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Comparative Study of Bronchial Responsiveness between Young Male Smokers and Non-Smokers

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ABSTRACT

Background: Cigarette smoking has been increasing at an alarming rate amongst the youth in India. This early and long term exposure could result in grave consequences later in life affecting their productivity and vitality. Although not clinically evident, bronchial hyper-responsiveness might be the earliest manifestation of a latent respiratory disorder.

Objectives: To record the PEFR in smokers and non-smokers before and after the exercise and to compare the bronchial responsiveness between the same two groups.

Materials & Method: The study included 30 smokers and 30 non-smokers aged 18-25 years. PEFR was recorded was recorded using Spirothor Wavefront Handheld Spirometer before and immediately after the performance of the Harvard Step Test.

Results: On statistical analysis of the recorded data it was observed that PEFR in smokers before exercise was 8.46±0.6 L/s and in non-smokers was 8.58±0.6 L/s (P value >0.05). PEFR in smokers after the exercise protocol was 5.04±0.38 L/s and in non-smokers it was 8.38±0.58 L/s (P value <0.001)

Conclusion: Although PEFR is comparable in smokers and non-smokers at rest, bronchial responsiveness is significantly increased in smokers. This early sign could be considered as a screening tool for a respiratory disorder in its latent phase. If smoking cessation is not implemented at this level it may lead to grave consequences.

Keywords: *Bronchial responsiveness, exercise, PEFR, smokers.*

INTRODUCTION

Every third person in an Indian city today is a youth. In about seven years, the median individual in India will be 29 years. By 2020, India is set to become the world's youngest country with 64 per cent of its population in the working age group.[1] So, the health and fitness of the youth is of prime concern.

Cigarette smoking is probably one of the most addictive and dependence producing self-gratification known to man. Tobacco smoking is a major risk factor for cardiovascular disease, chronic obstructive pulmonary disease and some cancers. The morbidity and mortality with tobacco use is entirely preventable[2]. India is the second largest consumer of tobacco products and third largest producer of tobacco in the world. The adult population of smokers in India is about 84.8 million and is almost equal to the population of Vietnam or Germany. The death toll from tobacco use is projected to rise from 5.4 million in 2004 to 8.3 million in 2030 [3].

Cigarette is the leading known risk factor for the development of chronic obstructive pulmonary disease and 50% of smokers develop clinically significant airflow obstruction[4]. The lung functions of cigarette smokers showed accelerated decline when compared with the non-smokers [5]. Tests of peak

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expiratory flow rate (PEFR) reflect changes in airways caliber[6]. Airflow obstruction in cigarette smokers is often diagnosed relatively late. Heightened bronchial responsiveness is an early manifestation of pulmonary disorder that is probably in its latent stage. Earlier detection of air-flow obstruction and smoking cessation may result in significant health gain[7]. If a cigarette smoker stops smoking, PEFR improves with the passage of time[8].

This study was undertaken to compare the PEFR in apparently healthy smokers and non-smokers at before and after an acute bout of exercise.

OBJECTIVES

1. To record the PEFR in smokers and non-smokers before exercise.
2. To record the PEFR in smokers and non-smokers after exercise.
3. To compare the bronchial responsiveness between the same two groups.

METHODOLOGY

This is a cross-sectional study done on 60 apparently healthy males of which 30 were smokers and 30 were non-smokers aged 18-25 years selected based on the eligibility criteria from the general population residing in residential areas of Bangalore city.

Inclusion criteria

- Males
- Age- 18-25 years
- Sedentary
- BMI- 18-25 kg/m^2
- Cases- smokers with smoking history >1 year
- Controls- Non-smokers

Exclusion Criteria

- Acute or chronic respiratory diseases
- Patients having oral lesions or any other abnormalities that prevent the performance of the test.
- Any history of cardiovascular or endocrine disorders
- Neuromuscular disorders
- Any long term medications that can affect lung functions

Prior to the study written informed consent was taken followed by relevant history taking and general physical examination of the selected participants. On history taking it was found that all the smokers who were selected for the study smoked less than 10 cigarettes per day (mild smokers) and had smoking history of less than 5 years.

Height was measured using a wall-mounted stadiometer (CMS Instruments, London, UK) and body weight was recorded in fasting state using Equinox BR-9201 weighing scale and Body Mass Index (BMI) was calculated using Quetelet's index [BMI = weight(kg)/height^2(m)].

Recording the PEFR

PEFR was recorded using a Spirothor Wavefront Hand held Spirometer in the morning hours after light breakfast.The procedure was explained and subjects were made to stand straight, They were made comfortable and familiarized with the procedure. Nose clip was applied. Forced spirometry was performed using Hand held Spirometer; subject asked to take deep maximum inspiration followed by forceful maximum expiration. Total 3 recordings were taken with the interval of 2 minutes. Best of the 3 recording was selected.

After an interval of ten minutes the participants were made to perform the Harvard Step Test

Exercise protocol-The **Harvard Step Test** [9]

The person who is taking the test steps up and down on a platform in a cycle of one step per two seconds. The platform is at a height of about 50 cm or 20 inches. The rate of 30 steps per minute must be held up for 5 minutes or until exhaustion. To ensure the right speed, a metronome is used. Exhaustion is the point at which the subject cannot maintain the stepping rate for 15 seconds.

Immediately post exercise PEFR was again recorded using the method mentioned above.

Statistical Analysis

Data are presented as mean ± standard deviation (SD) and the difference between two means was compared by student's t–test. A p value less than 0.05 ($p < 0.05$) was considered to be statistically significant.

RESULTS

Table 1- Baseline Characteristics of the Study Group

	SMOKERS (n=30)	NON-SMOKERS (n=30)	P VALUE
Age (in years)	22.5±2.14	22.53±2.02	0.95
Height (in m)	1.65±0.05	1.67±0.06	0.14
Weight (in kg)	59.8±8.23	63.17±8.9	0.14
BMI (in kg/ m^2)	21.88±2.22	22.52±2.3	0.28

Table 1 shows that the cases and control are age matched and have no statistically significant difference in their height, weight and BMI.

Table 2- Comparison of PEFR between Smokers and Non-smokers

	SMOKERS PEFR (in L/s)	NON-SMOKERS PEFR (in L/s)	P VALUE
Pre-exercise	8.47±0.64	8.58±0.59	0.5

Table 2 shows that PEFR values in smokers and non-smokers are not statistically significant

Table 3- Comparison of Bronchial Responsiveness in the Study Group

	PRE-EXERCISE PEFR (in L/s)	POST-EXERCISE PEFR (in L/s)	P VALUE
Smokers	6.76±0.51	5.04±0.38	<0.001*
Non-smokers	8.58±0.59	8.38±0.57	0.21

Table 3 shows that bronchial responsiveness is increased in smokers significantly with P value <0.001.

DISCUSSION

This study demonstrates that at rest PEFR in smokers and non-smokers is comparable. There is an increase in bronchial responsiveness in both the groups but the extent of increase was significantly greater in smokers compared to non-smokers. Therefore, from our study it can be implicated that smoking brings about a bronchial hyper-responsiveness.

Smoking produces inflammatory changes throughout the respiratory tree and lung parenchyma. The exact constituents of cigarette smoke that are responsible for this are still not clearly known[10]. Irrespective of this, inflammation in early stages is associated with increase in mediator cells such as neutrophils, macrophages and mast cells both within the airway lumen and airway wall. The resultant edema and proteoglycan deposition produces increased thickness of submucosal and adventitial tissue thereby compromising airflow[11]. In the later stages, there is progressive fibrosis of airway walls related to smooth muscle hypertrophy and hyperplasia with remodelling. Smoking also increases the number of mucosal goblet cells, resulting in increased secretion of mucus, formation of mucus plugs and obstruction of airways[12]. These are the pathological changes present in smokers during the latent phase of respiratory disorder. Exercise increases bronchial smooth muscle tone, mediated through cholinergic vagal pathways, and this bronchoconstricting effect explains the acute reduction in PEFR immediately after exercise. So, exercise acts as a stimulus resulting in manifestation of the earliest sign i.e. bronchial hyper-responsiveness.

Several studies have reported that PEFR was significantly lower in smokers than in non-smokers [13-17] . This study has strong clinical repercussions. Its findings can be linked to tip of an iceberg in the sense that most of the smokers have grave pulmonary consequences than just asymptomatic airflow limitation. It is just a matter of time before the ill effects become clinically evident. So, before it is too late smoking cessation should be implemented.

The strength of this study is that we have used a simple measure like PEFR to identify the deterioration of pulmonary functions in otherwise asymptomatic healthy smokers. This simple and economical method can be used as a screening tool as well as to educate the general population regarding the hazards of smoking. To the best of our knowledge this the first study to evaluate the effect of acute bout of exercise on PEFR of smokers and non-smokers. The

study is limited by its sample size and involvement of only males. Correlation of the decline in PEFR with the smoking history and evaluation of other pulmonary function tests could be the further scope for this study.

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Conflict of Interest: Nil

Source of Funding: Self

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Assessment of Knowledge, Attitude and Practice of Blood Donation among Medical Students in Bangalore Karnataka, India

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ABSTRACT

Context: Despite a requirement of over 8 million units of blood per year, data regarding awareness and practices of blood donation among students remains inadequate. A study on the knowledge, attitude and practice of blood donation among medical students will help highlight the importance of adopting effective measures to motivate voluntary blood donation among the population.

Aims: To assess the knowledge, attitude and practice of blood donation among medical students

Settings and Design: Observational, Cross Sectional Study

Methods and Material: A cross sectional study conducted, pre tested validated semi structured questionnaire was administered to medical students in Bangalore.

Statistical analysis used: The data was tabulated in Microsoft Excel version 10 and Descriptive statistics was applied using statistical package of social sciences, SPSS version 18. P value of < 0.05 was considered statistically significant.

Results: A total of 140 subjects participated in the study, of which 75(53.6%) were males and 65(46.4%) were females. The mean age in years was 18 years 97(68.8%). 86(63.2%) of the subjects were aware that diseases could be transmitted through blood and 57(40.7%) knew that blood was tested for infective agents before transfusion. 96(71.1%) of the subjects felt they are fit enough to donate blood. Only 15(10.7%) received education on blood donation. Majority answered incorrectly for the use of one unit of donated blood for the number of patients and this difference was found to be statistically significant. Only 25(17.8%) have attitude towards voluntary blood donation, while 6(1.4%) had really donated blood.

Conclusions: study emphasizes that the knowledge, attitude and practices regarding voluntary blood donation is not adequate among first year medical students. Measures have to be taken to raise awareness and abolish the myths and fears linked to voluntary blood donation. The lack of awareness and misconceptions concerning donation may contribute to the lack of initiative for voluntary donation.

Key-words: *Blood donation, knowledge, attitude, practice.*

Key message: Good knowledge about blood donation translates into a more positive attitude however this has not necessarily translated into practice. The lack of awareness and misconceptions concerning donation may contribute to the lack of initiative for voluntary donation.

INTRODUCTION

Human blood is an essential element of human life and there are no substitutes to blood as yet.[1] Millions of lives are saved each year through blood transfusions, but the concern is quality and safety of blood transfusion particularly in the developing countries.[2] Blood will be safe if there is a nationally coordinated blood transfusion service, collection of blood only from voluntary non-remunerated donors, testing of blood for transfusion transmissible infection and transfusion of the right blood to the right patient through the appropriate clinical use of blood.[3] The role of youth in promoting and actively participating in blood donation is significant. According to the WHO, an estimated 38% of reported voluntary blood donations are by people under the age of 25. [4] The WHO has also suggested that all developing countries focus on the youth, in trying to achieve 100 percent voluntary blood donation.[5] Hence, this study was undertaken to assess the knowledge, attitudes and practices regarding blood donation among first year students in a medical college in Bangalore.

MATERIALS & METHODOLOGY

Study Design: Observational, Cross Sectional Study

Duration of Study: November and December, 2014

Source of Data: All first year medical students in a medical college in Bangalore, Karnataka were included for the study. Those who were not willing to participate were excluded from the study and final sample size was 140 students

Study Technique: A semi structured questionnaire was administered, to assess their knowledge, attitude and practice about blood donation after taking informed consent. The questionnaire comprised of four sections: on demographic data, knowledge, attitude and practice about blood donation. Knowledge was assessed by questions on blood groups, the blood donation process and situation requiring blood donation. Attitude was assessed by questions on harmful effects of blood donation, time and necessity of blood donation. Practices were assessed based on whether the subject or his/her family members have donated or received blood in the past and their participation or willingness to participate in blood donation camps.

Ethical clearance was obtained from the institutional ethical committee.

Statistical analysis: The data was tabulated in Microsoft Excel version 10 and Descriptive statistics was applied using statistical package of social sciences, SPSS version 18. P value of < 0.05 was considered statistically significant.

RESULTS

A total of 140 subjects participated in the study, of which 75(53.6%) were males and 65(46.4%) were females. The mean age was 18 years 97(68.8%). Of the 140 subjects, 103(74.1%) had knowledge of their own blood group. 84(60.0%) of them knew minimum age for blood donation but only 15(10.8%) knew maximum age limit for blood donation. 86(63.2%)of the subjects were aware that diseases could be transmitted through blood and 57(40.7%) knew that blood was tested for infective agents before transfusion. 96(71.1%) of the subjects felt that blood donation was a generous act and that they were fit enough to donate blood, but as many as 104(74.2%) of the subjects felt that it could be harmful to the donor. Only 15(10.7%) had received awareness education on blood donation previously. Majority answered incorrectly for the use of one unit of donated blood for the number of patients used and this difference was found to be statistically significant($p<0.05$). Statistically there were no significant differences among genders in knowledge and attitude regarding blood donation. (Table 1).Only 25(17.8%) have attitude towards voluntary blood donation, whereas 18(12.8%) showed a willingness to donate blood in case of an emergency. About 110(78.6%) felt that their family does not support blood donation (Table 2). While 10(7.1%) of the subjects had encouraged blood donation in the past among their family and friends and only 6(1.4%) had actually donated blood in the past (Table 3).

DISCUSSION

The percentage of the population that had donated blood at least once in the past was 6(1.4%) which is lower than a study conducted in a Delhi, where 7.7% had donated blood. [6] Gender did not arise as a significant factor for knowledge and attitude towards blood donation, which is similar to a study carried out in a South Indian university [7] and in a rural Bangalore study. [8]

About 71.1% were aware of the eligibility for blood donation. The lack of awareness and misconceptions concerning blood donation may contribute to the lack of initiative for voluntary donation. This is similar to the findings in the studies done before that all quote fear, lack of opportunity, ignorance and lack of family support as the main reasons for not donating blood.[6,9,10] Majority felt that blood donation was a good act. This has not necessarily translated into practice, similar findings were observed in a rural Bangalore study. [8]

It is surprising that 104(25.90%) of the study subjects were not aware of their own blood group. This is higher compared to the study done by Sabu et al that found that 4.1% did not know their own blood group. [11]

More than half of the subjects, 80 (60%) in the present study knew correct age limit for donating blood. Shahshahani *et al* reported that 45% in the general population had correct knowledge regarding minimum age requirement for blood donation.[12] In other studies however, it was correctly known by only 3-6% of the respondents.[13,14] Correct knowledge regarding the minimum gap between two donations was recorded in 48(34.3%) of the subjects, as also observed in other studies.[14,15,16]

Based on the findings of the study, health education programs regarding the importance of voluntary blood donation, the safety procedures followed and the beneficial aspects of blood donation to self and society must be implemented with greater force among the youths. It must target all age groups, the youth in particular, as they would be more eligible to donate.

CONCLUSION

A lack of information results in fears and misconceptions about blood donation. This can be prevented by conducting awareness programs. Educating medical students also has the added benefit that they play a key role in spreading awareness among other members in the community especially family members and relatives. The misconceptions concerning blood donation may be one of the contributing factors to the lack of initiative for voluntary donation. This study emphasizes the importance of knowledge, attitude and practice regarding voluntary blood donation among the students.

Table 1 . Knowledge regarding blood donation according to gender characteristics:

Sl. no.	Particulars	Correct response n(%)		Total n(%)
		Male 75	Female 65	
1	Knowledge of their own blood group	51(68.0)	52(80.0)	103(74.1)
2.	Minimum age for blood donation (correct=18yrs)	41(54.7)	43(66.2)	84(60.0)
3.	Maximum age for blood donation (correct=60yrs)	7(9.5)	8(12.3)	15(10.8)
4.	Minimum spacing between donations (Correct=3 months)	21(28.0)	27(41.5)	48(34.3)
5.	One unit of donated blood is used for how many patients*(Correct= 4) P=0.01*	17(22.7)	5(7.8)	22(15.8)
6.	Testing for infective agents before transfusion	28(37.3)	29(44.6)	57(40.7)
7.	Can diseases be transmitted through blood	48(65.8)	38(60.3)	86(63.2)
8.	Are you fit enough to donate blood	54(76.1)	42(65.6)	96(71.1)
9	Is blood donation harmful to the donor	6(100)	98(73.1)	104(74.2)
10	Received education on blood donation	5(83.3)	10(7.4)	15(10.7)

*P value significant at <0.05

Table 2: Attitudinal response of the study population regarding blood donation

Sl no.	Particulars	Response n (%)	
		Yes	No.
1	Do you feel faint/weak if you donate blood	112(80.0)	28(20.0)
2	Do you get an infection if you donate blood	74(52.8)	66(47.2)
3	Will you voluntarily donate blood	25(17.8)	115(82.2)
4	Will you donate at the time of emergency	18(12.8)	122(87.2)
5	Do you encourage blood donation	28(20.0)	112(80.0)
6	Anybody has approached you for blood donation	17(12.2)	123(87.8)
7	Does your family support for blood donation	30(21.4)	110(78.6)

Table 3 . Blood donation practice among study population

Sl no.	Particulars	Response	Blood Donated n(%)		Total
			Yes	No	
1.	Ever donated blood		6(1.4)	134(98.6)	140
2.	Received blood in the past		2(1.4)	138(98.6)	140
3.	Have you taken part in blood donation camps	Yes	2(66.6)	2(1.4)	4(2.8)
		No	4(33.4)	132(98.6)	136(97.1)
4.	Have you encouraged Blood donation	Yes	4(33.3)	6(4.4)	10(7.1)
		No	2(66.4)	128(96.6)	130(92.8)

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A Comparative Study of Variations in Hematological Profiles in Different Trimesters of Normal Pregnancy

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ABSTRACT

Background: The haematological profile of an individual to a large extent reflects their general health and many studies have identified the haematological profile of the pregnant woman as one of the factors affecting pregnancy and its outcome. The study was aimed to determine the effect of pregnancy on haematological indices and compare the haematological indices at different trimesters of normal pregnancy

Objectives: To evaluate the values of some major hematological parameters at different trimesters of pregnancy.

Materials and Method: The research involved 30 healthy pregnant women as the study group and 10 non pregnant women as control .Age range of these women was 20-30 years.3 millilitres of venous blood collected from the median cubital vein with minimum stasis were put into EDTA bottle. The blood was properly mixed and analyzed for packed cell volume (PCV), total white cell count, Differential count and Erythrocyte sedimentation rate (ESR).

Results: The result showed that study group exhibited statistically significant lower values of PCV, monocyte and lymphocyte while WBC, eosinophil and ESR were not significantly changed. There was no significant difference in all hematological parameters among the three trimesters.

Conclusion: Healthy pregnancy may have effect on hematological parameters. Therefore there is a need to monitor these parameters during pregnancy. We also find that stages of pregnancy have no influence on hematological parameters.

Keywords: *Hematological Profiles, Trimesters, Pregnancy.*

INTRODUCTION

The haematological profile of an individual to a large extent reflects their general health and many studies have identified the haematological profile of the pregnant woman as one of the factors affecting pregnancy and its outcome.[1] The most commonly referred to of the haematological indices are the indicators of haemoglobin concentration, and low haemoglobin (anaemia) is the most widely identified haematological abnormality and is associated with adverse pregnancy outcome.[2] Anaemia in women is variously defined with the two most common being either as a haemoglobin concentration less than 11.0 g/dl or <5th percentile of the distribution of haemoglobin concentration or Haematocrit in a healthy reference population and is based on age, sex, and (among pregnant women) stage of pregnancy. According to the World Health Organisation, "anaemia is a common and serious problem in pregnancy" and needs to be addressed.[3].

The study was aimed to determine the effect of pregnancy on haematological indices and compare the haematological indices at different trimesters of normal pregnancy.

MATERIALS & METHOD

SUBJECT: Thirty pregnant women (10 in first

trimester, 10 in second trimesters and 10 in third trimesters) between the ages of 20-30 years were enrolled in the Antenatal Clinic of BMC, Chitradurga for the study. Ten non-pregnant age-matched women were used as control subjects. Ethical approval was obtained from the Research and Ethics Committee of the Hospital.

METHODOLOGY: Three milliliters of venous blood collected from the median cubital vein with minimum stasis were put into EDTA bottle. The blood was properly mixed and analyzed

for PCV, total white cell count, differential counts and ESR. Hematology was done according to the standard methods.

STATISTICAL ANALYSIS: All calculations were done using the SPSS-V15 statistical software package for analysis of the data. The data were presented as Mean± SD, and statistical analysis was carried out using the student's paired t-test and ANOVA. Differences were considered to be statistically significant at an error probability of less than 0.05 ($P \leq 0.05$).

RESULTS

Table 1 showed the comparison of mean hematological indices between pregnant women and non pregnant women where differences in PCV, WBC, eosinophil, monocyte, lymphocyte and ESR were found to be statistically significant.

Table 1: Comparison of hematological indices in pregnant women and control (Mean±SD)

Parameters	Pregnant women	Control
Age	28 ±3.40	24±2.10
PCV (%)	31.72±4.30*	38.75±3.70
WBC($\times 10^9$/L)	7.29±3.00*	4.93±0.90
Neutrophil (%)	52.91±13.90	44.63±13.4
Eosinophil (%)	10.35±4.30*	6.32±3.40
Monocyte (%)	1.41±0.30*	4.16±1.90
Basophil (%)	1.00±0.00	1.30±0.52
Lymphocyte (%)	35.68±14.50*	44.86±12.50
ESR (mm/hr)	31.46±8.90*	11.07±4.70

*P ≤0.05 comparing with the control group.

Table 2 showed the mean hematological values between the three trimesters of pregnancy. All the parameters were compared with each other between trimesters and none of the values were found to be statistically significant.

Table 2: Hematological values over the three trimesters in pregnant women (Mean ±SD).

Parameters	Trimester 1	Trimester 2	Trimester 3	P-Value		
				1st&2nd	1st&3rd	2nd & 3rd
PCV (%)	30.88±2.61	32.45±4.38	31.70±5.52	0.364	0.647	0.664
WBC($\times 10^9$/L)	6.22±1.79	7.52±2.74	8.11±4.13	0.233	0.098	0.583
Neutrophil (%)	55.17±9.24	48.97±17.96	55.32±12.17	0.277	0.980	0.266
Eosinophil (%)	10.53±5.21	9.72±3.10	10.90±4.91	0.639	0.835	0.494
Monocyte (%)	1.82±0.67	0.85±0.43	1.37±0.77	0.318	0.601	0.591
Basophil (%)	1.00±0.00	_	_	_	_	_
Lymphocyte (%)	33.09±6.44	41.05±19.37	31.94±12.57	0.165	0.845	0.113
ESR(mm/hr)	35.64±23.97	31.38±14.75	27.36±18.36	0.528	0.241	0.551

DISCUSSION

The aim of this study was to evaluate the hematological profile of pregnant women at different trimesters and to compare hematological parameters of pregnant and non pregnant women. There is a statistical difference in the PCV of pregnant women (31.72±4.30) % compared with the control (38.75±3.70) %. This correlates with findings in other studies .The decrease in PCV may be due to increase in plasma volume during pregnancy causing hemo-dilution, infections e.g. malaria, hormonal changes that

increase fluid retention and iron deficiency.[4]

There is no statistically significant difference in the value of PCV throughout the whole process of pregnancy, although there was variation in actual numeric values. A study showed marked decrease in PCV in the third trimester. This was attributed to maternal diabetes. [5]

White blood cells are responsible for body defense. During pregnancy, WBC is reported to be elevated.[6] In this study, the leucocytes count was significant higher compared to that of the controls. This agrees with previous work by Roy et al that reported a total leukocyte count rising in early pregnancy which remained elevated throughout pregnancy. This may be as a result of the body building the immunity of the fetus and it is achieved by a state of selective immune tolerance, immunosuppression and immunomodulation in the presence of a strong antimicrobial immunity. There is also down-regulation of potentially dangerous T-cell-mediated immune responses, while activating certain components of the innate immune system, such as neutrophils. This unique dysregulation between different components of the immune system plays a central role in the maternal adaptation to pregnancy.[7]

The elevation of total WBC is accounted for neutrophil. [6] There is no statistical difference between the value of neutrophil in both the study and control groups, but the value is higher in the studied group than the control group. Lymphocyte and monocyte counts were lower while eosinophil count was significantly higher in studied group than in control. [6, 7] Similar observations have been made in previous studies. Lurie et al reported no significant increase in eosinophil count. [8]

The erythrocyte sedimentation rate is one of the measurements of acute phase response. It is helpful in detecting the presence of inflammation and its response to treatment. In the studied group, the value of ESR is significantly increased compared with control group. It is said that ESR can increase to as much as 2-3 times normal values during pregnancy. [9] This may be as a result of anemic state of the studied group due to plasma expansion and decrease in packed cell volume in healthy pregnancy: it may also be due to marked increase in circulating fibrinogen in pregnancy. [10] There is no significant difference in the value of all the parameters analyzed when compared at different stages of the pregnancy. This disagrees with the report that there is significant difference across the trimesters in the value of WBC and PCV as reported by James et al. [4] The limitation of this study is we have not considered iron supplementation of pregnant women in all three trimesters as iron supplementation increases PCV level in the blood.

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Conflict of Interest: The authors declare they have no conflict of interest

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Inter-relationship between Computer Exposure, Exercise and Sleep Quality in IT Professionals of Bangalore

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ABSTRACT

Background : Information and Communication Technology (ICT) has been constituted as a pivotal role in our society. Newer computers and TV screens are now frequently equipped with light-emitting diodes (LED), which peak in the short-wavelength region and which are primarily responsible for a variety of nonvisual light responses, in particular, resetting the timing of the circadian pacemaker, suppressing melatonin production, improving alertness and performance, and elevating brain activation, as assessed from EEG-derived correlates of arousal. Physical Exercise is recommended by the American Sleep Disorders Association as a non-pharmacological intervention to improve sleep. Hence we examined whether exercise may help to improve the sleep quality among IT professionals.

Aim : 1. To find the interrelationship between hours of computer exposure,exercise and sleep quality in information technology professionals of Bangalore.

Method : This study includes 40 IT professionls. The practice of physical activities, use of computers during weekdays and participants' demographic information such as gender and age were collected. Quality and quantity of sleep was evaluated by Pittsburgh Sleep Quality Index (PSQI) questionnaire.

Results : Mean age was 25.9±2.2 yrs, computer exposure 8.45±2.3 hrs and mean PSQI score is 5.02±1.8 and is indicative of poor quality of sleep.There was significant positive association between computer exposure and PSQI and negative association between exercise and PSQI.

Conclusion : The results of the present study indicate a negative association between level of physical activity and computer exposure among IT professionlas of Bangalore.

Keywords: *Sleep, IT Professionals, Exercise.*

INTRODUCTION

Information and Communication Technology (ICT) has been constituted as a pivotal role in our society. IT firms in Bangalore employ about 35% of India's pool of 2.5 million IT professionals and account for the highest IT-related exports in the country[1].

There are approximately six-computers/1000 population with an installation of 18 million Personal Computers (PCs) and their number increasing all the time. This has also ushered in a new genre of occupational health problem i.e. of computer related health problems[2] .Newer computers and TV screens are now frequently equipped with light-emitting diodes (LED), which peak in the short-wavelength region (i.e., the blue range at ~460 nm). There is ample evidence that a novel, short-wavelength-sensitive photoreceptor system is primarily responsible for a variety of nonvisual light responses, in particular, resetting the timing of the circadian pacemaker, suppressing melatonin production, improving alertness and

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performance, and elevating brain activation, as assessed from EEG-derived correlates of arousal[3].Furthermore, bright light exposure and exposure to monochromatic blue light in the evening lengthens sleep latency and reduces initial EEG delta activity, a marker of slow-wave sleep .Thus the frequent use of LED sources could have ramifications on human behavior, since light is the most important synchronizer of our biological clock. In a study of 10,000 16 to 19-year-olds, researchers in Norway found that the longer a young person spent looking at an electronic screen before going to bed, the worse quality sleep and those who spent more than four hours a day looking at screens had a 49 per cent greater risk of taking longer than an hour to fall asleep and were three and a half times more likely to sleep for under five hours a night[4].

The practice of Physical Exercise (PE) is advocated as a promoter of good quality sleep because it reduces sleep latency increases total sleeping time and improves alertness during wakefulness[5]. Hence, PE is recommended by the American Sleep Disorders Association as a non-pharmacological intervention to improve sleep[6].

Based on the above evidence we hypothesize that increased number of computer usage is associated with sleep disturbances and and exercise may help to improve the sleep quality among IT professionals.

AIMS OF THE STUDY

1. To find the interrelationship between hours of computer exposure,exercise and sleep quality in information technology professionals of Bangalore.

SUBJECTS AND METHOD

Subjects: The primary service providers in Information technology (IT) industry are grouped into: IT software industry, IT enabled service, Internet and e-commerce. The IT professionals working in different sectors were identified and representative sample was taken to complete the sample size of 40. All participants signed an informed consent form before inclusion in the study.

Inclusion criteria:

1) Subject should be working in the current job for past six months.

2) He/she should be working on the computer for at least 3 hours/day or 15 hours/week.

Exclusion criteria: 1) unable to give consent 2) smokers 3) alcoholics 4) Drugs which affect sleep

STUDY TOOLS

General information questionnaire: The practice of physical activities(mininmum30 min), use of computers during weekdays and participants' demographic information such as gender and age were evaluated.

Research tool: In all patients, quality of sleep was evaluated by administering the PSQI through an interview. The PSQI is a self-report questionnaire that assesses sleep quality and quantity over a month long period. The questionnaire consists of 19 self-rated questions. The 19 questions were categorized into 7 components, which are graded on a scale that ranges from 0 to 3. The PSQI components are as follows: subjective sleep quality (C1), sleep latency (C2), sleep duration (C3), habitual sleep efficiency (C4), sleep disturbances (C5), use of sleeping medication (C6), and daytime dysfunction (C7). The sum of scores for these 7 components yields one global PSQI score, which ranges from 0 to 21, where the highest score indicates the worst sleep quality. A global PSQI ≥ 5 has a diagnostic sensitivity of 89.6 and specificity of 86.5 in distinguishing poor sleepers (PSQI ≥5) from good sleepers (PSQI ≤5). Patients with GQ (PSQI score ≤ 5) were compared with patients with PQ (PSQI ≥ 6).

RESULTS

1) General information: Table 1 gives baseline characteristics.Out of 40 Table 2 shows that about 65% people exercised and 25% of people had good quality sleep who used computers for 4-8hrs.17.5% had good quality sleep even though they used computer for prolonged period but exercised.

Table 1: Baseline caracteristics

Sl.no	Parameters	Mean±SD
1	Age(yrs)	25.9±2.2
2	Computer exposure(hrs)	8.45±2.3
3	PSQI score	5.02±1.8

Table 2: Hours of computer exposure,sleep quality and exercise

	Exercise(65%)		**Non-exercise(35%)**	
Computer exposure	Good quality(<5)	Poor quality(≥5)	Good quality(<5)	Poor quality(>5)
4-8 hrs	25%	12.5%	5%	7.5%
9-14hrs	17.5%	5.41%	10%	20%

2) Relationship between hours of computer exposure,exercise and PSQI :

There was significant positive association between hours of computer exposure and PSQI (r=0.6;p=<0.01) and negative association between exercise and PSQI(r = -0.46;p=<0.01). To test the hypothesis that a PSQI scoring of IT professionlas is a function of these variables, the hours of computer exposure and exercise ,interaction between hours of computer exposure and exercise ,multiple regression analysis was performed. Beta coefficients for the three predictors were hours of computer exposure, β = .473, t =4.5, p < .001; exercise, β = -1.8, t = -3.3, p < .001; and computer exposure and exercise, β = -.11, t = -1.76, p = .081, n.s. The best fitting model for predicting PSQI is a linear combination of the increase in hours of computer exposure and interaction between computer hours and exercise (R^2 =0 .54, F= 21.8, p < .001)

DISCUSSION

The impact of light on the circadian timing system is classically measured by suppression of the "darkness hormone" melatonin, a key circadian phase marker secreted only during the night[7].These responses are mediated by a subset of retinal ganglion cells containing the photopigment melanopsin, most sensitive to wavelengths within the blue light spectrum around 480 nm , which transmit light signals via the retinohypothalamic tract directly to the suprachiasmatic nuclei[8,9] .

Intuitively speaking, the earth rotates around the sun every day. This phenomenon causes day and night, or light and darkness. The human have adapted to this phenomenon. Human sleeps at night. This period is no light. It does not suppress the melatonin production then human falling asleep. In the morning, human wakes up by the light. This period contains red light more than any light. It stimulates alertness and suppresses melatonin production. During a day, this period contains blue light more than any light. It suppresses melatonin production. In the dusk, this period contains red light more than any light again. It induces the melatonin production more than blue light then human falling asleep. In conclusion, blue light has effect on melatonin production more than red light. Red light stimulates positive affect and anger more than blue light. It also has effect on melatonin production. Finally, this conclusion can be used to design and develop the electrical display devices used before sleep that cannot stimulate alerting or stress and suppress melatonin production[10].According to the study done by Figueiro et al two hour exposure to light from self- luminous electronic displays can suppress melatonin by about 22%[11].

PE is recommended by the American Sleep Disorders Association as a non-pharmacological intervention to improve sleep[6]. Several authors have observed that the effects of PE on sleep are associated to thermoregulatory hypotheses, energy conservation and body restoration[12,13]. Similarly, Moreover, a literature review conducted by Kubitz et al.concluded that active individuals not only fall asleep faster, but also sleep for longer periods and more deeply compared to inactive individuals[14].

Our study revealed that a lower sleep quality was associated with longer exposure to computer and nonexercising professionals according to Table 2. 17.5% had good quality sleep even though they used computer for prolonged period but exercised.According to National Sleep Foundation if an individual is inactive, adding a 10 minute walk every day could improve your likelihood of a good night's sleep[15].

CONCLUSIONS

In this study, a high proportion of IT professionals were found to have disturbed sleep.The data support an association between prolonged hours of computer exposure and sleep disturbances. exercise may help to improve the sleep quality among IT professionals sleep quality and quantity.

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Effect of Short Term Pranayama Practice on Autonomic Function in Young Healthy Female

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ABSTRACT

Aim: To study the effect of short term practice (one week) of Nadishodhan Pranayama, a slow breathing exercise on parasympathetic and sympathetic reactivity test in young healthy females.

Material and Method: I[st] year MBBS female medical students in the age group of 17-22 years were chosen for the study. Group 1 Students practiced Pranayama for 20 minutes. The subjects were taught "Nadishodhan Pranayama" (alternate nostril breathing). Parasympathetic reactivity test were done using E: I ratio, 30:15 ratio and Sympathetic reactivity test was assessed using Blood pressure response to sustained Handgrip. All the assessment was done at the start of the study and at the end of one week's session of Pranayama. Group 2 students acted as control.

Result: We observed an increase in E: I ratio and 30:15 ratio indicating increase in the parasympathetic activity. Increase in the DBP in response to the Isometric exercise was found to be decreased indicating a decrease in the Sympathetic activity.

Conclusion: Short term Practice of Nadishodhan Pranayama leads to decrease in sympathetic activity and increase in parasympathetic activity.

Keyword: *Pranayama, Parasympathetic Reactivity test, Sympathetic Reactivity test, Autonomic function.*

INTRODUCTION

The word Pranayama is deep and powerful. Prana means breath, wind, life, vitality, energy or strength. It also implies soul as opposed to body. Ayama means length, expansion, stretching, or restraint. The two used together in Pranayama mean extension and control of the breath. Pranayama is both a powerful tool, and an end in itself[1].

Research has shown that practice of Pranayama modulates autonomic balance by increasing parasympathetic tone and decreasing sympathetic activity. Regular practice of pranayama has been shown to increase baroreflex sensitivity and reduce chemoreflex activation, and to reduce systolic, diastolic and mean blood pressures as well as heart rate variation [2,3,4]. Yogic breathing techniques have shown to effect autonomic neuropathy and hypertension.[5,6].

Pal GK studied the effect of breathing exercises on autonomic functions in young volunteers in the age group of 17-19 yr. A total of 60 male undergraduate medical students were randomly divided into two groups: slow breathing group and the fast breathing group. The breathing exercises were practiced for a period of three months. Auto nomic function tests were performed before and after the practice of breathing exercises. Increased parasympathetic activity and decreased sympathetic activity were observed in slow breathing group, whereas no significant change in autonomic functions was observed in the fast breathing group [7].

Voluntary slow deep breathing functionally resets the autonomic nervous system through stretch-induced inhibitory signals and hyperpolarization currents propagated through both neural and non-neural tissue which synchronizes neural elements in the heart, lungs, limbic system and cortex. During inspiration, stretching of lung tissue produces inhibitory signals by action of slowly adapting stretch receptors (SARs) and hyperpolarization current by action of fibroblasts. Both inhibitory impulses and hyperpolarization current are known to synchronize neural elements leading to the modulation of the nervous system and decreased metabolic activity indicative of the parasympathetic state [8].

The aim of the present study was to study the effect of short term practice (one week) of Nadishodhan Pranayama, a slow breathing exercise, on parasympathetic and sympathetic reactivity test in young healthy females.

MATERIAL & METHOD

Twenty healthy I[st] year MBBS female medical student in the age group of 17-22 years were chosen for the study. The study was explained to the students and their informed consent was taken. They filled semi structured Performa which recorded their age , height weight and history of present or past illness including hypertension, heart disease, diabetes, asthma, cardiovascular risk factor like smoking and family history of cardiovascular disease, asthma, diabetes etc. The subjects were evaluated twice, first at the start of the study and second at the end of one week session of Pranayama.

Criteria for selection:

- Not suffering from any illness like diabetes mellitus, hypertension, CKD, Psychiatric disorder, Neurological disease or any other illness which is known to impair Autonomic function.
- Not practicing Yoga, Meditation, Biofeed back technique or any other relaxation technique.
- Not on any medication known to effect autonomic nervous system.

Study Design: The volunteers were randomly divided into two groups, each group consisting of 20 subjects.

Group1(P): Practiced Pranayama for twenty minutes for one week.

Group 2 (C): Acted as a control and did not practicing Yoga, Meditation, Pranayama, Biofeed back technique or any other relaxation technique.

To ensure regularity and uniformity in Pranayama practices, the training was given in the Department of Physiology, Lady Hardinge Medical College, New Delhi under the supervision of a trained Yoga teacher from Morarji Desai National Institute of Yoga, New Delhi.

Group 1 Students practiced Pranayama for 20 minutes. The subjects were taught "Nadi Shodhan Pranayama" (alternate nostril breathing). They were asked to sit in Padmasana keeping the body erect without stiffness and no part of the body having any trace of tension. Before starting Pranayama they were asked to breathe normally.

Technique: They were first asked to exhale completely. Then they closed their right nostril with thumb of their right hand. Then they were asked to inhale through the left nostril slowly, steadily and deeply as long as possible (POORAKA). When the breath was completed the left nostril was closed with the third finger and held in this state of KUMBHAKA for a few second. Then the thumb was released from right nostril and asked to exhale slowly and completely (RECHAKA), feeling movement of air. The process was then repeated by inhaling through the right nostril, thus completing one round of Nadi Shodhan Pranayama. The cycles were repeated initially for 5-6 round for practice, then students were asked to set a ratio of 1:4:2 i.e. 5 second for inhalation and then retain for 20 second and exhale for 10 second.

Autonomic Assessment

Parasympathetic reactivity test were done using E: I ratio and 30:15 ratio. Sympathetic reactivity was assessed using Blood pressure response to sustained Handgrip. All the assessment was done at the start of the study and at the end of one week's session of Pranayama.

Parasympathetic Reactivity Tests

E: I Ratio

E: I Ratio based on the phenomenon of respiratory arrhythmia, which is most pronounced

at the respiration rate of 6 breaths per minute. The subject is asked to breathe at this rate (with 5 s of inhalation and 5 s of exhalation per breath). The expiratory-inspiratory ratio (E: I ratio), which is the ratio of the longest RR interval during expiration and the shortest RR interval during inspiration from 5 cycles was determined. The E: I ratio in young person should be higher than 1.2.

$$\text{E:I ratio} = \frac{\text{Longest R -R interval during expiration}}{\text{Shortest R -R interval during inspiration}}$$

30:15 Ratio

During the postural change from lying to standing a characteristic immediate rapid increase in heart rate occur which maximal at about the 15th beat after standing is followed by a relative overshoot bradycardia maximal at about the 30th beat. To perform this test the subject is asked to lie quietly on a couch and then to stand up unaided. The characteristic heart rate response can be expressed by the 30 : 15 ratio, which is the ratio of the longest R-R interval around the 30th beat after starting to stand up to the shortest R-R interval around the 15th beat. The 30:15 ratio should be at least1.04. It was calculated by following formula:

$$\text{30:15 ratio} = \frac{\text{R-R interval at beat 30 after assuming erect posture}}{\text{R-R interval at beat 15 after assuming erect posture}}$$

Sympathetic Reactivity Tests

Blood pressure response to sustained handgrip

A rise in diastolic blood pressure is determined during isometric pressing of a handgrip dynamometer at approximately one third of the maximum contraction strength for 3-5 min. Blood pressure measurements are taken at the other arm at 1 min interval. An increase in diastolic blood pressure is a result of heart rate acceleration without an increase of peripheral vascular resistance. The test result is presented as the difference between the highest diastolic pressure during the examination and the average diastolic pressure at rest. It should normally be higher than 15 mmHg.

Statistical Analysis:

The data collected was evaluated using appropriate statistical technique:

a) Intragroup comparison was done using paired't' Test.

b) Intergroup comparison was done using Students't' test.

c) Adherence to following 'p' value was followed

p>0.05: Not significant

p<0.05: Significant

p<0.01: Highly Significant

p<0.001: Very Highly Significant

Table 1: Distribution of Age, Height, and Weight in Study group.

Parameter	Group 1	Group 2
Age(Years)	18.85±1.08	18.85±0.82
Height(cms)	159.40±5.74	160.50±4.80
Weight(kgs)	50.20±6.04	53.30±6.41

Table 2: Baseline comparison of Resting Heart Rate, Respiratory rate, SBP, DBP in Pranayama and control Group

Parameters	Group 1(n=20)	Group 2(n=20)	p-Value
Heart Rate	89.70±8.95	90.00±8.46	NS
Respiratory Rate	16.00±1.34	15.90±1.17	NS
Systolic Blood Pressure (SBP)	109.80±9.17	109.70±11.07	NS
Diastolic Blood pressure (DBP)	72.60±7.29	72.70±76.23	NS

All results are expressed as Mean ± standard deviation, p< 0.05 is significant

Table 3: Baseline comparison of Autonomic Function Tests in Pranayama and control Group

Parameters	Group 1(n=20)	Group 2(n=20)	p-Value
E: I Ratio	1.38±0.14	1.41±0.16	NS
30:15 ratio	1.18±0.13	1.13±0.14	NS
Change in DBP due to Isometric Exercise(IE)	21.40±5.39	21.60±4.89	NS

All results are expressed as Mean ± standard deviation, p< 0.05 is significant

Table 4: Comparison of Heart Rate, Respiratory rate, SBP, DBP in Pranayama group at Baseline and One Week

Parameters	Baseline	One Week	p-Value
Heart Rate	89.70±8.95	81.40±7.95	P<0.001
Respiratory Rate	16.00±1.34	14.20±0.83	P<0.001
Systolic Blood Pressure (SBP)	109.80±9.17	105.90±6.41	P<0.05
Diastolic Blood pressure (DBP)	72.60±7.29	65.30±5.00	P<0.001

All results are expressed as Mean ± standard deviation, p< 0.05 is significant

Table 5: Comparison of Autonomic Function Tests in Pranayama group at Baseline and One Week.

Parameters	Baseline	One Week	p-Value
E: I Ratio	1.38±0.14	1.85±0.28	P<0.001
30:15 ratio	1.18±0.13	1.44±0.19	P<0.001
Change in DBP due to Isometric Exercise(IE)	21.40±5.39	15.60±2.11	P<0.001

All results are expressed as Mean ± standard deviation, p< 0.05 is significant

Table 6: Comparison of Heart Rate, Respiratory rate, SBP, DBP in Control group at Baseline and One Week.

Parameters	Baseline	One Week	p-Value
Heart Rate	90.00±8.46	89.70±5.48	NS
Respiratory Rate	15.90±1.17	16.20±1.96	NS
Systolic Blood Pressure (SBP)	109.70±11.07	110.20±8.05	NS
Diastolic Blood pressure (DBP)	72.70±76.23	72.60±6.18	NS

All results are expressed as Mean ± standard deviation, p< 0.05 is significant

Table 7: Comparison of Autonomic Function Tests in Control group at Baseline and One Week.

Parameters	Baseline	One Week	p-Value
E: I Ratio	1.38±0.14	1.40±0.17	NS
30:15 ratio	1.18±0.13	1.17±0.12	NS
Change in DBP due to Isometric Exercise(IE)	21.40±5.39	21.20±4.92	NS

All results are expressed as Mean ± standard deviation, p< 0.05 is significant

RESULT

Comparison of Resting Heart Rate, Respiratory rate, SBP, DBP in Pranayama and control Group

The Resting Heart rate at baseline in Control group was 90.00±8.46 and at one week was 90.00±8.46. The change was not significant when compared to the baseline value but in case of the Pranayama group a decrease in the heart rate was observed when baseline recording (89.70±8.95) was compared at the end of one week (81.40±7.95) and this difference was statistically significant. Similar observation was made for Resting SBP, DBP and Respiratory rate. A

statistically significant decrease from baseline value was observed in the Pranayama group at the end of one week.

Comparison of Autonomic Function Test in Pranayama and control Group

Parasympathetic Reactivity Tests

The E: I ratio at baseline in Control group was1.41±0.16 and at one week was 1.40±0.17. The change was not significant when compared to the baseline value but in case of the Pranayama group a increase in the E:I ratio was observed when baseline recording (1.38±0.14) was compared to recording at the end of one week (1.85±0.28) and this difference was statistically significant. Similar observation was made for other Parasympathetic reactivity test like 30:15 ratio. A statistically significant increase from baseline value was observed in the Pranayama group at the end of one week signifying increase in the parasympathetic tone.

Sympathetic Reactivity Test

The pranayama group showed a decrease in the rise of SBP and DBP(105.90±6.41, 65.30±5.00) with isometric exercise at the end of one week when compared to base line recording(109.80±9.17, 72.60±7.29) and it was statistically significant, pointing towards a decrease in sympathetic activity. The control group didn't show any statistically signicant difference at the end of one week.

DISCUSSION

Literature is abundant with studies involving long term regular practice of Pranayama on Autonomic function. We specifically designed the present study to observe a very short term (One week) practice of Pranayama on autonomic function.

Resting heart rate, SBP and DBP

In our study a decrease in the Resting heart rate, Respiratory rate, SBP and DBP was observed in the group practicing Pranayama.

Sharma et al. compared the effects of commonly practiced slow and fast pranayama on cardiovascular functions in young health-care students. Significant decrease in HR, DBP and RPP was seen in subjects practicing Nadishodhan Pranayama. We also came to the same conclusion[9].

Pramanik T et al. evaluated the immediate effect of Bhramari Pranayama, a slow breathing exercise for 5 minutes on heart rate and blood pressure. Both the systolic and diastolic blood pressure was found to be decreased. Their findings are comparable to ours[10].

Effect of practice of Nadishodhan on autonomic parameter was also studied by Bhargava R et al. They observed a decrease in heart rate systolic and diastolic blood pressure and decrease in galvanic skin resistance (GSR) [4].

Autonomic Function Test

We observed an increase in E: I ratio and 30: 15 ratio indicating increase in the parasympathetic activity. Increase in the DBP in response to the Isometric exercise was found to be decreased after one week practice of Nadishodhan Pranayama, indicating a decrease in the Sympathetic activity.

A study similar to our study was done by Pal GK et al. where he observed the effect of breathing exercises on autonomic functions in young volunteers in the age group of 17-19 yr. Increased parasympathetic activity and decreased sympathetic activity were observed in slow breathing group[7].

Mourya M et al. also studied the effect of breathing exercise on autonomic function and their findings are in agreement to ours. They observed S/L ratio, 30: 15 ratio, E: I ratio and BP response in the hand grip and cold pressor test showed significant change in patients practicing the slow-breathing exercise[5].

Udapa et al. studied the effect of Pranayama training on cardiac function in normal young volunteers. They arrived at a conclusion that three months of Pranayama training modulates ventricular performance by increasing parasympathetic activity and decreasing sympathetic activity[11].

CONCLUSION

Short term Practice of Nadishodhan Pranayama leads to decrease in sympathetic activity and increase in parasympathetic activity. It could be due to normalization of autonomic cardiovascular rhythm as a result of increased vagal modulation and improved baroreceptor reflex sensitivity. Further study are needed to understand in depth mechanism.

Source of Funding: Self

Ethical Clearance: Taken

Conflict of Interest: None

Acknowledgement: Nil

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A Study of Thyroid Hormone Levels (T3, T4 & TSH), in Normal Pregnant Females and Pregnancy Induced Hypertensive Patients

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ABSTRACT

Background: Thyroid disorders are commonly encountered in pregnancy. Thyroid disorder during pregnancy are associated with adverse health outcome for both mother and child, including increased risk of miscarriage, gestational hypertension, preterm delivery, placental abruption, low birth weight and fetal death.

Method: Three ml of venous blood was collected and serum was separated and stored in deep freezer. Total T3, T4 and TSH were measured by Chemiluminiscent Microparticles Immunoassay (CMIA) technology with flexible assay protocol, referred to as Chemiflex**.**

Results*:* The mean thyroid levels in patients without hypertension (group I) was total T3-1.03±0.18 ng/mL, total T4- 9.49±2.45 μg/dl & TSH-2.28±1.67 μIU/mL and in patients with hypertension (group II) was total T3-1.02±0.16 ng/ml, total T4-9.83±2.08 μg/dL & TSH-2.87±1.85 μIU/mL

Conclusions: There was no statistically significant difference found in Total T3,T4 & TSH levels in patients without hypertension (group I) and patients with hypertension (group II).

Keywords: *Thyroid hormones , pregnancy, pregnancy-induced hypertension.*

INTRODUCTION

Thyroid disorders are commonly encountered in pregnancy. Interest in thyroid dysfunction complicating pregnancy has increased greatly during the decade. Normal pregnancy is associated with significant changes in maternal thyroid physiology. Serum thyroid-stimulating hormone (TSH) concentration is the initial and most reliable test for assessing thyroid function in pregnancy.

Serum TSH testing is relatively inexpensive, readily available, and is a reliable test in pregnancy, assuming that trimester-specific reference ranges are applied. There is moderate thyroid enlargement as a result of pregnancy hormone - induced glandular hyperplasia and increased vascularity. This enlargement is not pathologic. Thyroid function tests during pregnancy are also affected by estrogen-mediated increases in the level of thyroxin binding globulins (TBG). Total T3 and T4 levels increase starting in early pregnancy, due to the increased TBG levels, so that the upper limit of normal for total T3 and T4 in pregnancy is approximately 1.5-fold the upper limit of the non pregnancy reference range. Free T4 assays may be unreliable in pregnancy due to interference by the high TBG levels.

MATERIAL & METHOD

This case control study was conducted on 18-40 years old pregnant women with single pregnancy with gestational age of 20 weeks or more (based on first trimester sonography) and who presented at OPD & IPD of Upper India Sugar Exchange Maternity hospital (Obstetrics & Gynaecology department), G.S.V.M. Medical College, Kanpur. The

study group consisted of 35 normal pregnant females and 35 pregnancy induced hypertensive patients, who were selected randomly from the population with diagnosis of gestational hypertension.

CRITERIA FOR INCLUSION:

- Antenatal patients with gestational age 20 or more than 20 weeks.
- Primigravidae and multigravidae.
- With singleton pregnancy.

Gestational age of the patient was calculated from the first day of the last menstrual period (LMP) and if it was not known, then by available ultrasonographic parameters preferably in first trimester by crown rump length (CRL) and in second trimester by biparietal diameter and femoral length etc.

CRITERIA FOR EXCLUSION: Subjects with following complication were not included in the present study:

- Patient with chronic hypertension.
- Twin in present pregnancy.
- Molar pregnancy in present pregnancy.
- Chromosomally abnormal foetus.
- Diabetic patient
- Chronic renal disease patient.
- Patient with autoimmune disorder.
- Patient with history of hypertension, thyroid disorder, proteinuria, pre-ecclampsia or excessive weight gain in previous pregnancies.
- Patients with family history of diabetes, hypertension, or any other cardiovascular disease or history of pre-ecclampsia or ecclampsia.
- Patients in habit of illicit drug abuse like smoking, tobacco, chewing etc.
- Tobacco chewing etc.

In our study following criteria were taken for the diagnosis:

GESTATIONAL HYPERTENSION

American College of Obstetritician and Gynecologist in 2002, NHBPEP 2000: Gestational hypertension is defined as: "new hypertension (systolic blood pressure ≥ 140 mm Hg OR diastolic blood pressure ≥ 90 mm Hg or both) presenting at or after 20 weeks gestation without proteinuria or other features of preeclampsia," this terminology replaces the term "Pregnancy-Induced Hypertension".

SPECIMEN COLLECTION & PREPARATION: Three ml of venous blood was collected and serum was separated and stored in deep freezer.Total T3, T4 and TSH were measured by Chemiluminiscent Microparticles Immunoassay (CMIA) technology with flexible assay protocol, referred to as Chemiflex.

Normal range of T3-0.6-1.81 ng/mL Normal range of T4-4.5–10.9μg/dl Normal range of TSH-0.35-4.94μIU/mL .

STATISTICAL ANALYSIS: The significance between the standard errors of means of different sets of observation would be assessed by student't' test and 95% level of confidence.

OBSERVATION AND RESULT: The study was conducted on 70 pregnant females between age 20-38 years who attended the outpatient department of obstetrics and Gynaecology department of G.S.V.M. Medical College Kanpur. They were divided into two groups on the basis of blood pressure and presence of protein in urine (by dipstick method).

GROUP I: Comprised of 35 pregnant women of age group 20-38 years having normal blood pressure and no proteinuria

GROUP II: Comprised of 35 Hypertensive pregnant women of age group 20-38 years having blood pressure ≥ 140/90 mm of Hg and no proteinuria whom were diagnosed as pregnancy induced hypertensive patient.

The parameters estimated were Serum Protein (Total & Albumin), Serum Total

T3, Serum Total T4, Serum TSH.

Table-1: Serum Protein Total In Patients Without Hypertension (Group-I) And Patients With Hypertension (Group-II)

STATUS OF PERSON	PREGNANCY WITOUT HTN (Gr.I)		PREGNANCY WITH HTN (Gr.II)	
	N=35	%	N=35	%
DECREASED	5	4.29	4	11.43
NORMAL	30	85.71	29	82.86
INCREASED	0	0	2	5.71
MEAN ± SD	6.71 ± 0.87		6.120 ± 1.45	

(Total S. Protein level is 6.0-8.3 gm/dl)

The total S. Protein in group II had lower level as compared to group I.

The total serum protein levels in group I and group I, there was no statistically

significant difference.

The total S. Albumin in group II had lower level as compared to group I.

The total serum albumin levels in group I and group I, there was no statistically significant difference.

Table-2: Serum Total T3 Levels In Patients Without Hypertension (Group-I) And Patients With Hypertension (Group-II)

STATUS OF PERSON	PREGNANCY WITHOUT HTN (Gr.I)		PREGNA NCY WITH HTN (Gr.II)	
	N=35	%	N=35	%
NORMAL	35	100	35	100
MEAN ± SD	1.02 ± 0.18		1.02 ± 0.16	

Normal T3 Level = 0.6-1.81ng/mL)

The mean thyroid levels in group I was 1.03±0.18 ng/mL for total T3, in group II was 1.02±0.16 ng/ml. There was no statistically significant difference found in between Thyroid Hormones levels total T3 in group I and group II.($p > 0.05$).

Table-3: Serum Total T4 Levels In Patients Without Hypertension (Group-I) And PatientsWith Hypertension (Group-II)

STATUS OF PERSON	PREGNANCY WITHOUT HTN (Gr.I)		PREGNANCY WITH HTN (Gr.II)	
	N=35	%	N=35	%
NORMAL	24	68.57	22	62.87
INCREASED	11	31.43	13	37.14
MEAN ± SD	9.48 ± 2.44		9.83 ± 2.08	

(Normal Total T4 level is 4.5 – 10.9µg/dl)

The mean thyroid levels in group I was 9.49±2.45 µg/dl and in group II was 9.83±2.08 µg/dL for total T4.There was no statistically significant difference found in between Total T4 in group I and group II.($p > 0.05$).

Table-4: Serum TSH Levels In Patients Without Hypertension (Group-I)

And Patients With Hypertension (Group-II)

STATUS OF PERSON	PREGNANCY WITHOUT HTN (Gr.I)		PREGNANCY WITH HTN (Gr.II)	
	N=35	%	N=35	%
NORMAL	34	97.92	34	97.92
INCREASED	1	2.85	1	2.85
MEAN ± SD	2.28 ± 1.67		2.86 ± 1.84	

(Normal TSH 0.35-5.5μIU/mL)

The mean TSH levels in group I was 2.28±1.67 μIU/mL and in group II 2.87±1.85 μIU/mL. There was no statistically significant difference found in TSH levels in group I and group II. ($p > 0.05$).

DISCUSSION

Pre-eclampsia and eclampsia are severe complication of pregnancy and have an important role in pregnancy outcome. Thus this present study was designed to evaluate the role of thyroid hormone levels (T3, T4 and TSH) and thyroid autoantibodies (anti TPO-ab and anti TG-ab) in predicting pre-eclampsia.

In our study, mean Serum protein levels were found 6.71±0.88 gm/dl in patients without hypertension and 6.13±1.46 gm/dl in patients with hypertension which were similar to findings of **Deflamingh JP et al (1984), Rashid PM et al (2005) and Imoru M et al.** They found no significant difference between the normotensive and hypertensive group as found in our study.

In our study, the mean Serum albumin levels 2.49±0.90gm/dl in patients without hypertension (group-I) and 2.06±0.95gm/dl in patients with hypertension (group-II) there were no statistically significant association was found. **Donovan A et al (2009)** had similar findings as in our study. There was contradictory finding observed by **Salako et al (2009)** who reported that mean serum albumin levels (4.06±0.06 Vs 3.71±0.33gm/dL) were significantly higher in pre-eclampsia($p-<0.05$) but as evident from their values, the serum albumin levels in both the groups were within the normal range and the most number of subjects were normotensive and only 5 out of 23 subjects had pre-eclampsia. Thus as in our study group II, pregnant females with hypertension, was large in comparison to above mentioned study i.e. 35 patients. So the difference in s. albumin values may be due to difference in sample size in the two studies.

In our study, the mean thyroid levels in patients without hypertension (group-I) was 1.03±0.18 ng/mL for total T3,9.49±2.45 μg/dL for total T4 and 2.28±1.67 μIU/mL for TSH and the mean thyroid levels in patients with hypertension (group-II) were 1.02±0.16 ng/mL for total T3, 9.83±2.08 μg/dL for total T4 and 2.87±1.85 μIU/mL for TSH which was similar to findings of **Azin Alavi et al (2012)** who found no significant difference in thyroid hormone levels and thyroid antibodies levels in the 4 groups of cases including gestational hypertension, mild preeclampsia, severe preeclampsia and eclampsia which were similar to findings of our study.

Larijani et al.(2004) evaluated thyroid hormone alteration in pre eclamptic pregnant women on 39 pre-eclamptic patients and 42 healthy controls and reported, increased TSH levels and decreased free and total levels of T4 and T3 compared to healthy controls, that supports **Kaya et al (1994) and Tolino et al.(1985**) studies. None of them studied thyroid autoantibodies.

Glinoer et al. (1997) prospectively evaluated changes in thyroid function found to have a doubling in the rate of premature delivery. Specifically, 8% of controls had Preterm Delivery (PTD) compared with 16% of the auto immune thyroid disease (AITD) group

The serum protein levels (total and serum Albumin) in group I and group II there was no statistically significant difference found but group II

had lower serum protein and serum albumin levels as compared to group I while both are in normal range.

The mean thyroid levels in group I was 1.03±0.18 ng/mL for total T3, 9.49±2.45 μg/dL for total T4 and 2.28±1.67 μIU/mL for TSH while the mean thyroid levels in group II was 1.02±0.16 ng/mL for total T3, 9.83±2.08 μg/dL for total T4 and 2.87±1.85 μIU/mL for TSH further there was no statistically significant difference found in between Thyroid Hormones levels (total T3, total T4 and TSH) in group I and group II. ($p > 0.05$).

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Conflict of Interest: None

Source of Funding: Self

Ethical Clearance: The study was started after obtaining with consent from the patient. The study was cleared from Ethical Committee of Institute.

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A Study of Changes in Blood Glucose and Lipid Profile in Women after Menopause

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ABSTRACT

It is established that Menopause is accompanied by metabolic changes in women. These result in increased cardiovascular risk in post-menopausal women, which often progresses rapidly and has worse outcome. Studies exploring metabolic changes following menopause, have produced varying results. However, screening and monitoring of post-menopausal women for CVD risk factors have been suggested. This study attempts to compare Blood sugar levels and Lipid profile in post-menopausal women with those of pre-menopausal women, as these parameters can be easily assayed, monitored and can be targeted for intervention, in order to reduce CVD risk.

Method: 30 Pre-menopausal and 30 Post-menopausal women were selected from general population of Bengaluru city. Anthropometric data was collected. Fasting blood sugar and Lipid profile were assayed in all the subjects and results compared statistically.

Results: Post-menopausal women had significantly higher BMI (p<).05), FBS (p<0.05), Total Cholesterol (p<0.05), LDL Cholesterol (p<0.05) and Triglycerides (p<0.05) compared to pre-menopausal women.

Conclusion: Blood glucose levels and Lipid profile parameters are unfavourably altered in post-menopausal women.

Keywords: *Pre-menopausal women, Post-menopausal women, Cardiovascular disease, BMI, Fasting Blood sugar, Lipid profile*

BACKGROUND

Cardiovascular disease (CVD) and in particular coronary heart disease (CHD) is the leading cause of morbidity and mortality in women. The relative impact of impaired vasoreactivity of the coronary artery, increased viscosity of blood and dysregulation of automaticity and arrhythmia, is greater in women than in men. Women, more than men have their initial manifestation of CHD as angina pectoris; are likely to be referred for diagnostic tests at a more advanced stage of disease; and are less likely than men to have corrective invasive procedures. The overall morbidity and mortality following the initial ischaemic heart event is worse in women, and the case fatality rate is greater in women than men[1]. So it cannot be sufficiently emphasised that all efforts should be made to prevent such events.

During the fertile period women enjoy special cardiovascular protection. Estrogen plays a significant role in conferring this protection. Menopause though a physiological phenomenon, is marked by an abrupt interruption of estrogenic support. This results in withdrawal of the protection and causes unmasking of a new cardiovascular risk profile. This cardiovascular risk profile progresses rapidly in a hitherto unprepared organism, the post-menopausal woman.

Hyperinsulinemia, chronic hyperglycemia, insulin resistance and diabetes mellitus are independent risk factors for CVD, and are considered

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causative factors of premature mortality from CHD. Blood glucose levels are graded as an important risk factor for CVD in women. The risk of CVD deaths among women with diabetes is 3-fold greater than that of non-diabetic women[2]. Women with diabetes mellitus lose the female gender related advantage aganist CVD over men[3].

CHD is caused by an adverse compliment of the plasma lipids. High levels of total cholesterol, Low density lipoprotein cholesterol, Triglycerides, and a reduction in High density lipoprotein cholesterol all contribute to atherogenesis.

Though there is agreement on the fact that menopause is characterized by unfavourable alterations in lipid and glucose metabolism, what serum parameters are markedly altered, is yet unsettled. Also, the effect of confounding factors such as ageing and obesity has to be considered. Different studies conducted have brought out varying results. Yet it is agreed that menopause results in significantly higher risk of CVD in women. Emphasis has been laid on screening and monitoring of post-menopausal women for presence of CVD risk factors. L. N. Achie et al recommend the establishment of menopause clinics for early identification of women at risk and hence commencement of intervention[4]. So it is important to identify markers that are easily detectable and amenable to modification.

This study attempts to explore changes in parameters of glycidic and lipid metabolism after menopause, by comparing these in samples of pre and post-menopausal women.

OBJECTIVES

To compare blood glucose levels and lipid profile in samples of Pre-menopausal and Post-menopausal women.

To examine whether significant difference exists in glucose levels and lipid profile of these two groups of women.

METHODOLOGY

A comparative study was undertaken with random samples of women selected from general population of Bengaluru city. Age group ranged from 34 years to 56 years. Pre-menopausal group consisted of 30 women who were still having their regular menstrual cycles. Post-menopausal consisted of 30 women, who gave history of amenorrhoea for more than 12 months. Menopausal status was ascertained by estimation of serum levels of Follicle Stimulating Hormone. FSH > 30 mIU/ml was considered evidence of post-menopause status[5].

Exclusion criteria were pregnant status and history of irregular menstrual cycles (for pre-menopausal women), pre-existing cardiac disease, chronic liver, jaundice, hormonal disorders, hormone therapy or use of oral contraceptives (for both groups).

All subjects were explained about the study protocol and written informed consent was taken. Thorough history was obtained from each subject and clinical examination was done. Anthropometric measures of height, weight, BMI, and waist-hip ratio were recorded.

Biochemical parameters: Human Follicle stimulating hormone (hFSH) assay:

Chemiluminiscent immunoassay (CLIA) was used for the quantitative determination of hFSH levels. Levels more than 30 mIU/ml was considered evidence of post-menopausal status[5].

Fasting blood glucose: Blood sugar level was measured after 12 hour overnight fast, by Trinders method. According to American Diabetes Association guidelines, women with FBS level more than 126 mg/dL were considered diabetic[6].

Lipid profile: Blood sample collected in the morning after 12 hour fast was used for estimation of serum Total Cholesterol (TC), Low density lipoprotein (LDL), High density lipoprotein (HDL), and serum Triglycerides (TGL). CHOD-PAP (Cholesterol oxidase methopd for cholesterol, GPO-TRINDER (Glycerol phosphatase oxidase) method for TGL and Phosphotungstic acid method for HDL was used. Serum LDL was calculated using Friedwald's formula:

Serum LDL cholesterol = Total Cholesterol – (HDL cholesterol + Triglyceride/5)

NCEP ATPIII(National Cholesterol Education Program- Adult Treatment Panel III)[7] guidelines for each lipid parameter were used to reckon normalcy: TC < 200 mg/dl; LDL < 100 mg/dl; HDL > 50 mg/dl; TGL < 150 mg/dl.

Statistical analysis: Chi-square and Fisher exact test were used to test the significance of proportions between Pre and Post-menopausal women. Student t test was used to find the significance of difference in blood glucose, lipid parameters and hFSH between the two groups. p value < 0.05 was considered significant.

RESULTS

Table 1: Comparison Anthropometry parameters between two groups

Anthropometry parameters	Pre-menopause (Mean ± SD)	Post-menopause (Mean ± SD)	p value
Age in years	40.80±5.92	52.00±5.68	0.001*
HIP circumference in cm	87.33±24.98	101.47±15.19	0.002*
Waist circumference in cm	75.43±24.05	90.45±14.37	0.005*
Waist-Hip Ratio	0.84±0.08	0.85±0.05	0.5
BMI (kg/m^2)	24.84±4.56	27.29±4.90	<0.05*

* Significance at 5% student t test

It can be observed from the table that post-menopausal women were of older age, had significantly higher BMI, larger waist and hip circumferences, but WHR was not significantly different.

Table 2: Serum Follicle stimulating hormone levels in the two groups

Follicle stimulating hormone (mg/L)	(Mean ± SD)
Pre-menopause	9.17±5.8
Post-menopause	55.40±15.62
Significance	P<0.001

FSH level was significantly higher in post-menopausal women.

Table 3: Comparison of presenting features between groups

Presenting Factors	Pre menopause (n=30)	Post menopause (n=30)	p value
Hypertension	5(16.7%)	13(43.3%)	0.024*
Diabetes Mellitus	4(13.3%)	13(43.3%)	0.010*
Family history (Diabetes/Hypertension/Ischeamic Heart disease)	13(43.3%)	10(33.3%)	0.426

* Significant at 5% by Chi-square and Fisher Exact test

By subjects' self-reporting in clinical history, it was found that proportion of Hypertensives and Diabetics was significantly higher among post-menopausal women than pre-menopausal women.

Table 4: Comparison of Fasting blood sugar level in two groups

Haematology	Pre-menopause (Mean ± SD)	Post-menopause (Mean ± SD)	p value
FBS	101.80±82.64	134.50±95.38	**<0.05**

Fasting blood glucose level was higher among post–menopausal women compared to pre-menopausal women. The difference was statistically significant.

Table 5: Comparison of Lipid parameters in two groups

Lipid Parameters	Pre-menopause (Mean ± SD)	Post-menopause (Mean ± SD)	p value
Total Cholesterol	207.60±47.29	221.00±45.30	<0.05*
HDL-Cholesterol	41.93±3.74	39.03±3.69	0.7
LDL-Cholesterol	126.06±29.56	141.35±41.75	<0.05*
Triglycerides	209.85±50.81	201.00±40.58	<0.05*

*Significant at 5% by student t test

Lipid Parameters: It can be observed that TC, LDL and TGL were elevated in post-menopausal women compared to pre-menopausal women. HDL levels were not significantly different.

DISCUSSION

Our comparative study revealed the following results: After menopause, women gained weight. They had significantly larger waist circumference and greater BMI. Their FBS, TC, LDL and TGL were increased, compared to their pre-menopausal counterparts.

Changes in fasting blood sugar: Findings similar to ours have been reported by earlier investigators. Nese Ozbey et al found that FBS was significantly higher in post-menopausal women[8]. Jouyandeh Z et al also found higher FBS in post-menopausal women, especially in association with other components of metabolic syndrome[9].

Alka M Kanaya et al found in their study that women with diabetes had an approximately 75% increased risk for all cardiovascular outcomes compared with women without diabetes.[10]

Diabetes may cause a microvascular disease with a consequent silent ischaemic heart disease and diastolic failure, commonly seen in elderly women. Diabetes is an established cause of endothelial dysfunction, micro and macro-angiopathy and vessel calcifications, which may be more detrimental to females because of anatomically smaller and more fragile coronary and peripheral arteries[11].

Changes in lipid profile: There is wide variation in results of earlier studies done with similar purpose. Nese Ozbey et al found that only Total cholesterol was significantly higher in post-menopausal women, compared to age-matched pre-menopausals[8]. Akahoshi M et al, concluded from their study that only serum cholesterol increased significantly at the time of menopause[12]. Pao-Ling Torng et al concluded that of the changes in lipid profile that occur during the transition to menopause, the increase in TC levels was the only significant change resulting from menopause. Elevation of TC though influenced by age, was found to be an independent anthropometric variable which changed with menopausal status. They also reported that levels of LDL were dependent on age and BMI, apart from menstruation status[13]. On the other hand, Do KA, et al reported that HDL cholesterol appeared to be the only biological risk factor for CVD that changes as a direct consequence of menopause[14].

Al- Dahhan et al, in their study found that TC and LDL cholesterol were significantly higher in women with natural and surgical menopause compared to pre and peri menopausal women. They found no significant intergroup difference in HDL[15]. Igweh JC et al study found no significant difference in TC and TGL levels in pre and post-menopausal women. HDL and VLDL levels significantly decreased whereas LDL levels were significantly increased in post-menopausal group[16].

Results of our study are comparable to those of Shende S et al who found that TC, TGL, LDL and VLDL levels were significantly higher and HDL levels significantly lower in post-menopausal women[17]; Maulik S. Varu et al found significant increase in TC and LDL with significant decrease in HDL in post-menopausal women[18]; Geetanjali Bade et al found that serum levels of TC, TG and LDL-C were significantly higher in post-menopausal women in comparison to their pre-menopausal counterparts, irrespective of BMI. Similarly, HDL-C levels were significantly

lower in post-menopausal women as compared to pre-menopausal women of similar BMI. Since they found similar changes in women of different BMIs, they supposed that the difference in hormonal status is the probable cause of altered lipid profile[19].

Estrogen has been said to confer cardiovascular protection in pre-menopausal women through several mechanisms. Estrogen exerts cardioprotective action by maintaining high level of high density lipoprotein cholesterol and lowering the low density lipoprotein cholesterol and triglycerides[20]. A major effect of estrogen on lipid metabolism is up-regulation of LDL receptors, resulting in increased clearance of LDL particles by hepatocytes and reduction in plasma LDL-C. Estrogen can reduce VLDL and Chylomicron remnant concentrations by increased elimination through hepatic clearance. Estrogen increases HDL cholesterol by several mechanisms, which mainly includes increased hepatic production of apolipoprotein A and decreased hepatic elimination of HDL2 cholesterol by reducing activity of hepatic Lipase[21].

Abrupt withdrawal of Estrogen support in the post-menopausal period reverses these beneficial effects and result in an adverse metabolic profile characterised by increased TC and LDL level and decreased HDL level.

It is suggested that loss of ovarian function also induces a reduction in resting metabolic rate, fat free mass and an increase in fat mass and abdominal adipose tissue accumulation.

The point of common agreement for all studies is that menopause is accompanied by significant increase in cardiovascular risk for women. It is further noteworthy that alterations in lipid and glucose metabolism are interlinked, and form components of a larger constellation of CVD risk factors. This increased CVD risk is of multifactorial causation, involving alterations in body weight, body fat distribution, altered vascular function resulting in hypertension, altered glucose and lipid metabolism, which together constitute the so called "metabolic syndrome".

A high prevalence of metabolic syndrome has been seen in post-menopausal women[9].

Marie-Eve Piche et al study in post-menopausal women suggested that visceral fat and insulin resistance both mediate the metabolic risk profile and that the combination of high visceral adipose tissue and high insulin resistance in post-menopausal women is the most detrimental combination of factors for the metabolic health of these women[22].

As all the above risk factors tend to occur in cluster, CVD risk increases multiple-fold in post-menopausal women. It can be safely proposed that screening and monitoring of post-menopausal women by simple haematological assays such as blood sugar and lipid profile will help identify those at greater risk of CVD. Early intervention targeting modifiable risk factors, in the form of patient education, diet changes, increased physical activity, and pharmacotherapy where indicated, may go a long way in prevention of cardio-vascular morbidity and mortality in this particularly vulnerable population.

CONCLUSIONS

Glucose and Lipid metabolism are adversely affected in post-menopausal women. This manifests as higher fasting blood sugar, Cholesterol, LDL and TGL in post-menopausal women compared to pre-menopausal women.

These metabolic changes place post-menopausal women at a greater risk of Cardiovascular morbidity and mortality.

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A Comparative Study of Blood Pressure, Oral Glucose Tolerance Test and Lipid Profile between Non Diabetic Obese and Non-Obese Women

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ABSTRACT

Obesity had been the most prevalent disorder in developed countries, but now considered to be the common nutritional disorder in both developing and developed countries. To compare blood pressure, glucose levels and lipid profile between non diabetic obese women in comparison to non-diabetic non obese women, 50 obese non diabetic and 50 non obese non diabetic women residing in Tirupathi were selected and were subjected to Blood pressure measurement, OGTT and Lipid profile. There was significant increase in the systolic blood pressure, FBS, PPBS, triglycerides and total cholesterol levels in obese non diabetic women compared to non obese non diabetic women($p<0.05$). However there was significant decrease in HDL levels in obese women. Results suggests that obese non diabetic women had increased risk of developing metabolic disorders compared to non obese non diabetic women.

Keywords: *Obesity, blood pressure, lipid profile.*

INTRODUCTION

Obesity had been the most prevalent disorder in developed countries, but now obesity is the common nutritional disorder in both developing and developed countries. It is considered as a chronic modifiable disorder, which could prevent major diseases like Ischemic Heart Disease. It is estimated that 20 to 40% of the adults and 10-20% of children and adolescents are obese[1-3]. Clinical observations have long suggested, a connection between obesity and a variety of illnesses. For a given height & weight women have 10% more fat than men and thus they are more prone for obesity related complications. The prevalence of Diabetes Mellitus is three times higher in obese people than non obese people.The syndrome complex of Obesity, Hyperglycemia, Hyperinsulinemia, dyslipidemia and Hypertension is now considered as Metabolic Syndrome, which is the prediabetic condition, and the individuals are more prone to develop atherosclerosis, hypertension and other cardiovascular diseases. Obese people have hyperinsulinemia & Insulin resistance, the resistance due to defect in insulin receptors and post receptor defects [4-6]. Hence the present study was undertaken to study Oral glucose tolerance test, Estimation of levels of cholesterol, triglycerides, LDL, HDL & Recording Blood Pressure in nondiabetic obese women in comparison to non-diabetic non obese women.

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METHOD

The study is a hospital based, observational study with the primary objective to compare a few physiological parameters among obese and non-obese women. The study was conducted in Tirupati Municipality of Chittoor District in A.P. Institutional Ethics Committee's approval was obtained prior start of study. Informed consent from subjects were obtained. Subjects were randomly selected between age groups of 25 to 45 years. 50 non obese and 50 obese women were included in the study. All the women were Non diabetic & Non-hypertensive. They did not have any significant illness or other

acute symptoms and were not taking medications with a known effect on blood glucose and lipids (including oral contraceptives and other hormones). Obese women with known endocrinal disorders were not included. Based on the Height and weight, by using Quetlet's index BMI is calculated. Obesity was defined as a body mass index (BMI) >30kg/m^2 .

Each subject was advised to take normal carbohydrate diet 3 days prior to oral glucose tolerance test. The test was performed after overnight fasting for 10 to 14 hours. A fasting blood sample was taken. The samples were estimated for glucose by Somogyi Nelson method.

Fasting blood samples were taken from all the subjects and lipid profile was done as follows: (i) Total serum cholesterol (CHOD-POD Method); (ii) Serum triglycerides (GPO/PAP Method); (iii) Serum High density lipoproteins (HDL) (Phosphotungstic Acid Method); (iv) Serum Low density lipoproteins (LDL) (Freidwald equation) . B.P. is recorded by using sphygmomanometer tied to the left arm, first by palpatory method, followed by auscultatory method. Statistical analysis was carried out in SPSS version 11.0. Student t-test was used to compare the variables between controls. The difference was considered significant if the p-value was <0.05 & highly significant if the p-value was <0.001. The data was analyzed and valid conclusions were drawn.

RESULTS

The data was analyzed and represented as mean and SD. Table 1 shows the age and BMI profile of obese and non obese subjects. As per table 2, blood sugar was significantly higher in non diabetic obese women compared with non diabetic-non obese women (p<0.005).TC, HDL, TGL and LDL were significantly higher in non diabetic obese women compared with non diabetic-non obese women (Table 3). Even the SBP was higher in obese women compared with non obese women, which was statistically significant (Table 4).

DISSCUSSION

The morbidity and mortality associated with being over weight or obese have been known to the medical profession for more than 2000 years[1] . Excessive body weight has become a major problem in industrialized and developed countries, where it has reached the proportion of an epidemic[2,3]. Individuals from disadvantaged communities are also not exempted and are at a substantial risk of obesity and its complications[4]. A number of large epidemiologic studies have proved that mortality increases with obesity[5–7]. Obese individuals are prone to many cardiovascular risk factors. Type 2 DM is strongly associated with overweight and obesity[8,9]. Lipid metabolism is also adversely affected in obesity[3,10]. The Prevalence of these risk factors substantially increases with increasing BMI[11].

The present study shows significantly decreased levels of HDL in obese compared to non-obese women. Similar findings was suggested by previous study (Bertsias et al., 2003) [12].Total cholesterol, triglycerides and LDL was higher in obese women compared to non obese women. This is similar to the previous studies (Hu et al., 2000[13], Van Pelt et al., 2002 [14]), but the strength of statistical significance differed in different studies, which may be attributed to different age distribution of subjects as well as to the ethnic variations in fat distribution. In obese individuals the accompanying hyperinsulinaemia due to insulin resistance may be responsible for changes in lipid and lipoprotein concentration(Goran and Gower, 1999 [15]). Lipid mobilization from the fat depots and release of FFA is mainly regulated by catecholamines and insulin.

Catecholamines regulate lipolysis in human adipocytes through stimulatory β (mainly β_3) and inhibitory $\alpha_2$receptors. Insulin has an inhibitory effect on lipolysis. Central fat depots show higher density and sensitivity to stimulatory β receptors, while lower density and sensitivity to inhibitory α2 receptors. These are also less sensitive to the anti-lipolytic effect of insulin. Thus all these factors combined together may explain the altered lipid profile level in individuals with central obesity. Moreover in terms of blood glucose levels, an increase value was observed in non diabetic obese women compared to non obese women. These findings are in accordance with the results from other western studies [16,17]. Several studies on Asian population also showed the same findings [18,19]. These result indicate that irrespective of age obesity plays an important role in development of glucose intolerance. Results also showed that mean SBP and DBP were higher in obese women compared to non obese women. It is proven that metabolic syndrome is defined as a

group of cardiovascular disease risk factors including impaired glucosetolerance, dyslipidaemia and hypertension.

In our study mean systolic BP showed a significant difference between obese and non obese women. The non diabetic obese women had higher recording compared to non obese. These findings are in accordance with the results from other westernstudies [22,23]. Further , Overweight and obesity lead to many complications, including diabetes, dyslipidemia and hypertension.

Most of the landmark studies[10] revealed that obesity was an independent risk factor for incidents of cardiovascular mortality in men and women. Obesity especially abdominal obesity is associated with an atherogenic lipid profile. It is expected that as the BMI increase, the frequency of these complications will also increase. As Central distribution of body fat, particularly intra-abdominal fat is more a risk factor for obesity related ill health than peripheral distribution(WHO, 2000). Thus it can be concluded by our study that, central obesity is associated with the abnormal lipid profile in non diabetic obese women . It is documented that Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) measure the fat distribution more correctly but their high cost and radiation hazards prevent their use in large scale epidemiological studies, clinical study and self assessment. So this investigation was also a limitation of the study.

Moreover postmenopausal women have increased prevalence of many diseases due to physical changes the later middle ages compare to men. Steady exercise has been emphasized to prevent obesity and proper body composition. Like western nations our population is alsoat risk of obesity. BMI should be routinely checked in clinical practice and epidemiological surveys. So women need formal guidance about healthy life style especially about diet and exercise

Table-1 Age and BMI of obese and non obese women

	Type	Mean	Std. Deviation
Age	Obese	24	9.45
	Normal	25	7.67
BMI (kg/m^2)	Obese	35.50	3.20
	Normal	21.32	2.59

Table 2 Glucose Tolerence Test in obese and non obese women

	Type	Mean	Std. Division	p-value
FBS (mg/ dl)	Obese	94.43	13.16	0.062^{NS}
	Normal	88.09	8.43	
1 Hr	Obese	136.70	9.53	0.005^{*}
	Normal	129.27	7.26	
2 Hr	Obese	111.53	9.46	0.282^{NS}
	Normal	107.64	7.82	

Table 3 Lipid profile of obese and non obese women

	Type	Mean	Std. Division	p-value
TC (mg/dl)	Obese	195.76	23.66	0.002^{*}
	Normal	169.64	23.72	
HDL (mg/dl)	Obese	36.36	7.43	0.044^{*}
	Normal	41.61	4.12	
TGL (mg/dl)	Obese	152.22	29.11	0.045^{*}
	Normal	131.05	37.43	
LDL (mg/dl) `	Obese	126.39	22.22	0.006^{*}
	Normal	108.55	19.42	

Table-4 Systolic (SBP)and Diastolic (DBP) blood pressure of obese and non obese women

	Type	Mean	Std. Division	p-value
SBP(mm of Hg)	Obese	122.44	10.67	0.009^{*}
	Normal	113.91	9.96	
DBP(mm of Hg)	Obese	78.44	5.66	0.489^{NS}
	Normal	76.46	3.56	

CONCLUSION

This study concludes that obese non diabetic women are at increased risk of developing metabolic disorders like impaired glucose tolerance, hyperlipidemias and hypertension compared to non obese non diabetic women.

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Conflict of Interest: We declare that there is no conflict of interest.

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Can Hypertension be a Cause of Irritable Mood in Middle-Aged Women?

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ABSTRACT

Background: Hypertension in middle-age can lead to greater drop in mental functions later in life. Not many studies have been done to evaluate hypertension as a cause of mood disorders manifesting with irritability as a symptom. An attempt has been made to study and score irritability in hypertensive and normotensive middle-aged women and assess a possible association between hypertension and irritable mood.

Objectives: To test the hypothesis that irritability score is higher among middle-aged hypertensive females compared to the normotensive controls.

Materials & Method: Study group consisted of 42 hypertensive females (mean SBP=152±8, mean DBP=92±4 mm Hg) and 44 normotensive females (mean SBP=118±6, mean DBP=80±4 mm Hg) aged 35-50 years. Irritability was scored using Born-Steiner self –rating Irritability Scale. It has 2 components- Irritability scoring and visual analogue scale.

Results: Statistical analysis shows that irritability scoring is significantly higher ($p < 0.0001$) in hypertensive middle-aged women (21.67± 8.6) compared to controls (9.6±6.1). Visual analogue scale shows that irritability is causing a problem to oneself and in relation with family, friends and community. Also, the severity of irritability as a trait and transient state are both significantly higher in cases compared to controls.

Conclusion: Irritability is higher in hypertensive middle-aged females compared to controls. Haemorrhagic and ischaemic cerebrovascular changes in the supratentorial region & brain metabolism in hypertension reducing neurotransmitter activity may be the reasons for altered mood manifesting with high irritability score.

Keywords: *Irritability, Hypertension, Females.*

INTRODUCTION

Hypertension is an important public health challenge worldwide. Globally, the overall prevalence of raised Blood Pressure in adults aged ± 25 years was around 40% in 2008.

Irritability is defined as a proneness to anger, annoyance or impatience.[1] It is characterized by a state of physical and psychological tension that may suddenly and rapidly escalate and may include reduced control over temper, a heightened or excessive sensitivity to external stimuli and irascible verbal or behavioural outbursts — even explosive aggressiveness.[2] It is a prominent feature associated with hypertension and can be an important predisposing factor for the development of hypertension. Hypertension and irritability in fact form a vicious cycle. As per review of reports of study from FDA 0.34% of hypertensives have irritability of which 66.56% are females.[3]

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Previous studies have also shown that hypertension in middle-age leads to greater drop in mental functions later in life.[4,5.6]

The current study is intended to score and compare the irritability scores of hypertensive and normotensive middle-aged women. It is hypothesized that middle-aged hypertensive females have higher irritability scores compared to the normotensive controls.

OBJECTIVES

1. To score the irritability using Born-Steiner self-rating Irritability scale in hypertensive and normotensive middle-aged females

2. To compare the scoring between hypertensive and normotensive middle-aged females.

METHODOLOGY

This work is a cross-sectional study done on 86 middle-aged females from Bangalore who were selected based on the eligibility criteria from the general population.

Inclusion criteria

- Females
- 35-50 years
- Housewives
- Cases: Known cases of hypertension
- Controls: Normotensives

Exclusion criteria

- Working women
- Pre-menstrual phase
- Uncontrolled hypertension (BP >180/ 100mmHg)
- Established cardiovascular diseases like stroke and IHD
- Antihypertensives other than beta-blockers, calcium channel blockers, ACE inhibitors and diuretics.
- Psychiatric illnesses like schizophrenia, bipolar disorders, anxiety disorder
- Head trauma or brain tumours
- Neurological disorders like epilepsy and migraine
- Chronic medical conditions like diabetes mellitus, hypothyroidism or tuberculosis
- Arthritis or other chronic pain disorders
- Antipsychotics, antidepressants, sedatives and hypnotics
- Smoking, alcohol or other forms of drug abuse

The study group consisted of 42 hypertensive females (mean SBP=152±8, mean DBP=92±4 mm Hg) and 44 normotensive (mean SBP=118±6, mean DBP=80±4 mm Hg) middle-aged females fulfilling the eligibility criteria. Written informed consent was taken followed by relevant history taking and general physical examination. Born-Steiner self-rating irritability scale[7] was administered to all the age matched participants.

Scale contained sl no 1-14 likerd questions which were specially designed for females and sl no 15-21 visual analogue scale which shows percentage of affection. The scale used is easy to use and interpret.

Sl no 1-14 questions which has scoring for how you are feeling in the past week is answered as not at all, a little or some of time, often and most of the time. They are scored 0, 1, 2 and 3 respectively. Maximum score being 42 and least being 0. If their score is between 1-14 it is considered mildly irritable,15-28 moderately irritable and 29-42 severely irritable.

Serial no 15-21 have visual scale which has 100mm horizontal line for each criteria . Subject has to mark percentage of affection on that horizontal line, starting point on the line is 0% affection and the end point of line being 100% affection .Later using a ruler scale we can divide the line into 10 segments, each segment represents 10% .Using this the percentage of affection is decided.T his scale shows affection of relationships with family, affection of daily activities, their ability to deal with frustration, affect on their self-esteem, affect on their social relationship, how they are feeling at that moment and how they rate their 'usual self'.

Statistical treatment was given to the collected data by using Mean, Standard deviation, percentage and also the p-value to analyse the significance. ($p < 0.05$ considered significant)

Table 1 – Age distribution in cases and controls

	HYPERTENSIVE FEMALES	NORMOTENSIVE FEMALES	P VALUE
AGE (in years)	41.09±3	39.9±3.2	0.08

Table 2- Irritability scoring (ISCR) of the subjects

	HYPERTENSIVE FEMALES (mean±SD)	NORMOTENSIVE FEMALES (mean±SD)	P VALUE
ISCR	21.67±8.6	9.6±6.1	<0.0001*

Table 3- Percentage of subjects in different levels of irritability

SCORE	0	1-14	15-28	29-42
RATING	NOT IRRITABLE	MILD IRRITABLITY	MODERATE IRRITABILITY	SEVERE IRRITABILITY
HYPERTENSIVE FEMALES	0	8 (19%)	26 (62%)	8 (19%)
NORMOTENSIVE FEMALES	0	36 (82%)	8 (18%)	0

Table 4- Visual Analogue Scores showing Percentage Affect with respect to different criteria

Criteria	Hypertensive women (mean ±SD)	Normotensive women (mean ±SD)	P value
Relationships with family?	60±35.38	11.26±12.35	<0.0001*
Daily activities?	55.24±39.45	23.5±24	0.003*
Ability to deal with frustration?	71.43±27.47	20.58±20.29	<0.0001*
Self-esteem?	69.14±32.01	14.18±18.04	<0.0001*
Social relationships?	47.76±32.48	13.23±16.28	<0.0001*
How would you rate yourself AT THIS MOMENT?	38.38±36.88	17.09±21.43	0.002*
How would you rate your USUAL SELF?	59.33±29.6	27.67±25.31	<0.0001*

RESULTS

Tab 1 shows that cases and control are age-matched with p value 0.08.

Tab 2 show mean Irritability Score of Hypertensive middle aged women & normotensive controls. It is seen that hypertensive women have significantly higher score (21.67±8.6) compared to normotensive controls (9.45±6.2) with p <0.0001. Tab 3 shows the frequency distribution of the study group with different scoring. It is seen that 62% of hypertensive women are moderately irritable compared to 18% of controls. Also, 19% of the hypertensive females have severe irritability while none of the normotensive females fell into this category. Most of the controls (82%) were only mildly irritable. Tab 4 with visual analogue scoring showed irritability has affected hypertensive female relationship with family, their daily activities, their ability to deal with frustration, their self-esteem and their social relationship. It also showed that hypertensive females rate themselves on higher limit of irritable scale.

DISCUSSION

We have found that irritability scores are significantly higher among middle aged hypertensive females compared to the normotensive controls. Also, the Visual Analogue Score (Tab 4) highlights the percentage burden of irritability which is significantly higher among the hypertensive group. From a pathogenetic perspective, our observation offers evidence for implicating hypertension as a cause of mood disorders manifesting with irritability.

Siever and Davis hypothesized that affective disorders are syndromes of neurotransmitter dysregulation.[8] Fujishima et al in their study on Cerebral Blood Flow (CBF) and brain function found that CBF was significantly lower in hypertensives than normotensives in the supratentorial regions such as cortex, striatum and thalamus. Cerebral metabolic rate for oxygen in these areas was also significantly lower in the hypertensive group suggesting that neuronal function or neurotransmitter activity is reduced in these patients.[9] Other studies on morphological changes in the brains of hypertensives suggest that long-standing hypertension per se is commonly associated with white matter lesions which correspond to demyelination and arteriosclerosis in the brain and seem to be responsible for a decline in intellectual function in elderly hypertensives.[10,11] Wei et al found that cerebral glucose utilisation was lower in adult spontaneously hypertensive rats than normotensive rats.[12] Hypertension is a major risk factor for atherosclerosis and vascular remodelling that potentiates cerebrovascular haemorrhage or silent cerebral infarction.[13]

Mood is related to amount of norepinephrine and dopamine available at synapses of brain.[14] Antihypertensive drugs can also cause affective disorders by depletion of neurotransmitters.[15] Taken together these results indicate that all these factors cumulatively lead to hypertension induced irritability.

Limitations of this study include small sample size and possible bias due to missing data on socio-demographic history. These limitations allows only explorative analyses from this study. It can be concluded from our study that hypertensive women are more irritable and irritability is negatively affecting their relation with their family and community. These finding may provide background for investigations of the relationship between vascular lesions and specific affective and cognitive symptoms in hypertension.

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The Study on Probable Relationship between Blood Group and Hypertension

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ABSTRACT

Hypertension is considered as one of the most important cardiovascular risk factors which precipitates the cardiovascular disorder. There are some reports indicating the probable relationship between ABO blood group with coronary artery disease. But such reports demonstrating the relationship between ABO blood group and hypertension are still uncertain. So the objective of the present study is to find out whether there is any relation of blood group in occurrence of hypertension to recommend the appropriate preventive measure for this deadly disease.

The hypertensive patients of both sexes of the age in between 18 to 55 of Haldia and Tamluk subdivision reporting to our associated Dr. B. C. Roy Hospital, Haldia are randomly selected for this study. The Blood group and the Blood pressure was measured and the result was compared with that of the normotensive subjects and also with reference population of this belt. It is observed that there is no significant relationship between blood group antigens and the occurrence of hypertension in the studied population.

Keywords; *Hypertension , Blood group, cardiovascular disease.*

INTRODUCTION

Cardiovascular diseases are the most important causes of morbidity and mortality especially in the developing country and hypertension is considered as one of the most important cardiovascular risk factors[1],[2]. Since uncomplicated hypertension is generally asymptotic peoples remain unaware about this even if the existence of this disease. But as the hypertension and its deadly complication imply a large burden on our healthcare system diagnosis of hypertension is important as well as its prevention or management.

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It is now well established that the blood group exhibits some relation with some diseases like nasopharyngeal carcinoma[3], duodenal ulcer [4], gastric cancer [5,6,7], epitaxis[8],anemia [9] and even cardiovascular diseases like coronary artery diseases and cardiac ischemia [10,11,12]. Several others also investigated the probable association between blood group and hypertension with conflicting results [13,14,15]. Whereas some other did not find any correlation between blood group with occurrence of hypertension[16]. In view of these conflicting results we conducted this cross sectional study to determine the possible association between ABO & Rh antigens with hypertension as any association ship between particular blood group and hypertension may be helpful for prevention or management of this deadly disease .

MATERIALS & METHOD

For this study the Type II hypertensive patients of both sexes of the age in between 18-55 years of Haldia and Tamluk subdivision reporting to our associated

Dr. B. C. Roy Hospital, Haldia are randomly selected. The inclusion criteria of the hypertensive patients of both sexes are- more than 18 years of age, non pregnant, non smokers, non alcoholic and economically similar group. The patients of DM and subjects suffering from renal hypertension whose serum creatinin levels more than 1.5 mg/dl and also thyrotoxicosis pateints were not included in the study[17]. Each subject filled the consent form containing self reported information about their sex, age, physical activity, smoking habits, drinking habits, and history of coronary artery disease, ischemic heart disease, diagnosed renal hypertension, thyrotoxicosis etc if any.

The blood pressure of the subjects was measured by sphygmomanometer in sitting positions after 10 min rest. The mean of two readings of systolic and diastolic pressure recorded in two occasions was taken as the final measure. As per WHO criteria a systolic pressure >140 mmHg and /or diastolic pressure >90 mm Hg was considered hypertension and included under the test group. The known hypertensive subjects under antihypertensive medication are also included in our study as test group. Whereas the normotensive subjects having systolic pressure < 140 mm Hg and Diastolic pressure within the range of 60-88 and without any antihypertensive medication are considered as control group. The work is approved by the Institutional ethical committee.

With all aseptic precaution the whole blood of suspected anemic patients was collected in fasting condition by venipuncture using disposable syringes. The ABO blood group and Rh factor of the subjects were determined using the **Tile or Slide testing method** [18] with the help of antisera A, antisera-B and antisera-D (Span Diagnostics Ltd. Surat,India) and finally the frequency of occurrence of anemia in relation to ABO blood group and Rh factor was assessed statistically. As per the standard protocol the result was expressed as percentage which is considered as frequency distribution of each ABO blood group and Rh factor. To establish the relationship in between the blood group and hypertension the frequency distribution (Observed frequency) of blood group among the entire hypertensive population was compared with that of general normotensive population by Chi Square test [19] and a P value of 0.05 was considered significant for all the statistical test conducted.

RESULT

The study indicates that neither the frequency distribution of ABO group nor the Rh factor of hypertensive subjects significantly differ from the normotensive group (Fig.1). Statistical analysis by Chi Square test reflects that the Chi Square value at degree of freedom 3 is 0.58 which is far less than the 5% level of significance (0.05). Hence the frequency distribution of hypertensive group does fit to the frequency distribution of normotensive group or in otherwise there is no significant difference between the frequency distribution of hypertensive group and frequency distribution of non hypertensive population.

There is also no significant difference between the frequency distribution of blood group of hypertensive subjects from that of reference population of this belt (Table-1). It is also observed that there is no significant difference between frequency distribution of ABO and Rh factor of hypertensive male and female with that of normotensive group (Table-2).

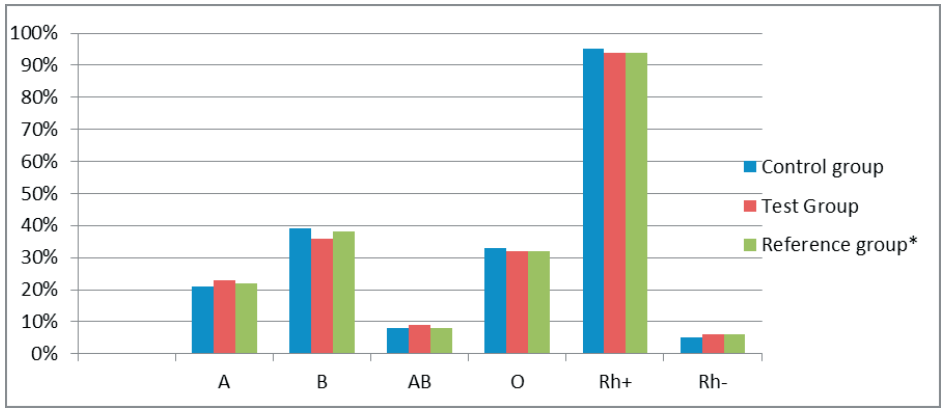

Fig.1 : Bar diagram showing frequency distribution of blood group among different groups

Table-1 : frequency distribution of blood group among different groups

Blood group	Control group(286)		Test Group(243)		Reference group (21)*	
	N	distribution	N	distribution	distribution	
A	59	21%	55	23%	22%	
B	111	39%	88	36%	38%	
AB	24	8%	23	9%	8%	
O	93	33%	77	32%	32%	
Rh+	272	95%	228	94%	94%	
Rh-	14	5%	15	6%	6%	
N= Absolute number of individuals			**(21) Basak et.al 2014***			

Table 2: Sexwise distribution of blood group among control and test group

	Control group (286)				Test Group (243)			
Blood group	**Male**		**Female**		**Male**		**Female**	
	N	**distribution**	**N**	**distribution**	**N**	**distribution**	**N**	**distribution**
A	32	21%	27	20%	30	23%	25	22%
B	60	39%	51	38%	46	35%	42	38%
AB	12	8%	12	9%	13	10%	10	9%
O	48	32%	45	34%	42	32%	35	31%
Rh+	152	94%	120	94%	124	93%	104	95%
Rh-	10	6%	7	6%	9	7%	6	5%
N= Absolute number of individuals								

DISCUSSION

Numerous previous studies have demonstrated the association between ABO blood groups particularly non O blood groups and cardiovascular diseases[20,13]. Whereas some others reported that occurrence of cardiovascular diseases like Coronary Artery Disease (CAD) and ischemia is high among O blood group individuals. But our study reveals no significant association ship between any blood group with hypertension which corroborate with other study[17]. It is also indicated that there is no significant difference between the sex wise frequency distribution among hypertensive and normotensive subjects and the frequency of occurrence of hypertension among the studied population matches with the reference frequency distribution of blood group of the general population of this belt.

It is therefore concluded that there is no significant relationship between blood group antigens and the occurrence of hypertension. The positive correlation found by some others with CAD and cardiac ischemia which is due to lower activated partial thromboplastin, VWF factor in non O blood group individuals and the positive correlation found in between ABO blood group and hypertension may be due to the family history and type of the individuals or other factors like stress, strain and etc.

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Effect of Duration and Quality of Sleep on Glycemic Control in Type 2 Diabetes Mellitus

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ABSTRACT

Background : The sleep disorder plays a pivotal role in the occurrence and development of diabetes via neuro-endocrine metabolic pathway. People suffering from a sleep disorder - sleep quality or sleep quantity, experience reduction in the insulin sensitivity and consequently, elevated blood glucose, aggravating the progress of diabetes.Hence we examined whether short or poor sleep is associated with glycemic control in Type 2 DM.

Aims and Objectives :

1.To assess the duration and quality of sleep on glycemic control in Type 2 DM.

2.To compare the same in males and females Type 2 DM.

Method : This study includes 40 Type 2 DM subjects with 20 males and 20 females. For each patient, data regarding age, gender, duration of diabetes, and use of medications were recorded. A detailed physical examination was performed, glycosylated hemoglobin (HbA1c) values and comorbid conditions were noted. Quality and quantity of sleep was evaluated by Pittsburgh Sleep Quality Index (PSQI) questionnaire.

Results : Mean age was 51.3 yrs, mean HbA1C was 9.8(±1.6)%.The mean difference between preferred and actual sleep was 2.4(±0.9)hrs.The mean PSQI score is 12.02(±1.7) and is indicative of poor quality of sleep.There was significant positive association between HbAIC and PSQI (r=0.47;p=<0.01) and between HbA1C and sleep debt (r=0.6;p=<0.01).

Conclusion : A statistically significant association between both quality and quantity of sleep in Type 2 DM subjects with poor glycemic control and also high perceieved sleep debt among females was found.These findings suggest that sleep hygiene should be a part of diabetes management.

Keywords: *Sleep duration,Glycemic control,Diabetes Mellitus.*

INTRODUCTION

Diabetes is a global pandemic with significant human, social, and economic impacts. According to the International Diabetes Federation (IDF),more than 371 million people across globe have diabetes and this figure is predicted to raise over 592 million by 2035[(1)]. It is estimated that 61.3 million people aged 20-79 years live with diabetes in India (2011 estimates) and this number is expected to increase to 101.2 million by 2030[(2)].

Poor sleep is a common feature of type 2 diabetes.[(3)] Difficulty initiating and maintaining sleep, daytime sleepiness, and poor sleep quality have been reported by persons with type 2 diabetes[(4)]. The Physiological data suggest that short-term partial sleep restriction leads to striking alterations in metabolic and endocrine function including

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decreased carbohydrate tolerance, insulin resistance, increased sympathetic tone, and elevated cortisol concentrations[5].

Based on the above evidence we hypothesize that reduced sleep duration or quality increases the severity of existing diabetic condition.

We therefore performed this study to assess the effect of quality and duration of sleep on glycemic control in Type 2 DM.

AIMS AND OBJECTIVES OF THE STUDY

1. To assess the duration and quality of sleep on glycemic control in Type 2 DM.

2. To compare the same in males and females Type 2 DM.

SUBJECTS AND METHOD

Subjects: The study was carried out at the BMCRI,Bangalore.The study population was comprised of type 2 diabetes patients attending the Outpatient Clinic. The total study sample included 30 patients. All these subjects were not recently diagnosed and had blood tests within 90 days of the interview. All participants signed an informed consent form before inclusion in the study.

Inclusion criteria: 1)Type 2 DM 2) Age : 30 – 50yrs

Exclusion criteria: 1) Type 1 DM 2) unable to give consent 3) smokers 4) alcoholics 5) weight loss > 6kgs in past 6months 6) Hepatic and Renal impairment 7) Ischemic Heart Diseases 8) Subjects on hypo-lipidemic drugs 9) Hypertensives.

STUDY TOOLS

General information questionnaire : Participants' demographic information such as gender, age and life style (smoking, drinking and exercise) were evaluated. Additionally, data on chronic diabetic complications and family history were collected to assess their disease status. Physiological and biochemical indicators such as HbA1c were obtained using case sheet.

Research tool: In all patients, quality of sleep was evaluated by administering the PSQI through an interview. The PSQI is a self-report questionnaire that assesses sleep quality and quantity over a month long period. The questionnaire consists of 19 self-rated questions. The 19 questions were categorized into 7 components, which are graded on a scale that ranges from 0 to 3. The PSQI components are as follows: subjective sleep quality (C1), sleep latency (C2), sleep duration (C3), habitual sleep efficiency (C4), sleep disturbances (C5), use of sleeping medication (C6), and daytime dysfunction (C7). The sum of scores for these 7 components yields one global PSQI score, which ranges from 0 to 21, where the highest score indicates the worst sleep quality. A global PSQI ≥ 5 has a diagnostic sensitivity of 89.6 and specificity of 86.5 in distinguishing poor sleepers (PSQI ≥5) from good sleepers (PSQI ≤5) . HbAlc values less than 7.0% were considered to be in the normal range. Patients with GQ (PSQI score ≤ 5) were compared with patients with PQ (PSQI ≥ 6).After extracting sleep duration from the PSQI questionnaire we categorized patients into 3 groups based on their sleep duration within the last one month: short (SSD, < 6 hours daily) medium (MSD, 6–8 hours daily) and long sleep durations (LSD, > 8 hours daily) and comparisons between the three groups were made.

RESULTS

1) General information: Mean age 51.3 yrs, mean HbA1C 9.8(±1.6)%, mean difference between preferred and actual sleep 2.4(±0.9)hrs, mean PSQI score is 12.02(±1.7) is indicative of poor quality of sleep.There was significant positive association between HbAIC and PSQI (r=0.47;p=<0.01) and between HbA1C and sleep debt (r=0.6;p=<0.01) and short sleep duration(r = -0.6;p=<0.01).

Table 1: Baseline characteristics of subjects

Sl.no	Parameters	Mean ±SD
1	Mean age(yrs)	51.3±4.5
2	HbA1C(%)	9.8±1.6
3	PSQI	12±3.3
4	Sleep debt(hrs)	2.4±0.8
5	Sleep duration(hrs)	4.5±0.83

2) Relationship between sleep duration,PSQI and sleep debt with glycated hemoglobin: There was significant positive association between HbAIC and PSQI (r=0.47;p=<0.01) and between HbA1C and sleep debt (r=0.6;p=<0.01).

Table 2: Correlation between glycated hemoglobin, sleep duration,PSQI and sleep debt.

Correlation	r	p
HbA1C and sleep duration	-0.59	0.01*
HbA1C and PSQI	0.7	0.01*
HbA1C and sleep debt	0.6	0.01*

3) Comparision between short and medium sleep duration in males and females: Table 3 shows baseline characteristics with 77% of diabetics with short sleep duration. Short sleep duration is associated with higher HbA1C in both males and females which was statistically significant.

Table 3: Baseline characteristics based on sleep duration in males and females

Gender	Parameters	Medium sleep duration (>6hrs)	Short sleep duration (<6)hrs	P-value
Male	Age	53±5.6	54.15±2.5	0.5
	HbA1c	8.3±1.6	9.86±1.1	0.02*
	PSQI	4±0.51	11.2±1.26	0.01*
Female	Age	50.5±10.6	48.7±3.9	0.6
	HbA1c	6.7±0.42	10.68±1.24	0.0005*
	PSQI	5	13.8±1.09	0.001*

4) Comparision of PSQI components between males and females: Subjective sleep quality, sleep efficiency and sleep latency were significantly worse in females than in males (Table 4). The PSQI global score was also poorer in females which is significant.

Table 4: Comparision of PSQI components between males and females

PSQI Component	Male	Female	p value
Subjective sleep quality	1.93±0.79	2.6±0.4	0.05*
Sleep latency	2.06±0.79	2.83±0.38	0.001*
Sleep duration	2.46±0.51	2.83±0.38	0.01*
Sleep efficiency	2.06±0.25	2.88±0.32	0.001*
Sleep disturbance	1±.35	0.94±0.22	0.5
Hypnotics	0	0	0
Day time dysfunction	1.46±0.64	1.5±0.5	0.6
Global PSQI	11.2±1.26	13.8±1.09	0.02*
HbA1C	9.86±1.1	10.68±1.24	0.05*

Table 5: Relationship between sleep duration,PSQI and sleep debt with glycated hemoglobin in males and females:

Correlation	Male	p-value	Female	p-value
HbA1C and sleep duration	-0.2	0.05*	-0.31	0.001*
HbA1C and PSQI	0.09	0.0001*	0.4	0.001*
HbA1C and sleep debt	0.2	0.34	0.3	0.001*

DISCUSSION

A good sleep is one of the most satisfying human experiences with a role to play in maintaining a good mood and cognitive acuity as well as in promoting physiologic balance and resilience. The sleep disorder plays a pivotal role in the occurrence and development of diabetes via neuro - endocrine metabolic pathway[6].Sleep disorder can facilitate the hypothalamic-pituitary-adrenocortical system to release extra Glucocorticoid[7].

The present study address this hypothesis by examining self-reported sleep duration and quality and HbA1c levels in patients with type 2 diabetes.

Our study reveal that a higher perceived sleep debt or lower sleep quality are associated with poorer glucose control. Table 2 shows significant positive association between HbAIC and PSQI, between HbA1C and sleep debt and negative association between HbA1C and sleep duration. Sleep deprivation stimulates the cerebral cortex, cerebral limbic system and hypothalamus, which induces the secretion of catecholamines from the sympathetic ganglion and adrenal medulla and of cortisol from the pituitary–adrenal system. Increasing sympathetic nervous system activity impair glucose regulation via the lipolytic effects of adrenergic stimulation of visceral adipose tissue,decreasing levels of the satiety hormone leptin and increasing the hunger hormone ghrelin, thereby increasing hunger and food intake. Sleep restriction is also significantly associated with increases in cortisol, interleukin -6, and tumor necrosis factor - α.In addition to these pathways, a decrease in brain glucose utilization after sleep deprivation might lead to an increase in the glycemic level[5,8,9]. There is evidence showing that a lack of 3 h sleep could lead to 1.1% elevation of HbA1c during one single night. With 0.5 increase in PSQI global score, the HbA1c can increase by 1.9%.[6]

Table 3 shows that short sleep duration is associated with higher HbA1C in both males and females which was statistically significant. The PSQI global score was also poorer in females which is significant. Subjective sleep quality, sleep efficiency and sleep latency were significantly worse in females than in males (Table 4).

The mechanisms by which sex may influence the prevalence of poor sleep quality are still unknown. The possible explanations for this phenomenon is that there are differences in peroxisome proliferator-activated receptor (PPAR)-a expression levels between males and females. Because PPARa is associated with obesity, insulin resistance, and type 2 diabetes , this receptor may also influence sleep quality. PPAR -α is under estradiol modulation and that cross-talk between this receptor and the estrogen receptor possibly exists[10]. Some researchers proposed that improving sleep quality, treating sleep disorder, and optimizing sleep duration could be used as a regimen to indirectly promote the glycemic control.

CONCLUSIONS

In this study, a high proportion of Type 2 diabetic subjects were found to have disturbed and reduced sleep. A statistically significant association between both quality and quantity of sleep in Type 2 DM subjects with poor glycemic control and also high perceieved sleep debt among females was found.These findings suggest that sleep hygiene should be a part of diabetes management.

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HLA Antigen Distribution in Renal Transplant Patients & Donors Visiting Tertiary Care Hospital of Karnataka State in South India

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ABSTRACT

Renal transplantation is the most successful treatment option for chronic renal failure patients. But success of this depends on HLA antigen matching between the renal recipient & donor. This study is the first report from Karnataka state of South India to find out distribution of HLA-A and B antigens in live renal transplant recipients and donors visiting a tertiary care hospital.In a retrospective study, HLA-A and B allele frequencies were studied in 345 renal transplant recipients & donors visiting our hospital, between 2005-2013. HLA tissue typing was done by polymerase chain reaction using sequence specific primers. Then Allele frequencies were obtained & analysed. A total of 32 HLA-A and 40 HLA-B alleles were identified in the live renal transplant recipients & donors. High frequency A alleles found in our patients & donors were- A*24(16%),A*33(13.6%),A*02(13.5%),A*11(11.4%),A*01(9.6%). The most frequent alleles in the HLA-B locus were B*40(15%), B*07(13.4%), B*35(12.7%), B*51(8.8%), B*44(7.6%). Also we have found that females dominated among donors. The results showed significant heterogeneity in both HLA-A and B antigens. By knowing the frequency of HLA antigens in population,it is easy to find HLA matched organ transplant recipient & donors from different ethnic background, without a blood relationship.

Keywords: *Allele, renal transplantation, HLA*

INTRODUCTION

Renal transplant has become a permanent and final option for patients with fatal kidney diseases and success of which depends on the degree of HLA (Human leucocyte antigen) matching between patients & donors. Since HLA system exhibits high polymorphicity, finding a suitable donor to needy renal transplant patient is indeed a difficult task. HLA system is the name of the locus of genes that encode for major histocompatibility complex (MHC) in humans. Locus of genes that are present on short arm of chromosome 6, encodes for cell-surface antigen-presenting proteins which is involved in immune system's recognition of self and non-self tissues.

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Since HLA antigens are involved in allograft rejection, when matched donor is not found in a family then search may be done to find out unrelated donors. Therefore the documentation of information about frequencies with which a particular HLA haplotype occurs in a population of a specific region or locality is important which in turn might help the organ transplant team to find a suitable donors.

HLA profiles of different populations are well documented from various parts of world. Studied the pattern of HLA allele distribution among various ethnic groups of Iranian renal transplant recipient population and showed that A2 & B35 are the most predominant HLA A & B antigens in Iranian population.[1]

similarly Singh it was found that A02 and A11 are uniformly high among most of the Indian population including Siliguri and adjoining areas of West Bengal. This study also documents B37 is most observed

frequency specifically this Bengali population compared to B07 and B08 with the rest of the India.[19]

A most recent study by[18] Tuladhar in live related renal transplant recipient and donors of Nepal showed that there exist a considerable heterogeneity in both HLA class I and class II antigens and A11 & B15 are more relatively common among the Nepali population.

[5]Babita have showed that the phenotypic frequencies of HLA-A10, B5, and B8 were found to be increased while the frequencies of HLA-A19, A28, B35 and B40 were found to be decreased in Sikh population when compared to another north Indian population.

Studies on population genetics with HLA antigen distribution have demonstrated that the Dravidian non tribal population is highly diverse and more likely to have admixed origin [17].

Studies have also documented that HLA A1 &B 35 are more common in Iyer , a tamil Brahmin community [6] and similarly HLA B-14 are common in Toto population a tribal community of Himalayan region [8].

But there is no such study reported from Karnataka state. Therefore the present study has been carried out to study the HLA class I (A & B) profile frequency distribution in the renal transplant patients & donors admitted in Kasturba Hospital, Manipal , one of the leading tertiary care hospital in this region.

In our present study the distribution of HLA-A and B are obtained in the live related renal transplant donors and recipient of population coming to Kasturba tertiary care hospital to determine HLA antigen frequencies.

Aim & objective: To determine the most frequent class I HLA antigens (A & B) in live related renal transplant patient & donors of coastal Karnataka & Kerala visiting the Nephrology department of Kasturba Hospital, Manipal.

PATIENTS AND METHOD

This retrospective cross sectional study, was duly started after obtaining mandatory clearance from Institutional Ethical Committee of Kasturba Hospital(--IEC-372/2014). Only live donors and recipients of both sexes from the year 2005-2013 of Indian ethnicity are considered for this study. HLA typing in 345 persons were done and the information and data collected are strictly maintained as confidential and they are further coded and analyzed for the frequency distribution.

Briefly in this study we located patient's case reports who have undergone renal transplant surgery in our Kasturaba Hospital from MRD and we collected the data of HLA typing results of both potential donors and patient to analyze the frequency distribution of the commonly occurring alleles. A total number of 345 renal transplant patients & their donor's HLA-A,B phenotypes were analyzed.

DNA extraction and HLA typing:

DNA extraction was done using whole blood (QIAGEN DNA extraction mini kit). Low resolution molecular typing using polymerase chain reaction-sequence specific primer (PCR-SSP) technique (One Lamda Inc,USA) was performed for detecting HLA class I alleles. After PCR, agarose gel electrophoresis is done & gel picture is taken using UV gel documentation machine which are used for analyzing the results. HLA class I alleles are reported based on the DNA bands appearing in the particular well of the gel & the literature supplied by the company.

Statistical analysis: HLA Phenotypic frequencies were calculated by direct count and the results are expressed as percentage of frequency distribution.

The gender specific HLA phenotypic distribution are also be expressed in percentage

Finally an attempt is also made to correlate the HLA allele with degree of relation between the donor and recipient.

RESULTS

From our study, we have observed that males (83%) outnumbered females(17%) among recipients, whereas females (76%) outnumbered males (24%) among donors (Tables 1&2).

Table 1: Gender Frequency of recipients (n=169)

gender	N	%
males	141	83
females	28	17

Table 2: Gender frequency of donors(n=176)

gender	N	%
females	135	76
males	41	24

From Table 3,Further analyzing the donor's profile and their relationship with the recipient it was found that it was the mother who was donor most of times (38%) followed by wife(18%), sister(14%), brother(12.5%), father(5.6%), other relatives like cousin, aunt, friend, uncle, nephew(10%). Most of the time, donor & recipients are parents Vs offspring (43.6%), siblings (26.5%), spousal donors (18%), offsprings Vs parents(0.5%). Unrelated donors as friends constituted around 1.7%.

Another interesting observation that followed was out of 176 donors there were not even single husband nor a son were reported as donors(0%).

Table 3. HLA donors frequency(n=176)

relationship	N	%
mother	68	38
wife	32	18
sister	25	14
brother	22	12.5
father	10	5.6
cousin	4	2.2
friend	3	1.7
aunt	3	1.7
uncle	2	1.1
2nd cousin	1	0.5
daughter	1	0.5
Distant relative	1	0.5
Grand mother	1	0.5
Sister-in-law	1	0.5
nephew	1	0.5
Step-mother	1	0.5

Allele frequencies: We have observed 32 HLA-A & 40 HLA-B alleles in our population.

Table :4

HLA-A	Frequency-N	%	HLA-B	N	%
24	107	16	40	99	15
33	91	13.6	07	88	13.4
02	90	13.5	35	83	12.7
11	76	11.4	51	58	8.8
01	64	9.6	44	50	7.6
03	57	8.5	58	41	6.2
30	39	5.8	15	36	5.5
26	32	4.8	52	29	4.4
32	25	3.7	57	25	3.8
29	15	2.2	37	24	3.6
68	13	1.9	13	16	2.4
02/92	10	1.5	40	2	0.3
31	8	1.2	67	1	0.1
36	5	0.7	07/81	1	0.1
11/34	5	0.7	08	7	1
02/68	4	0.6	08/42	1	0.1
23	4	0.6	15/95	3	0.4
29/31	4	0.6	18	14	2.1
01/11/23	2	0.3	18/35	1	0.1
07	2	0.3	23	1	0.1
01/03	1	0.1	27	7	1
01/11/24	1	0.1	35/40	4	0.6
03/02/92	1	0.1	37/53	1	0.1
08	1	0.1	38	8	1.2
23/24	1	0.1	39	5	0.7
26/32	1	0.1	39/48/56	1	0.1
34	1	0.1	41	1	0.1
35	1	0.1	48	6	0.9
38	1	0.1	49	1	0.1
68/02	1	0.1	50	1	0.1
68/92	1	0.1	53	2	0.3
69	1	0.1	54	2	0.3
			54/55	1	0.1
			54/55/56	1	0.1
			55	19	2.9
			55/56	2	0.3
			56	5	0.7
			78	1	0.1
			81	2	0.3
			95	3	0.4

(From above table 4),The most frequent alleles in the HLA-A locus were A*24(16%), A*33(13.6%), A*02(13.5%), A*11(11.4%), A*01(9.6%) and A*03(8.5%).

HLA B allele frequencies

(From Table 4)The most frequent alleles in the HLA-B locus were B*40(15%), B*07(13.4%), B*35(12.7%), B*51(8.8%), B*44(7.6%)

Table 5: HLA-A allele frequency in 182 males(n=665)

HLA-A	N	%
24	55	8.2
02	50	7.5
33	45	6.7
01	36	5.4
11	32	4.8
03	30	4.5
30	27	4

Table : 6 HLA-A allele frequency in 162 females(n=665)

HLA-A	N	%
24	52	7.8
33	46	6.9
11	44	6.6
02	40	6
01	29	4.3
03	27	4
30	12	1.8

(From Tables 5 & 6), Males show slightly higher frequency of A*24 & A*02 than females, whereas females show slightly higher number of A*33 & A*11 than males.

Table 7: HLA-B allele frequency in 182 males (n=653)

HLA-B	N	%
40	57	8.7
07	51	7.8
35	37	5.6
51	29	4.4
44	26	3.9
52	15	2.2
58	26	3.9

Table : 8 HLA-B allele frequency in 162 females (n=653)

HLA-B	N	%
35	45	6.8
40	42	6.4
07	38	5.8
51	29	4.4
44	20	3
58	15	2.2
52	14	2.1

(From Tables 7 & 8),B*40 is predominant in males,but it occupies second place in females. B*35 antigen is present in high percentage in females, but it occupies 3rd place in males .

DISCUSSION

Previous studies in the histocompatibility complex of Indian population had been done mainly as a part of population genetics to understand the origin, migration and degree of admixture of population. Currently not much documented studies are available on frequency distribution of HLA type allele in Karnataka population to guide the renal transplant surgeon as what to expect in cadaver, live related and live unrelated donors HLA typing.

Our study has shown large number of HLA-A alleles (total 32) & HLA-B alleles (total 40) in our population, much more than any of the other studies. This may be due to wide geographical distribution of our patients.

Studies have proved that there is positive correlation between the degree of HLA compatibility between recipients and donors and the outcomes of transplant reactions.

Our center performs live renal transplant's surgeries with HLA typing. Most of the time the donor and recipient are mother vs offspring in live related and wife vs husband in live unrelated planned surgeries. Siblings as donor almost falls third next to mother and wife, findings contradicts to previously studies done in Nepal [18].

Despite the patients of Kasturba hospital who opt for renal transplant surgeries come from three different states (coastal Karnataka, coastal Kerala and Goa) surprisingly there seems to be homogeneity in few HLA antigens (both in HLA-A and HLA-B) , but

more pronounced in HLA-A) indicating a fact that the Malabar coast/ west coastal population would have originated from an common region and later would have migrated far and wide from coastal Kerala to Goa.

Most striking observation found was females outnumbered males in case of donors, may be because males are mostly bread winners & females are mostly housewives. Greater income of men may encourage females to be donors. Similar observation found in patients undergoing permanent sterilization, where females undergoing tubectomy outnumbered males undergoing vasectomy. Among female donors, mother dominates proves the universally accepted fact that she is the living God. Many studies have shown the gender inequality in renal transplantation. Possible causes for paucity of male to female donations are: more female population in this region, ambivalence among men about donation, high incidence of hypertension & coronary artery disease among men, military obligations. Another cause for the lower percentage of male-female donations is the presence of high levels of preformed anti-HLA antibodies in females due to previous pregnancy/ blood transfusion. This may be an important barrier to transplant wives using their husband's kidneys [20].Also awareness & changes in the attitudes of the public are needed to eliminate this gender bias.

The introduction of less invasive laparoscopic donor nephrectomy has become more popular because of less post operative pain & quick recovery. Patients who underwent laparoscopic nephrectomy demonstrated less morbidity & shorter hospital stays than conventional open nephrectomy patients. This may attract more males to become donors.

Males outnumbered females in recipients.[21] showed 58% of CKD(chronic kidney disease) & ESRD(end stage renal disease) patients are males & most common cause for it is diabetic nephropathy (44%). Therefore we can say that males could be more genetically predisposed to kidney diseases than females.

HLA-B27,which is associated with Ankylosing Spondylitis, is observed in 7 cases(1%).

HLA-B*40 is also found in high frequency in Maharashtrian population [10],[7], Nepali population[18].

CONCLUSION

These results show the presence of wide variety of HLA –A & B alleles in both renal transplant recipient & donors. This high degree of polymorphism in the HLA system is useful in anthropological studies. Population with same HLA antigen frequencies might have derived from common origin. By knowing the frequency of HLA antigens in population,it is easy to find HLA matched organ transplant recipient & donors from different ethnic background, without a blood relationship. If some alleles are present in high frequency in a population,they might be the cause of some disease in that population. This gives information about genetic predisposition to some diseases.

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Estimation of Thyroid Auto-antibodies Levels in Normal Pregnant Females and Pregnancy Induced Hypertensive Patients

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ABSTRACT

Background:-Autoimmune thyroid disorders are characterized by presence of antithyroid antibodies, specifically Antithyroglobulin (Tg-ab), Antithyroid peroxidase (TPO-ab) and TSH receptor antibodies. Anti-TPO antibodies are the most common anti-thyroid autoantibody Anti-TPO antibodies are present in 99% of cases where thyroglobulin antibodies are present, however only 35% of anti-TPO antibody positive cases also demonstrate thyroglobulin antibodies.

Method: - Three ml of venous blood was collected and serum was separated and stored in deep freezer. Thyroid autoantibodies levels were measured by ELISA method.

Results: - In group I; the mean anti-thyroperoxidase (TPO) levels were 0.14 ± 0.5 IU/mL and anti-thyroglobulin (TG) levels 0.06 ± 0.24 IU/mL .In group II; the mean anti- thyroperoxidase (TPO) levels were 0.06 ± 0.24 IU/mL and anti-thyroglobulin (TG) levels 0.03 ± 0.17 IU/mL.

Conclusions:-There were no statistically significant relationship found in between group I and group II in relation to pregnancy induced hypertension and thyroid antibodies levels. ($p > 0.05$).

Keywords:- *Thyroid auto-antibodies , pregnancy ,pregnancy-induced hypertension.*

INTRODUCTION

Thyroid dysfunction and autoimmunity are relatively common among women of reproductive age with prevalence of 2-3% during pregnancy. Although during reproductive age, thyroid antibodies are found in 5-15% of women, they are not necessarily accompanied by thyroid dysfunction. It has been suggested that antithyroid antibodies are independent markers of at-risk in pregnancy. TPO and thyroglobulin (TG) auto-antibodies can be detected in 10–20% of women of child bearing age. The majority of women who test positive for thyroid auto antibodies are euthyroid.Irrespective of maternal thyroid function, the function of Tg-ab is associated with higher rate of obstetrical complications. Tg-ab positive mothers had higher prevalence of gestational hypertension compared to Tg-ab negative mothers. No differences were seen in rates of gestational hypertension between thyroid dysfunction group and reference group or TPO-ab positive and negative mother.

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MATERIAL AND METHOD

This case control study was conducted on 18-40 years old pregnant women with single pregnancy with gestational age of 20 weeks or more (based on first trimester sonography) and

who presented at OPD & IPD of Upper India Sugar Exchange Maternity hospital (Obstetrics & Gynaecology department), G.S.V.M. Medical College, Kanpur. The study group consisted of 35 normal pregnant females and 35 pregnancy induced hypertensive patients, who were selected randomly from the population with diagnosis of gestational hypertension.

CRITERIA FOR INCLUSION:

- Antenatal patients with gestational age 20 or more than 20 weeks.
- Primigravidae and multigravidae.
- With singleton pregnancy

CRITERIA FOR EXCLUSION:

Patient with chronic hypertension,Twin pregnancy, Molar pregnancy, Patient with history of hypertension, thyroid disorder, proteinuria, pre-ecclampsia or excessive weight gain in previous pregnancies. In our study following criteria were taken for the diagnosis:

GESTATIONAL HYPERTENSION: American College of Obstetritician and Gynecologist in 2002, NHBPEP 2000: Gestational hypertension is defined as: "new hypertension (systolic blood pressure ≥ 140 mm Hg OR diastolic blood pressure ≥ 90 mm Hg or both) presenting at or after 20 weeks gestation without proteinuria or other features of preeclampsia," this terminology replaces the term "Pregnancy-Induced Hypertension".

SPECIMEN COLLECTION & PREPARATION

Three ml of venous blood was collected and serum was separated and stored in deep freezer. Thyroid autoantibodies levels (Thyroid peroxidase & Thyroglobulin) were measured by ELISA method. Converting of Ab Index to IU/mL as an option**, TPO Ab** index may be converted to IU/mL by multiplying Ab index value by 50. International units may then be interpreted as follows:

< 50 IU/mL: Negative

50-75 IU/mL: Borderline positive

> 75 IU/mL: Positive.

Converting of Ab Index to IU/mL as an option**, TG Ab** index may be converted to IU/mL by multiplying Ab index value by 100. International units may then be interpreted as follows:

< 100 IU/mL: Negative

100-150 IU/mL: Borderline positive

> 150 IU/mL: Positive.

STATISTICAL ANALYSIS: The significance between the standard errors of means of different sets of observation would be assessed by student't' test and 95% level of confidence.

OBSERVATION AND RESULT

The study was conducted on 70 pregnant females between age 20-38 years who attended the outpatient department of obstetrics and Gynaecology department of G.S.V.M. Medical College Kanpur. They were divided into two groups on the basis of blood pressure and presence of protein in urine (by dipstick method).

GROUP I: Comprised of 35 pregnant women of age group 20-38 years having normal blood pressure and no proteinuria.

GROUP II: Comprised of 35 Hypertensive pregnant women of age group 20-38 years having blood pressure ≥ 140/90 mm of Hg and no proteinuria whom were diagnosed as pregnancy induced hypertensive patient.

The parameters estimated were Thyroid autoantibodies (TPO&TG), SGPT, SGOT, Serum bilirubin, Serum Alkaline Phosphatase levels.

Table- 1: Anti- Thyroid Antibody (Anti TPO-Ab) Levels In Patients Without Hypertension (Group-I) And Patients With Hypertension (Group-II)

STATUS OF PERSON	PREGNANCY WITH OUT HTN (Gr .I)		PREGNANCY WITH HTN (Gr.II)	
	N=35	%	N=35	%
NEGATIVE	32	91.43	33	94.39
BORDERLINE POSITIVE	1	2.86	2	5.71
POSITIVE	2	5.71	0	0
MEAN ± SD	0.14 ± 0.49		0.06 ± 0.23	

(Normal Anti TPO-Ab Level, < 50 IU/mL –Negative, 50-75 IU/mL-Borderline Positive

and >75 IU/mL is Positive). positive for anti TPO antibodies

Table-2 : Anti Thyroid Antibody (Anti TG-Ab) Levels In Patients Without Hypertension (Group-I) And Patients With Hypertension (Group-II)

STATUS OF PERSON	PREGNANCY WITHOUT HTN (Gr.I)		PREGNANCY WITH HTN (Gr.II)	
	N=35	%	N=35	%
NEGATIVE	33	94.39	34	97.14
BORDERLINE POSITIVE	2	5.71	1	2.85
MEAN ± SD	0.06 ± 0.23		0.03 ± 0.169	

In group I the mean anti Thyroperoxidase (TPO) levels were 0.14 ± 0.5 and the group I had 5.71 % of positive and 2.86 % patients were found borderline positive for anti TPO and no positive and group II mean TPO levels was 0.06 ± 0.23 and had 5.71% were borderline (Normal Anti TG-Ab Level, <100 IU/mL –Negative,

100-150 IU / mL- Borderline Positive and >150 IU / mL –Positive)

In group I the mean anti Thyroglobulin (TG) levels 0.06 ± 0.24.The group I had 5.71 % were borderline positive and group II mean TG levels was 0.03 ± 0.169 and had 2.85% were borderline positive for anti TG antibodies. There were no statistically significant relationship found in between group I and group II in relation to pregnancy induced hypertension and thyroid antibodies Anti TG & Anti TPO levels. ($p > 0.05$).

Table- 3: Alkaline Phosphatase Levels In Patients Without Hypertension (Group-I) And Patients With Hypertension (Group-II)

STATUS OF PERSON	PREGNANCY WITHOUT HTN (Gr.I)		PREGNANCY WITH HTN (Gr.II)	
	N=35	%	N=35	%
NORMAL	29	82.86	9	25.71
INCREASED	16	17.14	26	74.28
MEAN ± SD	233.17 ± 107.4		339.11 ± 154.13	

(Normal S. Alkaline Phosphatase Level = 50-240 IU/ml).

There was statistically significant difference was found in serum alkaline phosphatase levels in group I mean 233.17 ± 107.4 and group II mean 339.11 ± 154.13 at $p < 0.05$.

Table- 4: SGPT In Patients Without Hypertension (Group-I) And Patients With Hypertension (Group-II)

STATUS OF PERSON	PREGNANCY WITHOUT HTN (Gr.I) N=35		PREGNANCY WITH HTN (Gr.II) N=35	
	N=35	%	N=35	%
NORMAL	29	82.86	22	62.86
INCREASED	6	17.14	13	37.14
MEAN ± SD	27.51 ± 11.37		53.48±52.73	

(Normal SGPT level = 0-40 IU/mL)

The mean SGPT level of group I was 27.52 ± 11.38 and group II was 53.49 ± 52.73 which was statistically significant ($p < 0.05$).

Table- 5: SGOT In Patients Without Hypertension (Group-I) And Patients With Hypertension (Group-II)				
STATUS OF PERSON	**PREGNANCY WITHOUT HTN (Gr.I)**		**PREGNANCY WITH HTN (Gr.II)**	
	N=35	**%**	**N=35**	**%**
NORMAL	25	71.42	11	31.42
INCREASED	10	28.57	24	68.57
MEAN ± SD	32.28 ± 15.68		64.11 ± 59.41	

(Normal SGOT level = 0-32 IU /ml)

The mean SGOT level of group I was 32.29 ± 15.69 and group II was 66.11 ± 59.42 which was statistically significant ($p < 0.05$).

Table-6: Serum Bilirubin Total In Patients Without Hypertension (Group-I) And Patients With Hypertension (Group-II)				
STATUS OF PERSON	**PREGNANCY WITHOUT HTN (Gr.I)**		**PREGNANCY WITH HTN (Gr.II)**	
	N=35	**%**	**N=35**	**%**
NORMAL	33	92.29	28	80
INCREASED	2	5.71	7	20
MEAN ± SD	0.75 ± 0.3		0.86 ± 0.4	

(Normal S. Bilirubin level = 0.2-1.0 mg / dl)

Majority of both group had normal serum bilirubin levels. There was no statistically significant difference found in between Serum biliribin total in group I and group II.($p > 0.05$).

DISCUSSION

The mean S.bilirubin (total, direct & indirect) in our study was 0.75 ± 0.31mg/dl, 0.35 ± 0.20mg/dl and 0.40 ± 0.17mg/dl in patients without hypertension (group-I) and 0.86 ± 0.40mg/dl, 0.40 ± 0.25mg/dl and 0.44 ± 0.18mg/dl in patients with hypertension (group-II) respectively and our findings were similar to findings of **Deflamingh JP et al (1984).** They found no significant difference between the normotensive and hypertensive group.

In our study, the mean SGOT, SGPT and S. Alkaline Phosphatase were 32.29±5.69 IU/L, 27.52±11.38 IU/L and 233.17±107.4 IU/L in patients without hypertension (group-I) and 66.11±59.42 IU/L, 53.49±52.73 IU/L and 339.11±154.13IU/L in patients with hypertension (group-II) respectively and found significant association between group I and group II (p0.05). The findings of our study were similar to findings of **J. C. Girling et al (1997**), **Anna L. David et al (2000), Dilip Kumar Bhowmik et al (2013) and Sonagra AD et al (2012).** Alkaline phosphatase (ALP) is a nonspecific enzyme which hydrolyses aliphatic, aromatic or heterocyclic compounds. It has several isoenzyme forms, of which α2 heat stable ALP is of placental origin. Normal serum level of placental isoform is only 1% of total ALP but in pregnancy and pregnancy associated disorders its level raises enormously. The elevated levels of these enzymes indicate that increased levels of these parameters are seen as the disease severity increases.

Azin Alavi et al (2012) who found no significant difference in thyroid hormone levels and thyroid antibodies levels in the 4 groups of cases including gestational hypertension, mild preeclampsia, severe preeclampsia and eclampsia which were similar to findings of our study**. Ghafoor et al. in 2006** evaluated pregnancy outcome and TPO antibody status in Pakistani women found significant increase in preterm delivery compared with antibody-negative women , however Thyroglobulin antibody was not evaluated in this study.

Acknowledgement: Faculty and staff of Physiology, Department of Pathology, and Obstetrics & Gynaecology Departments.

Conflict of Interest: None

Source of Funding: Self

Ethical Clearance: The study was started after obtaining consent from the patient. The study was cleared from Ethical Committee of Institute.

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Study of Change in Intraocular Pressure during different Trimesters of Pregnancy in Rural Area

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ABSTRACT

Background: During pregnancy, many physiological changes occurs in all the organs due to interactions between fetus and mother including Eyes. It is seen that it has beneficial effect in glaucoma.

Objectives: To study change in intra ocular pressure (IOP) during First, Second and Third trimesters of pregnancy and to compare with age-matched non-pregnant women.

Method : IOP was measured in 50 pregnant women between 20-30 years of age, using Schiotz Tonometer in First, Second and Third Trimesters and was compared with 50 non-pregnant women where IOP was measured thrice at interval of 12 weeks.

Results: Data was analysed statistically using ANVOA. There was no statistically significant decrease in mean IOP in first trimester as compared to control group ($p>0.05$). Mean IOPs in second trimester and third trimester were significantly decreased compared to control group ($p<0.05$). Mean IOP differences between first and second, second and third and first and third trimesters of pregnancy were statistically significant ($p<0.05$).

Discussion : Present study suggests that IOP decreases with the progression of pregnancy under hormonal influence . Knowledge of normal level of IOP in different trimesters of pregnancy helps in glaucoma screening.

Keywords*: Pregnant woman, Intra ocular pressure, Trimester, Schiotz tonometer.*

INTRODUCTION

Pregnancy and child birth is a wonderful phenomenon associated with progressive anatomical and physiological changes. These changes are not only confined to the reproductive organs, but also to all the systems of the body including eyes [1,2]. While pregnancy can worsen pre-existing ocular conditions such as diabetic retinopathy, it can also has beneficial effects in women with glaucoma and uveitis.

Glaucoma is a common ophthalmic disease in India and is a second leading cause of visual impairment and blindness and is responsible for 23% of all cases[3]. Few reports are available, on effect of pregnancy on the intra ocular pressure (IOP) changes [1] in Indian women.

Knowledge of the normal level of intraocular pressure in various stages of pregnancy and routine screening of IOP during an antenatal check up may be useful in glaucoma screening[1].

Hence, the present study, was undertaken to observe the physiological changes of intraocular

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pressure in different trimesters of pregnancy in normal Indian women.

MATERIALS & METHOD

Present study was conducted in Kamineni Institute of Medical Sciences, Narketpally, Nalgonda District. It was a case controlled prospective study conducted on 100 subjects during December 2011 to December 2013 over a period of 24 months.

Pregnant and non pregnant women of age between 20-30 years were included in the study.

Patients with history of Glaucoma , Eye surgery, Ocular injury, Toxemia of pregnancy, Systemic disease such as hypertension, diabetes mellitus, Known hypersensitivity to Lignocaine, those taking Oral contraceptives pills, antihypertensive, β blockers, diuretics were excluded from the study.

100 subjects were divided in two groups, Group A & Group B . Group A consisted of fifty pregnant women between 20-30 years in first trimester.

Group B (n = 50) consist of 50 age matched non pregnant women. All subjects were informed in detail about aim , objectives and the procedure of the study; and written consent was taken for conduct of study. Age, height, weight, pulse rate and blood pressure were recorded .

In present study a total of 66 pregnant women were studied during first trimester of pregnancy, out of which 50 were followed upto third trimester. The rest 16 were dropped out from the study due to various reasons like abortion, development of pregnancy induced hypertension and lack of follow up.

This study was performed in 3 sessions : First between 10-12 weeks of gestation . Second between 24-28 weeks of gestation & Third between 34-36 weeks of gestation . Fifty age matched non pregnant women were included as controls and IOP was measured thrice at an interval of 12 weeks . IOP was recorded using Schiotz tonometer in the morning hours between 10 AM to 1 PM to prevent any diurnal variation in IOP .

STATISTICAL ANALYSIS

The data were analysed using one way analysis of variance (ANOVA) to compare the data within the group and student's t- test to compare the data between the groups. 'p'- value < 0.05 was considered to be significant.

RESULTS

Demographic profile and haemodynamic parameters were comparable in both groups. There was no statistically significant decrease in mean IOP in first trimester as compared to control group (p>0.05). Mean IOPs in second trimester and third trimester were significantly decreased compared to control group (p<0.05). Mean IOP differences between first and second, second and third and first and third trimesters of pregnancy were statistically significant (p<0.05) .

Table-I: Comparison of Mean Intra Ocular Pressure of Control and Study groups (N = 100)

	Mean IOP (mmHg ± SD)		
Reading / Trimester	**Control Group (n = 50)**	**Study Group (n = 50)**	**'p' value**
First	15.39 ± 1.43	15.26 ± 1.58	> 0.05
Second	15.31 ± 1.47	13.04 ± 1.25	< 0.05
Third	15.36 ± 1.21	12.13 ± 1.11	< 0.05

'*p*' value: <0.05 – significant.

There was statistically significant decrease in mean IOP in study group as compared to control group ('p' value < 0.05).

In study group IOP was found to be progressively decreasing from first trimester to third trimester in both the eyes. The mean difference in IOPs between first to second , second to third and first to third trimesters of pregnancy were statistically significant (p value <0.05) in both the eyes.

DISCUSSION

TABLE II: Comparison of Mean IOP of Present study with Other studies

Mean IOP (mmHg ± SD)							
		Phillips et al [4] (1985) (N=97)	**Qureshi et al [5] (2000) (N=200)**	**Pilas et al[6] (2004) (N=182)**	**P Jyothi et al[1] (2011) (N=60)**	**Ebeighe Jet al[7] (2011) (N=217)**	**Present study (N=100)**
Control Group		14.8 ± 1.7	15.1 ± 0.2	15.6 ± 1.81	-	15.0±1.7	15.35 ± 1.37
Study Group Trimester	I	14.1 ± 2.6	-	15.33 ± 1.98	-	14.7 ± 2.2	15.26 ± 1.58
	II	-	-	14.52 ± 2.37	12.48 ± 1.0	13.2 ± 2.0	13.04 ± 1.25
	III	12.1 ± 1.7	13.5 ± 0.3	12.5 ± 1.96	11.44 ± 0.90	11 ± 1.3	12.13 ± 1.11

The finding that the third trimester of pregnancy has an ocular hypotensive effect is consistent with other studies.

The decreased IOP in the pregnant women explains improvement in glaucoma during pregnancy as reported by previous studies.[2]

The physiological mechanism responsible for the decreased in IOP during pregnancy is not well known. A number of mechanisms have been postulated.

It is well documented that levels of progesterone and oestrogen increases during pregnancy. Thus it can be speculated that the decreased IOP in pregnant women might be due to hormonal changes. Moreover, literature shows the influence of sex hormones on IOP. This is because sex hormones are steroids and they causes salt and water retention. This leads to increase in total body fluid content which builds up in the spaces between cells causing water retention [8,9].

Increased levels of progesterone and oestrogen that occur in pregnancy causes dilatation of the vessels leading to generalized reduction in the peripheral vascular resistance[1] and decreased arterial pressure and thus a reduction in aqueous humour production [6].

Wilke[10] demonstrated a decrease in episcleral venous pressure during pregnancy.

Qureshi et al[5] suggested that a decrease in IOP during pregnancy is due to effect of hormones on transport enzymes namely Na+K+ATPase and carbonic anhydrase which are involved in aqueous humour secretion. Hormonal changes and metabolites produced during pregnancy acts as antagonists of these enzyme and hence reduces the intraocular pressure.

Pregnancy also induces a slight metabolic acidosis that contributes to decrease in IOP through osmotic gradient [2].

The effects of relaxin could be another possibility. It causes a relaxation of the pelvic ligaments in pregnant women, so that the sacroiliac joints become relatively soft and relaxed and the symphysis pubis becomes elastic. These changes facilitate easier passage of the fetus through the birth canal.

Philips and Gore[4] suggested that this softening of ligaments in late pregnancy might extend to the ligament of the corneo-scleral envelope and reduces corneo-scleral rigidity, improved uveoscleral outflow and therefore cause a fall in IOP.

CONCLUSIONS

From present study it is concluded that there is a progressive and statistically significant fall in intraocular pressure as the pregnancy advances. This knowledge of intraocular pressure in different trimesters of pregnancy will also help in glaucoma screening . Improved understanding of change in intraocular pressure in pregnancy and impact of pregnancy on the course of glaucoma offers an opportunity for meaningful counselling of glaucoma patients who are pregnant and those who are planning for pregnancy. Hence along with the routine antenatal investigations , pregnant women with glaucoma should be managed in conjunction

with their Ophthalmologists so as to meticulously monitor and titrate the antiglaucoma medication throughout their pregnancy.

Also, this study has established baseline data on the pattern of intraocular pressure changes in pregnant and non pregnant Indian women.

The limitation of present study is small study group. Therefore further larger studies are required to confirm the results of present study .

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Ethical Clearance - Taken from Institutional Ethical Committee

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Conflict of Interest - Nil

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To Estimate the Excretion of Urinary Electrolytes in Type 2 Diabetes Mellitus in Industrial Area Population

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ABSTRACT

Objective: Disturbance of electrolyte balance along with metabolic derangement in diabetes mellitus may results in further development of future hypertension, cardiovascular diseases, osteopenia, renal failure and many other complications. Estimation of urinary Na^+, K^+ & Ca^{++} can be useful marker for diabetes mellitus as well as may be helpful in preventing other fatal diseases like hypertension, Chronic renal failure etc in diabetic patients.

Material & Method: Newly diagnosed 37 patients with Type 2 diabetes mellitus compared with 30 age & sex matched healthy controls was evaluated for the estimation of urinary electrolytes. Fasting serum glucose & serum creatinine was estimated by GOD-POD & Jaffe's method respectively. Urinary electrolytes were estimated by using flame photometer.

Results: In this study serum fasting glucose was significantly (179.85 ± 40.182 vs 73.95 ± 5.97) higher in diabetic group. Serum creatinine level was significantly (0.9125 ± 0.16 vs 1.015 ±0.17) lower in patients group when compared to control group. While in electrolytes retention of sodium occurs in diabetic group compared to other remaining electrolytes which were significantly higher in patients group.

Conclusion: This study suggested that diabetes mellitus is characterized by urinary electrolytes imbalance. This electrolyte imbalance can give rise to some other diseases in future.

Keywords*: Diabetes mellitus, hyponatriurea, hyperkaluria, hypercalciurea.*

INTRODUCTION

India is emerging as the diabetic capital of world with around 40.9 millions diabetics currently and expected to rise to 69.9 million by 2025.[1] Diabetes Mellitus is a group of metabolic diseases characterized by hyperglycemia resulting from defect in insulin secretion, insulin action or both.[2] cardiac function is decreased in DM patients compared with non-DM patients.[3,4] Chronic hyperglycemia of diabetes is associated with long term dysfunction, damage and failure of various organs especially eyes , kidneys, heart , blood vessels and nerves.[5] Moreover diabetes mellitus is associated with disturbance of electrolyte metabolism and acid base balance.[6] An increase in plasma insulin level within or slightly above the physiological range markedly reduces fractional sodium excretion.[7] Consequently Sodium (Na^+) retention occurs as a characteristic alteration in type 2 diabetes mellitus.[8] So Urinary excretion of sodium is significantly lower in patient with diabetes mellitus type 2 then in healthy subjects.[9] Similarly Potassium (K^+) homeostasis is abnormal in type 2 diabetes mellitus.[10] While renal tubular reabsorption of calcium (Ca^+) is reduced in diabetes mellitus resulting in increased urinary losses.[11] Disturbance of electrolyte balance along with metabolic derangement in diabetes mellitus may results in further development of future hypertension, cardiovascular diseases, osteopenia, renal failure and many other complications.[12,13] Estimation of urinary Na^+, K^+ & Ca^+ can be useful marker for diabetes mellitus as well as may be helpful in preventing other fatal diseases like hypertension, Chronic renal failure etc in diabetic patients.

MATERIAL & METHOD

Participants: A cross sectional study was conducted at VIMS & VH, Gajraula, & study group was consisted of 67 peoples including both male & female, in which 37 peoples were diabetic and they were compared with 30 ages and sex matched healthy controls. Volunteers with and without type 2 diabetes were enrolled *via* oral representation. The diagnosis of type 2 diabetes was made on the basis of long term elevated fasting serum glucose (≥126 mg/dl). Control subjects without diabetes were participants without a history of type 2 diabetes. Inclusion criteria was age >30 years and ability to provide informed consent. Excluded patients were pregnant women, individuals with a known history of kidney stone disease, chronic renal failure, and those who were treated with diuretics or alkali therapy. In the type 2 diabetes group, treatment with insulin and/or thiazolidinediones was an additional exclusion criterion.

Study Procedures: Each participant of the study group collected their 24-hour urine collections. Urine was collected and kept refrigerated until analysis. Fasting blood sample was obtained at the end of each collection. [14]

Method: Fasting blood sugar and serum creatinine was estimated by enzymatic GOD-POD and Jaffe's method respectively. Twenty-four-hour urine was analyzed for sodium, potassium, calcium using a flame photometer. Values of sodium and potassium were expressed in mill equivalent/ 24 hr, while calcium was expressed in mg/ 24 hr. [15]

Statistical Analysis: Results were expressed as mean ± SD. Student t test was used to differentiate between diabetic and control group. Pearson Correlation Coefficient was used to analyse the relation between fasting blood sugar and different electrolytes (sodium, potassium and calcium). P value < 0.05 was considered statistically significant.

RESULTS

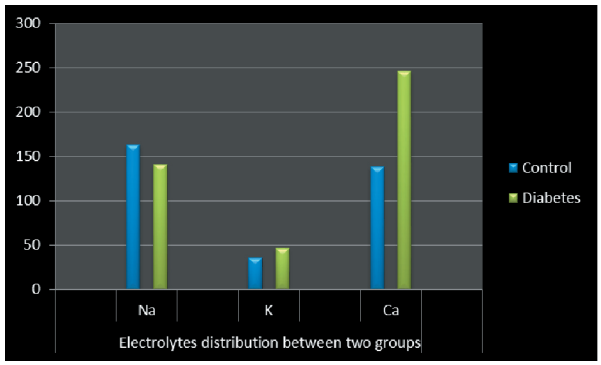
Fasting Blood sugar, serum creatinine and urinary Sodium, Potassium and Calcium all were normal for control group and were comparatively increased for diabetic patients except sodium, which was significantly retained in patients group. Table 1 shows the values of various parameters in diabetic as well as control group. Pearson Correlation Coefficient analysed a positive correlation between fasting blood sugar and different electrolytes (sodium, potassium and calcium), Table 2.

Table 1: Clinical and biochemical characteristics of patients with diabetes and control group

Parameters	Diabetic group(37)	Control group(30)	P value
Fasting blood sugar (mg/dl)	179.85 ± 40.182	73.95 ± 5.97	<0.05
Serum creatinine (mg/dl)	0.9125 ± 0.16	1.015 ±0.17	<0.05
Urinary sodium (mEq/ 24 hr)	140.75 ± 39.24	163.36 ± 49.44	<0.05
Urinary potassium (mEq/24 hr)	46.25 ±12.59	34.97 ± 11.62	<0.05
Urinary calcium (mg/ 24 hr)	246.06 ± 65.68	138.31 ± 44.45	<0.05

Table 2: Correlation of fasting blood sugar (FBS) and different electrolytes in diabetic group

	Correlation coefficient 'r'	P value
FBS – Sodium	0.31	<0.05
FBS – Potassium	0.3	<0.05
FBS – Calcium	0.19	<0.05



Electrolytes distribution between Control and diabetic Groups

DISCUSSION

This study confirm the notion that diabetes mellitus is characterised by disturbance in electrolyte balance of the body. In the present study, we found that Urinary Na^+ was inversely while urinary K^+ and Ca^{++} were directly correlated with fasting blood sugar in type 2 diabetic patients. There was decrease excretion of Na^+ while urinary K^+ and Ca^+ excretion were increased in type 2 diabetic patients in comparison to healthy individuals. Studies have suggested that diabetes mellitus is characterized by abnormalities of Na metabolism at all physiologic levels: whole-body, renal, and cellular. The most consistently described abnormality is an expansion of exchangeable Na, which seems to be closely associated with increased proximal renal tubular Na reabsorption and suppression of membrane sodium-potassium ATPase activity in circulating cells resulting in Na^+ retention moreover increases in blood glucose and insulin concentrations ensue in the stimulation of sodium reabsorption by the kidney.[16]Although the combined occurrence of hyperglycemia and hyperinsulinemia, frequently secondary to insulin resistance with regard to carbohydrate metabolism, is a hallmark of non-insulin dependent diabetes mellitus (NIDDM), the role of these abnormalities in determining an impaired natriuresis in NIDDM is not yet clear.[17] Our findings showed hyperkaluria and hypercalciurea in type 2 diabetes mellitus patients while previous researchers also found hypokalemia in NIDDM as potassium homeostasis is influenced by basal insulin levels and this effect of insulin is mediated via extra renal mechanisms of potassium disposal.[18]However it is thought that disturbance of K^+ hemostasis in type 2 diabetes mellitus is probably related to insulin and mineralocorticoid deficiency.[19] Renal excretion of calcium is increase in diabetes mellitus as the renal tubular reabsorption of calcium is reduced in diabetic patients resulting in increased urinary losses of the calcium.[20] Similarly diabetes mellitus is associated with a decrease in bone mineral content and increased urinary excretion of calcium.[21]

Hypothetically the urinary estimation of sodium, potassium and calcium may be used to diagnose impaired glucose tolerance (IGT) and pre diabetic state along with other parameters. Though further study with large samples size along with other parameters should be conducted on different types of diabetic patients to establish the fact.

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Conflict of Interest: Nil

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Hand Grip Strength, Endurance Time, Heart Rate and Blood Pressure Changes in Smokers

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ABSTRACT

Background: Smoking is a well-known risk factor for atherosclerosis. Loss of distensibility and compliance of large arteries may play a role in the onset of atherosclerosis. Smoking is reported to increase arterial stiffness. Light smoking increases arterial wall stiffness that might be harmful to the artery and increase the risk for plaque rupture. A study by Lucini et al group showed an increase in heart rate in smokers than the non-smokers. These facts gave us an impetus to verify the effects of smoking on autonomic functions in light and heavy smokers.

Method: We measured the BMI, hand grip strength, endurance time and blood pressure and assessed their relationship to smoking in light and heavy smokers and compared them with nonsmokers. Statistical analysis of our findings using students T- test and ANOVA were done.

Results: In light smokers endurance time increased significantly. BMI was significant amongst the groups. The systolic blood pressure change, handgrip strength and endurance time between smokers and non-smokers, on comparison were found to be significant.

Conclusion: Smoking leads to altered sympathetic activity leading to increased heart rate and Blood pressure at rest, during exercise and after exercise. The effects on health will help recommend cessation of smoking.

Keywords: *Smoking, nicotine, autonomic function, blood pressure, ageing.*

BACKGROUND

WHO states that there are 1.1 billion smokers in the world today and the number is expected to increase to 1.6 billion by the year 2025. S.K. Jindal et al [1] state that the prevalence of Smoking was 28.5% in men and 2.1% in women in India (2006). The Global Adult Tobacco Survey India 2009-2010 (GATS India) states that on an average a daily cigarette smoker in India smokes 6.2 cigarette sticks per day [2].

Cigarette smoke contains about 4,000 different chemicals that include at least 80 chemicals that can cause cancer including tar, arsenic, benzene, cadmium, formaldehyde, nicotine and hundreds of other poisons.

In men aged 47 to 55 years who are light smokers the prevalence of a major cardiac event during a 12-year period is 11% compared with 3.7%, in nonsmoking men [3, 4] and the risk of death from aortic aneurysm is nearly 3 times greater in them [5]. Low-dose exposure to nicotine on the brain suggests that

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this type of tobacco use may trigger up regulation of nicotinic acetylcholine receptors, resulting in a heightened physiological response to an occasional cigarette [6].

Cigarettes are ignited and inhaled, usually through a cellulose acetate filter, into the mouth and lungs. Pipe and Cigar smoke are not inhaled because of its higher alkalinity (pH 8.5) compared to cigarette smoke (pH 5.3). Nicotine absorption from cigar and pipe, is much less than that from cigarette smokes [7].

Nicotinic acetylcholine stimulation is not directly addictive. However, since dopamine-releasing neurons are abundant on nicotine receptors, dopamine is released and is associated with pleasure. There is formation of MAO inhibitor from acetaldehyde in tobacco smoke. It plays an important role in nicotine addiction probably, by facilitating dopamine release in nucleus accumbens [8].

Endurance is defined as the ability to perform prolonged muscular work at predetermined intensity without external signs of fatigue. Handgrip strength a physiological variable, is affected by a number of factors including age, gender and body size. Strong correlations between grip strength and anthropometric traits, (weight, height, hand length) were reported earlier (Malina et al. 1987; Rossand Rösblad 2002; Jurimae et al. 2009). There is altered autonomic activity with increased adrenergic activity in chronic smoking which predisposes to cardiovascular morbidity and mortality. Not many studies have been conducted for testing autonomic function tests in smokers using handgrip dynamometer and in evaluating the relationship between muscular strength and endurance time. These facts gave us an impetus to evaluate blood pressure changes, hand grip strength and endurance time in smokers and compare them with non-smokers.

MATERIALS & METHOD

This descriptive study was conducted in the clinical physiology lab in Indira Gandhi Medical College and research Institute, Kadirkamam, Pondicherry, India. The duration of the study was for two months. All male smokers and nonsmokers in this institute were included in this study. Hundred and forty four subjects were included comprising of sixty one smokers and eighty three non smokers for comparison, were enrolled in the study. Individuals who were smoking a similar brand [Gold flake/wills] of cigarette, non-alcoholics, not on any medications and apparently healthy were selected. All women and those who smoked for less than 5 years were excluded. Individuals with hypertension, diabetes, chronic obstructive pulmonary disease, altered pulmonary functions, myocardial infarction and stroke were excluded from the study. They were grouped as light smokers-who smoked less than 15 cigarettes /day and heavy smokers-those who smoked more than 15 cigarettes /day [9]. The common brand gold flake and wills, released about 1.5 to 2 mgs of nicotine/Gm of tobacco [10].

Subjects were seated comfortably, explained about the procedure. A written informed consent was obtained from all of them.

Weighing scale that could measure to the nearest 0.1kg was used to record weight and height was measured using a stadiometer. Their BMI was calculated based on Quetelet index [11].

Autonomic function was evaluated using hand grip dynamometer for endurance, isometric muscle tension and the sympathetic response for which the subjects were seated comfortably. The subjects were asked to put maximum force on the dynamometer thrice from the dominant hand. The maximum value was recorded in kilograms. Anthropometric equipment and handgrip dynamometer were calibrated before each assessment. All subjects were tested thrice and the best of three attempts was recorded. Thirty seconds time interval was maintained between each handgrip strength testing [12]. 1/3rd of the hand grip was calculated from the above value and the endurance time at maintaining 1/3rd of the hand grip was measured. Baseline Pulse was recorded by palpating the radial artery and expressed as rate or beats /minute. Respiratory rate was recorded with the subject in supine position (expressed as rate/minute). Blood pressure was recorded at rest, during and after the procedure using a mercury sphygmomanometer .Change in blood pressure (Blood pressure during the procedure minus base line BP) was recorded. Data were collected in the morning between 8 AM. To 12 noon.

The study was approved by the institute ethics committee. Data was entered in MS excel SPSS software version 17.0 and students T- test and ANOVA were used to compare the means between various groups.

RESULTS

Sixty one smokers and eighty three nonsmokers were included in the study. The mean age in smokers was 28years and that of nonsmokers was 30 years. The baseline pulse, respiratory rate, diastolic blood pressure and height were comparable (Table-1). Baseline systolic BP in the smokers was high in smokers than that in the nonsmokers and was statistically significant. Similarly weight and BMI were significantly lower in the smokers than the non smokers. This difference can be attributed to smoking per se. The hand grip strength and endurance time in the light and heavy smokers though statistically not significant, shows a higher mean value (Table-2). Baseline diastolic BP in light smokers shows a higher mean than heavy smokers (Table-2). The Diastolic blood pressure change amongst the heavy smokers had a higher mean value and was not statistically significant.

DISCUSSION

By 2020, the WHO expects the worldwide death toll to reach 10 million, causing 17.7 per cent of all deaths in developed countries..

The mean value for pulse rate was higher in smokers due to the effect of sympathetic stimulation (table -1). The mean BMI values in the light and heavy smokers were less than the controls due to the fact that smoking decreases the lean body mass. An increase in the mean systolic BP in them could be attributed to the increased sympathetic response. Hand grip strength and endurance time in the light and heavy smokers though statistically not significant, shows a higher mean which may be due to the neuropharmacological effects of nicotine on the blood vessels thereby increasing the blood supply to the exercising muscles within physiological limits (Table-2). Baseline diastolic BP in light smokers shows a higher mean than heavy smokers, which may be due to increased peripheral vascular resistance. The BP change (both systolic and diastolic) amongst the light and heavy smokers appears to be due to the sympathetic over activity due to smoking.

The mean BMI of the nonsmokers was slightly higher than the subjects as 75% of them were overweight. The resting heart rate, pulse, systolic and diastolic blood pressure in light and heavy smokers as compared to control were higher and seems to be consistent with the findings by Kotamaki. M [13], Kim JW et al [14] and Kool M J et al [15] which stated that smoking caused short term increase in arterial wall stiffness and plaque rupture. Increased oxidative stress also causes atherogenesis contributing to altered heart rate and blood pressure in smokers. This supports the theory of smoker's paradox. Kunz F et al [16] pointed out that compared to nonsmokers, smokers had lower LDL cholesterol, greater hand grip strength and better performance on bicycle ergo meter. Better handgrip strength and endurance in smokers in our study is attributed to the stimulatory effects of nicotine and its reinforcement effect on the brain. Nicotine acts on the nicotinic acetylcholine receptors, specifically the ganglion type nicotinic receptor and one on the CNS nicotinic receptor. In humans nicotine induced improvement of rapid information process is well explained [17].

Nicotine improves performance by increasing the attention resources available for such strategic processing [18]. Different processes including attention stimulus evaluation and response selection appear to be involved on human information processing[19].Hence our study suggest that nicotine has positive effects on central nervous system which facilitates human attention, memory and sensorimotor function similar to a study by Neil Sherwood[20]. Formation of MAO inhibitor from the acetaldehyde in tobacco smoke plays a major role in nicotinic addiction, probably by facilitating a dopamine release in the nucleus accumbens as a response to nicotine stimuli. Thus the reinforcement effect on the brain and nicotine mediated – neuromuscular response is responsible for better handgrip in the smokers especially, heavy smokers [16].

In our study the smokers had higher diastolic blood pressure levels after exercise in hand grip strength suggesting altered parasympathetic control over blood pressure in smokers. The tachycardic effect elicited by smoking is accompanied by acute changes in heart rate spectral components. Therefore the cardiac autonomic control is altered by smoking during rest and exercise [21]. Andrikopoulos et al reported that smoking causes an acute and constant decrease in vagal cardiac control [22]. The diastolic blood pressure components (baseline DBP and Diastolic blood pressure change) in heavy smokers would have a statistically significant response if more number were included. Nicotine mediated response at neuromuscular junction and reinforcement effect of nicotine is responsible for better hand grip strength

and endurance time in our subjects.

Thus we conclude that smoking leads to altered sympathetic activity leading to increased heart rate and Blood pressure at rest, during exercise and after exercise. The smoker's paradox is an astounding mystery till date.

TABLES

TABLE - 1 Baseline data of the study population

Variable	Workers Mean ± SD	Controls Mean ± SD	P value
Age in yrs	28.42± 8.450	30.35±10.821	0.249
Pulse /min	81±4.97	80.75±5.423	NS
Respiratory rate/min	19.77±10.25	17.78±2.096	0.08
Systolic BP in mmHg	114.62±12.69	110.22±10.94	0.027
Diastolic BPin mmHg	77.64±6.875	76.35±6.93	0.270
Weight in kg	57.27±9.144	60.97±11. 510	0.040
Height in mts	1.650± 0.0575	1.65±0.0649	NS
BMI	20.98±2.78	22.318±3.84	0.023

TABLE - 2 Comparison of variables between light smokers, heavy smokers & Non-Smokers

Variable	Light smokers (Mean±SD)	Heavy smokers (Mean±SD)	Control (Mean±SD)	P value
AGE	27.09 ± 8.051	30.95±8.808	30.35±10.82	NS
BMI	21.67± 2.94	19.6765±1.87023	22.31±3.841	.007
HAND GRIP	27.77 ± 5.22	30.47±4.379	30.13 ± 6.583	0.092
1/3RD OF HAND GRIP	9.23± 1.738	10.14±1.448	10.04 ± 2.186	0.084
ENDURANCE TIME(sec)	44.58 ± 22.64	54.50±86.60	36.94±18.44	NS
BASELINE SYSTOLIC BP	113.75±8.912	116.29±18.000	110.22 ±10.946	0.064
BASELINE DIASTOLIC BP	78.15±5.404	76.67±9.129	76.35 ± 6.932	NS
DURING PROCEDURE SBP	128.15±10.480	134.71 ±17.88	127.59±18.005	NS
DURING PROCEDURE DBP	89.78± 15.318	91.86±11.744	90.73±8.803	NS
SYSTOLIC BP CHANGE	14.40±7.899	18.43±2.481	17.37±13.919	0.306
DIASTOLIC BP CHANGE	11.63±4.124	15.19±5.938	14.27±7.545	0.060

P value <0.05(level of significance)

FIGURES

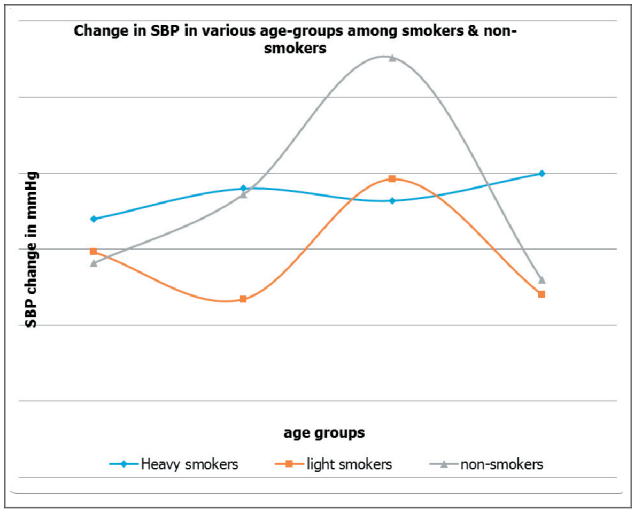

Figure: 1- Change in systolic blood pressure in various age-groups among smokers and non-smokers.

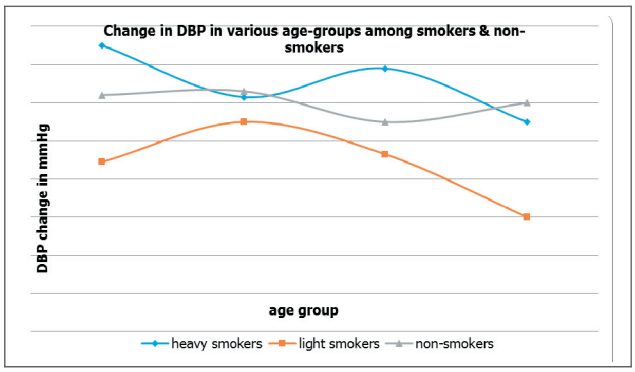

Figure: 2 - Change in Diastolic blood pressure in various age- groups among smokers and non-smokers.

CONCLUSION / SUGGESTIONS

Young people are especially vulnerable because of pressure from their peers and the image that smoking is part of growing up. Just trying a few cigarettes can be enough to become addicted. It takes on an average of about six cigarettes before nicotine receptors in the brain are switched on, generating a craving for nicotine which may continue for the rest of the person's life. Although the health risks of smoking are cumulative, giving up can yield health benefits, regardless of the age of the patient. There is substantial evidence that cigarette smoking causes a sympathetic overdrive leading to sudden cardiac death. People should be adviced to join smoking-cessation service and be aided with all available help including medication and counselling.

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A Descriptive Study of Sleep and its Relation to Body Mass Index (BMI) in First Year Medical Students of Bangalore Medical College and Research Institute

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ABSTRACT

The objective of this study was to observe the sleep latency, day dysfunction, actual sleep hours and sleep efficiency in first year medical students and its relation to their BMI.

A total of 68 first year medical students answered the Pittsburgh Sleep Quality Index (PSQI) Questionnaire. Height and weight of the participants were measured and Body Mass Index (BMI) calculated.

A suggestive significance of P=0.08 for sleep efficiency score across various category of BMI indicates that higher BMI is associated with worse sleep efficiency scores.

Medical students should maintain their BMI in the normal BMI range by taking nutritious food and engaging in adequate physical activity as normal BMI is associated with good sleep.

Keywords: *Sleep, PSQI, BMI, medical students.*

INTRODUCTION

According to the recommendations of the National Sleep Foundation, seven to eight hours of quality sleep every night is required for a young adult aged 18-25 years old[1]. First year medical students are no exception. However, medical students spend a large amount of hours for academic activities as a result of which many of them may be sleep deprived. Obesity and overweight add to the above problem. It is well known that overweight and obese suffer from sleep apnea that may impair sleep in them[2]. The objective of this study was to observe the sleep latency, sleep disturbances, actual sleep hours and sleep efficiency in first year medical students and its relation to their BMI.

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MATERIALS & METHOD

The research project was conducted in the physiology department of Bangalore Medical college and research institute. After obtaining informed consent, eighty-seven healthy I year medical students were enrolled in the study. Height was measured with a measuring tape stuck to the wall and recorded to the nearest to 0.1cm.Weight of the students was recorded to nearest to 0.5 kg using a weighing scale with light clothing and without shoes. Students were then asked to answer the Pittsburgh sleep quality index questionnaire.

The Pittsburgh Sleep Quality Index questionnaire was used to examine the sleep habits of the individual. It had 10 questions. The first question was on sleep duration. Further questions elicited response regarding sleep latency(The time required to fall asleep), sleep disturbances, sleep medication use, sleep quality, daytime dysfunction that resulted due to inadequate or disturbed sleep and so on.

Students filled the questionnaire recollecting

their sleep pattern over the last one month. The questionnaire was then assessed using the PSQI scoring available at the website of PSQI.

Based on the PSQI scoring instructions, Sleep duration, sleep latency, sleep efficiency and day dysfunction, sleep disturbances, medications taken to induce sleep, sleep quality were scored on a scale of 0,1,2,3.zero indicated better and 3 was for worse. The sum of these scores provided us with a Global sleep score. A Global sleep score of 5 or less than 5 implied that the subject had good sleep. A score of value >5 implied poor sleeper.Students were classified into normal BMI (18.5-24.9Kg/m^2), less than normal or unsderweight BMI(<18.5 Kg/m^2) , overweight BMI(BMI between 25 -30 kg/metre^2) and obese (With BMI greater than 30Kg/m^2)

68 students completed the questionnaire. Data was entered in Microsoft excel and was analyzed by SPSS 15 and Microsoft excel.

Statistical Methods: Descriptive and inferential statistical analysis was carried out in the present study. Results on categorical measurements were presented in Number (%). Significance was assessed at 5 % level of significance. Fisher Exact test was used to find the significance of study parameters on categorical scale between two or more groups.

FINDINGS

Table-1 Sleep latency according to different category of BMI

Sleep Latency	BMI (kg/m^2)				Total
	<18.5	18.5-24.9	**25-30**	**>30**	
0	7(58.3%)	17(37%)	6(85.7%)	1(33.3%)	31(45.6%)
1	2(16.7%)	15(32.6%)	1(14.3%)	2(66.7%)	20(29.4%)
2	3(25%)	10(21.7%)	0(0%)	0(0%)	13(19.1%)
3	0(0%)	4(8.7%)	0(0%)	0(0%)	4(5.9%)
Total	12(100%)	46(100%)	7(100%)	3(100%)	68(100%)

P=0.406, Not significant, Fisher Exact test

Table 2: Day time-Dysfunction according to different category of BMI

Day Dysfunction	BMI (kg/m^2)				Total
	<18.5	18.5-24.9	25-30	**>30**	
0	2(16.7%)	9(19.6%)	1(14.3%)	2(66.7%)	14(20.6%)
1	7(58.3%)	23(50%)	5(71.4%)	0(0%)	35(51.5%)
2	3(25%)	13(28.3%)	0(0%)	1(33.3%)	17(25%)
3	0(0%)	1(2.2%)	1(14.3%)	0(0%)	2(2.9%)
Total	12(100%)	46(100%)	7(100%)	3(100%)	68(100%)

P=0.235, Not significant, Fisher Exact test

Table 3: Actual sleep hours according to different category of BMI

Actual sleep (in hours)	**BMI (kg/m^2)**				**Total**
	<18.5	**18.5-24.9**	**25-30**	**>30**	
1-5	3(25%)	11(23.9%)	3(42.9%)	1(33.3%)	18(26.5%)
6-10	9(75%)	35(76.1%)	4(57.1%)	2(66.7%)	50(73.5%)
Total	12(100%)	46(100%)	7(100%)	3(100%)	68(100%)

P=0.646, Not significant, Fisher Exact test

Table 4: Sleep efficiency according to different category of BMI

Sleep Efficiency Score	BMI (kg/m^2)				Total
	<18.5	**18.5-24.9**	**25-30**	**>30**	
0	11(91.7%)	41(89.1%)	5(71.4%)	2(66.7%)	59(86.8%)
1	0(0%)	5(10.9%)	2(28.6%)	1(33.3%)	8(11.8%)
2	0(0%)	0(0%)	0(0%)	0(0%)	0(0%)
3	1(8.3%)	0(0%)	0(0%)	0(0%)	1(1.5%)
Total	12(100%)	46(100%)	7(100%)	3(100%)	68(100%)

P=0.088+, suggestive significant, Fisher Exact test

Table 5: Global Sleep score according to different category of BMI

Global Sleep Score	**BMI (kg/m^2)**				**Total**
	<18.5	**18.5-24.9**	**25-30**	**>30**	
Good	8(66.7%)	25(54.3%)	4(57.1%)	2(66.7%)	39(57.4%)
Poor	4(33.3%)	21(45.7%)	3(42.9%)	1(33.3%)	29(42.6%)
Total	12(100%)	46(100%)	7(100%)	3(100%)	68(100%)

P=0.937, Not significant, Fisher Exact test

DISCUSSION

Sleep latency is defined as time required for falling asleep by a person after he goes to bed. A sleep latency of <15 minutes is scored zero. There are two questions in the PSQI regarding sleep latency. The second question in PSQI enquires about" How much time in minutes it has taken for the individual to fall asleep after he goes to bed ?" and the question 5A enquires about" How often the individual had trouble sleeping and could not get to sleep within 30 minutes. The individual scores for these two questions is added to give final score for sleep latency[3].

Day dysfunction reflects on daytime incapacitation of the individual usually a consequence of poor night sleep. 51.5% of the sample had day dysfunction score of one. 58.3% of less than 18.5kg/m^2 BMI students had a day dysfunction. This is contrary to the belief that day dysfunction is associated with overweight and obese due to the coexisting disorder of Obstructive sleep apnoea generally among the overweight and obese. We understand from the table that being underweight(less than 18.5kg/m^2)is not protective against sleep disorders. Among overweight, 71.4% had a score of one. A study conducted in a International Malaysian Medical school by Zailinawati et al showed that of the 799 students who participated in the study, there was a prevalence of 35.5% of the student sample who were experiencing day time sleepiness. However, in this study, the daytime sleepiness was assessed not by PSQI but by the Epsworth sleepiness scale[4].

Observing Table 3 shows that 73.5% of the sample had an actual sleep of 6 hours to 10 hours.While only 26.5% of the sample had sleep of 5 and less than 5 hours. According to Bjorn Bjorvatn , their study confirmed a clear association between short sleep duration and elevated BMI and obesity[5] However in our research project, we did not get significance on Fischer Exact test.

Table 4 shows sleep efficiency score in various category of BMI represented as cross tables..Sleep efficiency is fraction of "actual sleep" to "time spent in bed" expressed as percentage. Of the 59 students who had a score of zero, 41 of them were in normal BMI group, 11 were in less than normal BMI and 5 were in overweight and 2 were in obese group. Fisher

exact test showed suggestive significance of P=0.088. This mean that there were higher proportion of those with zero score (better than 1,2 or 3) in normal BMI than in other groups.

According to a study by Moraes et al, normal BMI individuals tended to sleep longer and had higher sleep efficiency than obese or overweight individuals[6].

A global score of 5 and <5 implies good sleepers and score of >5 implies poor sleepers. 57.4% of the sample were good sleepers while 42.6% were poor sleepers.

CONCLUSION

A suggestive significance of P=0.08 for the table on sleep efficiency score across various category of BMI indicates that higher BMI is associated with higher or worse sleep efficiency scores.

Acknowledgement: I thank the statistician, Dr KP Suresh for having done the statistical analysis.

Conflict of Interest: None

Source of Funding: Self

Ethical Clearance: Obtained

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Assessment of Cardiac Functions during Immersion of Face in Water in Humans

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ABSTRACT

Context: Studies on cardiovascular changes during breath-hold diving at various depths have shown that there is significant reduction in heart rate. A decrease in cardiac output is a known response during immersion, while a coexisting bradycardia may partly contribute to a fall in cardiac output. But the contribution of changes in end diastolic volume, if any have received lesser attention. Hence, this study was aimed at filling this lacuna by measuring changes in end diastolic volume during immersion of face in water using echocardiography.

Objective: To observe the changes in the following parameters during breath-hold face immersion in water using Lead 11 ECG and 2D Echo

1. Heart rate (HR)
2. Left Ventricular End Diastolic Volume (LVEDV)
3. Left Ventricular End Systolic Volume (LVESV)
4. Left Ventricular Stroke Volume (LVSV)

Method: 15 healthy male swimmers of the age group 20 to 35 years, who swim at least two hours per week were selected for the study.

Results: There was a statistically significant reduction in HR, as well as a statistically significant increase in LVEDV and LVESV. The increase in LVSV was not of statistical significance.

Conclusion: Bradycardia is a common feature observed in both actual diving and breath-hold face immersion in water. An increase in the LVEDV can be attributed to the coexistent bradycardia .

A slight increase in the LVSV observed in this study is contrary to our expectation, since the LVESV showed a significant increase. This would mean, a reduction in myocardial contractility might have contributed to an increase in the LVESV. Earlier studies have shown that stimulation of trigeminal receptors on the face during diving can influence the vasomotor centre and stimulate parasympathetic influence on the heart. This explains both, the bradycardia and an increase in LVESV observed in the present study.

Keywords *: Cardiac function, immersion of face in water, End Diastolic volume, End Systolic volume.*

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INTRODUCTION

The Diving reflex in man has been observed in professional divers. Popular interest in breath hold diving with its danger of drowning has stimulated much research on untrained subjects in diving and swimming. The diving reflex is an oxygen conserving reflex consisting of striking bradycardia, peripheral

vasoconstriction and increased blood supply to such vital organs as the brain and heart[1].

Studies on cardiovascular changes during breath-hold diving at various depths have shown that there is significant reduction in heart rate[2]. A decrease in cardiac output is a known response during immersion, while a coexisting bradycardia may partly contribute to a fall in cardiac output. But the contribution of changes in end diastolic volume, if any have received lesser attention. This study was aimed at filling this lacuna by measuring changes in end diastolic volume using echocardiography.

India has a very long coastal belt. There are large number of people who are involved in water based activities mainly for their livelihood and also for leisure and hobby. In addition swimming pools are provided in some hotels, residential apartments, educational institutions and city muncipalities. The instances of death due to drowning are not uncommon. Professional divers are also required to rescue many of these victims. A study of this kind would be appropriate to scientifically assess the competency of professional divers as well as people who are willing to explore various water based activities. In view of paucity of Indian research publications in this area we decided to study the cardiac changes during immersion of face in water in our laboratory.

MATERIALS & METHOD

Study sample : The subjects chosen for this study were experienced active breath hold swimmers of age group 20 to 35 years, who can communicate in English. They were members of a swimming pool in Manipal, Karnataka

Sample size : 15

Inclusion criteria:

a. Male subjects aged 20 – 35 years

b. Those who were swimming at least two hours per week

Exclusion criteria

a. Male subjects aged < 20 and >35 years

b. Non swimmers

c. Those with cardiovascular or respiratory diseases.

Note: The absence of female subjects was casual and not due to a selection criterion.

PROCEDURE

Equipment and supplies

1 plastic basin, 1 towel, Tap water

1 stop watch, ECG leads

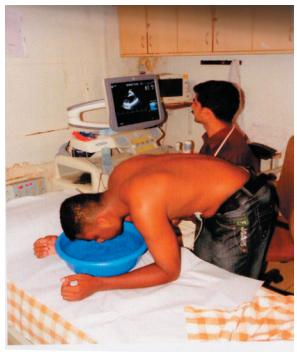
Echocardiographic equipment -
Echocardiographic examination performed using the instrument – VIVID 7, GE Germany.

Each subject was made to sit comfortably in a chair and the full procedure was explained to him. Only after obtaining a written consent, the procedure was started. The test was conducted in a posture such that the subject had to lean over the cot with elbows resting on the cot and the head down. A plastic basin filled with water at room temperature was kept on the cot.

The procedure consists of holding the breath and immersion of face in water kept in the basin for 20 seconds. The subject should immerse the face up to the temples. The following parameters were selected to assess cardiac function.

1. Heart rate.

2. Left ventricular End Diastolic Volume and End Systolic Volume



Photograph showing a subject connected to 2D-Echocardiograph ready to immerse his face in water

Heart rate was recorded before and during the procedure using Lead II ECG. Left ventricular End Diastolic volume and End Systolic volume were recorded before and during the procedure using 2D-Echocardiograph (calculated by area-length method with the help of an inbuilt software). Left ventricular stroke volume was calculated as the difference between the left ventricular end diastolic and the end systolic volumes

RESULTS

The study was conducted in 15 healthy male swimmers between the age groups of 20 – 35 years.

Table 1: Showing the changes in the selected cardiac parameters before and during the immersion of face.

Sl. No of subjects	HR (bpm)		LV EDV (ml)		LV ESV (ml)		LVSV (ml)	
	BI	DI	BI	DI	BI	DI	BI	DI
1.	77	63	69	72	26	34	43	38
2.	108	97	97	101	46	54	51	47
3.	71	69	104	126	33	42	71	84
4.	95	72	74	82	30	40	44	42
5.	87	85	94	128	40	63	54	65
6.	63	60	113	119	55	57	58	62
7.	61	56	64	86	28	38	36	48
8.	89	63	69	85	34	37	35	48
9.	75	68	70	80	33	40	37	40
10.	79	78	79	90	29	46	50	44
11.	65	63	76	97	32	43	44	54
12.	66	59	78	90	32	40	46	50
13.	84	76	53	55	19	15	34	40
14.	76	64	69	72	26	34	43	38
15.	98	86	97	101	46	54	51	47

(HR – Heart rate, LVEDV – Left ventricular end diastolic volume, LVESV – Left ventricular end systolic volume, LVSV – Left ventricular stoke volume)

(BI – Before immersion, DI – During immersion)

Statistical analysis:

To test the significance of the difference between the cardiac changes during basal condition and breath hold face immersion in water, the Non parametric test (Wilcoxon Signed Ranks test) for paired data was used

Table 2: Comparison of the values obtained in the basal and during breath-hold face immersion in water using Non Parametric test.

No	Parameter	Median		Inter-quartile range		p-value
		Basal	DI	Basal	DI	
1.	Heart rate (bpm)	77	68	(66,89)	(63,78)	0.001*
2.	End Diastolic Volume (ml)	76	90	(69,97)	(80,101)	0.001*
3.	End Systolic Volume (ml)	32	40	(28,40)	(37,54)	0.001*
4.	Stroke Volume (ml)	44	47	(37,51)	(49,54)	0.146

(DI – During immersion, *p value <0.05 is taken as significant)Box plots showing the median values for the cardiac parameters during basal and breath-hold face immersion in water is given below

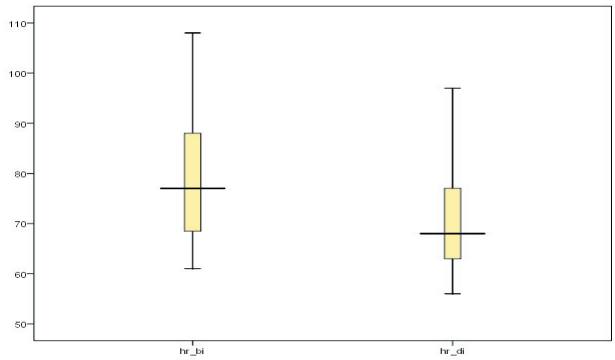


Fig 1: Comparison of median value for basal heart rate (hr_bl) and during breath-hold face immersion (hr_di)

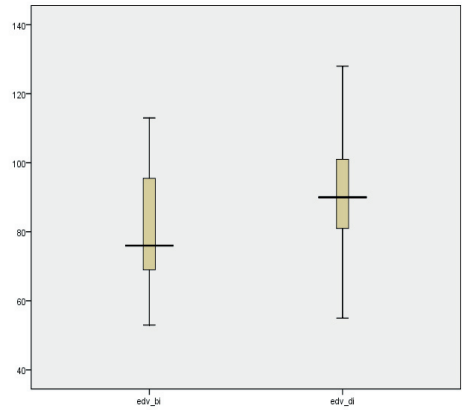


Fig 2: Comparison of the median value for basal left ventricular end diastolic volume (edv_bl) and during breath-hold face immersion (edv_di).

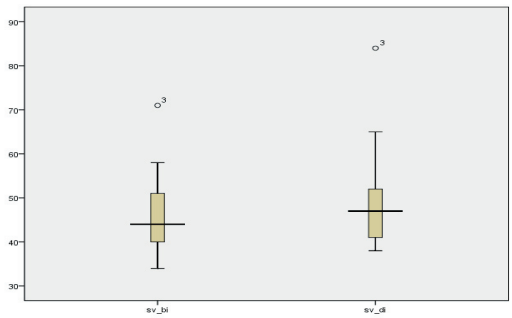


Fig 3: Comparison of the median value for basal LVESV (esv_bl) and during breath-hold face immersion (esv_ di)

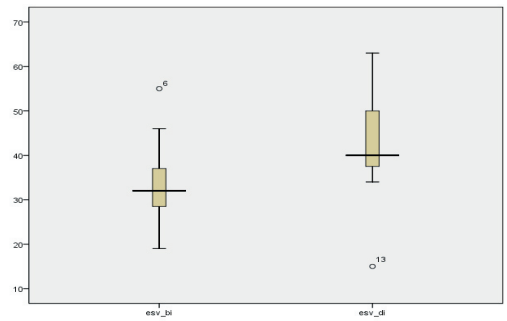


Fig 4: Comparison of the median value for basal LVSV (sv_bl) and during breath-hold face immersion (sv_di).

After analysis of the data for the 15 subjects, the following changes were observed during 'breath-hold face immersion in water' when compared to those of basal condition.

1. There is a statistically significant reduction in heart rate.
2. There is a statistically significant increase in Left Ventricular End Diastolic Volume as well as Left Ventricular End Systolic Volume.
3. Even though there is an increase in the calculated Left Ventricular Stroke Volume, it is found to be of NO statistical significance.

DISCUSSION

In the present study, the cardiac response to facial immersion in water was assessed using 2D-Echocardiograph in 15 healthy male swimmers. It was observed that there is a significant reduction in heart rate, as seen in the previous studies[3]. The main finding of this study was a significant increase in the left ventricular end diastolic volume and end systolic volume. An increase in the left ventricular end diastolic volume can be attributed to the coexistent bradycardia which allows more time for ventricular filling[4]. It is well known, that the left ventricular stroke volume is the difference between the left ventricular end diastolic volume and left ventricular end systolic volume[5] . A slight increase in the left ventricular stroke volume observed in this study (statistically insignificant) is contrary to our expectation since the left ventricular end systolic volume (means the ventricle has contracted to a lesser extent during systole) showed a significant increase. This would mean, a reduction in myocardial contractility might have contributed to an increase in the left ventricular end systolic volume[6]. Earlier studies have shown that stimulation of trigeminal receptors on the face during diving are integrated at the cardiovascular centers in the medulla oblongata and stimulate parasympathetic influence on the heart[7,8,9]. The increased parasympathetic tone on the heart, thus can conveniently explain both bradycardia and an increase in end systolic volume observed in the present study[10,11] .

CONCLUSION

In conclusion, while bradycardia is a common feature observed in both actual diving and breath hold face immersion in water, the significant increase

observed in the left ventricular end diastolic volume in the present study, differs with some of the previous observations of actual diving. So it may be difficult to extrapolate the results of this study to those of actual diving. However more studies comparing the changes between the actual diving and breath hold face immersion in water may help to decide the correlation between the two. Once this correlation is established, the efficacy of the diving reflex may be conveniently assessed in prospective divers using the method employed in this study.

Acknowledgement: We would like to thank all the subjects and the technical people who participated in the study

Conflict of Interest – Nil

Funding – Self

Ethical Clearance - obtained from the institutional ethical committee.

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Comparative Study of Pure Tone Audiometry (PTA) and Brain Stem Auditory Evoked Potentials (BAER) in Type 2 Diabetes Mellitus with Duration of More than 5 Years

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ABSTRACT

Background: Prevalence of Diabetes Mellitus (DM) is increasing worldwide and it is more pronounced in India. Chronic complications are attributed to number of changes occurring at variable time period involving the vascular system, nerves, skin and lens. These complications are responsible for negative effect on quality of life with diabetes mellitus. Hence it has become important that chronic complications are recognized early and necessary interventions are made.

Aim: This study was carried out to compare pure tone audiometry and brainstem auditory evoked potentials in type 2 diabetes mellitus patients with duration of more than 5 yrs and also in age, sex matched controls.

Material and Method: 30 type 2 Diabetics with duration of more than 5yrs and 30 age, sex matched controls were subjected to Pure Tone Audiometry (PTA) and Brainstem Auditory Evoked Responses (BAER).

Results : PTA - Avarage hearing loss with mean value is 30.6600 (p value is .018).

A – B gap with mean value is 5.02006. (p value is .005) .

BAER - Left side latencies III is decreased (p value 0.012). left IPL III -V is decreased (p value 0.012), right IPL III – V decreased (p value of 0.010).

Amplitudes – I and V were not significant on booth sides .

Conclusion*:* To conclude our study showed unilateral deafness in PTA and bilateral deafness in BAER. This indicates use of more than one modality of electrophysiological tests to buffer the fallacies of a single mode, in evaluating central neuropathy in patients with type2DM. Earlier diagnosis of central neuropathy is recommended to offer an early opportunity for proper management.

Keywords: *Diabetes mellitus, Pure Tone Audiometry, Brainstem Auditory Evoked Potentials, Neuropathy.*

INTRODUCTION

The constellation of abnormalities caused by insulin deficiency is called Diabetes Mellitus [- 1]

Diabetes Mellitus is a group of diseases marked by high levels of blood glucose resulting from defects in insulin production , insulin action or both. Link between diabetes mellitus and hearing loss has been debated since 1960. [- 2]

According to the International Diabetes Federation(IDF), in 1985, 30 million people had diabetes worldwide. The number rose to 150 million in 2000,285 million in 2010 and it is estimated to be 435 million by 2030 (7.8 % of the adult world population). India has the highest number of diabetics, in the world. By next year, the country will be home to 50.8 million Diabetics, making it the worlds unchallenged

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Diabetes capital. The number is expected to go up to 87 million (8.4 % of the country's adult population by 2030). [-3]

In diabetes mellitus patients all cells of the body are exposed to high levels of glucose but it is observed that symptoms of complications are arising only in few cell types. This may be because many of such complications are unrecognized or only particular cells are affected by hyperglycemia. The sense organ of hearing the organ of corti has complex components that are arranged in such a way that makes it a potential target for hyperglycemia. Damage to any part of hearing mechanism can lead to hearing loss. Hearing loss is one of the chronic conditions which are highly prevalent and associated with depression and functional decline. Still it is one of the chronic conditions which is often underdiagonosed. [4]

The hearing tests like PTA and BAER which are non invasive tests measures hearing sensitivity and central conduction time. PTA measures hearing sensitivity across a range of sound frequencies whereas BAER latencies and interpeak latencies indicative of increased conduction time.

This study was carried out to compare PTA and BAER reports in patients with type 2 diabetes mellitus with duration of more than 5 yrs and also in age, sex matched controls.

AIM

This study was carried out to compare pure tone audiometry and brainstem auditory evoked potentials in type 2 diabetes mellitus patients with duration of more than 5 yrs and also in age, sex matched controls.

MATERIALS & METHOD

Study type - Case control study.

Study location - Narayana medical college and hospital , Nellore.

Study duration - 12 months from Aug 2010 to 2011.

The test group were type 2 DM patients , who were attending outpatient department . Informed consent was taken. All the subjects underwent a detailed clinical examination before being included in the study as per the study protocol. Institutional ethical committee clearance was taken.

PARTICIPANTS

Case group - 30 Diabetics with duration of more than 5 yrs including both sex, aged between 40- 60 yrs of age were included.

Control group - 30 healthy individuals between 40 - 60 yrs of age, with no history of diabetes mellitus.

There was no past history of consumption of ototoxic drugs, ear surgeries, infections of ear, nose and throat in both case and control group.

Occupational history (where noise pollution is more) such as factory workers, traffic police were excluded in both groups.

PTA and BAER were evaluated in both groups.

[1] Pure Tone Audiometry (PTA)

It is an audiological investigations in which we test the hearing sensitivity of a subject only for pure tone sounds across a range of sound frequency.

Principle: Audiometry is an objective & accurate method to assess the degree of deafness and frequency range at which it manifests. This was done by using an audiometer which is an electro acoustic device .

REQURIMENTS

1)Electronic oscillator - It can generate pure tones of frequencies ranging from low to high.

2) Intensity dial - It helps to adjust the threshold intensity of hearing for each tone. 3)A headphone - Helps to deliver the pure tones of various frequencies to each ear separately.

Apparatus :- Diagnostic Audiometry

Elkon, Elkon private limited

E M - 060399, 2006.

Procedure: The test was conducted in a sound-proof room. Each ear was tested separately. The instrument used for this is an electronic device, the pure tone audiometer.

It consists of an audio oscillator which generates pure tone sounds of various frequencies usually

at regular steps of 125 , 250, 500, 1000, 1500, 2000, 3000, 4000, 6000, 8000Hz. Each tone can be separately amplified to a maximum of 100 or 120dB in most frequencies. The audiometer was connected to standard and specified earphones or to a bone conduction vibrator through which the sound is presented to the subject ear. The subject was asked to wear headphone and instructed to raise his finger whenever he hears a sound. - 5

METHOD

First, the patient was familiarized with tone.

1. Following a satisfactory response , tone was reduced by 10dB, until no further response occurred.
2. Tone level was increased to 5dB, until a response has obtained.
3. Once we get responses, level was decreased by 10dB,followed by an another ascending 5dB series until the subject responded again .
4. This cycle continues until the subject responded to the same level (i.e 50% or more) on increasing the tone . This was the hearing threshold level .
5. Starting at a clearly audible level proceeded to next frequency by "10dB down, 5dB up method" until the threshold criteria was satisfied. [6]

Normal values of pure tone average

Pure tone average	Hearing Acuity
0 - 25 dB	Normal Hearing
25 - 40 dB	Mild Loss
40 - 60 dB	Moderate Loss
60 - 90 dB	Severe Loss
>90 dB	Profound Loss [7]

[2] Brainstem Auditory Evoked Responses(BAER)

Synonyms: Brainstem Evoked Response Audiometry(BERA), Auditory Brainstem Response(ABR), BAER (Brainstem Auditory Evoked Response).

Requirements- Cathode ray oscilloscope, pre-amplifier, ether or spirit, EEG Electrodes , EEG jelly, earphone.

Apparatus :- GSI Auder

Viasys Neurocare **.** INC

REF no - 200197XX

S NO - AT070353

Principle- A brief auditory stimulation generates action potentials in the auditory pathway .These potentials are recorded from the ear and vertex as BAER .

Procedure: The stimulus either in the form of click or tone was transmitted to the ear via a transducer placed in the ear phone or head phone. The wave forms of impulses generated at the level of brain stem were recorded by the placement of electrodes over the scalp.

Since the electrodes should be placed over the head, the hair must be oil free. The standard electrode configuration for BAER involves placing a non inverting electrode over the forehead, and inverting electrodes placed over the ear lobe or mastoid prominence. One more earthing electrode was placed over the forehead. This earthing electrode is important for proper functioning of preamplifier.

Since the potentials recorded are in far field, well displaced from the site of impulse generation, the wave forms recorded are very weak and they need to be amplified. This amplification is achieved by improving the signal : noise ratio.

Filtering: - This is employed to reduce the recording bandwidth so that only the importat components of the signal generated are record.

Repeated stimulation: This is done with synchronous time domain averaging to increase the amplitude of the components of the signal. In real time situations these two can be achieved by connecting the recording electrodes to a preamplifier, with appropriate filter settings. Polarity alteration: By altering the polarity of impulses recorded, the artifacts are cancelled making the brain stem waves stand out.

In auditory brain stem evoked response audiometry, the impulses are generated in the brain stem. These impulses when recorded contain a series of peaks and troughs. The positive peaks are referred by the Roman numerals I - V.

These peaks occur in response to click stimuli over a period of 1-10 milliseconds after the stimulus is given. [8]

A) Absolute latency : It is measured in msec from peak of the respective waves.

B) Absolute amplitude : It is measured in microvolts (uV) from the peak of the wave to the bottom of the wave .

C) Interpeak latency (IPL) :Commonly used IPL in clinical practice are I-V,I-III and III-V.

a) I-V IPL means latency difference between wave I and V. b) I-III IPL means latency difference between wave I and III.

c) III -V IPL means latency difference between wave III and V. [9]

Wave I - Generated from distal part of auditory nerve .

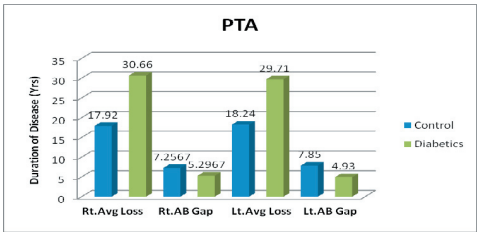
Wave II - Generated from proximal part of auditory nerve .

Wave III - Generated mainly from the cochlear nucleus.

Wave IV - Generated mainly from superior olivary complex & also from lateral leminiscus. Wave V - Generated mainly by lateral leminiscus (positive component) & inferior colliculus (negative components) [10]

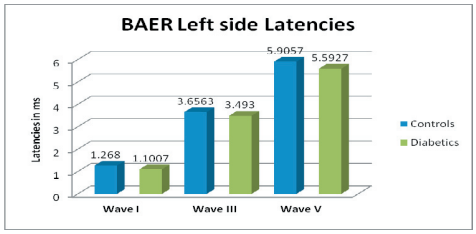
RESULTS

I) **Pure tone audiometry**

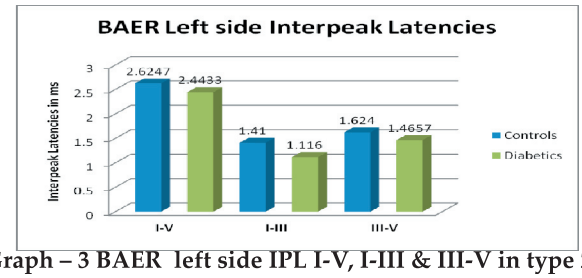


Graph - 1 Showing PTA in 30 type 2 DM and in controls.

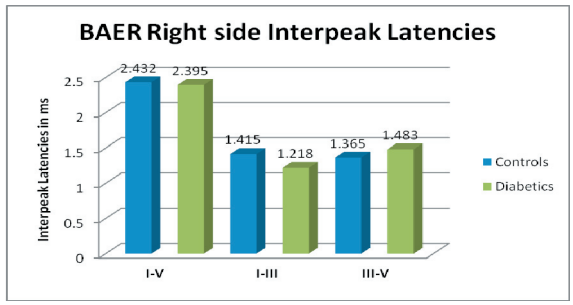
II) Brainstem auditory evoked potentials



Graph – 2 BAER Left side Latencies I , III ,V in 30 type 2 diabetics and controls



Graph – 3 BAER left side IPL I-V, I-III & III-V in type 2 diabetics and controls.



Graph - 4 BAER right side IPL I-V , I – III & III – V in type 2 diabetics and controls.

DISCUSSION

PTA and BAER reports were compared in 30 type 2 DM patients and also with healthy control group. The results were then subjected to statistical analysis. Following are our observations.

PTA measures hearing sensitivity across a range of sound frequencies.

BAER is a noninvasive test useful for earlier detection of damage in central neural axis in patients with diabetes mellitus.

PTA and BAER were normal in control group, where as in 30 Diabetics with duration of more than 5 yrs the results were as follows

PTA

Right Side shows avarage hearing loss with mean value 30.6600 (pvalue is .018). A – B gap with mean value 5.02006. (p –value is .005)

Left Avarage gap and A-B gap both were not significant (p value of = .062**).**

BAER

Left side

Latencies - Among I, III & V , latency III is decreased (p value 0.012).

Interpeak letencies - Among IPL I-III, I-V & III-V , left IPL III -V is

decreased (p value of 0.012).

Amplitudes – I and V were not significant .

Right side

Latencies - were notsignificant.

Interpeak letencies – Among IPL I –III , I – V & III – V, right IPL III – V is

decreased (p value of 0.010).

Amplitudes – I and V were not significant.

PTA - our study matches with the study done by Susan.T.frisina where they obsreved hearing loss in right ear was more when compared to left ear. In this study they have shown that in type2 DM the damage to vascular endothelial cells accelerates the age related decline in hearing in the right ear. Added to this there are asymetries in the blood supply to the right and left cochlea. As a result the vasculature to the right ear is more compromised with age in type 2 DM.[11]

But in some studies hearing loss was found in both ear.

BAER showed delay in left side latencies III and delay in both right and left side interpeak latencies III - V in DM patients. But amplitudes were not significant on both sides.

Three main mechanisms have been proposed to explain pathogenic mechanism of Diabetic neuropathy.

a) Alteration of endoneural vessels leading to widespread anoxia or multiple infarcts.

b) Metabolic abnormalities that include reduction of free myoinositol , Na+ K+ ATPase & rate of protein synthesis.

c) Direct alteration of protein by nonenzymatic glycosylation.

Each, or a combination of these mechanisms could lead to axonal degeneration by impairing axonal transport or causing a direct injury to axon.[12]

So patients with type 2 diabetes can have subclinical hearing loss & impaired auditory brainstem responses, independent of peripheral neuropathy.[13]

CONCLUSION

Central neuropathy in type 2 DM is not uncommon even in absence of peripheral neuropathy. It is related to patient's age, duration of DM, HbA1c level and diabetic neuropathy.

In our study PTA showed unilateral deafness, whereas BAER showed bilateral deafness. This indicates use of more than one modality of electrophysiological tests can buffer the fallacies of a single mode and is advisable in evaluating central neuropathy in patients with type2 DM. Earlier diagnosis of central neuropathy is recommended to offer an early opportunity for proper management.

Acknowledgement - Nil

Conflict of Interest - Nil

Source of Funding - Self

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A Simple, Inexpensive and Portable Model for Teaching Human Body Composition to Undergraduate Health Science Students

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ABSTRACT

The study of human body composition has several dimensions. This includes medical, anthropological, physical activity performance and aesthetic aspects. Understanding, in particular, of human body composition is very essential to medical and allied health science students. But, the teaching of this aspect to them is given little or no importance at the entry level in most situations. Body composition at two or three compartment levels that include body fat and fat free mass is adequate and appropriate for such students for a good understanding and clinical application. The teaching of this area of human body composition can be made easier, interesting and appealing by making use of a simple and economical physical model. Designing, constructing and use of such a model is presented in this communication.

Keywords: *Human body composition, teaching model, health faculty students.*

INTRODUCTION

Body composition studies have greater importance because of its closer relevance to obesity, diabetes, cancer, arthritic problems and related quality of life. It is very essential to measure body composition in metabolic and nutrition studies[1]. Hence, health faculty students should have a good knowledge and clear understanding of this subject. But, very little coverage is given or not at all in basic medical and health sciences degree curriculum for teaching this fascinating aspect. And moreover, students would be having difficulties as beginners to certain words as they sound similar and may not have a proper concept of body composition. There are several levels at which body composition estimations in humans are made[2]. Two or three compartmental level of body composition that depicts body fat and fat free mass/lean body tissue (including body water) is good enough for undergraduate teaching. This can be achieved and could be made very interesting, clear and easily understandable by making use of a simple physical model.

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METHOD

A four-member team embarked on this project of designing and producing the model under the guidance of the authors. Attention was given to keep the model simple, light in weight, portable, easy to use and more than all, to keep the cost low without losing the quality of contents that was to be demonstrated. The materials used were ceramic powder, plaster of Paris, modeling clay, used news paper, good quality glue and color paints. The first step involved was in making suitable drawings. Based on that, a basic mould was prepared using plaster of Paris and clay. Three impressions on the mould were taken with a paste of ceramic powder, paper pulp and glue after a thorough drying of the mould. Different components of body composition were effectively brought out on each dried impressions and the mould with appropriate color paintings by using suitable reference drawings or dissected specimen (see figures 1-4). While teaching/demonstrating, the three impressions can be lifted up to explain the body

composition in an orderly manner. Fat mass and body water (intracellular and extracellular that includes blood, cerebrospinal fluid, lymph and synovial fluid) and bone and skeletal muscle mass as components of fat free mass were depicted on these structures.

Following the use of the model for teaching in lecture classes and tutorial sessions (small group teaching) for medical, physiotherapy and laboratory technology course students, a set of questions were asked on the effectiveness of using the model in understanding the concept of body composition (such as the appropriateness of the model, level and clarity of understanding and perception and the sense/degree of satisfaction) to the students to get a feed back.

RESULTS

The model was tested for teaching both in a class room setting during the lecture and in small group during tutorial sessions. Students enjoyed particularly the tutorial hours and were highly impressed and satisfied. They reported that their understanding of the concept of body composition was much better by using the model. This was supported by the feedback from the students who rated very positively at an average of 4 points on a 1 to 5 scale starting from 1 as a least response.

DISCUSSION

This communication describes the developing of a suitable, portable, and a simple model for teaching human body composition to entry level health science students. The most important features of this model are that it is least expensive, affordable, quite light, easily dismantled and reassembled and transported. It is particularly good and useful for small group teaching. It is not a high-tech stuff, but provides the 'feel touch' sense and a good degree of satisfaction during the learning process.

It may not be inappropriate here to briefly discuss the body composition. Human body mass is primarily made up of 30 or more components which can be measured by employing various methods[3,4]. Body composition is important in understanding human growth process. It reflects various aspects of life style such as dietary habits, physical activity as body composition changes in diseases, altered nutritional status and at different degrees and styles of physical activity[1]. However, the two major components of the body can be grouped as fat and fat free mass (bone, water, muscle, connective and organ tissues). The major component (about 60%) of the human body is water. These components are effectively depicted in the model. When people gain or lose weight they will add or reduce the amount of fat mass and to a much lesser degree, fat-free mass. Excess body fat exposes a person to a higher risk for health problems such as high blood pressure, elevated blood cholesterol, diabetes and risk for heart disease and stroke and also to cancer, impaired immune function, gall bladder disease, kidney disease, skin problems and sleeping problems [5,6].

The major contribution of using the model is as follows. A learning style or preference is the complex manner in which learners perceive, process, store, and recall what they learn most efficiently and most effectively. VARK, a guide to learning styles classifies learning preferences based on sensory modalities in to four modes such as Visual (V) – seeing graphs, charts, flow diagrams, drawings, diagrams, pictures, colored word accents, demonstrations etc.; Auditory (A) – listening, interacting, discussing, speech; Reading-writing (R) – textual contents, reading books, word lists, writings, handouts; and Kinesthetic (K) – physical touch, manipulating objects or materials. Students will have their own individual learning style preferences[7]. It is also mentioned that the students will remember 20% of what they read, 30% of what they see, 50% of what they say and 60% of what they do[8]. In that sense visual and kinesthetic style of learning seems better that is met by using our model.

Therefore, it is important to recognize the other factors that influence learning; stimuli and preference for learning/processing new information because when information is presented using simple and effective learning style, not only teachers are better able to connect with students but students also achieve better levels of understanding and sense of satisfaction[7].

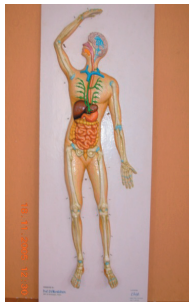

Figure 1.
First mould

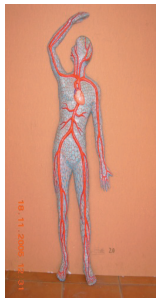

Figure 2.
Impression 1

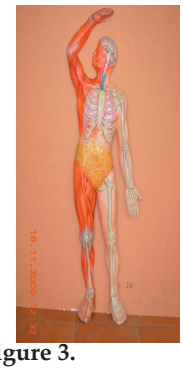

Figure 3.
Impression 2

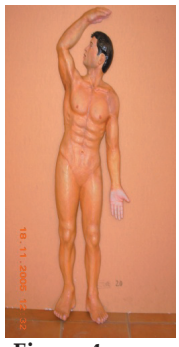

Figure 4.
Impression 3

Impressions 1, 2, and 3 are placed on the mould in that order while assembling and removed in reverse order while dismantling.

In the mould (Figure 1), transcellular fluid such as synovial fluid, cerebrospinal fluid, fluids in the gastrointestinal tract are represented. In figure 2, blood, intracellular and extracellular fluids are represented. In figure 3, skeletal muscle mass, visceral fat mass and bone mass is represented. Figure 4 represents the body surface covered by skin.

CONCLUSION

Since body mass index is becoming less relevant as an index of body fat, a strong recommendation has been made to measure actual body composition, particularly body fat in such studies as it would reflect a more realistic picture[9]. Since physiology deals with the functioning of the human body and its organs, it is good that they teach the concepts of body composition at the beginning of the course. And, thus the present model is hoped to help develop a fair and clear concept of the human body composition that can be termed simply as a 'bloodless dissection' of the human body.

There are web based and multimedia models for teaching human body composition[10]. Such facilities will have their own advantages and disadvantages and it may not be possible to be employed by all for various reasons. Our physical model is one that allows close observations for detailed study with kinesthetic aspect of touch and feel for a better learning and satisfaction.

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Conflicts of Interest: The authors declare no conflict of interest.

Ethical Clearance: Approved by the Institute's Research Ethical committee

Source of Funding: Self

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Association of Glycated Hemoglobin and Serum Lipid Profile in Type 2 Diabetes Mellitus Patients

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ABSTRACT

Objective : To find relationship between glycemic control and lipid profile Type 2 DM**.**

Material and Method: This study includes 48 Type 2 DM subjects. For each patient, data regarding age, gender, duration of diabetes, and use of medications were recorded. A detailed physical examination was performed, glycosylated hemoglobin (HbA1c) values serum total cholesterol, serum triglycerides and comorbid conditions were noted. Quality and quantity of sleep was evaluated by Pittsburgh Sleep Quality Index (PSQI) questionnaire**.**

Results: Plasma triglycerides and total cholesterol were significantly increased in diabetic patients compared with control group. HbA1c showed positive correlations with triglycerides and total Cholesterol ($r = 0.66$, $P = 0.01$ and $r = 0.32$, respectively).

Conclusion: It is concluded from the results of this study that HbA1c can be used as a predictor of dyslipidemia in patients with type II diabetes**.**

Keywords: Diabetes mellitus; Lipid profile; glycosylated Haemoglobin.

INTRODUCTION

Diabetes is a global endemic with rapidly increasing prevalence in both developing and developed countries. According to the International Diabetes Federation (IDF),more than 371 million people across global have diabetes and this figure is predicted to raise over 550 million by 2030[(1)].

Diabetes is commonly associated with abnormalities in plasma lipids and lipoproteins commonly referred to as "Dyslipidemia" which is most of the time unnoticed. Lipid abnormalities are common in Type 2 DM than Type 1. Type 2 diabetes mellitus is associated with the development of premature arteriosclerosis and a higher cardiovascular morbidity and mortality. Diabetic hyperlipidemia is believed to play an important role in the pathogenesis of accelerated atherosclerosis.[2,3] Silent myocardial ischemia has a reported prevalence of 10-20% in diabetic population as compared to 1-4% in non-diabetic population.[4] Dyslipidemia and hyperlipidemia generally coexist in diabetic patients with poor glucose control. Although they have been shown to be independent significant risk factors for vascular complications, the interaction of hyperglycemia and dyslipidemia increases the risk of macro and micro-vascular complications together. Dyslipidemia which includes both quantitative and qualitative abnormalities of lipoproteins can also play significant role in the pathogenesis of vascular complication in patients with type 2 diabetes.[5]

AIM OF THE STUDY

This study tests the hypothesis that there is a relationship between glycemic control in Type 2 diabetic subjects and lipid profile Type 2 DM.

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METHOD

Participants: The study was carried out at the BMCRI,Bangalore, South India.The study population was comprised of type 2 diabetes patients attending the Outpatient Clinic. The total study sample included 48 patients. All these subjects were not recently diagnosed and had blood tests within 90 days of the interview. All participants signed an informed consent form before inclusion in the study.

Inclusion criteria: Type 2 DM

Exclusion criteria: a) Type 1 DM b) unable to give consent c) smokers and alcoholic d)causes of secondary dyslipidemia were excluded e) weight loss > 6kgs in past 6months f)tuberculosis g) Asthma h)Hepatic and Renal impairment i) Ischemic Heart Diseases j) Subjects on hypo-lipidemic drugs.

DATA COLLECTION

For each patient, we recorded data regarding age, gender, duration of diabetes, and use of medications. A detailed physical examination was then performed. All vital signs and glycosylated hemoglobin (HbA1c) values,serum total cholesterol and serum triglycerides were available for all the patients and were recorded from their case sheets. All the study patients were on anti-diabetic medications, either insulin or oral hypoglycemic agents or both. The presence of major complications of diabetes (neuropathy, retinopathy, nephropathy, coronary artery disease, and peripheral vascular disease) was also assessed.

STATISTICAL ANALYSIS

Data are presented as mean ± standard error of mean (S.E.M.) Also co-relationship between glycated hemoglobin and lipid profile parameters was ascertained by Pearson's correlation coefficient(r).Two tale value of $P < 0.05$ was considered statistically significant for all analyses.

RESULTS

We divided the subjects based on their glycemic control into two groups; HbA1c≤ 7% as good, HbA1c>7% as worse glycemic control. Table 1 shows Mean±SD values of age,Total cholesterol(TC0 and Total triglycerides(TG) between groups. It was also observed that Total cholesterol and Total triglycerides were significantly higher in type 2 diabetics with poor glycemic control which was statistically significant.

Table 1:

Variable	HbA1C(≤7)	HbA1c(>7)	P value
Age(yrs)	53.8±5.8	51.6±7.6	0.26
Total cholesterol(mg/dl)	141±17.1	171.6±20.5	0.002*
Total triglycerides(mg/dl)	98±10.2	126±33	<0.01*

Table 2: Correlation between glycated hemoglobin and total cholesterol and triglycerides

HbA1c showed direct and significant correlations with serum cholesterol (r = 0.26) and TG levels (r=0.32).

Correlation between	r	P value
Total cholesterol(mg/dl) and HbA1C	0.26	0.001*
Total triglycerides(mg/dl) and HbA1C	0.32	0.001*

DISCUSSION

A high prevalence of dyslipidemia is well recognized in type 2 DM. Approximately 77.5% of type 2 diabetic patients exhibit dyslipidemias. Although the dyslipidemias in T2DM, particularly hypertriglyceridemia, are exacerbated by poor glycemic control, lipid abnormalities cannot be explained merely by hyperglycemia. The main cause of diabetic dyslipidemia is the increased free fatty acid release from insulin resistant fat cells. The increased influx of free fatty acids into the liver in the presence of adequate glycogen stores promotes TG production, which in turn stimulates the secretion of apolipoprotein B and VLDL cholesterol. Lipoprotein lipase (LPL) plays a central role in TG metabolism. LPL, if defective, could predispose to hypertriglyceridemia (6). The main disorder in lipid metabolism in our study were hypercholesterolemia and hypertriglyceridemia, which are significantly higher among patients with worse glycemic control.

Normally Insulin plays important role in inhibiting intracellular hormone sensitive lipases of adipose tissue and activating lipoprotein lipase because of lack of insulin action in Type-2 Diabetes Mellitus, the activity of lipoprotein lipase gets

depressed whereas the activity of hormone sensitive lipase increases. Also because of insulin deficiency in DM, glucose cannot be utilized for energy purposes by the cells. Thus there is increased lipolysis leading to increased FFA (Free Fatty Acids) which are then catabolized to acetylCoA in Liver and other tissues. Due to deficiency of acetyl CoA carboxylase in DM;(the Enzyme that converts acetyl CoA to malonyl CoA) there is no conversion of acetyl CoA to Malonyl CoA. Hence excess acetyl CoA gets converted to more & more cholesterol & its concentration in blood rises in Type-2 DM. VLDL & LDL increases either because of increased hepatic production of VLDL or decreased removal of VLDL and LDL from circulation[7]. Serum concentration of triglycerides also increases because of decreased removal from circulation. Hence Hyperglycemia is related with deranged Lipid Profile and this may lead to dyslipidemia

The significant increase in total cholesterol and triglycerides in patients with higher HbA1c value indicates that severity of dyslipidemia increases in patients with increased HbA1c value. This is found in agreement with findings of Khan, H.A et al. [8] and Rohlfing, C.L. et al. [9] Hence improving glycemic control can substantially reduce the risk of cardiovascular events in diabetics.

CONCLUSION

Dyslipidemia is associated with high HbA1C, but correlation of different parameters of lipid profile probably depends from genetic fond of population and differences specifically for various regions patterns of life.

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Conflict of Interest: Nil

Source of Funding: Self

Ethical Clearance: Not applicable

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Impact of Length of Visual Pathway on VEP Latency

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ABSTRACT

Introduction: Visual evoked potentials (VEPs) are electrical potentials evoked from visual stimuli and recorded from the human scalp.VEP latencies and amplitudes are affected by many variables like age, gender, core body temperature, hormones and head size. It was hypothesized that head size would indirectly reflects brain size and the length of the visual pathway and hence the conduction time. In this study P100 latency (P100L) of VEP was compared with head size for males and females to analyze the impact of length of the visual pathway on P100L of VEP.

Method: 34 subjects, 17 males and 17 females of age group 17-25 were participated. Head measurements like head circumference (HC), nasion to inion (AP-anteroposterior-measured across the top of the head), and right external auditory meatus to left (RL-also measured across the top of the head) were measured. Total head size was formed by adding all three measures.VEP was performed by checker board reversal stimuli system.

Results: Statistical analysis showed significant differences between males and females in head size (HC,AP,RLand total head size) with a P value of < 0.01.P100 L was prolonged in males than females with a significance of P value < 0.01.Pearson's correlation of head size with P100L showed that head size has a moderate positive influence on latencies.

Conclusion: In the present study significant positive correlation have been found between external measurements of head and VEP latency. This study support the theory that length of the visual pathway and hence the conduction time can be correlated to the size of the head. Thus head size should be considered for normal VEP recording. Further studies can also be carried out in different age groups.

Keywords: *VEP, Length of visual pathway, Head size, Gender.*

INTRODUCTION

Evoked potentials are the important non-invasive diagnostic tool used in the assessment of conduction of sensory impulses in the nervous system [(1)]. Visual evoked potentials (VEPs) are electrical potentials evoked from visual stimuli and recorded from the human scalp. It consists of 3 unpaired waves N70,P100 and N135 [(2)]. They are used to interrogate the functional integrity of the visual pathway from retina up to visual cortex[(3,4)]. P100 is the dominant wave and usually seen in all normal subjects[(1)]. other synonyms for VEPs include visually evoked response (VER) and visually evoked cortical potential(VECP)[(5)]. VEP latencies and amplitudes tend to vary with many factors like age,gender,core body temperature, hormones and head size [(3,6,7,8,9,10)]. It was hypothesized that head size would indirectly reflects brain size and hence the length and conduction time in the visual pathway[(11)]. certain studies have reported positive correlation between head size and latencies of VEP,[(8,11,12)] while others have found little or no significant relation[(13)]. Therefore this study was conducted to analyze, whether P100 L of VEP is influenced by different head sizes.

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MATERIALS & METHOD

34 subjects, 17 males and 17 females were selected. All were aged between 17-25. The study was done at physiology research laboratory, Thanjavur medical college, Thanjavur from April 2014 to Dec 2014.A detailed history and thorough clinical and ophthalmic examination was done to rule out any medical problem. Subjects with amblyopia, corneal opacity, squint, and colour blindness, history of ophthalmic surgery, ptosis, glaucoma, retinal pathology, those on myotic or mydriatics, neuromuscular disorder or other diseases that might affect visual acuity were excluded. Informed written consent was obtained from all the participants and experimental protocol was approved by the college ethical committee.

Head measurements like head circumference (HC), nasion to inion (AP-anteroposterior-measured across the top of the head), and right external auditory meatus to left (RL-also measured across the top of the head), were measured. Total head size was formed by adding all three measures.

The study was done with 4 channel Digital Polygraph. Digital index colour monitor, 17 inch model- no: IT-173SB.

VEP- Experimental Design and Recording:

VEPs were performed by checkerboard pattern reversal displayed on a TV monitor subtending 15°×12° at a viewing distance of 90cm and individual squares in the checkerboard pattern subtended a visual angle of 60°. The stimuli reversal rate was 2 per second. Electrode scalp placement and recording parameters were carried out according to the standard of the International Society for Clinical Electrophysiology of Vision (ISCEV) [14].

Standard disc EEG electrodes were placed at the Oz position (active electrode) and reference electrode was placed at Fz position & ground electrode on the patient's vertex (Cz).The subject was instructed to fix his gaze at the centre of the screen. The latency of P_{100} was measured.

VEP Pre test instructions [2,]

1. Informed about the procedure of the test and got informed consent.
2. To avoid hair spray or oil after the last hair wash.
3. The room parameters should be maintained constant throughout the experiment.
4. Not to use any eye drops (miotic/ mydriatics) 12 hrs before the test.

Statistical analysis

Head size and P100L between males and females were analyzed by unpaired student's t-test.P100L was correlated with head size in males and females by Pearson's correlation coefficient.Graphpad statistical software was used for data analysis.

RESULTS

The mean age for male and female was 17.8,17.3 respectively. Comparison of head size of 2 groups showed significance in HC,AP,RL and in total head size with significant P value of < 0.01.(Table1).P100L was prolonged in males than females with a significance of P value < 0.01(table2). Table 3 presents Pearson's correlation coefficient for whole group ,males and females. When total head size was correlated with P100 for whole group and females, there were moderate positive influence of head size on latency with a significance of P< 0.01, and P<0.05 respectively. Whereas in males it showed weak positive correlation with a significance of P<0.1.

Table 1: Head size of male and females

Head size (cm)		Mean	SD	P
HC	Male (n=17)	55.65	1.06	<0.0001
	Female (n=17)	54.06	0.75	
AP	Male	34.94	2.41	0.0011
	female	32.65	1.06	
RL	male	34.676	2.404	0.0003
	female	32.176	0.728	
Total Head size	male	125.265	5.197	<0.0001
	female	118.882	2.147	

Table 2 :VEP

P100-L(ms)	Mean	SD	P
male	102.71471	2.65329	0.0062
female	99.8529	3.0260	

Table 3: Pearson's correlation

	Whole group(n=34) Total head size		Male(n=17) Total head size		Female(n=17) Total head size	
P100L(ms)	Pearson's correlation (R)	P	Pearson's Correlation (R)	P	Pearson's Correlation (R)	P
	0.5749	0.000376***	0.4176	0.095338*	0.5759	0.01555**

*** P< 0.01 ** P < 0.05 *P<0.1

DISCUSSION

The present study analysed the impact of length of visual pathway on VEP latencies between males and females. Results found that males had bigger head measures (HC, AP, RLand total head size) than females with significance of P< 0.01. P100 latency was longer in males than females and it was statistically significant P<0.01. The gender difference seen in P100L could be because of bigger head size in males. Pearson correlation revealed a significant positive influence of head size on P100L. Hence head size should be considered for VEP study.

Allison T et al in 1983 reported that in adults the peak latencies of (BAEP) brain stem auditory evoked potential, VEP and somatosensory evoked potentials were significantly longer in males than females. The reason for the differences was explained by sex differences in brain size. However no significant differences in VEP latencies were observed in children [8].

Celesia GG et al in 1987 studied the VEP response with 15′4 and 31′4 checks. Shorter VEP latencies were observed in females for both checks. The effect of age on VEP was different for different size stimuli. It was revealed that, as the individual ages there was random neuronal cell loss in the visual pathways and the latency difference between gender may be related to anatomical brain size and hormonal influences [7] . J Lanrova (2012) found very small P100L differences between males and females[3]. Dion LA et al showed larger amplitude in girls but the effect on latency is negligible[13]. The effect of head size on VEP parameters is inconsistent across studies. The present study is consistent with Bruno Gregori et al.They observed that P100 L was slightly shorter in females than males P< 0.05, and found the difference was related to smaller head size in females than males.

CONCLUSION

In the present study significant positive correlation have been found between external measurements of head and VEP latency which revealed that length of the visual pathway and hence the conduction time can be correlated to the size of the head. Therefore based on the fact that the average male head size is bigger than the females, this study also support the theory that differences in the conduction time between males and females are due to the differences in actual brain size[8, 15]. Further research can also be carried out in different age groups.

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Comparison of Tread Mill and Ergometer Cycle and its Effects on Lipid Profile

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ABSTRACT

The aim of this study is to compare the tread mill and ergometer cycle and to identify the better instrument which favours the lipid profile. This study includes 40 subjects, and divided into two groups Group A consists of 20 subjects who underwent treadmill training and Group B consists of 20 subjects who underwent cycling. All subjects underwent aerobic training of moderate intensity for 12 weeks, five days in a week, one hour per day on tread mill for Group A individuals and five days in a week, fifteen minutes per day on the cycle for Group B individuals. The mean difference between pre and post tests of treadmill, cycle groups respectively were compared to know which one is the best instrument and gives favourable results for lipid profile. Treadmill group showed higher energy expenditure and more favourable results compared to cycle group

Keywords: *Tread mill, Bicycle Ergometer Aerobic exercise, Lipid profile.*

INTRODUCTION

Aerobic exercises are advised for health promotion and prevention of many cardiovascular diseases. To perform aerobic exercise many modes are available, of which in the modern busy life, the tread mill and ergometer cycle are the commonest indoor exercise machine used for this purpose. Kravitz et al[(1)], compared energy expenditure and VO_2 during treadmill running, cycle ergometer, simulated cross country sky. Results showed treadmill exercise is the modality of choice for individuals who try to improve cardio respiratory endurance and expend high energy expenditure. Ravikiran et al" [(2)] studied the effects of the treadmill and bicycle ergometer on cardiovascular responses, both treadmill and ergometer cycling have different group of muscles involved in exercise. Narges Argani'[(3)] compared the effects of different exercise intensities on bicycle ergometer. Eric C. Freese et al'[(4)] quantitatively reviewed the effects of prior exercise on postprandial lipidemia; in a meta-analytic review of literature and showed that postprandial lipemia depends upon on the type and intensity of the exercise.

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MATERIALS & METHOD

Study is a randomized control trial for 12 weeks, observing the effects of aerobic training on lipid profile. Subjects were recruited from Thanjavur Medical College Hospital and Raja Mirasudhar Hospital, Thanjavur, in the 25 to 35 years age group, study was conducted between January 2014 and June 2014. This study was conducted in the research laboratory, Department of physiology, Thanjavur Medical College, Thanjavur. Subjects were randomized into two groups Group A Treadmill group; Group B Ergometer Cycle group. This study included 40 subjects. Group A consisted of 20 subjects who underwent treadmill training and Group B consisted of 20 subjects who underwent cycling. Before starting our study, we obtained ethical committee approval and clearance from the college. Informed consent was obtained from all subjects who were participating in the study. Subjects included in our study were healthy individuals. Subjects with

history of diabetes, hypertension, hyperlipidemia, intake of drugs (lipid lowering drugs), smoking, alcoholism, coronary artery disease, pulmonary illness, endocrinal diseases, and orthopaedic limitation to physical activity were excluded from the study. If an individual was involved in any other exercise activities (including yoga) were also excluded from the study.

For tread mill group (A) subjects, hundred steps per minute for one hour per session, five days in a week for twelve weeks, in cycle group(B) subjects, sixty to seventy revolutions per minute(RPM), fourth resistance in tension adjuster, for fifteen minutes duration, five days in a week for twelve weeks were followed. Both of these protocols were qualified for the moderate exercise intensity as per WHO guidelines and AHA guidelines. Instruments used in the study are Treadmill (Cardiotrack 900 XL, Whispermill, Browndove Health care Ltd, Bangalore), Pedometer (Omron health care, Singapore, model No: HJ-005) –To calibrate number of steps in tread mill walking, fingertip pulse oximeter (Model No: MD 300C22 Nidek Medical India (P) LTD, Kolkata) - to monitor heart rate during aerobic training session, Ergometer cycle (Aerofit India, Hyderabad), Keragen Biosystem – Semi Auto analyser (Keragen Technologies Pvt. Ltd, Bangalore). All subjects who were participated in the treadmill group (Group A) wore a pedometer (to quantify the number of steps) in a belt strapped in their waist, before stepping on to the treadmill. Exercise was performed for one hour. Warm up was done on the treadmill itself for 10 minutes. To start with 1.1 km/hr was gradually increased, and finally attained the desired speed of 3.2 to3.7 km/hr (equivalent to 100 steps per minute in pedometer). Exercise intervention continued in treadmill, 100 steps per minute for 1 hour. This is followed by 10 minutes cool down with light stretching exercises. This qualifies for a moderate intensity exercise. Like this, exercise session was followed for 5 days in a week and for 12 weeks for all subjects who were in treadmill group. During the exercise, heart rate and oxygen saturation in the blood was monitored closely using finger pulse oximeter. Before starting the exercise in cycle group (Group B), seating arrangement was made comfortable and adjusted according to the height of the individual. To start the exercise session, warm up with slow pedalling of 30 to 40 RPM for 5 minutes with resistance level in tension controller is set to first level (equivalent to 1 Kilopond). After completing the warming up, the pedalling speed gradually increased to 60 to 70 RPM for 15 minutes with resistance being increased from 1 to 4 in the tension adjuster gradually. This is followed by 10 minutes cool down with light stretching exercises. Like this, exercise session continued for 5 days in a week for 12 weeks. Before starting the exercise intervention, in all subjects blood samples were collected for pre-test evaluation of lipid profile (base line sample). After 12 weeks of exercise intervention, blood samples were again collected for post-test evaluation. Blood was analysed in Keragen Biosystem – SemiAuto analyser in the biochemistry laboratory by standard enzymatic technique CHOD-PAP, End point method TC, TGL, and HDL noted from the analyser monitor, VLDL, LDL, TC/HDL, LDL/HDL were calculated.

RESULT

The mean difference between pre and post tests of treadmill and cycle groups respectively were compared to know which one is the best instrument and gives favourable results for lipid profile. Results showed more reduction of body Weight, TC, TGL, VLDL, LDL, and more elevation of HDL in treadmill group compared to cycle group indicating treadmill group had more energy expenditure**.** (Table no: 1)

Table No: 1: Comparison of mean difference between pre and post tests of treadmill, cycle groups and its inference.

Variable	Test type	Group A n = 20 TREADMILL Mean ± SD	MEAN DIFFERENCE (PRE AND POST TESTS) TREAD MILL	Group B n = 20 CYCLE Mean ± SD	MEAN DIFFERENCE (PRE AND POST TESTS) CYCLE	INFERENCE (BEST)
Weight	Pre	67.9 ± 13.92	3.70 ± 2.61	70.8 ± 15.34	3.60 ± 2.161	TREAD MILL
	Post	64.2 ± 11.96		67.2 ± 13.96		

Table No: 1: Comparison of mean difference between pre and post tests of treadmill, cycle groups and its inference. (Cont...)

TC	Pre	184.26 ± 21.99	6.96 ± 1.82	180.42 ± 20.24	6.22 ± 1.78	TREAD MILL
	Post	177.29 ± 22.42		174.19 ± 19.19		
TGL	Pre	128.33 ± 16.39	8.58 ± 11.59	129.93 ± 12.80	7.94 ± 2.75	TREAD MILL
	Post	119.74 ± 18.47		121.99 ± 12.59		
VLDL	Pre	25.66 ± 3.28	1.71 ± 1.13	25.98 ± 2.58	1.58 ± 0.55	TREAD MILL
	Post	23.94 ± 3.69		24.39 ± 2.51		
HDL	Pre	37.85 ± 4.59	-3.73 ± 1.88	37.14 ± 4.04	-3.58 ± 1.36	TREAD MILL
	Post	41.58 ± 4.67		40.72 ± 4.48		
LDL	Pre	120.74 ± 21.37	8.98 ± 3.23	117.29 ± 20.43	8.22 ± 2.51	TREAD MILL
	Post	111.76 ± 21.73		109.06 ± 19.23		
TC/HDL	Pre	4.94 ± 0.89	0.61 ± 0.246	4.90 ± 0.70	0.588 ± 0.20	TREAD MILL
	Post	4.32 ± 0.81		4.31 ± 0.579		
LDL/ HDL	Pre	3.25 ± 0.79	0.51 ± 0.28	3.19 ± 0.66	0.48 ± 0.181	TREAD MILL
	Post	2.74 ± 0.72		2.71 ± 0.55		

DISCUSSION

This study compared tread mill and ergometer cycle to identify the better instrument favouring the lipid profile. Thomas TR A. S et al'[5] studied the effects on fat and metabolic responses on four different modes of training. Their results showed treadmill and skiing group had more energy expenditure and fat utilization. These results are in accordance with our present study. Helan Carter Andrew[6] studied the physiological effects of oxygen uptake, comparison was done between treadmill and cycle group. Study results showed tread mill as high energy expenditure instrument compared to cycle. Similar results were found in our study.

In our study, Pre and post tests mean difference showed more reduction of TC, TGL, VLDL, LDL, TC/HDL ratio and LDL/HDL ratio in treadmill group and more elevation of HDL in treadmill group compared to cycle group. This is due to more energy expenditure in treadmill group than cycle group. As the mean value is a powerful and sensitive indicator, we conclude that the treadmill is better than the ergometer cycle in aerobic exercise related to lipid profile. Anne I. Zeni[7] studied the effects on indoor exercise machines, on energy expenditure which showed slight improvement in tread mill group compared to the cycle group. This is because of more energy expenditure in tread mill group. Similar results were observed in our study.

Comparing the treadmill and bicycle, in terms of energy expenditure, tread mill is ideal, as it exercises the core muscles of the body like abdominal, back and upper limbs (due to swinging of arms) and is the reason for more calorie burning on the treadmill than ergometer cycle[6,8,9,10]. In cycling, individual may fatigue easily and the energy expenditure is based on the pedalling, which is under the control of subject themselves and not on the observer's part as in tread mill. This may be a contributing factor for more energy expenditure in tread mill group compared to the cycle group.

CONCLUSION

In general, the physiological responses of both exercise modes i.e treadmill and ergometer cycle are almost similar and favour lipid profile. But treadmill

group showed higher energy expenditure and more favourable results (elevation of HDL and lowering of TC, TGL, VLDL, LDL, TC/HDL, LDL/HDL) than cycle group. Those differences which were observed are small; thus both treadmill and bicycle ergometer can be recommended for the favourable improvement of the lipid profile. The choice of instruments is individualized. If an obese or older individual is to be subjected to aerobic training, cycle is the better choice compared to treadmill. If the patient is young with no pre existing risks factor like obesity and joint arthritis, tread mill is preferable.

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Conflict of Interest: Nil

Source of Funding: Self

Ethical Clearance: Taken

ABBREVIATIONS USED IN THE STUDY

BMI - Body Mass Index

HDL - High Density Lipoprotein

LDL - Low Density Lipoprotein

VLDL - Very Low Density Lipoprotein

TC - Total Cholesterol

TGL - Triglycerides

RPM - Revolutions Per Minute

SD - Standard Deviation

AHA- American Heart Association

WHO - World Health Organization

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A Comparative Study on the Effect of Hemin on Gastric Induced Ulcer in Rats

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ABSTRACT

Background: Induction of HO_1 may act as a defensive mechanism to reduce inflammation and tissue injury in the gastrointestinal tract. **Aim:** can we use hemin (HO-1 inducer) as a new therapy in treatment of gastric ulcer? **Material and methods:** 48 male albino rats were divided into 6 groups. Group 1 (control): received 0.5 ml of the vehicle. Group 2: Hemin-treated group (H) received hemin one hour before the administration of Indomethacin (IND) (25 mg/kg b.w). Group 3: pre-treated with famotidine (3 mg/kg). Group 4: pre-treated with 20mg/kg of omeprazole**.** Group 5: pretreated with famotidine and hemin. Group 6: pretreated with omeprazole and hemin. **Results:** hemin produced a significant protective effect on gastric ulcer when used alone and highly significant changes when used with famotidine or omeprazole. **Conclusion:** hemin can be used as a new therapy in treating gastric ulcer.

Keywords: *Hemin – bilirubin - heme oxygenase – indomethacin – gastric ulcer.*

INTRODUCTION

Heme oxygenase-1 (HO-1) is a stress-inducible protein, which catalyzes oxidative degradation of heme [(1, 2)]. HO-1 has been identified in the gastric mucosa and participates in a number of cellular defense mechanisms[(3, 4 and 5)]. Induction of HO-1 in several animal models of diseases has been shown to protect tissues and cells against oxidative stress, inflammation, ischemia/reperfusion injury and apoptosis [(6 and 7)]. It has been demonstrated that gastric cytoprotection induced by polaprezinc, [(8)] eupatilin [(9)] and ketamine [(10)] against noxious agents is mediated by HO-1 induction. Recent study by **Takagi et al.,** [(11)] showed that lansoprazole, a gastric H+/K+ ATPase inhibitor, upregulates HO-1 expression in rat gastric epithelial cells and the up-regulated HO-1 has anti-inflammatory effects and that lansoprazole-induced HO-1 induction is mediated by the activation, phosphorylation and nuclear translocation of Nrf2[(11, 12)]. In addition to cytoprotection by HO-1, it has been shown that HO-1 exerts a modulatory role on gastric smooth muscle excitability via CO production[(13, 14)]**.** Using a diabetic gastroparesis model, **Choi et al.,** [(14, 15)] have demonstrated that Kit expression in interstitial cells of Cajal is lost during diabetic gastroparesis due to increased levels of oxidative stress caused by low levels of HO-1, and that CD206(+) M2 macrophages that express HO1 appear to be required for prevention of diabetes-induced delayed gastric emptying. For all these reports about the role of HO-1 in gastric protection, we investigated the effect of hemin (HO-1 inducer) on gastric induced ulcer in rats and we compared this effect with the commonest anti-ulcer drugs (H2 receptor blocker, famotidine and proton pump inhibitor, omeprazole)

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MATERIAL & METHOD

Animals: 48 male albino rats (11-13 weeks old, 180–220 g) were included in this study obtained from the Laboratory Animal Research Unit of Zagazig University, Egypt. Rats were housed in a 12:12-h light-dark cycle at 25 ± 2°C, and had free access to tap water and commercial rat chow for 7 days for acclimatization before entering the study. All

experiments were performed in accordance with the Institutional guidelines for the Care and Use of Animals for Scientific Purposes.

MATERIALS

Hemin (from Sigma, UT, USA) was freshly dissolved in 0.1 mol/L Na OH adjusted to pH 7.4 with 0.1 mol/L HCl and diluted with saline to the required volume (0.5 ml of this vehicle was given to control group). Hemin was prepared in darkness and protected from light [16].

Omeprazole: Omeprazole (purchased from EPICO, EGYPT). The dose administered was 20mg/kg b.w [17].

Indomethacin (was purchased from EPICO, EGPT). The dose used for ulcer induction was single intraperitoneal injection of indomethacin (30 mg/kg) after 24h fast [18].

EXPERIMENTAL PROTOCOL

Animals were fasted overnight but allowed free access to water until one hour before the experiment. All experiments were performed at the same time of the day to avoid diurnal variations of putative regulators of gastric functions.

Rats were divided into the following groups (each group, 8 rats):

Group 1 (control): each rat received 0.5 ml of the vehicle i.p. one hour before induction of ulcer.

Induction of gastric ulcer: Gastric ulceration was induced by a single intraperitoneal injection of indomethacin (30 mg/kg) after 24h fast.

Group 2 Hemin-treated group (H); each rat received hemin one hour before the administration of IND at a dose level of 25 mg/kg body weight, i.p.

Group 3 animals were pre-treated with famotidine (3 mg/kg [19].

Group 4: consist of animals pre-treated with 20mg/kg of omeprazole [20].

Group 5: animals were pretreated with famotidine and hemin.

Group 6: animals were pretreated with omeprazole and hemin.

The animals were sacrificed 6 h later by head trauma and their stomachs were opened along the great curvature and washed with tap water to remove gastric contents. Gastric lesions were counted and measured. The stomach of each animal was divided into two parts, the first part for nitric oxide (NO) and malondialdehyde (MDA) assays, and the second part for histopathological examinations [21].

The first part of the stomach was excised, immersed in saline, and immediately stored at -40°C for measurement of NO and MDA levels. Gastric tissues were homogenized in ten volumes of 150 mmol/L ice-cold KCl using a glass Teflon homogenizer (Ultra Turrax IKA T18 Basic) after cutting the tissues into small pieces with scissors (for 2 min at 5000 r/min). The homogenate was then centrifuged at 5000 × *g* for 15 min. The supernatant was used for analysis. High-performance liquid chromatographic analysis was performed using a Shimadzu HPLC system (Kyoto, Japan) with an MDA kit (Immundiagnostik AG, Bensheim, Germany). Spectrophotometric measurements of total antioxidant status (TAS) (Randox, Crumlin, UK) was performed using a Shimadzu UV-1601 (Kyoto, Japan) spectrophotometer. Serum nitric oxide levels (nitrite + nitrate) were measured, after conversion of nitrate to nitrite by copperized cadmium granules, by a spectrophotometer at 545 nm (Shimadzu, Tokyo, Japan). Protein assays were measured on an Advia 2400 chemistry analyzer (Bayer Healthcare Instruments, Tarrytown, NY, USA). Results were expressed as μmol/g protein for NO and nmol/g protein for MDA.

The other part of the stomach was fixed in 10% neutral formalin, embedded in paraffin, and cut into 5 μm sections. The sections were stained with hematoxylin eosin (HE) and examined under the light microscope by a blinded pathologist for histological changes. The stomach is examined under a dissecting microscope with square-grid eyepiece to assess the formation of ulcers. For each stomach, ulcerated and total areas were measured as mm^2. The ulcer index (UI) for each stomach was calculated using the following formula [22]:

UI= (ulcerated area/ total stomach area) X 100.

The ulcer inhibition rates (UIR) for each group were calculated as: UIR (%)= { (UI control- UI treated/ UI control)} X 100.

STATISTICAL ANALYSIS

The statistical analysis was performed using Statistical Package for the Social Sciences (SPSS) version 12.0 for Windows. All data are expressed as the mean ± SD. Unpaired T test and ANOVA were used for statistical analysis of data among all groups. $P < 0.05$ was considered as statistically significant.

RESULTS

Macroscopic analysis showed gastric mucosal lesions in all stomachs of the indomethacin 30 mg/kg treated groups. The mean ulcer area was 21.00 ± 2.35 in the control group. In our study we found that Gastric mucosal damage was significantly reduced by hemin 25mg/kg. We also found that famotidine 3 mg/kg and omeprazole 20 mg/kg pretreatments produced highly significant protective effects. In all groups, the mean count of ulceration and ulcer area were significantly lower than the control group. Gastric mucosal lesion areas were extremely highly significantly diminished in rats pretreated with hemin & famotidine group and hemin & omeprazole group, when compared with the control group. The mean ulcer area in the last two groups were 6.85 ± 0.88 and 5.75 ± 0.78, respectively.

Macroscopic evaluation of gastric mucosal lesion counts and gastric mucosal lesion areas for each group are presented in **Table 1**.

Table 1: Macroscopic evaluation of gastric mucosa (Data presented as mean ± SD)

Groups	Weight (gr)	GML count	GML area mm^2
Control	225.00 ± 13.77	6.25 ± 0.49	21.00 ± 2.35
Hemin 25 mg/kg	222.00 ± 14.34	5.12 ± 0.97*	18.62 ± 1.07*
Famotidine 3 mg/kg	223.25 ± 13.13	5.7 ± 0.97**	16.78 ± 1.97**
Omeprazole 20 mg/kg	224.75 ± 14.78	5.35 ± 0.43**	16.64 ± 1,95**
Hemin + Famotidine	221.12 ± 13.27	1.23 ± 0.38 ***	6.85 ± 0.88***
Hemin + omeprazole	224.12 ± 15.16	1.25 ± 0.35***	5.75 ± 0.78***
ANOVA		***	***

GML: Gastric mucosal lesion. The values are presented as mean ± SD, (min-max).

All groups were compared with the control group.

* = P < 0.05 = significant

**= P < 0.005 = highly significant

***= P < 0.001 = extremely highly significant

On histopathological examination, erosion, inflammation, hemorrhage, and necrosis were abundant in the control group. Hemin & Famotidine pretreatment were found to have extremely highly significant protective effect against indomethacin-induced gastric mucosal lesions. Minimal hemorrhage, minimal focal necrosis, and superficial erosions were observed in rats given 25 mg/kg hemin. Omeprazole and hemin were also found to have extremely highly significant protective effect against indomethacin-induced gastric mucosal lesions.

Lesions of the gastric mucosa in each group are shown in Figure 1.

In our study, we found that hemin reduced significantly MDA and NO levels (24.87 ± 1.97, 25.31 ± 1.27 respectively) and famotidine and omeprazole produced a significant lowering effect on MDA. However, famotidine and omeprazole were found to increase significantly NO levels. In our study we found that combination of hemin and famotidine or omeprazole increased NO levels significantly but less than that produced by hemin alone (25.89 ± 0.92, 26.12 ± 0.67 respectively). Tissue MDA and NO levels are presented in Table 2 for each group.

Table 2: MDA and NO levels in gastric tissues in each group:

Group	MDA (nmol/g protein)	NO (μmol/g protein)
Control	28.48 ± 1.51	27.20 ± 1.25
Hemin 25 mg/kg	24.87 ± 1.97**	25.31 ± 1.27**
Famotidine 3 mg/kg	25.97 ± 1.91*	41.01 ± 1.27
Omeprazole 20 mg/kg	25.86 ± 1.70**	33.55 ± 1.29
Hemin + Famotidine	16.21 ± 0.88 ***	25.89 ± 0.92*
Hemin + omeprazole	15.88 ± 0.60 ***	26.12 ± 0.67*
ANOVA	***	***

All groups were compared with the control group.

* = P < 0.05 = significant

**= P < 0.005 = highly significant

***= P < 0.001 = extremely highly significant

DISCUSSION

It has been demonstrated that gastric cytoprotection against noxious agents is mediated by HO-1 induction. In our study we found that Gastric mucosal damage was significantly reduced by hemin 25mg/kg. In all groups, the mean count of ulceration and the mean count of ulcer area were significantly lower than the control group. Gastric mucosal lesion areas were significantly diminished in rats pretreated with hemin 25 mg/kg, when compared with the control group. Gastric mucosal lesion areas were extremely highly significantly diminished in rats pretreated with hemin & famotidine group and hemin & omeprazole group, when compared with the control group. Our findings were supported by **Takagi et al.,** [11] who showed that lansoprazole, a gastric H+/K+ ATPase inhibitor, upregulates HO-1 expression in rat gastric epithelial cells, and the up-regulated HO-1 has anti-inflammatory effects, and that lansoprazole-induced HO-1 induction is mediated by the activation, phosphorylation and nuclear translocation of Nrf2 in accompaniment with the dissection of oxidized Keap1. This was also found to be supported by **Schulz-Geske et al.,** [23] who found that lansoprazole induced HO-1 in macrophages.

Our results were also supported by **Gomes Et al.,** [24] who found that Pre-treatment with hemin reduced gastric damage and MDA formation and increased GSH concentration in the gastric mucosa and they suggested that HO-1 pathway plays a protective role against ethanol-induced gastric damage. Our results were also in agreement with **Matsuoka et al.,** [25] who found that HO-1 expression is up-regulated in bladders with cyclophosphamide (CYP) -induced hemorrhagic cystitis, and this inducible enzyme plays cytoprotective roles in association with down-regulation of NO production and iNOS expression. They suggested that HO-1 induction might have therapeutic potential against inflammatory insults such as CYP-induced cystitis.

In our study, we also found that hemin reduced significantly MDA and NO levels and famotidine and omeprazole produced a significant lowering effect on MDA. However, famotidine and omeprazole were found to increase significantly NO levels. Our results were supported by **Duridanova et al.,** [26] who suggested that HO-1 induction (by hemin) might serve in the guinea-pig stomach as genetically determined defense mechanism aimed to combat toxic stress-related pathology in order to preserve the functional performance of the organ. In our study we found that combination of hemin and famotidine or omeprazole increased NO levels significantly but less

than that produced by hemin alone.

In conclusion: in our study we confirmed that hemin has a gastroprotective effect against indomethacin induced ulcer and this beneficial effect of hemin may be associated with decreased NO and MDA levels.

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Ethical Clearance: Taken from National Animal Committee.

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A Study of Correlation of C-reactive Protein, and Physical and Metabolic Indicators of Cardiovascular Risk in Menopausal Transition

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ABSTRACT

The metabolic changes accompanying menopauseare all established risk factors for cardiovascular morbidity. These factors are known to act in conjunction with each other, and inflammation is possibly a common mechanism which is responsible for atherogenesis. C-Reactive Protein (CRP) has been found by several workers to be a reliable marker of pro-inflammatory state and is also a predictor of cardiovascular risk. So CRP level estimation may add to predictive capacity in cardiovascular risk assessment. This study attempts to explore the physical and metabolic changes in post-menopausal women and to explore whether these parameters show significant correlation with CRP levels. If so, then CRP estimation can be used as a tool to assess cardiovascular risk in post-menopausal women.

Method: 45 Pre-menopausal and 45 Post-menopausal women were selected from general population of Bengaluru city. Anthropometric data was collected. Hb, Total Leucocyte count (TLC), Fasting blood sugar, Lipid profile, high sensitivity CRP (hs-CRP) were assayed in all the subjects and results compared statistically. Pearson's correlation coefficient was calculated for CRP and the other parameters.

Results: Post-menopausal women had significantly higher weight ($p= 0.045$), Hip circumference ($p= 0.002$), waist circumference ($p = 0.005$), BMI ($p<0.05$), TLC ($p = 0.041$). Also they were 5.09 times ($p = 0.038$) more likely to have elevated CRP levels compared to pre-menopausal women. FBS and lipid profile did not show significant difference in the two groups. In the post-menopause group, CRP showed significant positive correlation with BMI ($p=0.045$), WHR ($p=0.016$), LDL ($p=0.017$) and FBS ($p=0.004$); and significant negative correlation with HDL levels ($p=0.024$).

Conclusion: Post-menopausal women have tendency to gain weight and the obesity is of android type. CRP levels are significantly correlated with several parameters which are associated with cardiovascular risk. CRP estimation may prove to be a useful tool in identifying post-menopausal women who are at greater risk of CVD.

Keywords: *Pre-menopausal women, Post-menopausal women, Cardiovascular disease, BMI, Fasting Blood sugar, Lipid profile, C-Reactive protein, Inflammation,*

INTRODUCTION

Incidence of Coronary artery Heart disease (CHD) in women after menopause equals that in men. Further, the overall morbidity and mortality following the initial ischaemic heart event is worse in women, and the case fatality rate is greater in women than in men[1].

As numerous changes, physical and metabolic are usual accompaniments of menopause, several of these have been implicated in mediating increased CVD events in post-menopausal women. These include weight gain, altered body fat distribution, altered function of vascular endothelium and smooth

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muscles leading to hypertension, impaired glucose metabolism, insulin resistance and unfavourable alterations in lipid metabolism[2]. These factors may act synergistically in producing greater cardiovascular morbidity. An inflammatory process is believed to be the final common pathway for all of these to culminate in atherosclerosis.

The major injurious factors that promote atherogenesis are cigarette smoking, hypertension, atherogenic proteins, and hyperglycemia. These risk factors give rise to a variety of noxious stimuli that elicit the inflammatory process. Potential targets for assessment include pro-inflammatory risk factors, inflammatory markers like C-Reactive Protein (CRP) and finally cellular responses to inflammation. The ability of hs-CRP to add to the predictive capacity of other established CVD risk factors has been examined and proven by several studies[3].

Serum markers of inflammation provide an avenue of insight into the pathophysiology of atherosclerosis and its complications. hs-CRP, a non-specific marker of low-grade systemic inflammation, has received much attention, and several studies now support a strong link between baseline elevations of hs-CRP and risk of coronary events[4].

In post-menopausal women, since all the classical risk factors for CVD are found to increase, the possibility of pro-inflammatory state is high. This can be ascertained by estimation of CRP which is a global marker for inflammation. Hence this study was undertaken to study the changes occurring in women during menopausal transition, and to examine possible correlation between these changes and CRP levels.

OBJECTIVES

To compare parameters of obesity, glucose and lipid metabolism, and CRP levels in pre and post-menopausal women.

To test whether a significant correlation exists between these parameters and CRP levels inpost-menopausal women.

METHODOLGY

A comparative study involving women selected randomly from female population of Bengaluru city. Subjects were all asymptomatic females in the age range of 35 to 61 years. They were grouped asPre-menopausal (45 women who were still having their regular menstrual cycles) and Post-menopausal (45 women, with history of amenorrhoea for more than 12 months, and serum levels of Follicle Stimulating Hormone > 30 mIU/ml)[5].

Exclusion criteria: pregnant status, history of irregular menstrual cycles (for pre-menopausal women), pre-existing cardiac disease, chronic liver disease, jaundice, hormonal disorders, acute or chronic infections, inflammatory diseases, hormone therapy or use of oral contraceptives, anti-inflammatory agents, and steroids (for both groups).

Thorough history was obtained from each subject and clinical examination was done.

Height and Weight were measured. Waist circumference was measured at the level of the mid-point between lower costal margin and iliac crest. Hip circumference was around the maximal bulge of gluteus maximus. Waist-Hip ratio (WHR) was calculated.

Haemoglobin (Hb), Total Leucocyte count (TLC), Differential count (DLC), were estimated.

Biochemical parameters: Human Follicle stimulating hormone (hFSH) assay:

Chemiluminiscent immunoassay (CLIA) was used for the quantitative determination of hFSH levels. Levels more than 30 mIU/ml was considered evidence of post-menopausal status[5].

Fasting blood glucose: Blood sugar level was measured after 12 hour overnight fast, by Trinders method.

Lipid profile: Blood sample collected in the morning after 12 hour fast was used for estimation of serum Total Cholesterol (TC), Low density lipoprotein (LDL), High density lipoprotein (HDL), and serum Triglycerides (TGL). CHOD-PAP (Cholesterol oxidase method for cholesterol, GPO-TRINDER (Glycerol phosphatase oxidase) method for TGL and Phosphotungstic acid method for HDL was used. Serum LDL was calculated using Friedwald's formula:

Serum LDL cholesterol = Total Cholesterol – (HDL cholesterol + Triglyceride/5)

hs-CRP assay: Quantia CRP-US was used for ultrasensitive determination of CRP by turbidometric immunoassay by instrument Systronics 625.

RESULTS

Table 1: Comparison Anthropometry parameters between two groups

Anthropometry parameters	Pre-menopause (Mean ± SD)	Post-menopause (Mean ± SD)	p value
Age in years	40.80±5.92	52.00±5.68	0.000*
Height in meters	1.53±0.05	1.51±0.06	0.055
Weight in kg	58.00±10.81	61.78±9.89	0.045*
HIP circumference in cm	87.33±24.98	10.47±15.19	0.002*
Waist circumference in cm	75.43±24.05	90.45±14.37	0.005*
Waist-Hip Ratio	0.84±0.08	0.85±0.05	0.480
BMI (kg/m^2)	24.84±4.56	27.29±4.90	0.050*

* Significant at 5% by student t test

In post-menopausal women, weight, hip circumference, waist circumference and BMI were significantly higher than in pre-menopausal women. WHR was not significantly higher.

Table 2: Follicle stimulating hormone (hFSH) level in two groups

Follicle stimulating hormone (mg/L)	(Mean ± SD)	95% CI
Pre-menopause	7.07±4.33	6.25-9.89
Post-menopause	52.50±13.76	44.97-56.04
Significance	P<0.001**	

** Significant at 1% by student t test

Table 3: Comparison of Haematological parameters between the two groups

Haematology	Pre-menopause (Mean ± SD)	Post-menopause (Mean ± SD)	p value
Hemoglobin gm%	11.55±1.46	12.57±1.31	0.152
Total count	7782.76±2096.27	9565.52±1755.47	0.041*
FBS	101.80±82.64	124.50±95.38	0.345

* Significant at 1% by student t test

TLC in the post-menopausal women was increased significantly.

The fasting blood glucose level was slightly higher among post-menopausal women compared to pre-menopausal women, but not statistically significant.

Table 4: Comparison of Lipid parameters between two groups

Lipid Parameters	Pre-menopause (Mean ± SD)	Post-menopause (Mean ± SD)	p value
Total Cholesterol	207.60±47.29	221.00±55.30	0.317
HDL-Cholesterol	39.93±3.74	39.03±3.69	0.323
LDL-Cholesterol	126.06±29.56	141.35±41.75	0.450
Triglycerides	201.00±140.58	209.85±150.81	0.125

Postmenopausal women had higher levels of lipids when compared to Premenopausal women, but difference was not statisticallysignificant.

Table 5: Comparison of hs-CRP levels in the two groups

CRP mg/L	(Mean ± SD)	95% CI
Pre-menopause	0.34±0.85	0.30-0.66
Post-menopause	0.68±1.32	0.19-1.18
Significance	P=0.237.	

Mean hs-CRP level was not significantly different between two groups.

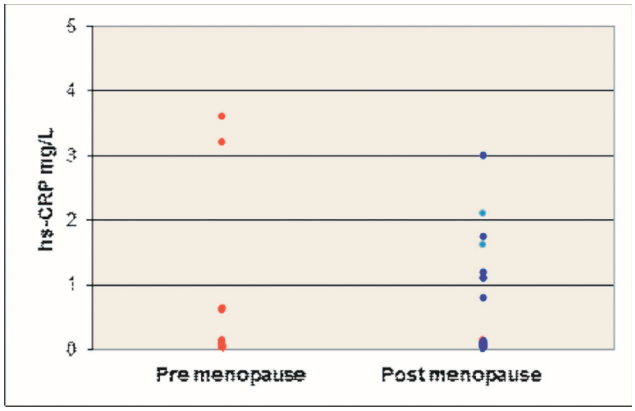

Figure 5a: Distribution of hs-CRP levels in the two groups

Only two cases seemed to be outliers in the pre menopause group, excluding these two indicated clear elevation of hs-CRP in post menopause group

Table 5b: Frequency classification of hs-CRP

Groups	CRP mg/L (<1.0)	CRP mg/L (>1.0)
Pre-menopause	42 (93.3%)	3(6.7%)
Post-menopause	33 (73.3%)	12 (26.7%)
Significance	P=0.038 * significant by Fisher Exact test	

Frequency distribution analysis showed that Post-menopausal women were 5.09 times more likely to have elevated hs-CRP levels when compared to pre-menopausal women.

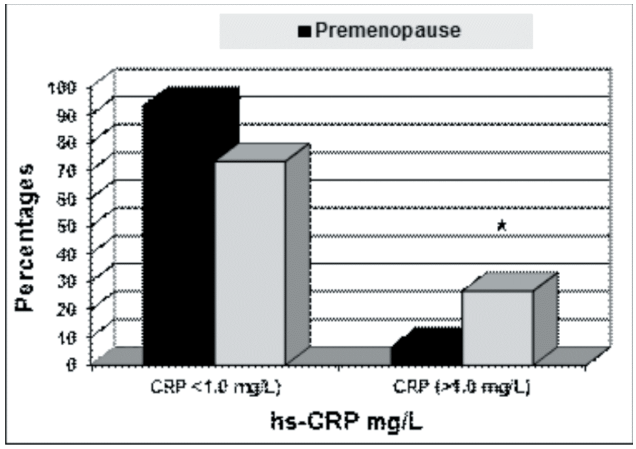

Figure 5b Frequency classification of hs-CRP

*significant at 5%

Pearson correlation coefficient test was done to correlate the relationship between hsCRP level and with other cardiovascular risk factors.

Table 6: Correlation of CRP with Anthropometry parameters

Anthropometry Parameters with CRP	Pearson Correlation	
	Pre - menopause	Post-menopause
Age	r= 0.177 p=0.072	r=0.121 p=0.077
BMI	r=0.256 p=0.163	r=0.456 p=0.045*
Waist-Hip Ratio	r=0.166 p=0.381	r=0.435 p=0.016*

*significant at 5%

There was significant positive correlation between hs-CRP levels and BMI and hs-CRP levels and WHR in post-menopausal women.

Table 7: Correlation of hs-CRP with Lipid Parameters and FBS

Lipid Parameters And FBS	Pearson Correlation	
	Pre -menopause	Post-menopause
Cholesterol	r=0.009 p=0.963	r=0.249 p=0.184
HDL	r=0.285 p=0.134	r= -0.168 p=0.024*
LDL	r=0.177 p=0.383	r=0.412 p=0.017*
Triglycerides	r=0.137 p=0.471	r=0.183 p=0.334
FBS	r=0.494 p=0.006**	r=0.522 p=0.004**

* Significant at 5% ** Significant at 1%

Significant positive correlation was observed betweenhs-CRP and LDL, hs-CRP and FBS. Significant negative correlation was seen between hs-CRP and HDL in the postmenopausal group.

DISCUSSION

In our study, it was found that in post-menopausal women, weight, BMI, Waist circumference and BMI were all increased. This indicates post-menopausal tendency towards obesity and also redistribution of body fat to a more android configuration. Some workers have argued that waist circumference is a better indicator of visceral obesity, which is associated with unfavourable alterations in metabolism[6].

Post-menopausal women had significantly higher total leucocyte count which probably indicates a tendency towards inflammatory state. Margolis KL et al showed by their study that the WBC count, a stable, well-standardized, widely available and inexpensive measure of systemic inflammation, is an independent predictor of CVD events and all-cause mortality in postmenopausal women. They opined that WBC counts greater than 6.7 x 10^9 cells/L may identify high-risk individuals who are not currently identified by traditional CVD risk factors[7].

In our study, mean FBS and lipid profile did not show significant difference in the two groups. Also, mean values of hs-CRP was not elevated in post-menopausal group. But on frequency distribution analysis, post-menopausal women were 5.09 times more likely to have elevated hs-CRP when compared to pre-menopausal women. Post-menopausal tendency towards inflammation can be explained due to the removal of estrogen's regulatory influence on inflammatory mediators. Post-menopausal changes in CRP levels and their association with inflammation and CVD risk have been found in earlier studies[8].

On correlation analysis, we found that hs-CRP levels showed significant correlation with several cardiovascular risk factors in post-menopausal women.

BMI and WHR had positive correlation in post-menopausal women. This is similar to findings of Cynthia K. Sites et al, who also found that though hs-CRP alone did not show any difference between pre and post-menopausal women, there was positive correlation with hs-CRP and intraabdominal fat in post-menopausal women[9]. Mark Woodward et al found elevated hs-CRP levels and positive correlation with BMI in post-menopausal women[10]. Fats are involved in production of inflammatory mediators like hs-CRP, explaining the positive correlation.

hs-CRP levels were strongly correlated to FBS level, signifying the increased risk for CVD. Cynthia K. Sites et al study showed that higher CRP level was associated with lower insulin-mediated glucose disposal[9]. In a study by Lemieux S et al, it was

observed that compared with normal women having low amounts of visceral adipose tissue, women with Type 2Diabetes, women with Impaired Glucose Tolerance plus high amounts of visceral adipose tissue demonstrated a worse CVD risk profile: they had lower insulin sensitivity, higher triglyceride levels, higher levels of hsCRP[11]. These findings highlight the co-occurrence of visceral obesity and impaired glucose metabolism, important components of "metabolic syndrome" are associated with inflammatory state which might contribute significantly to atherogenesis and hence pose a greater risk of CVD.

On correlation of hs-CRP with lipid parameters, we found significant positive correlation with LDL and negative correlation with HDL levels post-menopausally. This is similar to study by Michelle A et al, who found modest correlation with Triglycerides, and a small decrease in mean CRP levels with increase in HDL[12].

Christie Ballantyne et al found individuals with low LDL but high CRP may have greater atherogenicity from LDL, than would be expected by the absolute level of LDL. They proposed that high levels of CRP which is an acute phase reactant, may also provoke vascular inflammation and CRP may preferentially bind to oxidized LDL causing increased expression of adhesion molecules, thus enhancing atherogenecity of LDL[13].

In a study by Aleksandra N. Klisik, et al, significantly higher triglyceride and hsCRP levels together with lower HDL levels were found in overweight compared to normal weight women. In the overweight group, positive correlations of hsCRP were observed with age, body mass index and WC, and a negative correlation was observed with HDL. In the normal weight group, positive correlations were found for hsCRP with age and WC. They concluded that elevated hsCRP levels in conjunction with abnormal lipid profiles may be strongly associated with weight gain in postmenopausal women. Efforts to reduce obesity and inflammation in this group may help correct abnormal levels of hsCRP and lipids[14].

In light of the above facts, it can be safely proposed that menopausal transition is associated with metabolic changes which in combination with each other, give rise to pro-inflammatory mechanisms which in turn contributes to atherogenesis, which may form the basis of increased cardiovascular events in post-menopausal women.

Abundant laboratory and experimental evidence demonstrate that atherothrombosis, in addition to being a disease of lipid accumulation, also represents a chronic inflammatory process. In terms of clinical application, CRP seems to be a stronger predictor of cardiovascular events than LDL-cholesterol, and it adds prognostic information at all levels of calculated Framingham Risk and at all levels of metabolic syndrome. The addition of CRP estimation to standard cholesterol evaluation may thus provide a simple and inexpensive method to improve global risk prediction and compliance with preventive approaches[3].

CRP is a global marker of inflammation. Since inflammation is crucial in the genesis and progress of atherosclerosis, CRP estimation may identify asymptomatic patients at risk of CAD events. About half of CHD events occur in people without marked LDL increase[15]. In such cases, CRP estimation may be clinically useful in targeting treatment to the individuals at risk.

CONCLUSION

We conclude from our findings that in post-menopausal women, there is tendency towards obesity and altered body fat distribution. There is a greater likelihood of elevated CRP levels and a significant correlation exists between elevated hs-CRP levels and adverse metabolic profile, pointing towards role of inflammatory mechanism in CVD morbidity seen after menopause.

hs-CRP estimation may prove to be useful in identifying post-menopausal women who are at greater cardiovascular risk, even when metabolic parameters seem to be unaltered.

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Ethical Clearance: Obtained from Institutional Ethical Committee

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