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EDİTÖRE MEKTUP LETTER TO EDITOR

ST SEGMENT YÜKSELMESİ OLMAYAN HASTANE DIŞI KARDİYAK ARREST OLGULARINDA ERKEN KORONER GİRİŞİM GEREKLİ Mİ?

IS EARLY CORONARY INTERVENTION NECESSARY IN OUT-OF-HOSPITAL CARDIAC ARREST CASES WITHOUT ST SEGMENT ELEVATION?

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ABSTRACT

Although advancements in technology and accumulated studies in the literature have led to progress in the management of patients with cardiac arrest, survival rates for those experiencing out-of-hospital cardiac arrest remain low. Despite various causes of cardiac arrest occurring outside the hospital, acute coronary syndrome remains the most common cause among adults who survive until admission to the intensive care unit. For patients who achieve return of spontaneous circulation after experiencing cardiac arrest outside the hospital, the electrocardiogram serves as a crucial tool in clinical decision-making. Existing literature recommends early coronary angiography and percutaneous coronary intervention in cases with accompanying ST-segment elevation, while in cases without ST-segment elevation, clinicians are advised to focus more on the medical management of the patient.

Keywords: *Cardiac arrest, Acute coronary syndrome,Electrocardiogram (ECG),Out-of-hospital cardiac arrest,Survival rate*

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Sayın editör;

İlerleyen teknoloji ve literatürde biriken çalışmalarla kardiyak arrest olan hastanın yönetiminde ilerlemelere rağmen, hastane dışı kardiyak arrest hastalarında halen sağ kalım oranları düşüktür. Hastane dışı kardiyak arrest birçok nedeni olsa da yoğun bakım ünitesine kabul edilene kadar hayatta kalan erişkinlerde halen akut koroner sendrom en yaygın nedendir (1). Hastane dışı kardiyak arrest sonrası sürekli spontan dolaşım dönüşü (ROSC) olan hastalar için mevcut kılavuz önerileri, erken hedeflenen sıcaklık yönetimi, koroner anjiyografiye (KAG) ve gerektiğinde perkütan koroner girişime (PKG) odaklamaktadır ve kılavuzlarda güncellenmeler ve öneriler temelde bu başlıklar üzerinde olmaktadır. Avrupa Resüsitasyon Konseyi (ERC), Amerikan Kalp Derneği (AHA) kılavuzları, kardiyak arrest sonrası ROSC sağlanan hastalarda EKG'de ST segment elevasyonu (STE) var ise mümkün olan en kısa sürede KAG ve PKG yapılmasını önermektedir. EKG'de STE olmayan hastalarda ise akılda bulundurulması gerektiği vurgulamıştır. Ancak bu ikinci hasta grubunda daha çok hastanın medikal yönetime odaklanılmasını öncelenmiştir (2). Bununla birlikte ROSC sağlanmasından sonra ilk 8 dakika içinde EKG'de gerek resüsitasyon sırasında uygulanılan adrenalin gerekse hastanın içinde olduğu klinik şok tablosu ile ilişkili olarak iskemik değişiklikler görülebileceği raporlanmıştır (3). COACT ve TOMAHAWK çalışmaları, STE olmayan resüsite edilmiş hastane dışı kardiyak arrest hastalarında en kısa sürede KAG ve PKG yapılmasının yararı olmadığını ve hastaların büyük çoğunluğu için gecikmiş veya seçici yaklaşımın mümkün ve güvenli olduğunu raporlamışlardır (4,5). Bu literatür ışığında STE dışında iskemik EKG değişiklikleri tek başına, erken KAG ve PKG kararı almak için yetersizdir. Bununla birlikte EKG'de STE olmaması negatif sonuçlanan EKG veya klinik yönetime katkısı olmayan EKG olarak yorumlanmamalıdır. Arrest etiyolojisinde yer alabilecek Wolf-Parkinson-White sendromu veya uzun QT sendromları gibi disritmiler hakkında bilgi verebilir. Ki bu sendromların erken tanınması hastanın kardiyak stabilizasyonu için tercih edilecek medikal ajanların değişmesine neden olabilir (6). Öte yandan EKG kardiyak etiyoloji dışında arrestin nedeni olabilecek hiperkalemi gibi elektrolit bozuklarının da erken tanınmasını sağlayabilir (7).

Sonuç olarak; hastane dışı kardiyak arrest sonrası ROSC sağlanan hastalarda EKG önemli bir klinik karar verme aracıdır. Mevcut literatür STE eşlik eden senaryolarda erken KAG ve PKG önerirken, eşlik etmediği senaryolarda klinisyenin daha çok hastanın medikal yönetimine odaklanmasını önermektedir.

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EDİTÖRE MEKTUP LETTER TO EDITOR

EDİTÖRE MEKTUP
Letter To The Editor

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Related publication: Kaya HB, Atik D.Gebelik kaşıntıları sadece alerjik midir?. IJOHSON. 2021;1(1):19-21.

ABSTRACT

Intrahepatic cholestatis is a pregnancy-specific liver disease that develops after the 2nd trimester of pregnancy and is characterized by high serum bile acid levels and pruritus. Pregnant individuals with pruritus should be suspected of having intrahepatic cholestasis. The condition may be associated with severe fetal problems. Potential maternal outcomes may be preeclampsia and gestational diabetes. Corticosteroids are recommended for lung maturation in patients with expected preterm birth. It has been reported that fetal mortality in patients with this disease is lower in those delivered at the 36th week.

Keywords: *emergency, cholestasis, pregnancy*

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Dear Editor,

We have thoroughly read the article titled "Gebelik kaşıntıları sadece alerjik midir?" authored by Kaya HB and Atik D, which was published in the first issue of your journal in 2021 (1). We want to emphasize adverse pregnancy outcomes and the management of intrahepatic cholestasis in pregnancy.

Intrahepatic cholestasis of pregnancy is a disorder that occurs in 0.2-2% of pregnancies. It leads to pruritus and elevated levels of serum bile acids, liver transaminases, and sometimes bilirubin(2). It has been correlated with serious negative outcomes during pregnancy, such as fetal distress, premature birth caused by iatrogenic or spontaneously, and stillbirth, for which there is now no recognized effective treatment(2,3).

The diagnosis of intrahepatic cholestasis of pregnancy is verified by the presence of high levels of nonfasting total serum bile acids (4). Fetal death occurs when the concentration of bile acids exceeds 100 μmol/L (5). Nonstress testing and ultrasonography for fetal surveillance do not have the ability to predict or prevent stillbirth resulting from intrahepatic cholestasis of pregnancy. Weekly measurements of bile acids during serial fasting should be conducted, and delivery should be scheduled based on the levels of bile acids and risk factors.

The 2011 guidelines published by the Royal College of Obstetricians and Gynaecologists advocate for transparent communication with women regarding the limited evidence supporting early term delivery as a means to reduce the risk of stillbirth (6). Nevertheless, following authors have employed decision analytic tools to propose delivering the baby at 36 weeks (7).

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DERLEME REVİEW

LITHIUM INTOXICATION

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ABSTRACT

Lithium has been tried in the treatment of different diseases from past to present. However, due to the reporting of toxication and the discovery of alternative treatment methods with less side effects, it has started to be used only in the treatment of mania. Clinically, 10 hours after the last oral intake due to the prolonged distribution phase. It is recommended to measure the lithium blood level after a minimum of 3 half-lives after dosing and after regular lithium dosing. Lithium is one of the drugs with a narrow therapeutic index(55). Lithium is similar to sodium in that it is distributed in the total body fluid and excreted by the kidneys. In cases of decreased kidney function, one should be alert for lithium intoxication. In addition, an increase in the amount of accumulated lithium can also be observed in the use of angiotensin receptor bloc-kers, ACE inhibitors and nonsteroidal anti-inflammatory drugs that reduce the glomerular filt-ration rate(56). Therefore, even in normal use, lithium may cause intoxication as an overdose. There is no molecule that is an antidote to lithium. There are 2 methods that we can follow in lithium intoxication.

1. Medical applications to reduce the concentration of lithium in the body.

2. Extracorporeal Toxin Removal (ECRT)

ECRT, ie dialysis method, is the most effective method. The only method in pa-tients with renal failure is hemodialysis(62). Although there are no definite indications for ECRT, kidney function tests and creatinine level are key points for dialysis decision. When the serum lithium level returns to normal, conditions such as arrhythmia and diabetes insipidus improve, while neurological symptoms such as impaired cognitive functions, cerebellar dysfunctions, and tre-mor may be sequelae. Due to the most and most permanent effect of the central nervous system in lithium intoxication, impaired consciousness may also be a determinant for dialysis depen-ding on the doctor of the person.

***Keywords**: Lithium poisoning,Diagnosis and treatment of lithium poisoning.*

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The element lithium, which has the lowest density among the alkali metals, was discovered in 1817 by Johan August Arfwedson(1). Lithium, which is the first solid element in the periodic table, is chemically similar to the next monovalent metals, sodium and potassium (2). The pharmacological introduction of lithium into our lives was when Alexander Ure suggested in 1843 that lithium could be used in the treatment of urinary tract stones(3). This was followed in 1859 by Alfred Garrod's description of its use in gout and related neurological disorders. Garrod showed that lithium carbonate had a dissolving effect on uric acid, especially in the fingers of patients with gout(2). Lange brothers reported the use of lithium salts in patients with recurrent depression in 1894(3). In these years, water and soda containing lithium became widespread, and it was even advocated that it be used as table salt in low sodium diets of individuals with heart failure(3), but intoxication and deaths caused by this widespread use. eliminated the popularity of lithium(2). By 1949, John Cade, who discovered that the lethality decreased when he gave urine samples of manic patients to pigs to which he had added lithium carbonate, tried lithium in the treatment of mania and had a successful result. With this study, lithium proved its use in psychiatry(4,5). In 1954, with the first controlled study of Danish Mogens Schou on patients with mania, lithium began to be talked about all over the world(2). Although lithium prophylaxis in platftorms is controversial, it received FDA approval in 1970 as a maintenance treatment for acute mania treatment and in 1974 for mania prophylaxis(6).

Although lithium is much less soluble in water than potassium, its suppressive effects on the myocardium and central nervous system are similar(1). As soluble salt compounds (often preferred because the most stable form is lithium carbonate(1)), almost all of the lithium taken orally is passively in the small intestines. It is absorbed by diffusion (7) and Na-K channels in an average of 6-8 hours. Absorbed lithium is distributed almost equally between the intracellular and extracellular compartments (8). It takes 2-4 hours to reach the maximum concentration in plasma(9). It crosses the blood-brain barrier approximately 24 hours after oral ingestion, thus showing its therapeutic effect(7). The final volume of distribution is almost equal to the total body volume (0.79 L/kg), but the sampled Ion concentrations may differ depending on the tissue(7). Autopsy studies have shown that the highest lithium uptake is in the cerebellum, brain and kidneys, respectively. Again, although the reason is not known exactly, it has been observed that there are 10-20% higher lithium levels in the cerebellum, brain and kidneys in women than men, while 13% less lithium levels in the pancreas.

There was no significant difference between the sexes in lithium levels in organs such as liver, lung, and thyroid (10). Since it is water-soluble, its transport in the blood does not depend on albumin(2). Its levels in erythrocyte, CSF, and brain are lower than plasma levels, while the opposite is true in the thyroid and kidney. (2). The lithium level measured from the scalp is a noninvasive method of showing the dietary lithium, but since it does not include the pharmacological lithium levels, it does not show the compliance of the patients with the drug (11). Since there is no metabolite, the lithium taken into the body is excreted unchanged. While almost all of its excretion is through the renal system, 4-5% is excreted in sweat and 1% in faeces(12). The mean

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renal clearance of lithium is 10-30 ml/min and it is excreted in the urine within 15 minutes after oral ingestion(13). Lithium filtered from the glomerulus is reabsorbed mostly from the proximal tubules, in addition to the distal collecting ducts(14). Its excretion from the central nervous system is 6- It is 12 hours slower, therefore, neurotoxicity continues even if the lithium concentrations in the plasma decrease to subtherapeutic levels in intoxications(7). Oliguria, hypomania, renal or cardiac diseases, low sodium diet, dehydration, and intercurrent infections can be counted among the causes that decrease clearance(15). In healthy humans, the lithium half-life is 24 hours, and steady blood concentration is reached in 3-4 days. Old age and kidney failure may prolong this period (16,17).

Lithium has been tried in the treatment of different diseases from past to present. Cardiac diseases, migraine, agranulocytosis, hyperthyroidism, diabetes insipidus, tardive dyskinesia, seborrheic dermatitis, familial Mediterranean fever can be given as examples of these diseases. However, due to the reporting of toxication and the discovery of alternative treatment methods with less side effects, it has started to be used only in the treatment of mania (30,31,32).

MECHANISM OF LITHIUM

Lithium acts in our body with different mechanisms of action. Lithium has a macroscopic as well as a cellular mechanism of action (22). Although it is still not known precisely and is open to investigation, the known mechanisms of action are via glutamate, inositol monophosphate, glycogen synthetase kinase 3, insulin, sodium, magnesium, calcium, thyroid functions, adenosine receptor, endoplasmic reticulum stress proteins (23).

Glutamate is the most common excitatory neurotransmitter in our nervous system (24). Glutamate is released into the synaptic gap and acts by binding to surface receptors. α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptors, kainate receptors, N-methyl-D-aspartate (NMDA) receptors, glutamate ionotropic receptors(25). When glutamate binds to these receptors, ion channels are opened, allowing ions such as sodium, calcium, potassium, and chlorine to pass through the membrane. The metabolic receptors of glutamate belong to the G protein family and show their effects through secondary messenger systems. Some metabolic receptors exert their effects by activating phospholipase c and increasing inositol triphosphate and diacyl glycerol, while others inhibit adenylate cyclase and decrease cAMP levels. Glutamate plays a role in long-term memory and learning through NMDA receptors in synaptic plasticity. In addition, the glutamatergic system is involved in anxiolytic and antidepressant mechanisms (24). The CNS role of lithium is to increase glutamate reuptake from the synaptic gap (26). Thus, the possibility of excitotoxicity decreases. Dopamine is a stimulating neurotransmitter just like glutamate. It also increases the amount of neurotransmitters such as lithium, serotonin, dopamine, noradrenaline, and shows antidepressant properties by increasing the well-being of the person (22).

Lithium also accelerates insulin-mediated glucose transport (27). Glycogen synthase kinase-3 beta (GSK3β) plays an important role in metabolic events, formation of neurofibrillary

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tangles, inflammation, regulation of cognitive functions, synaptic plasticity, axonal growth, regulation of cell signaling mechanism and cell death. Lithium inactivates GSK3β. GSK-3 plays a role in the pathophysiology of many neuropsychiatric diseases (28). Lithium inhibits potassium channels in the synaptic gap and thus inhibits the neuromuscular transmission system (29). Lithium decreases the amount of excitatory neurotransmitters in the synaptic cleft, while increasing the amount of suppressive neurotransmitters in the synaptic cleft. Since glutamate is an excitatory neurotransmitter, it increases the reuptake of glutamate from the synaptic gap and decreases its amount in the synaptic gap.

Lithium is an inhibitor of inositol monophosphatase and reduces the amount of inostol. It affects secondary news systems. It inhibits GSK-3. It decreases protein kinase C activity. In this way, it regulates mood. It inhibits proapoptotic genes and increases the resistance of the cell. It has a cytoprotective effect. Considering the objective parameters that can be used when evaluating lithium, gray matter thickness, N-acetyl aspartate amount, myoinositol level, EEG can be considered. There is an increase in the amount of gray matter in the brain in people using lithium. While N-acetyl aspartate amounts increased, myoinositol levels decreased. An increase in the size of the waves is observed in the EEG(22).

INTOXICATION

A 300 mg lithium carbonate tablet contains 8.1 mEq of lithium, resulting in an average increase in serum concentration of 0.2-0.4 mEq/L(1). Clinically, 10 hours after the last oral intake due to the prolonged distribution phase. It is recommended to measure the lithium blood level after a minimum of 3 half-lives after dosing and after regular lithium dosing(7). There is controversy about the therapeutic plasma lithium level(18). Schou and Baastrup suggested this level as 0.6–1.5 mEq/L in their study in 1967(19). The latest guidelines in Europe (NICE) recommend the steady-state concentration as 0.6–0.8 mEq/L in the USA. Recent publications in , recommend slow titration in the range of 0.5–1.2 mEq/L(20,21).

In addition to the narrow therapeutic index, the presence of other predisposing factors also facilitates intoxication. These predisposing factors can be listed as follows(33,34):

1. Presence of comorbid diseases: hypertension, renal failure, neurological diseases, hypothyroidism, cardiovascular diseases, Addison's disease, diabetes, schizophrenia

2. Gastrointestinal disorders: diarrhea, vomiting, dehydration, anorexia, decreased dietary sodium intake

3. Additional drug treatments: NSAIDs, diuretics, ACEIs, calcium antagonists, neuroleptics

4. Drug overdose

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5. History of previous lithium intoxication

6. Advanced age

Schou et al. classify lithium-related side effects in 3 groups: undamaged initial side effects, resistant undamaged side effects, and intoxication prodromes(35). Nausea, soft stools, fine tremor(36), lethargy, muscle weakness, polyuria and polydipsia are side effects that are seen in the first period and cannot be considered as a sign of toxicity (37). Of these, tremor and polyuria may persist persistently and may cause weight gain with the development of edema in some patients(38,39). Although Schou does not see persistent side effects as toxicity, they are side effects that should be carefully considered and investigated when they occur. After all, lithium is an electrolyte, and with its exogenous intake, it causes an imbalance in the intercellular fluid and affects the ions in the intracellular compartment (40). Therefore, excessive water intake is important for these patients. Many studies have shown that lithium treatment can cause hypothyroidism(41,42). Lithium competes with ADH for its receptors in the kidney, so nephrogenic diabetes insipidus is among the side effects of lithium treatment(43). Rarely, signs of acute renal injury may occur (44). Lithium affects cellular processes by inhibiting the prostatic acid phosphatase (PAP) enzyme, and this increase in PAP is responsible for most of the peripheral side effects (21). Another mechanism contributing to the side effects is the inhibition of glycogen synthase kinase-3, an enzyme in the signal transduction pathway (45).

Lithium poisonings can also be examined in 3 groups: acute poisonings, acute poisonings on a chronic basis, and chronic poisonings(46,47). lithium toxicity; It may occur by mechanisms such as cumulative overdose in a patient receiving lithium treatment, decreased lithium excretion due to dehydration or intercurrent infections in a patient with subtoxic overdose, and acute overdose caused by accidental or suicidal lithium intake(33). Considering that lithium is absorbed from the gastrointestinal tract, it can be understood that gastrointestinal system side effects such as nausea, vomiting and diarrhea are at the forefront in acute poisonings (47). In the clinic of chronic poisoning in case of increased toxicity; Neurological symptoms such as speech disorder, tremor, mood disorders and confusion become dominant and the patient may experience convulsions and stupor(48,49,50). The developing neurotoxicity may be reversible or irreversible. Cerebellar symptoms such as ataxia, dysarthria and prolonged exposure to toxicity are in favor of irreversible damage(48).

Persistent neurological symptoms were first described in 1980 as SILENT (The syndrome of irreversible effectuated neurotoxicity syndrome) syndrome (51). Acute poisonings on a chronic basis can be thought of as mechanisms that may lead to acute poisoning in patients with chronic lithium use, and side effects may occur in both acute and chronic poisonings.

Table 1: Clinical findings of lithium intoxication(41).

LITHIUM INTOXICATION			
	Mild	Moderate	Severe
Cardiovascular System	• T wave inversion • Arrhythmias due to intraventricular conduction defects • Sinus bradycardia	• Arrhythmias due to intraventricular conduction defects • T wave changes	• ST segment depression • Cardiovascular collapse • Hypotension • Sick sinus syndrome
Peripheral and Central Nervous System	• Apathy • Hyperreflexia • Tiredness • Fine tremor • Muscle weakness	• Hypertonia • Myoclomus • Ataxia • Coarse tremor • Dysatris	• Epileptic seizure • Stupor • Coma • Paraesthesia • Paralysis • Spasticity • In rigidity
Renal	• Polyuria • Polydipsia	• Kidney damage	• Acute kidney failure
Gastrointestinal System	• Nausea • Vomiting • Diarrhea	• Nausea • Vomiting • Diarrhea	• Nausea • Vomiting • Diarrhea

In 1978, Hansen and Amdisen formed cohort groups as mild toxicity when serum Li levels are above 2.5 mmol/L, severe toxicity when serum Li levels are above 2.5-3.5 mmol/L, and life-threatening toxicity when serum Li levels are above 3.5 mmol/L. concluded that there is no definite relationship between serum lithium levels(52). On the other hand, intracellular lithium levels and intoxication clinic are closely related(53). To assess the severity of poisoning, assessing the severity of the patient's clinic gives more accurate information than serum lithium concentration. Especially in acute poisonings, serum lithium levels and the degree of toxicity show a weak correlation until there is a balance between the tissue and plasma lithium levels (40). In cases of chronic poisoning, the ratio of serum lithium level to reflect intracellular lithium level is more reliable(54).

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TREATMENT

Lithium is one of the drugs with a narrow therapeutic index(55). Lithium is similar to sodium in that it is distributed in the total body fluid and excreted by the kidneys. In cases of decreased kidney function, one should be alert for lithium intoxication. In addition, an increase in the amount of accumulated lithium can also be observed in the use of angiotensin receptor blockers, ACE inhibitors and nonsteroidal anti-inflammatory drugs that reduce the glomerular filtration rate(56). Therefore, even in normal use, lithium may cause intoxication as an overdose. In addition, thiazide diuretics and spironolactone increase the concentration of lithium by increasing renal tubular reabsorption. Calcium channel blockers are known to increase the lithium level. and may even lead to clinical pictures up to death. Vomiting may reduce the amount of lithium both as excreted and by reducing its absorption(57). In such cases, our main goal in treatment should be symptomatic and supportive. There is no molecule that is an antidote to lithium. There are 2 methods that we can follow in lithium intoxication.

1. Medical applications to reduce the concentration of lithium in the body.

2. Extracorporeal Toxin Removal (ECRT)

As a supportive treatment, intravenous administration of 0.9% serum physiology will be beneficial in increasing lithium excretion and reducing lithium reabsorption(58). Airway management and gastrointestinal decontamination can be utilized(59). Rapid emptying of the column can be achieved with polyethylene glycol. The dose of polyethylene should be 1-2 / lt per hour. There are also studies showing that the application of kayexalate reduces the maximum lithium dose to be reached (60,61).

ECRT, ie dialysis method, is the most effective method(63). The only method in patients with renal failure is hemodialysis(62). Although there are no definite indications for ECRT, kidney function tests and creatinine level are key points for dialysis decision. According to a study, a serum lithium concentration of ≥5.2 mmol/L or a serum creatinine concentration of ≥200 µmol/l constitute an indication for emergency dialysis(63). When the serum lithium level returns to normal, conditions such as arrhythmia and diabetes insipidus improve, while neurological symptoms such as impaired cognitive functions, cerebellar dysfunctions, and tremor may be sequelae. Due to the most and most permanent effect of the central nervous system in lithium intoxication, impaired consciousness may also be a determinant for dialysis depending on the doctor of the person(64,65). Hemodialysis should continue until the serum level is below 1 mEq/L(62). Renal function tests should be used to determine the treatment prognosis(58).

Lithium is a pharmacological agent with a narrow therapeutic index, and it is an agent that needs to be taken care of because it can result in morbidity and mortality when intoxication occurs. However, lithium is still used in many psychiatric areas such as the maintenance treatment of bipolar disorder and resistant depression cases, especially in the manic episode period of bipolar

disorder, despite various difficulties in use due to side effects that may be fatal (65). Clinical doctors; should question the use of lithium, correctly determine the severity of the clinic in cases of poisoning, take a holistic approach in terms of complications that may arise, and choose the appropriate treatment. It should be kept in mind that serum lithium levels are not indicative of clinical severity, especially in acute poisonings. In patients prescribed lithium, the presence of comorbid conditions and additional drug use should be considered, families and patients should be warned about possible side effects and even educated about the prodromal signs of poisoning(66). It is possible to obtain satisfactory results in many psychiatric diseases with lithium treatment if precautions are taken against poisonings, early and correct diagnosis is made and appropriate treatment is given in poisoning cases.

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ARAŞTIRMA MAKALESİ RESEARCH ARTICLE

RETROSPECTIVE EVALUATION OF THE EFFECTS OF SERUM CALCIUM AND MAGNESIUM LEVELS ON HANDGRIP AND PINCH STRENGTH DURING PREGNANCY

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ABSTRACT

Purpose: *The assessment of upper extremity and hand performance widely recognizes handgrip and pinch strength measurements as functional parameters. Calcium and magnesium elements play crucial roles in several processes of the body, including muscular contraction. The goal of the present study is to investigate the impact of serum calcium and magnesium levels on pregnant women's handgrip and pinch strength measurements during the first and second trimesters of pregnancy.* ***Material and Methods:*** *The study included 48 pregnant women who came for regular follow-up during the first and second trimesters (age: 26.9 ± 4.9 years, height: 161.2 ± 6.2 cm, weight: 65.7 ± 12.5 kg). The laboratory measurements of serum calcium and magnesium were gathered from the local university hospital data. The Baseline® hydraulic hand dynamometer measured the handgrip strength and the Baseline® hydraulic pinch meter quantified the lateral (key) and tip pinch strength of the participants. The Tanita MC-580 was used a bioelectrical impedance measurement device, to measure the body composition of pregnant women. The open-source software (Jamovi, Sidney, Australia, https://www.jamovi.org, v. 1.6.21) was used for the statistical analysis of the data. The level of significance was set at $p < 0.05$.* ***Results:*** *It was found that statistically significant decrease in both serum calcium and magnesium levels were detected in the second trimester ($p<0.001$, $p<0.001$ respectively). Right-left handgrip, lateral (key) and tip pinch calculations revealed no statistically significant difference between the first and second trimesters ($p>0.001$). When investigating the correlation between serum calcium, magnesium, and muscular strength and muscle mass in upper extremities, it was found that serum magnesium leveles did not impact muscle strength during both trimesters. Moreover, the negative influence of serum calcium levels were only observed in the second trimester, specifically in the key pinch strength ($p=0.029$).* ***Conclusion:*** *The handgrip and pinch strength of pregnant women is not influenced by serum magnesium levels during their first and second trimesters. Serum calcium levels has not affect muscle strength in the first trimester. But it has effects in the second trimester on key pinch strength.*

Keywords*: Pregnancy, serum calcium and magnesium levels, handgrip sterngth, pinch strength*

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INTRODUCTION

Handgrip strength is a strong flexion movement that occurs when the biokinetic conditions necessary for a normal movement are met and occurs with the participation of all finge joints and many muscles in the hand. HGS serves as a gauge for the overall health of the body, and its measurement serves as a diagnostic tool due to its non-invasive nature, easy of application, and cost-effectiveness [1]. HGS is accepted as an objective measurement in the evaluation of upper extremity performance and is measured with a device called 'hand dynamometer' [2]. Variable ssuch as height, age, body weight, bone mineral density and gender relate to handgrip strength [1,2,3]. Physicians, trainer and athletes frequently use HGS, an easy applicable and non-invasive method, as an indicator of individuals' physical strength and muscle performance, nutritional levels, and health [4–7]. Similarly, pinch strength is known as a determinant of the functional integrity of the hand with the thumb because the thumb is responsible for approximately 70% of the function of the hand.

A device known as a 'pinchmeter' quantifies key pinch and tip pinch. medical and sport professionals mostly use a simple and non-intrusive technique to assess pinch strength, which is similar to handgrip strength, to evaluate the athlete's hand compression, grip, physical strength, and muscular performance [8,9].

Furthermore, studies have observed a notable reduction in handgrip strength in individuals with vascular comorbidities [10], diabetes [11], communities characterized by low socioeconomic status, and individuals suffering from depression [12].

Calcium (Ca^{2+}) plays a crucial role in numerous intra- and extracellular reactions in the body. The majority of Ca^{2+} in the body, around 99%, are located in bones, with the remaining 1% present in the plasma. The normal concentration of this element in the circulation ranges from 8.5 to 10.2 milligrams per deciliter (mg/dL). It serves crucial functions in the body, including muscular contraction, neuron function, blood clotting, and bone health [13]. Ca^{2+} metabolism plays a crucial role in maintaining a healthy pregnancy.

Magnesium (Mg^{2+}), a vital, positively chargedion, plays an essential role as a coenzyme in any reaction that ATP facilitates. It has a crucial function in regulating any action that involves calcium. The normal blood concentration of Mg^{2+} ranges from 1.7 to 2.2 mg/dL. It has crucial functions in the neurological system, regulating heart rhythm, facilitating muscle contraction, and aiding in protein synthesis [13].

During pregnancy, it is crucial to have well-developed flexor and extensor muscles in the forearms and upper arms, as well as strong muscle groups in the hand area. This is important for ensuring a healthy pregnancy and minimizing the risk of weakness and injuries in the upper extremities.

We anticipate that a healthy pregnancy requires adequate hand grip strength and a proper balance of Ca^{2+} and Mg^{2+} levels. Given this information, we formulated a hypothesis suggesting a potential correlation between handgrip and pinch strength, and the levels of serum Ca^{2+} and Mg^{2+} during both first and second trimesters of pregnancy.

Recently, there has been a lack of study in the available literature that investigates the influence of serum Ca^{2+} and Mg^{2+} levels in mothers on handgrip and pinch strength and throughout both first and second trimesters of pregnancy.

Aim

The main objective of this study is to conduct a retrospective assessment of the impact of serum Ca^{2+} and Mg^{2+} levels during the first and second trimesters of healthy pregnant women who visit our hospital of gynecology and obstetrics clinic for routine controls.

We want to investigate how these blood electrolits levels affect handgrip and pinch muscle strength of pregnant. The secondary aim of this study is to analyze and compare the impacts of these blood electrolits across trimesters.

Material sand Methods

Ethics

The instant study was carried out with the permission of the Local Scientific Medical Research Ethics Committee of Karamanoğlu Mehmetbey University Faculty of Medicine (project no: 07-2024/08; date: June 11, 2024).

Study Design

Our study was planned retrospectively, and pregnant patients who applied to Karamanoğlu Mehmetbey University Karaman Education and Research Gynecology and Obstetrics Clinic between 01.01.2021 and 01.01.2023 were included in the study. Karaman Training and Research Hospital serves the region with a population of approximately 350,000. Our hospital receives between 5500-6000 pregnant applications annually.

Participants

Our study included living single to pregnant women who were over the age of 18, had a known last menstrual date, came for regular polyclinic follow-ups in the first and second trimesters, and signed the informed consent form.

The study excluded pregnant women under the age of 18, pregnancies terminated by abortion, premature birth, intrauterine exitus, multiple pregnancies, and pregnancies with anomalies. However, the research also excluded pregnant women who did not attend regular check-ups during the first and two trimesters, engaged in extreme cardiovascular exercises, participated in resistance training, were Professional athletes, and consumed Ca^{2+} and Mg^{2+} supplements.

Data Collection

Obstetric information of pregnant women (gravide, parity), age, height, weight, examination findings, body mass index (BMI), first and second trimester Ca^{2+} and Mg^{2+} laboratory findings, right and left handgrip and pinch strength, measurement results were recorded by scanning the hospital data base. The 1^{st} trimester test results were obtained with the data between the 6^{th} and 13^{th} weeks

of pregnancy, the 2nd trimester test results were obtained with the data from the 24th and 28th weeks of pregnancy of the same pregnant women.

Pinch Strength Measurements

The American Association of Hand Therapists, as implemented by Mathiowetz et al., established the criteria for the participants' pinch strength measurements. The Baseline® hydraulic pinchmeter, (manufactured by Fabrication Enterprises in the USA), was utilized to quantify the lateral (key) (as shown in Figure 1) and tip pinch (as shown in Figure 2) strength of the participants. The participants' pinch strength was measured while they were sitting, with their elbow flexed at a 90° angle and their forearm and wrist in a neutral posture, using the method published by Mathiowetz et al. [14]. Lateral compression, also known as key compression, refers to the action of placing the thumb on the side of the index finger, specifically between the distal and proximal inter phalangeal joints. Tip compression refers to the technique of placing the thumb's tip in contact with the index finger's tip. The researcher lightly held the dynamometer during the measurement to ensure it was in the correct position. The researcher performed pinch strength measurements on the right and left hands of the participants three times at 1-minute intervals, recording the highest value as the maximum pinch strength in kg [9,14,15].

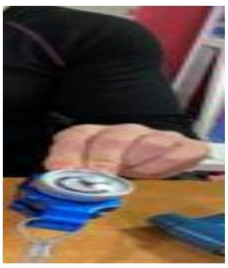

Figure 1. Lateral (key) pinch strength measurement Figure 2. Tip pinch strength measurement

Handgrip Strength Measurement

We conducted the handgrip strength measurements of the participants using the guidelines established by the American Hand Therapists Association [16]. The hand grip strength of the subjects

was measured using the Baseline® hydraulic hand dynamometer (90 kg) (manufactured by Fabrication Enterprises in White Plains, New York 10602, USA) (as shown in Fıgure 3). The participants were seated on a chair to evaluate their handgrip strength. The measurements were taken with the upper arm positioned close to the body, the elbow bent at a 90° angle, and the forearm and wrist in a neutral posture. Participants received verbal encouragement during the measurements. Three consecutive trials conducted for each hand grip strength measurement test and recorded the scores for each hand. The maximum weight recorded for each hand in kilograms. A minimum rest interval of one minute implemented between each measurement to prevent fatigue [17, 18].

Figure 3: Handgrip strength measurument

Bioelectrical İmpedance Measurement

The Tanita MC-580 used, a bioelectrical impedance measurement device, to measure the body composition of participants. The measurement conducted with out any foot wear. Participants instructed to place their feet on the device's metal electrodes, maintain an uprigh tposition, and grasp the hand electrodes during the measurement.

Prior to the measurement, all metallic accessories, such as watches, rings, necklaces, etc., excluded. As a result of this measurement, the right and left muscle masses derived from the body muscle distribution were reported.

Statistical Analysis

Open-source software (Jamovi, Sidney, Australia, https://www.jamovi.org, v. 1.6.21) was used for the statistical analyses of the data. The Shapiro-Wilk test was used to test the suitability of the data for normal distribution, and the results showed that some variables did not comply with the normal distribution ($p< 0.05$). Student t test was used to determine differences between groups. Continuous data were expressed as median ± (interquartile range (IQR)), Categorical data were presented as percentages and numbers. The pairs of correlations were analyzed by the Pearson test. A p-value of < 0.05 was considered statistically significant.

Results

A 48 (forty-eight) pregnant women were included in this study, and the average of participants were 26.9 years. When the calculations of right and left handgrip, right-left key & tip pinch were examined, no statistically significant difference was detected between the first and second trimester ($p=0.487$, $p=0.673$, $p=0.444$, $p=0.336$, $p=0.715$, $p=0.414$, respectively). When Ca^{2+} and Mg^{2+} values were examined, a statistically significant decrease in both Ca^{2+} and Mg^{2+} values were detected in the second trimester ($p < 0.001$, $p < 0.001$ respectively). The muscle mass, muscle strength and laboratory results of participants are shown in Table.1.

Table.1 Muscle strength and laboratory parameters in the first and second trimesters

Dependent: trimester	**1st trimester**	**2nd trimester**	**Total**	**p value**
Total N (%)	48 (50.0)	48 (50.0)	96	
Age (Mean±SD) (years)	26.9 (4.9)	26.9 (4.9)	26.9 (4.8)	1.000
Weight (Mean±SD) (kg)	65.7 (12.5)	72.5 (11.3)	69.1 (12.3)	0.007
Height (Mean±SD) (cm)	161.2 (6.2)	161.2 (6.2)	161.2 (6.2)	1.000
BMI (Mean±SD)(kg/m^2)	25.4 (5.1)	28.0 (4.7)	26.7 (5.1)	0.009
Right handgrip (Mean±SD) (kg)	27.9 (4.9)	28.6 (5.6)	28.2 (5.2)	0.487
Left handgrip (Mean±SD) (kg)	26.8 (5.1)	26.3 (5.5)	26.6 (5.3)	0.673
Right key pinch (Mean±SD) (kg)	6.2 (1.2)	6.4 (1.3)	6.3 (1.2)	0.444
Left keypinch (Mean±SD) (kg)	6.2 (1.3)	6.5 (1.4)	6.4 (1.4)	0.336

Right tip pinch (Mean±SD) (kg)	6.4 (1.6)	6.5 (1.5)	6.4 (1.5)	0.715
Left tip pinch (Mean±SD) (kg)	6.0 (1.5)	6.3 (1.7)	6.1 (1.6)	0.414
Total MM* (Mean±SD) (kg)	42.8 (4.9)	44.8 (4.9)	43.8 (5.0)	0.046
Right arm MM* (Mean±SD)	2.1 (0.3)	2.2 (0.3)	2.2 (0.3)	0.008
Left arm MM(Mean±SD)	2.1 (0.3)	2.2 (0.3)	2.1 (0.3)	0.007
Na** (Mean±SD) (mEq/dL)	137.2 (1.6)	137.1 (1.2)	137.2 (1.4)	0.886
K*** (Mean±SD) (mEq/dL)	4.1 (0.3)	4.2 (0.3)	4.1 (0.3)	0.368
Ca**** (Mean±SD) (md/dL)	9.4 (0.3)	9.0 (0.3)	9.2 (0.4)	<0.001
Mg***** (Mean±SD) (mg/dL)	2.0 (0.2)	1.8 (0.1)	1.9 (0.2)	<0.001

MM*; Muscle mass (kg); **Sodium; ***Potassium; ****Calcium; *****Magnesium

The correlation matrix in the study shows the relationships between serum Ca^{2+} and Mg^{2+} levels and other variables with Pearson correlation coefficient and p-values. We evaluated the correlation between variables and serum Ca^{2+} and Mg^{2+} levels in primiparous and multiparous pregnant women and in the first and second trimesters. There was a moderately positive and significant relationship between serum Ca^{2+} and Mg^{2+} levels in primiparous pregnant women (r: 0.431, p: 0.045). However, we found that right and left handgrip and pinch strength, strength, muscle mass measurement results did not provide a significant correlation with both serum Ca^{2+} and Mg^{2+} levels. The correlation matrix for primiparous pregnant women is shown in Table.2.

Table.2. Correlation analysis of primiparous pregnancy parameters

		Ca^{2+}	Mg^{2+}	GW	RHG	LHG	RKP	LKP	RPP	LPP	Total MM*	RAMM	LAMM
Ca^{2+}	Pearson's r	—											
	p-value	—											
Mg^{2+}	Pearson's r	0.431	—										
	p-value	0.045	—										

Gestationalweek(GW)	Pearson's r	-0.406	-0.097	—									
	p-value	0.061	0.669	—									
Right hand grip(RHG)	Pearson's r	0.093	0.341	-0.008	—								
	p-value	0.681	0.120	0.971	—								
Lefthand grip(LHG)	Pearson's r	-0.046	0.303	0.022	0.622	—							
	p-value	0.840	0.170	0.923	0.002	—							
Right key_pinch (RKP)	Pearson's r	0.029	0.192	0.247	0.524	0.358	—						
	p-value	0.898	0.392	0.268	0.012	0.102	—						
Left-key_pinch (LKP)	Pearson's r	-0.025	0.213	0.222	0.491	0.285	0.809	—					
	p-value	0.913	0.342	0.321	0.020	0.198	<0 .001	—					
Right_tip_pinch(RTP)	Pearson's r	-0.269	-0.219	0.375	-0.010	-0.058	0.101	0.238	—				
	p-value	0.226	0.328	0.085	0.966	0.798	0.653	0.287	—				
Left tip pinch(LTP)	Pearson's r	-0.402	-0.044	0.478	-0.088	-0.016	0.291	0.282	0.741	—			
	p-value	0.063	0.847	0.024	0.697	0.942	0.189	0.204	<0 .001	—			
Total MM*	Pearson's r	0.362	0.335	-0.180	0.143	0.262	0.473	0.452	-0.171	0.019	—		
	p-value	0.097	0.127	0.424	0.526	0.239	0.026	0.035	0.446	0.934	—		
Right arm_MM(RAMM)	Pearson's r	0.355	0.288	-0.134	0.271	0.331	0.402	0.343	-0.269	-0.090	0.916	—	
	p-value	0.105	0.194	0.551	0.223	0.133	0.064	0.118	0.227	0.690	<0 .001	—	
Left-arm_MM (LAMM)	Pearson's r	0.277	0.286	-0.140	0.245	0.373	0.474	0.425	-0.127	0.012	0.908	0.928	—
	p-value	0.212	0.197	0.535	0.272	0.087	0.026	0.049	0.573	0.959	<0 .001	<0 .001	—
BMI**	Pearson's r	0.112	0.279	-0.021	-0.046	0.212	0.095	0.050	-0.045	0.127	0.651	0.680	0.764
	p-value	0.618	0.209	0.927	0.839	0.345	0.674	0.825	0.843	0.572	0.001	<0 .001	<0 .001

*Muscle mass (kg)
**Body mass index (BMI) (kg/m^2), Gestationalweek(GW), Right hand grip(RHG), Lefthand grip(LHG), Right key_pinch(RKP), Left-key_pinch(LKP), Right_tip_pinch(RTP), Left tip pinch(LTP), Right arm_MM(RAMM), Left arm_MM(LAMM).

There was a moderately positive and significant relationship between serum Ca^{2+} and Mg^{2+} levels in multiparous pregnant women (r: 0.405, p: 0.040). There was no significant correlation between

our muscle mass and muscle strength measurement parameters and both serum Ca^{2+} and Mg^{2+} values. The correlation matrix for multiparous pregnant women is shown in Table.3.

Table. 3.Correlation analysis of multiparous pregnancy parameters

		Ca^{2+}	**Mg^{2+}**	**GW**	**RHG**	**LHG**	**RKP**	**LKP**	**RTP**	**LTP**	**Total MM***	**RAMM**	**LA MM**
Ca^{2+}	Pearson's r	—											
	p-value	—											
Mg^{2+}	Pearson's r	0.405	—										
	p-value	0.040	—										
GW	Pearson's r	-0.253	-0.484	—									
	p-value	0.212	0.012	—									
RHG	Pearson's r	0.229	-0.047	0.19	—								
	p-value	0.260	0.820	0.927	—								
LHG	Pearson's r	0.113	0.002	0.057	0.766	—							
	p-value	0.582	0.991	0.780	<0 .001	—							
RKP	Pearson's r	-0.062	-0.248	0.294	0.405	0.517	—						
	p-value	0.763	0.222	0.146	0.40	0.007	—						
LKP	Pearson's r	-0.061	-0.218	0.289	0.557	0.579	0.845	—					
	p-value	0.766	0.286	0.153	0.003	0.002	<0 .001	—					
RTP	Pearson's r	0.016	-0.238	0.302	0.551	0.477	0.777	0.800	—				
	p-value	0.937	0.241	0.134	0.004	0.014	<0 .001	<0 .001	—				
LTP	Pearson's r	0.015	-0.177	0.190	0.679	0.585	0.747	0.880	0.855	—			
	p-value	0.943	0.388	0.353	<0 .001	0.002	<0 .001	<0 .001	<0 .001	—			
Total MM*	Pearson's r	0.068	0.240	-0.258	0.083	0.086	-0.077	-0.071	-0.238	0.093	—		
	p-value	0.746	0.249	0.213	0.692	0.682	0.715	0.735	0.253	0.658	—		
RAMM*	Pearson's r	0.094	0.219	-0.244	0.144	0.104	-0.056	-0.043	-0.173	0.144	0.976	—	
	p-value	0.655	0.293	0.240	0.492	0.621	0.791	0.738	0.408	0.491	<0 .001	—	
LAMM*	Pearson's r	0.080	0.281	-0.260	0.060	0.068	-0.108	-0.076	-0.247	0.068	0.959	0.977	—
	p-value	0.702	0.173	0.209	0.774	0.747	0.607	0.720	0.233	0.746	<0 .001	<0 .001	—
BMI**	Pearson's r	0.077	0.216	-0.173	0.067	-0.132	-0.208	-0.030	-0.085	0.139	0.702	0.711	0.691
	p-value	0.708	0.290	0.399	0.747	0.520	0.309	0.884	0.681	0.497	<0 .001	<0 .001	<0 .001

Muscle mass (kg)
*Body mass index (BMI) (kg/m^2), Gestationalweek(GW), Right hand grip(RHG), Lefthand grip(LHG), Right key_pinch(RKP), Left-key_pinch(LKP), Right_tip_pinch(RTP), Left tip pinch(LTP), Right arm_MM(RAMM), Left arm_MM(LAMM).

There was a highly positive and significant correlation between serum Ca^{2+} and Mg^{2+} levels in the first trimester (r: 0.392, p: 0.006). There was no statistically significant correlation between right and left handgrip and pinch strength, muscle mass measurement results and serum Ca^{2+} and Mg^{2+} levels. The correlation matrix in the first trimester is shown in Table.4.

Table. 4 Correlation analysis of first trimester parameters

		Ca^{2+}	Mg^{2+}	GW	RHG	LHG	RKP	LKP	RTP	LTP	Total MM	RAMM*	LAMM*
Ca^{2+}	Pearson's r	—											
	p-value	—											
Mg^{2+}	Pearson's r	0.392	—										
	p-value	0.006	—										
GW	Pearson's r	-0.348	-0.267	—									
	p-value	0.015	0.067	—									
RHG	Pearson's r	0.138	0.134	0.043	—								
	p-value	0.350	0.362	0.774	—								
LHG	Pearson's r	0.023	0.149	0.023	0.645	—							
	p-value	0.876	0.311	0.877	<0 .001	—							
RKP	Pearson's r	-0.046	-0.065	0.298	0.462	0.389	—						
	p-value	0.759	0.661	0.040	<0 .001	0.006	—						
LKP	Pearson's r	-0.048	-0.081	0.252	0.521	0.406	0.821	—					
	p-value	0.747	0.582	0.084	<0 .001	0.004	<0 .001	—					
RTP	Pearson's r	-0.080	-0.237	0.291	0.343	0.225	0.567	0.668	—				
	p-value	0.589	0.105	0.045	0.017	0.125	<0 .001	<0 .001	—				
LTP	Pearson's r	-0.152	-0.139	0.276	0.365	0.278	0.567	0.706	0.820	—			
	p-value	0.303	0.345	0.058	0.011	0.056	< 0.001	<0 .001	<0 .001	—			
Total MM*	Pearson's r	0.227	0.202	-0.277	0.032	0.178	0.020	0.054	-0.175	0.099	—		
	p-value	0.124	0.173	0.060	0.831	0.232	0.893	0.718	0.239	0.507	—		
RAMM*	Pearson's r	0.214	0.161	-0.255	0.098	0.193	-0.016	0.025	-0.155	0.109	0.956	—	
	p-value	0.149	0.280	0.084	0.514	0.194	0.916	0.867	0.298	0.464	<0 .001	—	
LAMM*	Pearson's r	0.182	0.203	-0.264	0.044	0.196	-0.026	0.023	-0.176	0.084	0.945	0.969	—
	p-value	0.221	0.172	0.073	0.769	0.187	0.860	0.878	0.236	0.572	<0 .001	< 0.001	—
BMI**	Pearson's r	0.111	0.220	-0.119	-0.013	0.081	-0.109	-0.006	-0.053	0.145	0.662	0.656	0.680
	p-value	0.454	0.133	0.422	0.930	0.582	0.461	0.969	0.722	0.326	<0 .001	< 0.001	<0 .001

*Muscle mass (kg)

*Body mass index (BMI) (kg/m²), Gestationalweek(GW), Right hand grip(RHG), Lefthand grip(LHG), Right key_pinch(RKP), Left-key_pinch(LKP), Right_tip_pinch(RTP), Left tip pinch(LTP), Right arm_MM(RAMM), Left arm_MM(LAMM).

There was no statistically significant correlation between serum Ca^{2+} and Mg^{2+} levels in the second trimester (r: 0.101, p: 0.495). A negative and significant correlation was detected between serum Ca^{2+} levels and left key pinch strength (r: -0.316, p: 0.029). No statistically significant relationship was detected between serum Ca^{2+} and right key pinch, right-left handgrip, right-left tip pinch and muscle masses. No statistically significant relationship was detected between serum Mg^{2+} levels and right-left key pinch, right-left handgrip, right-left tip pinch and muscle masses. The correlation matrix in the second trimester is shown in Table.5.

Table. 5. Correlation analysis of second trimester parameters

		Ca^{2+}	Mg^{2+}	GW	RHG	LHG	RKP	LKP	RTP	LTP	Total Muscle-mass	RA MM	LAMM
Ca^{2+}	Pearson's r	—											
	p-value	—											
Mg^{2+}	Pearson's r	0.101	—										
	p-value	0.495	—										
GW	Pearson's r	-0.151	-0.152	—									
	p-value	0.318	0.314	—									
RHG	Pearson's r	0.108	0.079	0.088	—								
	p-value	0.466	0.594	0.559	—								
LHG	Pearson's r	-0.028	0.070	-0.010	0.777	—							
	p-value	0.849	0.636	0.946	<0 .001	—							
RKP	Pearson's r	-0.098	-0.025	0.111	0.489	0.510	—						
	p-value	0.508	0.867	0.463	< 0.001	<0 .001	—						
LKP	Pearson's r	-0.316	-0.050	0.192	0.423	0.532	0.773	—					
	p-value	0.029	0.738	0.202	0.003	< 0.001	< 0.001	—					
RTP	Pearson's r	-0.122	0.086	0.089	0.467	0.568	0.575	0.537	—				
	p-value	0.407	0.563	0.555	<0 .001	<0 .001	<0 .001	< 0.001	—				

LTP	Pearson's r	-0.230	0.054	0.095	0.434	0.582	0.523	0.690	0.804	—			
	p-value	0.116	0.717	0.529	0.002	<0 .001	<0 .001	<0 .001	<0 .001	—			
Total Muscle-mass	Pearson's r	-0.003	0.022	0.194	0.313	0.224	0.280	0.249	0.076	0.116	—		
	p-value	0.985	0.880	0.196	0.030	0.126	0.054	0.088	0.606	0.431	—		
RAMM*	Pearson's r	-0.005	0.078	0.201	0.241	0.224	0.285	0.268	0.108	0.108	0.935	—	
	p-value	0.975	0.604	0.186	0.103	0.131	0.052	0.068	0.471	0.469	<0 .001	—	
LAMM*	Pearson's r	-0.023	0.067	0.225	0.245	0.261	0.312	0.293	0.170	0.183	0.919	0.950	—
	p-value	0.879	0.656	0.137	0.097	0.077	0.033	0.046	0.253	0.218	<0 .001	<0 .001	—
BMI**	Pearson's r	0.197	0.095	0.279	0.126	-0.038	0.012	0.008	-0.070	-0.086	0.556	0.565	0.528
	p-value	0.179	0.520	0.060	0.395	0.800	0.937	0.955	0.634	0.563	< 0.001	<0 .001	<0 .001

*Muscle mass (kg)
**Body mass index (BMI) (kg/m^2), Gestationalweek(GW), Right hand grip(RHG), Lefthand grip(LHG), Right key_pinch(RKP), Left-key_pinch(LKP), Right_tip_pinch(RTP), Left tip pinch(LTP), Right arm_MM(RAMM), Left arm_MM(LAMM).

Discussion

In our study, it was found that serum Ca^{2+} and Mg^{2+} levels were not correlated with right-left key & tip pinch, right-left handgrip and muscle masses in pregnant women in the first trimester. While Mg^{2+} can not correlate with right-left key & tip pinch, right-left handgrip and muscle masses in the second trimester; It was determined that serum Ca^{2+} levels showed a negative and significant correlation with left key pinch strength in the second trimester (r:-0.316, p:0.029). Although a statistically significant decrease was observed in serum Ca^{2+} and Mg^{2+} levels in the second trimester, it was determined that there was no significant change in the measurements of right-left key & tip pinch, right-left handgrip in the second trimester compared to the first trimester. We found that the decrease in serum Ca^{2+} and Mg^{2+} levels did not affect the measurements in the second trimester as expected. In the literature, muscle strength evaluations mostly consisted of studies conducted on athletes [8, 16,17]. As far as we can determine, there is no study in the literature that evaluates muscle strength in pregnant women.

In a study, in which weightlifters and wrestlers were included and the relationship between anthropometric measurements and handgrip strength was evaluated, no statistically significant difference was observed between weightlifters and wrestlers in terms of handgrip strength. A significant relationship was found between body mass index (BMI) and handgrip strength in both groups [1]. In a study comparing women weightlifting athletes and sedentary women, it was reported that right-left handgrip strengths were correlated with BMI in both groups [3]. In our study, BMI was statistically significantly higher in the second trimester, as expected. We found a statistically significant relationship between BMI and right arm, left arm and whole body muscle mass in both the first and second trimesters. However, there was no significant correlation between BMI and right-left key & pinch strength, right-left handgrip sterngth. It was observed that there was a statistically significant relationship between handgrip strength and sports ability in many sports activities [5]. Some studies evaluated male [6] and female [8] athletes separately. In a study comparing male athletes with a sedentary group, handgrip strength was statistically significantly higher in athletes than in the control group [17]. In a study involving an average of 2500 people, patients over and under the age of 65 and male and female patients were grouped. Handrgrip strength was inversely proportional to increasing age in both groups. Women over 65 years of age with osteoporosis; It was found that handrgrip strength was statistically significantly lower in women with osteopenia under the age of 65 [19]. In a study conducted on geriatric patients, a statistically significant relationship was found between magnesium levels and handgrip strength at the beginning and end of rehabilitation [20]. In a study conducted in children, no statistically significant relationship was found between calcium and muscle mass and muscle strength, and no significant relationship was found between Mg^{2+} and handgrip strength [21]. In our study, there was no significant correlation between serum Mg^{2+} levels and right-left handgrip, right-left key & pinch. In addition

to Mg^{2+} level, there are also studies in the literature on dietary Mg^{2+} intake [22, 23]. In a study conducted in geriatric patients, the relationship between diet and muscle strength was evaluated, and it was stated that there was a positive significant relationship between Mg^{2+} intake and grip strength [20]. In a study, comparing a group given Vitamin D and Mg^{2+} for eight weeks with a control group given a placebo, handgrip strength increased statistically significantly in the group given Vitamin D and Mg^{2+}. It was found that there was no significant increase in the placebo group [23]. Our study was the first to evaluate muscle strength in pregnant women, and it is important as it shows whether muscle strength can be related to serum Ca^{2+} and Mg^{2+} levels.

Limitations

The number of participants included in our study is limited. A limited number of participants were included in our study in order to ensure effective pregnancy follow-up and muscle strength measurements. The participants' diets and dietary Ca^{2+} or Mg^{2+} content are unknown. Prospective or multicentric studies will contribute to the literature in terms of both recording data and ensuring a high number of participants. Moreover, the lack of third trimester values of pregnant women is another limitation.

Conclusion

According to the results serum Mg^{2+} levels in the first and second trimesters of pregnant women do not have a significant effect on handgrip and pinch strength. Serum Ca^{2+} levels did not impact muscle strength during the first trimester. But, a negative correlation was detected between serum Ca^{2+} levels and left key pinch strength during the second trimester.

Conflicts of Interest

The authors declared that they have no conflicts of interest regarding this work.

Supporting Agencies

No funding agencies were reported by the authors.

Author Contributions

Conceptualization: ÖD, BI; methodology: BI, ÖD, HŞA; investigation: BI, ÖD; resources: BI, ÖD; data curation: BI, ÖD; formal analysis: HŞA, ÖD, BI; writing-original draft preparation: ÖD, BI, HŞA.; review & editing: BI, ÖD, HŞA

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VAKA TAKDİMİ CASE REPORT

POST-TRAUMA RENAL INFACT; CASE PRESENTATION

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ABSTRACT

*Although the kidney is the most frequently injured organ in the urinary system, kidney injuries constitute 1-5% of all injuries.We wanted to present this case because we think it is a good example of kidney damage after blunt trauma and the need for detailed examination even though there are no findings on high-energy post-trauma examination.An 18-year-old female patient was brought to our hospital by 112 after a traffic accident while crossing the road. Temperature on arrival vitals: 36.5 °C pulse: 80/50 mmHg at 100/min GCS: 15.In her first examination, there was a 1*6 cm open wound on the lower lip, an open wound on the chin, abrasions of various sizes in all 4 extremities, and a fracture in the left elbow. There was no tenderness on abdominal examination and no defensive rebound was observed.. In the tomography report: A few wedge-shaped, large hypodense areas were observed in the right kidney parenchyma and were primarily evaluated in favor of traumatic infarction.USG report: Contusion areas seen in the right kidney on tomography could not be distinguished sonographically. No pathology was detected in other organs.In recent years, conservative approaches have been preferred rather than interventional treatment approaches in renal trauma. In this selection,the patient's hemodynamic status, renal functions and vascularization status are taken into consideration. Our patient was discharged after 2 days of intensive care follow-up and 2 days of follow-up in the ward.Renal damage should be especially considered in the presence of macroscopic and microscopic hematuria, abdominal examination findings and hypotension, especially in patients who have experienced high-energy trauma. In our case, the examination was very comfortable, there was no macroscopic or microscopic hematuria, but there was hypotension. Imaging was our main tool in making the diagnosis. Therefore, we think that CT scanning should be performed in high-energy traumas, even if there are no examination findings. should be kept in mind that kidney damage may occur in the presence of hypotension, especially in high-energy traumas, even if there are no examination findings. The absence of hematuria, absence of additional organ injury and normal urea-creatine values do not exclude kidney damage.*

Key words: *Emergency Medıcıne , Renal Infact , Trauma*

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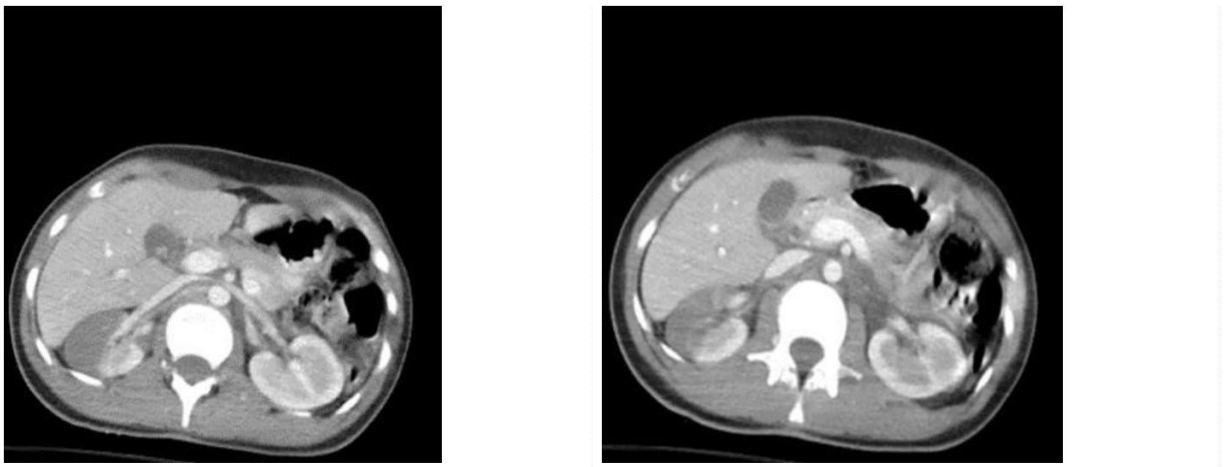
INTRODUCTION

Although the kidney is the most frequently injured organ in the urinary system, kidney injuries constitute 1-5% of all injuries (1,2). 80-90% of kidney traumas occur after blunt abdominal trauma(3). Acute kidney injury following blunt trauma is directly proportional to mortality and morbidity. (4) A single-center retrospective study showed that acute kidney injury developed in 23.8% of 1033 trauma patients in intensive care. Of these, 10% had to undergo renal transplantation (5,6). In one study, 9119 adult kidney injuries were examined, 63% of which occurred from motor vehicle accidents, 14% from falls, 11% from sports injuries, and 4% from non-vehicular traffic accidents (7). We wanted to present this case because we think it is a good example of kidney damage after blunt trauma and the need for detailed examination even though there are no findings on high-energy post-trauma examination.

CASE PRESENTATION

An 18-year-old female patient was brought to our hospital by 112 after a traffic accident while crossing the road. Temperature on arrival vitals: 36.5 °C pulse: 80/50 mmHg at 100/min GCS: 15

In her first examination, there was a 1*6 cm open wound on the lower lip, an open wound on the chin, abrasions of various sizes in all 4 extremities, and a fracture in the left elbow. There was no tenderness on abdominal examination and no defensive rebound was observed. The patient's laboratory tests were sent. In Biochemistry, Glucose: 139 mg/dL Urea: 28.5 mg/dL Creatinine: 0.92 mg/dL, AST: 246 u/L, ALT: 185 u/L, Calcium: 7.95 mg/dL, Total Bilirubin: 0.39 mg/dL, Direct Bilirubin: 0.08 mg/dL, Indirect Bilirubin: 0.31 mg/dL, CK: 276 u/L, CK-MB: 217.8 u/L, CRP: 0.2 mg/L, GGT: 15.5 u/L, Sodium: 132 mmol/L, Potassium: 3.49 mmol/L, Chlorine: 107 mmol/L, INR: 1.24, aPTT: 21.7 sec, PT : 13 sec.In the hemogram, WBC: 17.21 K/uL Hgb: 6.9 G/DL, HCT: 25%, MCV: 58.7 fL, MCH: 16.1 PG, RDW-CV: 20.2, RDW -SD:43.8 fL, Plt:430 K/uL, MPV:8.8 fL. It came as.Brain, cervical, thoracic, abdominal and pelvic CT scans were performed. In the tomography report: A few wedge-shaped, large hypodense areas were observed in the right kidney parenchyma and were primarily evaluated in favor of traumatic infarction. Although the right renal artery has a patent appearance, segmental and arcuate arteries cannot be evaluated. No significant hemorrhagic density was seen in the perirenal fatty planes.A bladder catheter was inserted into the patient. Hematuria was not observed. USG was performed. USG report: Contusion areas seen in the right kidney on tomography could not be distinguished sonographically. No pathology was detected in other organs. Antibiotics and fluid resuscitation were administered for traumatic injuries. The patient was started on 1 unit of erythrocyte suspension. The patient was consulted to urology, general surgery, orthopedics, cardiovascular surgery and plastic surgery. He was admitted to the 3rd stage surgical intensive care unit by the urologist.



Picture 1. Computerized tomography image of the patient

DISCUSSION

In recent years, conservative approaches have been preferred rather than interventional treatment approaches in renal trauma (8). In this selection, the patient's hemodynamic status, renal functions and vascularization status are taken into consideration (9). Our patient was discharged after 2 days of intensive care follow-up and 2 days of follow-up in the ward.

Renal damage should be especially considered in the presence of macroscopic and microscopic hematuria, abdominal examination findings and hypotension, especially in patients who have experienced high-energy trauma (8). In our case, the examination was very comfortable, there was no macroscopic or microscopic hematuria, but there was hypotension. Imaging was our main tool in making the diagnosis. Therefore, we think that CT scanning should be performed in high-energy traumas, even if there are no examination findings.

In a case report of 2 cases published by Zahoor Ahmed et al., it is mentioned that renovascular injuries are associated with non-renal organs (10). However, in our case, there was no accompanying organ injury and it was evaluated as isolated renal trauma.

It should be kept in mind that kidney damage may occur in the presence of hypotension, especially in high-energy traumas, even if there are no examination findings. The absence of hematuria, absence of additional organ injury and normal urea-creatine values do not exclude kidney damage.

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