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İÇİNDEKİLER / *CONTENTS*

DERLEME/REVIEW

VAKA TAKDİMİ / CASE REPORT

DERLEME REVİEW

THE ROLE OF IMPEDANCE CARDIOGRAPHY IN THE EVALUATION OF CARDIAC PARAMETERS IN WEIGHT-LIFTERS

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ABSTRACT

Weightlifting is a strength sport that puts intense pressure on the heart and cardiovascular system. In addition to performance data, cardiovascular and hemodynamic variables are also evaluated in the follow-up of athletes and the organization of training programs. These variables are of critical importance for both the health and performance of athletes. Methods such as the thermodilution measurement method, which is the gold standard used for evaluations, are invasive and expensive methods that require experts to apply. Introducing alternative methods to weightlifting that can be used for this purpose can facilitate both coaches' evaluations and financial managers. Thoracic Electrical Bioimpedance method is a non-invasive, inexpensive and operator-independent measurement method that enables the evaluation of cardiac and hemodynamic parameters. In this study, the evaluation of hemodynamic and cardiac parameters with the thoracic electrical bioimpedance method will be discussed as an alternative to inexpensive, expert-requiring and invasive methods.

Keywords: *Elite weightlifter, Cardiac output, Electrocardiography, Thoracic Electrical Impedance Cardiography, Quantitative Calculation*

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Methods

Keywords Selection

A comprehensive set of keywords was carefully selected for the research. The keywords consisted of the terms 'Weightlifting', 'cardiac', 'hemodynamic', 'performance', 'electrocardiography', 'echocardiography', 'thoracic electrical bioimpedance', 'thoracic bioimpedance', 'stroke volume', 'heart rate' and 'cardiac output'. Our search strategy involved using Boolean operators (AND, OR) and strategic combinations of keywords.

Database Search

A comprehensive search of major databases such as PubMed and Google Scholar was conducted. Only publications written in English were included in the searches.

Inclusion and Exclusion Criteria

Studies to be included in the review must have the following scope: *i*. Describe the thoracic electrical bioimpedance method. *ii*. Include hemodynamic and cardiac monitoring of weightlifters. *iii*. Compare the results obtained with the thoracic electrical bioimpedance method with the results obtained with accepted methods.

Publications published in languages other than English were excluded.

INTRODUCTION

The history of strength-based training, essential for survival, almost matches the beginning of human history. Since ancient times, weightlifting has been an all-round power sport which has expanded throughout all communities and continues to do so today (1). Olympic style weightlifting is a sport in which maximal weight is lifted using snatch and clean and jerk techniques. During exercise, the muscles work more with the anaerobic metabolic pathway, while the heart is faced with a pressure load rather than a volume load in weightlifting (2).

In many competitive strength sports, high-intensity training triggers different metabolic and cardiovascular physiological responses. These responses often represent a major effort to maintain whole-body homeostasis. In order to maintain homeostasis and overcome damage caused by training, many physiological adaptation mechanisms are involved, including metabolic processes related to both local and systemic inflammatory and repair mechanisms (3-9). Elite athletes who engage in prolonged overtraining are valued to evolve chronic myocardial adaptation. Eccentric hypertrophy is a result of endurance training, such as medium-distance or marathon running, where internal diameter of left ventricular increases relatively from the volume load during prolonged exercise (10). Training like bodybuilding and weightlifting put the myocardium under instantaneous high pressure, which results in concentric hypertrophy and thickens the myocardial wall more than the internal diameter of left ventricle (9-11). Degree of change in left ventricular hypertrophy and enlargement is dependent on physical training program, endurance or strength.

The remodeling in heart seen in athletes is related to particular hemodynamic demans of the exercise performed (12).

Long-term training may differ response to isometric exertion, reducing double product and stroke work. Time stands for some other variable which can influence responses, as stroke volume (SV) and ejection fraction recovery (partially) of the occurs following the first decrease and vascular resistance reduces. Perfusion of isometrically contracting skeletal muscles is related to the balance between regional metabolic variations that cause vasodilation and increases in intramuscular pressure that cause obstruction of blood flow; vascular resistance is affected by the interaction of both factors (11). Potent vasodilators such as histamine and bradykinin are produced in exercising muscles, and the effect of these metabolic factors leading to decreased vascular resistance will be even greater because of the large muscle mass involved in exercise. The muscle's connective tissue contains nerve ends called afferent fibers, which are able to sense changes in metabolism; in athletes, long-term muscle strength training can enhance circulatory function through down-regulating the detection threshold. Moreover, increased stroke volume with effort in athletes is an extra element that contributes to a reduction in vascular resistance. An additional mechanism which may clarify the enhanced cardiovascular adaptation of static athletes to isometric exercise is myothermal economy increasing which occurs along chronic pressure overload in experimental preparations. This is consistent with clinical results which measuring results of oxygen in hypertrophied hearts in hypertensive individuals specify an approximately 50% rise in productivity (11).

Cardiac adaptation is achieved by two main exercise forms: dynamic and static. During dynamic exercise, athletes may experience a small rise of arterial pressure, a rise in heart rate (HR), and cardiac output (CO) of up to 40 L/min. Eccentric hypertrophy may be experienced in these athletes; in athletes who perform short-term, high-intensity static exercise, such as weightlifters, a small rise in HR and CO is seen along with an increase in arterial pressure. In weightlifter athletes, arterial pressure may increase to 480/350 mmHg along training, corresponding to hypertrophy leading to wall thickening (concentric) in static exercises (13). Patients with myocardial infection, inflammatory myocardial diseases, idiopathic dilated cardiomyopathy, and chronic hypertension also exhibit this adaptability, although athletes typically exhibit compensation without myocardial dysfunction (10).

Myocardial deformity occurs in many different forms depending on the kind of training elite athletes undergo, however, the most frequent occurrence is cardiac hypertrophy. In elite athletes, pathological myocardial hypertrophy presents as hypertrophic cardiomyopathy, a leading reason of instantaneous death. Extreme myocardial hypertrophy causes rhythm disturbances and may have adverse impacts on heart mechanisms (14). In general, systolic mechanism of athletes is comparable to that of nonathletes. However, data on left ventricle diastolic mechanism in athletes is still lacking, and most assessments have been performed at rest (15, 16). The actual physiological responses of elite athletes during exercise may be assessed via examination of the structure and function of the myocardium not only at rest but also just following an optimum exercise

load test depending on the kind of training. Regular stretch training, whether or not combined with a schedule of endurance exercises, become an important factor in improving public health because of its proven beneficial effects on improving and maintaining physical fitness and cardiovascular health in adults with or without chronic disease or disability. On the other hand, the increasing popularity of continuous intense training schedule for lifting weights has led the researchers to have more information for the acute and chronic cardiovascular responses to intense strength training that cause the excessive but transient rise of cardiac afterloads and to examine cardiac characteristics of seen in well-trained weightlifter athletes (17).

It is possible to identify early abnormal circulation by continuously monitoring hemodynamic condition (18). Studies on heart of athlete primarily include echocardiographic assessment to identify changes in morphology and function. However, these studies have yielded conflicting results regarding changes in systolic and diastolic function in heart of athlete (12).

One of the primary evaluation methods used in studies is electrocardiography (ECG). The first effort when interpreting an ECG of athletes is specifying whether the ECG is ' standard' or 'nonstandard'; primer does not need further investigation, while second requires further assessment. Although, ECG examination should be performed in consultation with an expert who is sensitive to changes observed in heart and cardiac conditions of athletes related to instantaneous cardiac death. Changes associated with normal training include early repolarization, atrioventricular block, right bundle branch block left ventricular hypertrophy according to voltage specifications and sinus bradycardia/arrhythmia. Although these changes are significant, they are signs of physiological alterations in the morphology and mechanism of heart and therefore do not represent a higher risk for rhythm disturbances cases for athletes. Non-standard ECG differs contain T wave inversions, ST depression, pathologic Q waves, left axis deviation, and conduction latencies or deviations. Mentioned differences do not linked to physiological differs along exercising and can specify a pathologically underlying condition and therefore may pose a higher risk rhythm disturbance incidents, such as instantaneous cardiac death, for the athlete (19).

Various studies in athletes have revealed that the frequency of severe ECG nonstandard cases in public ranges between 4% and 9.6% (20-23). In a research that involved athletes training for Olympic games, ECG changes have been realized in athletes of 89% rate, whereas just 11% showed variations in ECG that were entirely normal. ‘Benign’ or common features (sinus bradycardia, atrioventricular block, early repolarization, right bundle branch block and isolated features of left ventricular hypertrophy) associated with athlete’s heart were detected in 65% of athletes, while ‘suspicious’ or uncommon features (left posterior fascicular hemiblock, complete bundle branch block, ventricular arrhythmia, T wave inversion or pathological QRS axis deviation) were reported in 23% (24). In a different observational comparative study, abnormal ECGs were seen in 34% of athletes (25).

Hemodynamic monitoring is important not only for improving the performance of athletes but also for their health. An instantaneous athlete death is a catastrophic incident that not only

causes nonrecoverable loss but also negatively impacts their counterpart in terms of training and performance. As a preventative action to lessen likelihood of this regrettable event, mostly sports organizations advise both an ECG and a thorough cardiovascular assessment. Sudden death from cardiovascular causes, particularly from arrhythmias, is the main reason why athletes die while participating in sports. Structural heart disease, such as Hypertrophic cardiomyopathy, Wolff–Parkinson–White syndrome, Brugada Syndrome, abnormal origin and course of the coronary arteries, or electrical heart disease predisposes athletes to enhance lethal rhythm disturbances along strenuous training or competitive sports. Because ECG differences in mentioned diseases resemble those observed in the physiologically adapted hearts of athletes, some athletes may be permitted to compete in sports although their hearts are structurally defective, thereby endangering their lives. Athletes' ECG variations are probably going to get worse as sports competitiveness rises (19).

One of the criteria evaluated in hemodynamic monitoring is CO, which is the primary factor influencing the supply of oxygen to the end organs, which is the HR and SV products (26). Techniques for measuring or evaluating CO can be categorized as noninvasive, minimally invasive, or invasive (27, 28). The clinical reference standard for invasive CO testing is still the intermittent pulmonary artery thermodilution method with a pulmonary artery catheter (29). Although pulmonary artery catheter placement and maintenance are related to uncommon yet possibly serious (30), pulmonary artery catheterization is still indicated. In order to measure CO and heart loading tensions (27). Alternative techniques for measuring CO have been reported; these techniques contain other indicator dilution methods which require invasive procedures (transcardiopulmonary thermodilution, lithium dilution), methods which require minimally invasive procedures (pulse wave analysis, esophageal Doppler) and several methods which do not require invasive procedure (28, 31-34). These non-invasive CO estimation techniques depend on various physical measuring concepts. These methods contain non-invasive pulse wave analysis, pulse wave transit time, thoracic bioreactivity, partial carbon dioxide rebreathing and thoracic electrical bioimpedance (TEB) (34-36).

Nowadays, functional hemodynamic parameters can be measured with different minimally invasive and noninvasive monitoring systems to guide treatment planning and improve patient care. Among them, the thoracic electrical bioimpedance technique is widely available (18).

TEB uses physical principles that assume that differs in intrathoracic blood volume along the cardiac cycle cause differs in electrical conductance and impedance of thorax and that these differs are primarily linked to differs in aortic volume (34-37). Differs in thoracic impedance are determined via electrodes that evaluate the variance between the applied voltage and the voltage determined following applied low-amplitude, high-frequency current to thorax (34-37). The bioimpedance method assumes a proportionate correlation between impedance (bioimpedance) variations and SV (estimated using the slope of the change in ventricular ejection time and aortic volume) (34-37). There are several factors that limit the thoracic bioimpedance method for CO

estimation in daily clinical practice. Quality of signal may be degraded because of person movement, electrical interference (electrocauterization in the operating room, etc.), rhythm disturbances, and mechanical ventilation (34-37). Additionally, the practical application of technologies is limited by a number of pathological situations since the underlying assumptions are no longer true (obesity, pleural effusion, pulmonary edema, aortic valve disease, marked changes in peripheral vascular resistance, aortic dissection, aortic prosthesis, etc.) (34-37). Numerous confirmation researches detail the use of bioimpedance for CO measuring in various clinical settings (35, 38, 39).

TEB involves delivering low-amplitude, high-frequency electric current through thorax. Sensor electrodes that measure impedance are flanked in the top and bottom thorax. TEB technique uses variations in thoracic electrical conductivity in relation to differences in thoracic aortic blood flow throughout the cardiac cycle to record hemodynamic characteristics (40, 41). Electrical current flows through both low- and high-conductivity channels in human bodies, and the electrical conductivity varies among bodily tissues (18). TEB takes advantage of the fact that various bodily tissues have varying electrical resistance or impedance characteristics. In this regard, blood has good electrical conduction, but fat, bones, lungs, and muscles have weak conductions. Variations in the blood's location within the thorax result in changes in the impedance of that region of the body. Blood is ejected into the aorta and pulmonary arteries throughout ventricular systole, enhancing their volume. This means a decrease in regional impedance. The recorded variations in impedance are used to calculate hemodynamic parameters (42).

The hemodynamic status can be reflected in the impedance variations that the TEB records (18). The test involves placing output electrodes on the patient's skin to apply high-frequency low-intensity alternating current to the person's chest and receiving electrodes to record voltage changes and obtain an ECG recording. The alternating current that was utilized to create the recording above is entirely safe, undetectable to the individual, and the test does not have any harmful or discomfort effect (42).

Using this method, in addition to CO, cardiac index (CI), SV, stroke index (SI), thoracic fluid content (TFC), systemic vascular resistance (SVR), systemic vascular resistance index (SVRI), velocity index (VI), acceleration index (ACI), left ventricular ejection time (LVET), pre-ejection period (PEP), systolic time ratio (STR) parameters can also be obtained. In addition, simultaneously recorded ECG and heart sounds collect the necessary data for hemodynamic evaluation (42).

Thermodilution measures, an invasive hemodynamic monitoring technique that has been employed in healthy volunteers for more than 40 years and is still the gold standard, have been utilized to validate the CO measurement by TEB (18); however, it is not commonly used in clinical settings and has been shown to be not reliable (43). Athletes' hearts are frequently identified using standard Doppler echocardiography (DE), which also helps to distinguish them from left ventricular diseases (11). Parashar et al. compared the results of TEB and DE. This research confirmed

that TEB and DE results are comparable (44). A study shows that there is a distinct trend correspondence between SV changes determined by thoracic echocardiography and TEB (43).

This technique has many limitations that may significantly decrease measurement accuracy, even if it has advantages and can monitor several hemodynamic characteristics. These consist of: having a very low body weight (less than 25 kg) or a high body weight (more than 220 kg), being short (less than 120 cm), being physically active at the time of recording, having a rhythm disturbance (frequent contractions, atrial fibrillation), having an intra-aortic counter pulsation, having a heart rate over 250 beats per minute, and having a serious insufficiency (defective closure) of the mitral or aortic valve (42).

It takes highly qualified healthcare professionals and extremely costly specific instruments to monitor alterations in cardiovascular hemodynamics. There is no inexpensive, noninvasive, and easy-to-use method available to assess the hemodynamics of the cardiovascular system, such as DE, magnetic resonance angiography, or radionuclide imaging. Impedance cardiography, or TEB, meets these criteria. It is noninvasive, which is a definite advantage over traditional methods that require catheterization. As a result, there is no risk of possible complications and the procedure is less costly and easier (42).

The TEB approach does not require a professional operator, therefore reduces the possibility of errors caused by operator variability. Compared to other approaches, the TEB technique has a reduced variation rate. The TEB approach has been utilized more frequently for evaluating hemodynamic condition since the 1990s, encouraging its application to a stage where the theory of the TEB technique serves as a guide for routine clinical practice. TEB monitoring has the ability of obtaining particular characteristics representing contraction of left ventricular that cannot be achieved with thermodilution technique and can provide important information to determine deterioration of left ventricular systolic mechanism, particularly for undesirable cardiovascular events and rate of death (18).

Although there are many researches in literature investigating morphological and physiological adaptation responses in weightlifters resulting from different weightlifting-specific training programs, the findings of existing studies provide limited physiological evidence in terms of hemodynamic parameters and impedance cardiography (45-49). The ultimate goal of weightlifting athletes and coaches is to implement the necessary and weightlifting-specific training programs to obtain the best weightlifting performance. Although, some physiological and hemodynamic differences which are a result of training in weightlifting athletes may negatively affect the maximal weightlifting performance expected from training. The type, duration and intensity of training applied to weightlifters are extremely important in terms of the reactions that will occur as a result of training and the physiological adaptation response.

CONCULUSION

Training an elite weightlifter is a challenging process for both the athlete and the coaches. During this process, many variables are examined and analyzed, and training programs are prepared according to the results of these analyses. A small criterion that may be overlooked may lead to irreversible outcomes in terms of both the athlete's health and performance. In a sport such as weightlifting, which requires intense strength and creates high pressure on the heart, hemodynamic and cardiac parameters are of vital importance. TEB is a reliable, valid, noninvasive and operator-independent medical measurement technique for measuring various functions of the cardiovascular system and some hemodynamic indices in elective medical settings, intensive care and laboratory environments. As mentioned above, most of the hemodynamic and cardiac parameters, which are valuable in evaluating the data required for both protecting the health of athletes and improving their performance, can be obtained with the TEB method. In addition, the necessary hemodynamic and cardiac data of the athletes can be recorded with the TEB method on a mat in the hall where the athletes train, without the need for a special place to record the data. These advantages can also provide advantages to the athlete and the coach in terms of time and money. In addition, with the cardiac and hemodynamic parameters obtained from the TEB records just before and after the training in the training hall, a clue about a negative development that may affect the athlete's health can be obtained early and the necessary health institution can be applied to immediately. This can prevent the occurrence of undesirable situations. This technique, which is being used in more and more application areas, has the potential to provide valuable information to coaches and athletes in terms of cardiac and hemodynamic monitoring of weightlifters. As a result, it can be concluded that TEB records taken before and after training during the training, training and competition preparation processes of weightlifting athletes can provide useful information both in preparing a training program for coaches and in preventing possible cardiac risks that may arise in the athlete's health.

LIMITATIONS

The use of TEB technique is increasing day by day both in research and in daily use. However, although there are many studies in the literature on the methods mentioned in the study used to determine hemodynamic and cardiac parameters in other segments of society and in research (human and animal), there are very limited numbers of researches in which the TEB technique was used in weightlifting athletes (or the other athletic branches) as far as can be reached. For this reason, in this review, rather than the measurements performed with the TEB technique on weightlifters, it has been interpreted that the TEB technique is recommended as a cheap, operator-independent and noninvasive technique for monitoring cardiac and hemodynamic parameters of weightlifters, using data obtained using other measurement methods on weightlifters, athletes in other branches and other segments of society in the literature, and using data that can also be obtained with the TEB technique.

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DERLEME REVİEW

RELATIONSHIP BETWEEN CHEMERIN, AN ADIPOKINE, AND CARDIOVASCULAR DISEASE; A REVIEW

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ABSTRACT

Statistical studies conducted for many years have found circulatory system diseases to be the leading cause of death. Turkey's 2023 statistics indicate circulatory system diseases as the cause of death with 33.4%. The World Health Organization (WHO) has defined cardiovascular diseases (CVDs) as diseases with high mortality rates due to serious economic problems, which continue to increase in the 21st century, and due to the level of morbidity and low quality of life. Obesity, which is one of the etiological factors, is becoming more common in children as well as adults and adolescents, and It is stated that the probability of becoming obese in later ages is approximately five times more in obese children than in normal weight children. Adipose tissue, liver, kidney, spleen, epithelial tissue, fibroblasts, chondrocytes, skin, lung, pancreas, placenta and adrenal glands can be counted as places where chemerin is synthesized. Retinoid Acid Receptor Response 2 (RARRES2) in other word Tazarotene Induced Gene 2 Protein (TIG2) was firstly detected at the gene level in the skin by Samson et al. in 1998, but it was only recently in 2007 that it was named chemerin, an adipokine. There are new findings related to adipokine family including, leptin, adiponectin, especially chemerin and tumor necrosis factor-α. (TNF-α.). Case-control studies have noted a parallelism between coronary artery disease (CAD) and serum chemerin levels and which can significantly impact defining the serious of CAD. This parallelism between chemerin, an adipokine, and CVDs with atherosclerosis, has been detected in autopsies. Togetherness was observed between chemerin excretion from perivascular adipose tissue (PVAT) and coronary atherosclerosis same like in aortic atherosclerosis. This study aims to encourage studies to be conducted on chemerin as a marker or follow-up criterion by drawing attention to the relationship between chemerin and CVDs.

***Keywords**: Adipokine, Chemerin, CVD*

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Background

This review focuses on the determination of high blood levels of chemerin, one of the endocrine functions of fat tissue in the physiological process, in people with increased fat tissue and/or metabolic syndrome (MetS), in cardiovascular system diseases and whether there is a positive correlation between them. It draws attention to the commonalities it shares with pathologies that may cause damage or decrease in the functions of the heart, coronary arteries and other vascular systems, which are elements of the cardiovascular system, and with diseases that may accompany it**,** as in the example of type 2 diabetes mellitus (DMII). In CVDs, it emphasizes the roles of increased body mass index (BMI), fat tissue around organs and plaques linked to intravascular endothelial damage and the association of chemerin. It is stated that vascular damage can affect vital organs such as kidneys and that kidney disease can also cause CVDs. The fact that CVDs are seen at an early age and have high morbidity and mortality also encourages research to clarify whether chemerin is a cause, effect or marker. This review aims to provide an effective research area to decrease the risk of CVD and to keep alive the perspective of reducing mortality and morbidity by using chemerin as a marker or chemerin antagonist as a treatment method in cardiovascular system diseases.

Aims and Objectives

The information provided by the review is expected to provide a projection for future studies on the subject. A structured search strategy was adopted to emphasize the roles of increased BMI, visceral fat, and plaques related with intravascular endothelial damage and the association of chemerin in CVDs.

Keywords Selection

A comprehensive set of keywords was carefully selected for the study. The keywords included the terms ‘endocrine’, ‘cytokine’, ‘adipokine’, ‘chemerin’, ‘CVD’ heart’, ‘health’, ‘heart failure’ and ‘coronary artery disease’. Our search strategy involved using Boolean operators (and/or) and strategic combinations of keywords.

Database Exploration

A comprehensive search was conducted through large databases such as PubMed and Google Scholar. Only publications written in English and Turkish were included in the searches**.**

Inclusion and Exclusion Criteria

The studies to be considered in the review should be within the following scope:

1. It should state the characteristics and endocrine function of fat tissues in the body 2. It should define adipokines and especially chemerin. 3. It should define CVDs and address their etiology 4. It should include studies that include explanatory approaches to the connections between CVDs and chemerin. 5. Since chemerin was recently defined, publications should be recent, but since CVDs are very old, important data should not be omitted.

Studies were excluded in the following cases:

1. Traumatic CVDs. 2. Studies not published in peer-reviewed journals. 3. Publications published outside of English and Turkish**.**

INTRODUCTION

The endocrine system is a very important organization for the growth-development of living beings. Therefore, the regulation of hormones release from endocrine organs must be understood correctly. Many studies have been conducted since the identification of hormones in humans and animals. Earlier researchers have primarily considered the endocrine properties of some organs. Therefore, the endocrine functions of organs such as the pituitary gland, pancreas, reproductive glands and adrenal glands are well known. But, the endocrine properties of liver, muscle and even fat tissue have not been precisely studied. Recent proofs have recognized that skeletal muscle, liver and fat tissue as members of endocrine system (1).

Cytokines were unveiled to be secreted proteins from endocrine system in the early 1980s. They have been reported a kind of hormones in other words cytokines secreted from endocrine system Previously, they were found to performance an important role in the defending system whose steadiness and reaction to hazardous pathogens. They take part to arrange cellular processes like increase in number and even change to become stronger in some tissues. (2). Some of cytokines, especially released from liver, fat tissue, and muscle tissue, adjust metabolism of carbohydrate, protein, and lipid. However, functional relationships between cytokines and abnormal secretions have been detected in many disorders, liver with fat, keto-acidosis (causing energy and alimentation disproportion), also anabolic and catabolic disorders (1).

The effects of myokines, hepatokines and adipokines have begun to be investigated in metabolic adaptation and chronic diseases. Statistical studies conducted for many years have found circulatory system diseases to be the important cause of death. CVDs have always been the important reason for death since the 1960s and have recently been detected as the cause of two out of every three deaths In Brazil. (3).

In the comparative study of TÜİK (Turkish Statistical Institute) between 2015 and 2016, it ranked first among the causes of death related to circulatory system diseases with 39.8%. It was determined that 40.5% of the deaths due to circulatory system diseases in 2016 were due to ischemic heart disease and 23.6% were due to cerebrovascular disease. According to the TÜİK Newsletter published on June 14, 2024; “When deaths were examined according to their causes, circulatory system diseases ranked first with 33.4% in 2023. This cause of death was followed by benign and malignant tumors with 15.0% and respiratory system diseases with 13.2%.”

The World Health Organization (WHO) defined CVDs as diseases with high mortality rates due to the level of morbidity and low quality of life, which continue to increase in the 21st century due to serious economic problems (4).

In the etiology of CVDs, factors such as DM, obesity, inactive lifestyle, etc, as well as year, sex, racial origin and genetic features play an important role (5, 6).

Obesity, one of the etiological factors, is common not only in the adults but also among adolescents and even children. The probability of becoming obese in later life is approximately five times more among children with obesity than in normal children (7).

In recent years, it has been encountered frequently with obesity in the children and resulting in critical physiological problems like enhanced metabolic syndrome (MetS), thus CVD, DMII (8). Excess fat and adipocyte abnormal function are strongly connected with metabolic diseases like obesity, DMII, and atherosclerosis (9, 10).

In case-control studies, it has been reported that there is a parallelism between coronary disease (CAD) and serum chemerin levels and that it has a duty in assign the violence of coronary pathologies (11, 12). Echocardiography studies have identified a relationship between CAD and around of pericardial fat tissues, which are answerable for a remarkable percentage of chemerin (13). The connection between CVD (accompanied by atherosclerosis) and chemerin has been especially identified in autopsies. Positive relation has been observed between chemerin discharge from perivascular tissue and major arteries and coronary lesions (14).

Fat Tissue, One of the Endocrine Organs and Adipokines

Current studies have figured out that fat tissue is an energy storage, an active endocrine organ as well. White fat tissue stores surplus energy in the give shape of triglycerides in the fat cell and can quickly release it into circulation when needed. Fat tissue is the major energy depot in the body, and the storage and secretion of energy in the fat cell are controlled by hormonal signals (insulin, catecholamines, glucocorticoids, etc.) (15, 16, 17,18).

White adipose tissue affects many body functions, a metabolically active and endocrine organ, such as energy and feeding regulation, carbohydrate and lipid metabolism, thermoregulation, neuronal endocrine function, semination, immunity and, most importantly, CV structure (15, 16, 17,18).

Subcutaneous white adipose tissue (scWAT) is connected with an insulin-sensitive body, however visceral white adipose tissue (vWAT) is related with obesity, DMII, dyslipidemia, and also insulin resistance (15, 16, 17,18). An inactive lifestyle causes fat to accumulate around the organs that is directly related to metabolic diseases. (19, 20, 21). For that reason, formulas of consuming energy immobile have been pointed out (24). Curiously, there are energy-exhausting

beige adipocytes that showing uncoupling protein 1 (UCP1) thermogenic capability like to brown fat tissue (22,23). These adipocytes are cells that state UCP1 in scWAT. They have been noted therapeutic objects for the remedy DMII and obesity, as do lipid- accumulating WAT (24).

WAT (white adipose tissue) is an insulin-responsive tissue that accumulating lipids and generates and excretes fat tissue-peculiar adipokine also it can fix power regulate in tissues (25). In this state WAT is territorial and hypodermic adipose tissue store with unique expression formwork of adipokine family.

Fat tissue is synthesized from endogenous glucose via gluconeogenesis, primarily from triglycerides (long-chain fatty acids), formed in the liver from propionate and acetic acid. Redundant energy is warehouse as triglycerol, which can be divided glycerol and fatty acids (26). In addition, when adipose tissue was investigated as an endocrine organ, cytokines were found and named as adipokines.

Adipokines, as endocrine factors, are proteins secreted from adipose tissue (27). With the growth due to various factors of adipose tissue, the expression and secretion of many adipokines have been shown to increase, and are thought to determine changes in long-term energy balance, arrange nutrition and metabolism, and sustain homeostasis in the body (27).

There are new findings concerning young members of the adipokine family leptin, chemerin, tumor necrosis factor (TNF)-α, adiponectin. (27).

Chemerin

Although chemerin, also named Retinoid Acid Receptor Response-2 (RARRES2) in other word TIG2, was first identified at the genetic level in the skin by Samson et al. in 1998, it was only recently in 2007 that it was named as an adipokine and chemerin (28). Today, chemerin is defined as a molecule can attached to the chemokine-like receptor-1 (CMKLR-1, Chem R23, DEZ, ChemerinR) protein and among the newly discovered adipokine groups, including vaspin, omentin-1 and lipocalin-2 adipokines (28, 29).

Chemerin is synthesized in the form of the 18 kDa, 163 amino acid preprochemerin and secreted in the form of inactive/less active prochemerin (30, 31, 32, 33). It has a statin-like folding domain in its structure (34). Conversion from the prochemerin form to the 157 amino acid, 16 kDa active chemerin form is achieved by removing the peptide parts at the carboxyl terminal by proteases (35, 36). In mature adipocytes or activated neutrophils, coagulation and fibrolytic proteases directly or stepwise proteolytic division of the carboxyl terminus of chemerin into active forms of 157 or 156 amino acids with different functions (31, 34, 36). These active forms of chemerin are full agonists against their receptors (31). However, it can become inactive when converted to the 154 amino acid form by the chymase enzyme. In *in-vitro* studies, it was observed that cathepsin G

removed 7 amino acids from prochemerin, elastase removed 6, 8 or 11 amino acids, plasmin removed 5 amino acids and tryptase removed 5 or 8 amino acids (31,34). Depending on where chemerin is isolated, for example, by removing 6 carboxyl-terminal amino acids in human ascites fluid, 8 in serum and 9 in hemofiltrate, chemerin isoforms with different functional properties are formed in different areas (31, 37).

The sites where chemerin is synthesized, its metabolic regulators, its active forms and its functions through the cmklr1 receptor can be summarized as follows (30, 37, 38, 39).

The sites, where chemerin is synthesized, are adipocytes, hepatocytes, kidney, spleen, epithelial tissue, fibroblasts, chondrocytes, skin, lung, pancreas, placenta and adrenal gland.

Retinoids, vitamin D, glucocorticoids, fatty acids, insulin, glucose, cytokines, lipopolysaccharides can be considered as metabolic regulators.

The active forms are Chem163S, Chem157S, Chem158 K, Chem156 F, Chem155 A, Chem154 F.

Some of the duties of chemerin are; acts as a chemotaxis agent, stimulates the immigration of dendritic cells from the skin to the blood in inflammation and damage, stimulates the adhesion of macrophages, reduces the immune response, regulates adipogenesis, increases adipocyte differentiation, increases lipolysis, regulates angiogenesis, regulates osteoblastogenesis, regulates myogenesis, regulates glucose homeostasis, increases glucose uptake, increases insulin release, inhibits the growth of bacteria.

If six amino acids (AA) are removed from the terminal-C tip of prochemerin, it becomes an agonist, high- interest of chemerin. The extracellular protease accountable for this activation is unknown, but a series of cells, inclusive CHO-K1 and COS-7 cells, adequately process prochemerin. This is an important process (40).

CMKLR1 antagonist, α-NETO (α-NETA, 2-(anaphthoyl) ethyl trimethylammonium iodide). The structure of CCX832 is not fully understood. ChemR23 was initially defined aspect a human G protein-coupled receptor reproduce from genomic DNA by PCR. The native ligand of this receptor was recently identified following the recognition of a specific biological movement in ascites likid repercussions to human ovarian carcinoma (41, 42).

The chemerin receptor is stated mainly on macrophages and undeveloped dendritic cells, proposing a play in the previous stage of promoting a defensive response (41). An In-vitro study, chemerin has been noted to provoke intracellular calcium discharge to stimulate chemotaxis of both macrophages, dendritic cells (41).

Chemerin, with an imaging method, as a retinoid-reactive gene on psoriatic piece was recognized in 1997 (43).

Chemerin, a recently discovered adipokine, noted as TIG2 and RARRES2, can say as a new adipokine (43).

A study by Roh et al. (2007) demonstrated that not only chemerin but chemerin receptor mRNA are highly released in fat tissues (44).

To identify trans-regulatory SNPs (single nucleotide polymorphisms), 495 identified patient samples were analyzed using the Cardio-Metabo Chip for association between cardiometabolic circulating chemerin and SNPs (45). It is an enlightening information.

Under certain criteria, RARRES2 gene is entirely marked with SNP rs3735167 and SNP rs4721. Genotyping bring to light that plasma chemerin amount in blood was significantly linking with rs3735167 ($p = -0.157$, $p = 0.001$), but no relationship was found with rs4721. The described transacting SNPs are nestled at field 15q15–23 (45).

On the other hand, polymorphisms in the genes encrypting play a role in angiogenesis, have been informed to be pointedly related with plasma chemerin amount (46).

Measurable chemerin levels are reduced during dietary weight loss and after bariatric surgery (47). However, the influences of dietary macronutrient combination are unnoted. In addition, bile acids manufactured by hepatocytes arrange body weight, insulin sensibility, and energy consumption in rodent models (48).

These metabolic hormones respond to diet with weight loss and changes in composition, although not much is known about their influences on weight loss or diet formulation in humans.

Chemerin and CVDs

Chemerin, found in nM levels in the circulation in both humans and rodents, is meaningfully higher in individuals with inflammation, obesity, MetS, and fatty liver than normal individuals. Current studies have indicated that chemerin is a member of adipokine family associated with fatty liver, insulin resistance, atherosclerosis, other diseases (49). Chemerin also participates in the regulation of angiogenesis, osteoblastogenesis, myogenesis and glucose homeostasis, bone homeostasis and pathophysiology (50).

While some adipokines; FGF21, adiponectin and CTRP9 possibly preservative in atherosclerosis, other members from adipokine family; resistin, leptin, chemerin and proinflammatory cytokines released from fat tissue may complicate disease good progression (51). Increased chemerin is related with systolic blood pressure, excursive proinflammatory cytokines (48, 52). Therefore, it is argued that chemerin may be a new factor in the regulation of blood pressure. This

protein also inhibits the growth of bacteria (29). As studies on the detection of high levels of chemerin in inflammatory fluids and atherosclerotic lesions and the decrease in the level of chemerin in serum with weight loss provided by exercise deepen, it is thought that it may be utility as an analyzing of the development of MetS (52).

Various statements have been made about the synthesis sites, regulators, active forms and functions of chemerin. It performs these functions through the CMLR1 receptor. Chemerin is effective in glucose and lipid metabolism regulation (53,54). It is expressed in a manner similar to the expression of adipocyte genes like glucose transporter-4, leptin, and adipokines, which regulate adipocyte differentiation and modulation (55). Therefore, it was identified as a novel adipokine in 2007.

In the study by Inci et al. (2016) effect of chemerin, in adipogenesis and adipocyte metabolism was confirmed (56). Firstly, Cash and colleagues were demonstrated chemerin are apply effects via the CMKLR1 receptor (57).

Chemerin is important in immune cell requirements and pathological progressions. It is thought to effective in all stages of inflammation. In inflammatory disorders, the increased chemerin activity may be protective or pathological (56).

Action of chemerin in the initial chemoattractant for defensive cites at sites of inflammation, was demonstrated by the expression of CMKLR1 in macrophage and effector cells of the immune mechanism (58). While all biological activities of chemerin were attributed to CMKLR1, two more receptors of chemerin, CCRL2 and GPR1, were identified (59). In later studies, it was found that the pro-inflammatory effect of chemerin was similar to TNFα (60, 61).

Its connection with systemic, inflammatory, chronic diseases and even cancers, both with the polymorphism in the gene locus and with its receptors and expression times, was wondered.

Studies in different processes, about cell development, organ function, and metabolism, have provided experimental evidence for additional functions of chemerin (62).

In systemic disease studies, obesity and MetS have been the primary targets for chemerin, adipokine and hepatokine. Since these diseases are also considered to be predisposing factors for other systemic diseases, along with type 2 DM, chemerin levels have also been measured in these diseases. It has also been related to chronic diseases like psoriasis. In addition, chemerin levels have been examined in relatively short-term but potentially fatal diseases such as preeclampsia and sleep apnea. Although there is a parallelism between chemerin levels in all these diseases and conditions, the highest levels have been detected in chronic hemodialysis patients (56).

Chemerin may affect the insulin receptor working method, leading to insulin resistance or increasing the body's natural insulin resistance (63).

It has been revealed when chemerin level increases in fat tissue, can stimulate serine/threonine kinas, decrescent tyrosine phosphorylation, preventing to move of glucose, give rise to insulin resistance in adipocytes (60).

Roh et al. summarized the physiological effects of adipokines in their studies and mentioned that chemerin increases insulin secretion (27). It was found that the chemerin amount in patients with fatty liver was meaningfully more than in normal persons, but decreased importantly following use metformin. This finding suggested that the event of NAFLD is strictly related to serum chemerin level and insulin resistance (64). In an investigation conducted on the Caucasian population, metabolite levels in serum were found to be very high level in populations with be prone to MetS (65). In a study, serum chemerin quantity were detected to be considerably increased and more severe in people with DMII and NAFLD compared to patients with simple DMII (66).

Newly, a high chemerin level has been shown to be a marker for T2DM (66, 67). From a clinical perspective, treating overabundant chemerin concentration to avert the onset of diabetes also kidney pathology may be a future mission to address, as rosiglitazone administration has been shown to ameliorate diabetic nephropathy by reducing chemerin expression (68).

Chemerin is a new actor that may cause various CVDs by triggering neovascularization, inflammation and contraction together with adipogenesis and also by affecting thermogenesis, steroidogenesis and insulin metabolism. While vit A, fat, carbohydrates and alcohol upregulate the amount of chemerin in the blood from nutrients, omega-3, salt and vit D suppress chemerin. Dietary measures rather than receptor antagonists as drugs may be a new perspective to prevent the harmful effects of chemerin (69).

Despite the advancing decline in CVD mortality among European population are highly prevalent causes of premature death and persistent disablement there. (70).

Many scientific proofs show that raised cholesterol amount have a big impact on the commencement and development of atherosclerotic plaques (71).

Long-term studies in some health research institutions in America have identified ensembles in the public population that do not smoke and do not have diabetes, have <120/80 mmHg arterial blood pressure, total cholesterol <200 mg/dL, and <25 kg/m^2 BMI without specific treatment. According to the risk profile, the group with these data has the minimum CVD and all-reason mortality. In addition, this group has the privilege of living the best in old age with lower healthcare costs (72).

Familial dyslipidemias are hereditary disorders defined by high plasma lipid section grade and frequently early happening of cardiovascular facts (71). One of the causes of CDV is withdrawal from society (73).

Jeopardy factors for CVD cause constructional and functional endothelial disorder (74). The endothelium is the principal regulator of vascular arrange and sustain the balance between vasodilation and vasoconstriction, encourage or prevention of smooth muscle multiplication and transmigration, thrombogenesis (75).

CVD are still the leading cause of death. high chemerin amount has been found to be important in increasing the risk of CVD in people (45).

Emerging proof recommends that adipokines may be very significant effect in the emergence, prognosis, and remission of atherosclerosis (51). As works rake profound into acute MI, the role of biomarkers is befitting progressively significant in both diagnosis and prognosis. Chemerin has the ability to be a valuable tool in predicting prognosis, not only as a marker of inflammation, but also by assessing the severity of CVD (76).

Chemerin has been shown to be related to peripheral arterial rigidity in cross-sectional studies (60). Adenovirus-mediated chemerin clampdown in high-fat diet-fed apoE −/− mice healed atherosclerosis result (77).

In case-control studies noted is between CAD and serum chemerin amount and that it plays a role in determining the seriously of coronary lesions (11, 78).

Echocardiography studies have identified a relationship between chemerin secretion and epicardial fat tissue, which is responsible for a important portion of CAD (13). mRNA levels in human epicardial fat tissue are positively related with TNF-α, BMI, waist size, fasting glucose level, and Gensini score, a mark of the serious of atherosclerosis (79).

A strong relationship has been found between chemerin and general metabolic characteristics like renal function. Chemerin amount may be a clue to renal failure. There is a positive relationship between high serum chemerin amount and CVD risk, independent of renal function (45). Considering that renal diseases predispose to CVDs; a connection can be established.

When chemerin in functional angiogenesis was investigated using in vitro assays, chemerin important stimulated the formation of capillary-like constructions, augmented length of tubule and the count of branches, and augmented the total count of microtubules in these training (46).

Isolated mice arteries express chemerin in PVAT (80). The effect of this is vasoconstriction. Vasoconstriction has been shown with the effect of chemerin without any stimulation or pathology.

Chen et al. (2022), they decided that chemerin blood level in coronary artery disease may be related to genetics and there may be an ordinary association between chemerin and coronary artery disease (81).

Independent of risk factors, Fabian et al. (2019) presented the first cohort study viewing a strong same direction relationship between chemerin and CVD. They were also able to show a same relationship between chemerin and T2D, but this relation was largely explained by BMI and waist size. In summary, the results in most studies have discovered immune-inflammatory causes in the growth of CVD and have highlighted chemerin as an important CVD risk factor (82).

CONCULUSION

The preferential etiological factor of CVDs is obesity caused by MetS and similar conditions. Other systemic and acute diseases can also lead to cardiovascular events. Visceral fat is prominent in CVDs. The endocrine organ feature of fat tissue has brought adipokines to the agenda and made the role of chemerin important.

In vitro and in vivo studies have isolated chemerin among the expressed substances by accepting the fat layer around the vessel as the fourth layer in addition to the endothelium, muscularis and adventitial layers in the vessels. A positive correlation has been found between the increase in epicardial and pericardial fat tissue around the heart and CVD and high chemerin levels.

Recent informations, chemerin is also seen as an angiogenetic factor among those that provide the vascularization needs of the increased fat tissue. It is brought to the agenda that chemerin does not only cause pathological events but may also have a healing aspect.

Although chemerin is not specific to CVDs, its high sensitivity keeps the research excitement alive.

The relationship of high chemerin amount with renal diseases reinforces the importance of chemerin in vascular diseases.

It makes chemerin more noticeable in the formation or stability of aortic and coronary atheroma plaques. The question of whether the proinflammatory chemoattractant effect is a healing effect of chemerin in damaged tissues is worth investigating.

Studies on its immunomodulatory effect and its role in angiogenesis are very limited and remain an issue to be resolved.

Accepting chemerin as a mark for diagnosis and treatment of CVDs will shed light on many issues, but further studies are needed.

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VAKA TAKDİMİ CASE REPORT

NON-COMMUNICATING RUDIMENTARY HORN PREGNANCY DETECTED WITHOUT RUPTURE: A CASE REPORT

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ABSTRACT

Rudimentary horn is a rare congenital uterine anomaly. Rudimentary horn pregnancies occur in approximately 1 in 100,000 cases and are often associated with severe complications. In this report, we present a case of a non-communicating rudimentary horn pregnancy diagnosed at 15 gestational weeks without rupture or detectable fetal cardiac activity, accompanied by a literature review.

A 33-year-old patient with an obstetric history of G5P3A1Y2 (two cesarean sections) was referred to our clinic from an external center. Ultrasound examination revealed the absence of an intrauterine gestational sac. Instead, a fetus and its appendages were observed in the left adnexal region, corresponding to 15 weeks of gestation, with no detectable fetal cardiac activity. The endometrial thickness was measured at 16 mm. The patient underwent laparotomy, during which a non-communicating rudimentary horn containing a 15-week fetus and its appendages was excised and sent for pathological examination.

Although rudimentary horn pregnancies are rare, they are difficult to diagnose via ultrasonography and carry a significant risk of morbidity and mortality. As pregnancy progresses, the risk of rupture increases considerably. Surgical excision is the recommended approach upon diagnosis.

Keywords: *non-communicating rudimentary horn, uterine rupture, ectopic pregnancy*

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INTRODUCTION

Unicornuate uterus is one of the rarest Müllerian anomalies. According to the classification by the American Society for Reproductive Medicine (ASRM), unicornuate uterus is divided into four subtypes: (1) unicornuate uterus with a communicating rudimentary horn, (2) unicornuate uterus with a non-communicating rudimentary horn, (3) isolated unicornuate uterus, and (4) unicornuate uterus with a non-communicating rudimentary horn without an endometrial cavity.

Rudimentary horn pregnancies are associated with complications such as early spontaneous miscarriages, ectopic pregnancies, and preterm labor. Due to the thin myometrial wall in the rudimentary horn, the risk of uterine rupture is significantly high (1).

Non-communicating rudimentary horn pregnancy is a rare condition, with an estimated incidence of 1 in 100,000–150,000 pregnancies (2). Early diagnosis is crucial, as patients presenting in the second trimester may experience severe complications with high maternal morbidity and mortality. However, many cases remain undiagnosed until later stages (3), and fetal survival rates are extremely low (4).

Case Presentation

A 33-year-old female patient, G5P3A1Y2 (two previous cesarean sections), was referred to our clinic with a preliminary diagnosis of extrauterine pregnancy. Upon admission, her vital signs were stable, and she exhibited mild abdominal tenderness. Laboratory results showed a hemoglobin level of 10.1 g/dL, platelet count of 280,000, normal INR, and no significant biochemical abnormalities. The patient had no known comorbidities or medication use, and no history of surgery other than two cesarean deliveries.

Ultrasound examination revealed no gestational sac within the intrauterine cavity. Instead, a well-defined gestational sac measuring 9 × 7.5 cm was identified in the superolateral left uterine region. The sac contained a fetus corresponding to 15 weeks of gestation without detectable fetal cardiac activity. Due to suspicion of an ovarian or tubal ectopic pregnancy, surgical intervention was planned, and necessary blood product preparations were coordinated.

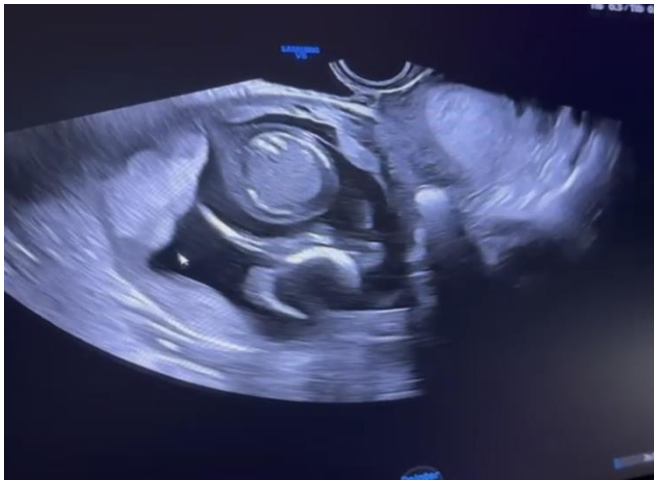

Figure 1. Ultrasound image showing the fetus and its appendages outside the thickened endometrial cavity

Following preoperative preparation, the patient underwent laparotomy via a Pfannenstiel incision. Intraoperatively, no free fluid was observed in the abdomen. A well-demarcated gestational sac was identified originating from the left uterine horn, along with an intact fallopian tube.

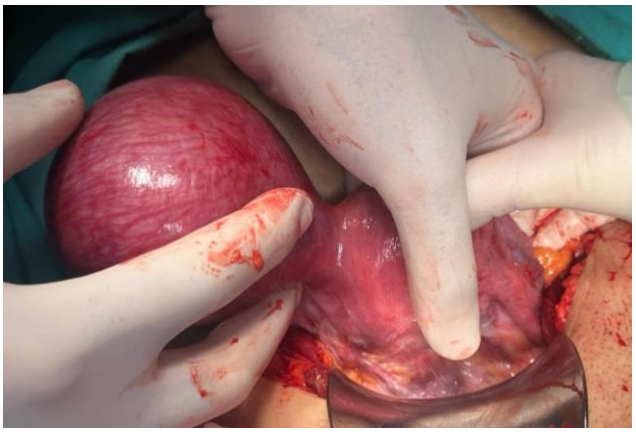

Figure 2. Intraoperative appearance of the gestational sac arising from the left rudimentary horn

The gestational sac and the left fallopian tube were excised together, while the left ovary was preserved. The surgical site was sutured with 0 Vicryl without complications. The excised specimen, including the 15-week fetus and its appendages, was sent for pathological examination.

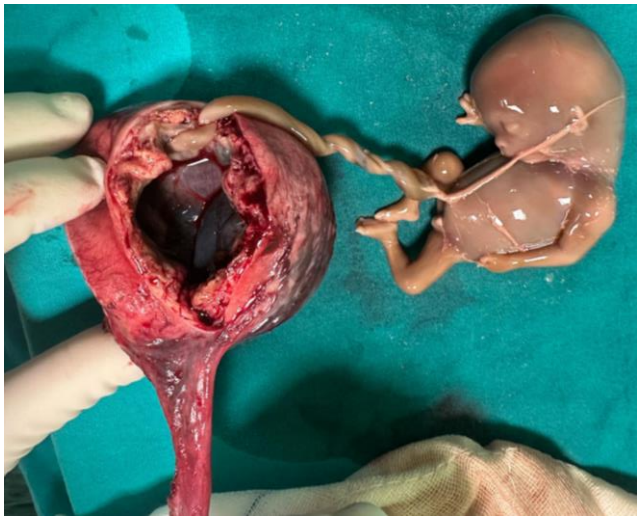

Figure 3. Non-communicating rudimentary horn pregnancy with fetus and appendages

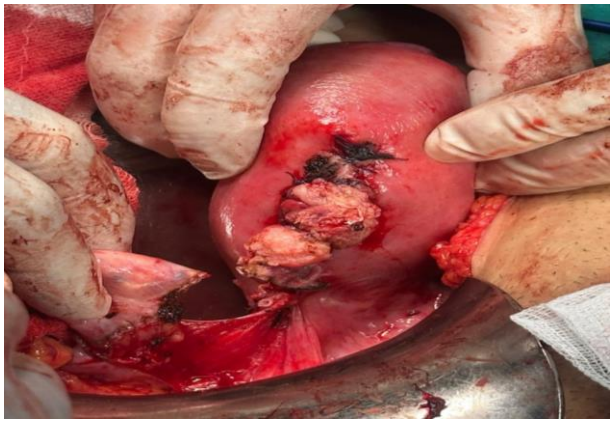

Figure 4. Final appearance of the uterus and left ovary post-excision

DISCUSSION

In cases of a communicating rudimentary horn, the mechanism of pregnancy is straightforward, as there is a direct connection with the functional uterine cavity. However, in non-communicating cases, pregnancy is believed to occur via transperitoneal sperm migration (5).

The majority of rudimentary horn pregnancies are diagnosed at the time of rupture, typically between 10 and 20 gestational weeks. Rupture often results in life-threatening hypovolemic hemorrhagic shock (6). Therefore, early diagnosis is crucial to prevent severe maternal morbidity and mortality.

Surgical intervention not only confirms the diagnosis but also prevents life-threatening complications. Magnetic resonance imaging (MRI) can assist in preoperative planning and help identify

concurrent urinary tract anomalies. The recommended surgical approach includes excision of the rudimentary horn and ipsilateral salpingectomy to prevent recurrent tubal ectopic pregnancies.

In recent years, laparoscopic approaches have been increasingly utilized in non-ruptured cases, demonstrating favorable outcomes (7). Studies suggest that surgical excision of the rudimentary horn does not adversely affect future obstetric outcomes or increase postoperative complications (8).

Conclusion

Rudimentary horn pregnancy is a rare and diagnostically challenging form of ectopic pregnancy associated with high morbidity and mortality. Early diagnosis significantly reduces maternal risks, and detection during early gestation can be life-saving. Given the risk of rupture and associated complications, surgical excision remains the preferred treatment approach.

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Ölçenoğlu M.

VAKA TAKDİMİ CASE REPORT

ENDOMETRIOMA AT THE ISTHMOCELE SITE: A CASE REPORT
İSTMOSEL HATTINDA ENDOMETRİOMA: OLGU SUNUMU

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ABSTRACT

Endometrioma at the isthmocele site is a clinically significant pathology characterized by ectopic localization of endometrial tissue within an isthmocele formed after a cesarean section (1). In this report, we aim to present a case of endometrioma located at the isthmocele site, in the context of the existing literature. A 43-year-old female patient, gravida 2, para 2, living 2 (one vaginal delivery, one cesarean section), presented to our clinic with complaints of groin pain, suprapubic tenderness, and postmenstrual spotting. Ultrasonography revealed a lesion suggestive of endometrioma, and the diagnosis was confirmed by magnetic resonance imaging (MRI) previously performed at an external center. Following endometrial biopsy and cyst aspiration, the cyst contents were evaluated histopathologically. Although endometrioma at the isthmocele site is rare, thorough history-taking and physical examination are crucial for diagnosis. Imaging modalities such as ultrasound and MRI can aid in the diagnostic process, which is ultimately confirmed through surgical intervention.

Keywords*: isthmocele, endometrioma, cesarean scar defect, surgical excision, diagnosis, pathophysiology*

ÖZET

İstmosel hattında endometrioma, sezaryen ameliyatı sonrası oluşan istmosel içinde endometriyal dokunun ektopik yerleşimi ile karakterize edilen klinik olarak önemli bir patolojidir.(1). Bu olguda istmosel hattında oluşan endometriomayı literatür eşliğinde sunmayı amaçladık. 43 yaşında G2P2Y2 (1 normal doğum,1 sezeryan) obstetrik öyküsü olan kadın hasta kasık ağrısı , suprapubik hassasiyetve postmenstruel lekelnme nedenı ile kliniğimize başvurdu. Yapılan usg de izlenen görüntünün endometrioma içeriğinde olması ve dış merkezde çekilen manyetik rezonans (MR) görüntülemesi ile tanının doğrulanmasından sonra endometrial biyopsi ile ,boşaltıldı, kist içeriiği patoloji ile değerlendrildi. İstmosel hattında endometrioma nadir görülse de tanı koymada detaylı anamnez ve fizik muayene önem taşır. usg ,mr gibi yöntemlerle tanısı desteklenip cerrahi yöntemle kesinleştirilir.

Anahtar Kelimeler: *İstmosel, endometrioma, sezaryen skar defekti, cerrahi eksizyon, tanı, patofizyoloji*

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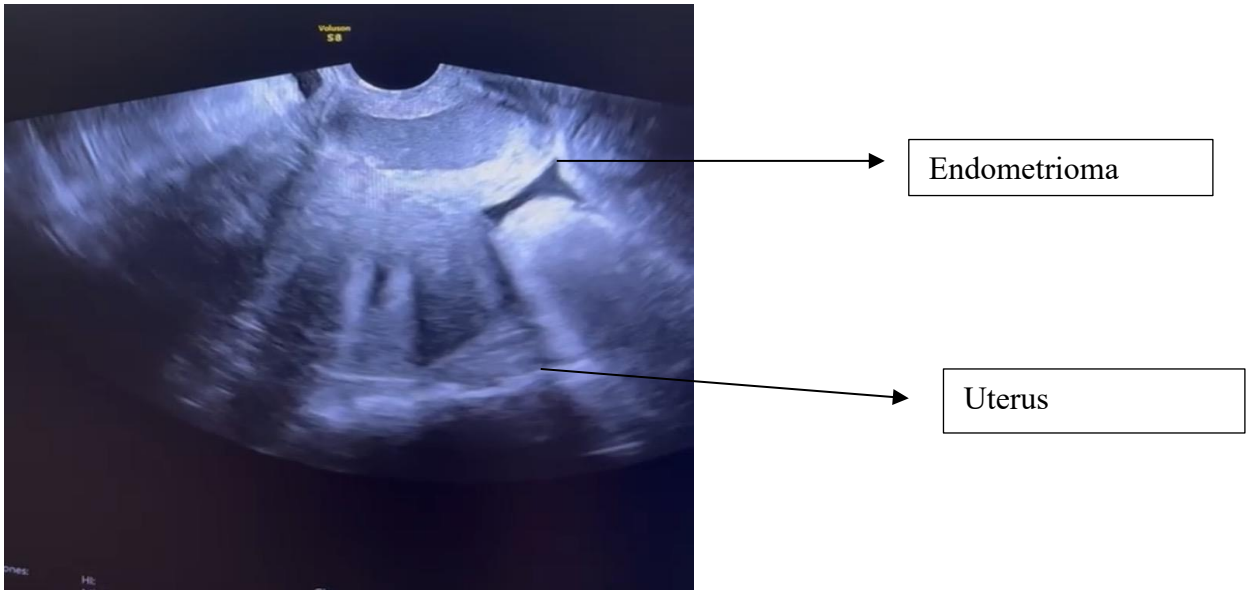
GİRİŞ

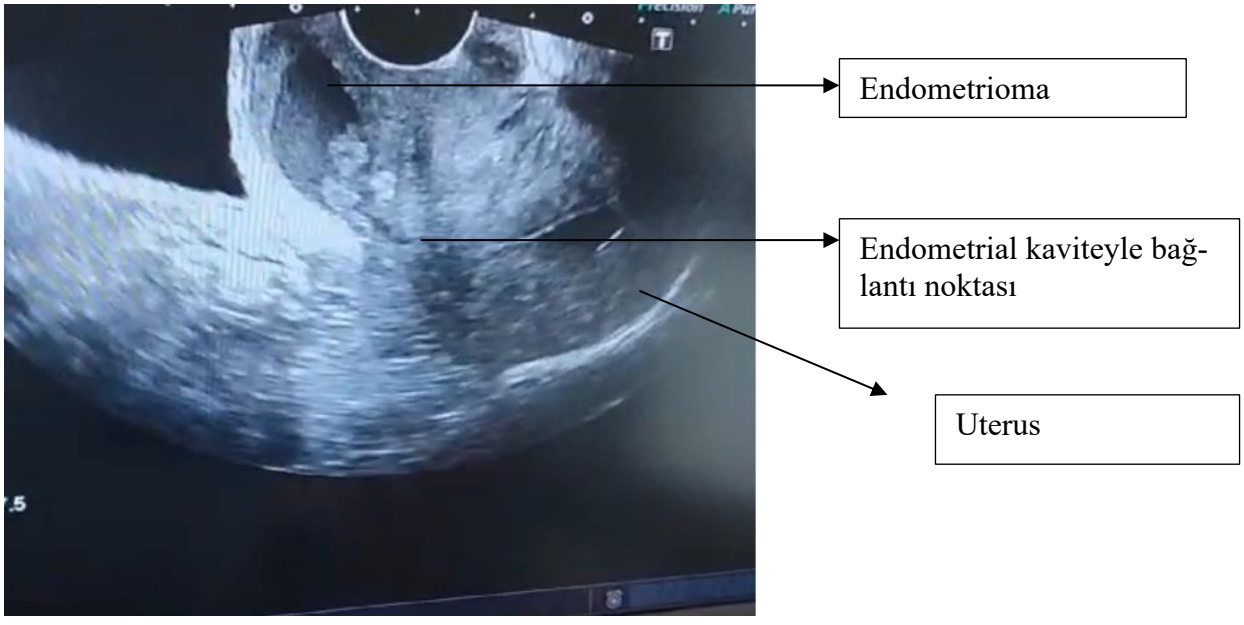
Endometrioma, endometriyal dokunun uterus dışında ektopik olarak bulunması ile karakterize edilen bir endometriozis formudur. İstmosel, sezaryen skar hattında meydana gelen, miyometriyal defekt sonucu oluşan bir cep şeklindeki yapıdır. İstmosel hattında endometrioma, bu alanda endometriyal dokunun heterotopik yerleşimi sonucu gelişen nadir bir klinik tablodur. Hastalar sıklıkla kronik pelvik ağrı, dismenore, infertilite ve anormal uterin kanama gibi semptomlarla başvururlar.İstmosel hattında endometriomanın patogenezi, iatrojenik endometriyal doku implantasyonu teorisi ile açıklanmaktadır (2). Sezaryen operasyonu sırasında endometriyal hücrelerin istmosel içine yerleşmesi ve burada proliferasyonu sonucunda lezyon gelişebilir. Risk faktörleri arasında tekrarlayan sezaryen ameliyatları, sezaryen insizyonunun düşük segmentten yapılması, cerrahi teknik ve yara iyileşme sürecinin bozulması yer almaktadır (3).Hastalar genellikle istmosel hattında lokalize, palpabl ve ağrılı bir kitle şikayeti ile başvururlar. Ağrı genellikle menstrüasyon dönemlerinde artış gösterir. Bunun yanı sıra, sunduğumuz vakada görüldüğü gibi postmenstrüel lekelenme, kronik pelvik ağrı gibi semptomlar da görülebilir, ultrasonografi ile istmosel içinde kistik ya da solid yapıların varlığı saptanabilir.Tanı koymada detaylı anamnez ve fizik muayene büyük önem taşımaktadır. Transvajinal ultrasonografi (TVUSG), manyetik rezonans görüntüleme (MRG) ve histerosalpingografi gibi görüntüleme yöntemleri, endometriomanın saptanmasında önemli rol oynar (4). Kesin tanı, cerrahi eksizyon sonrası histopatolojik inceleme ile konulmaktadır. İstmosel hattında endometriomanın en etkili tedavi yöntemi cerrahi eksizyondur (5). Hormonal tedaviler (GnRH agonistleri, oral kontraseptifler), semptomların hafifletilmesine yardımcı olabilir ancak kesin tedavi sağlamadığı için eksizyon önerilir. Bu olgu sunumu; ,sezeryan ameliyatı geçirmiş hastalarda insizyon hattında endometrioma olasılığını akılda tutulması amacıyla yapılımıştır.

OLGU SUNUMU

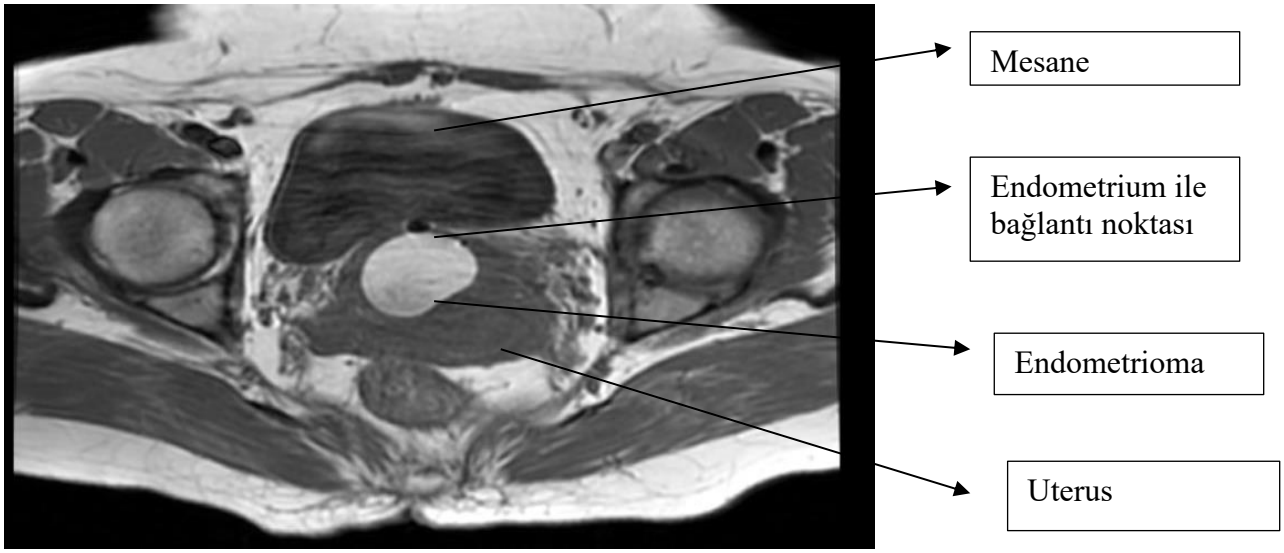
43 yaşında kadın hasta ,kliniğimize 1 yılı aşkın süredir devam eden kasık ağrısı , suprapubik hassasiyetve postmenstruel lekelnme nedenı ile başvurdu. Obstetrik öyküsünde G2P2Y2, 1 normal doğum,1 sezeryan olan hasta son sezeryanı 6 yıl önce idi. Hastanın fizik muayenesinde suprapubik bölgede mesane üzerine denk gelen alana uyumlu hassasiyet mevcut idi. Ele gelen kitle benzeri lezyon saptanmadı . Hormon panelinde LH :50, FSH : 40 .ESTRADIOL :94 ; tiroit fonksiyon testleri normal olarak saptandı. Yapılan TVUSG (transvajinal ultrasonografi) görüntüsü , endometrioma ile uyumlu olup, hastanın farklı bir merkezde yapılan pelvik MR(manyetik rezonans) görüntüsü de endometriomayı destekler nitelikte idi. Bu evrede hastaya diagnostik amaçlı endometrial biyopsi yapıldı ve kist içeriği ile endometriumdan alınan doku örnekleri patolojiye gönderildi. Sonuç, endometrioma içeriği ile uyumlu izlendi



Şekil 1. TVUSG de endometrioma görüntüsü



Şekil 2. TVUSG de endometrioma görüntüsü ve endometrial kaviteyle bağlantısı



Şekil 2. MR (manyetik rezonans) daki endometrioma görüntüsü ve endometrial kaviteyle bağlantısı

TARTIŞMA

Endometriozis ve/veya endometrioma, endometriyumun ektopik oluşumu olarak tanımlanır.Dismenore, düzensiz menstrüasyon, devamlı kasık ağrısı, endometrioma alanında hassasiyet endometriomada yaygın görülen şikayetler arasında bulunur(5)son yıllarda artan sezeryan sayısına bağlı olarak endometrioma gelişimi artmıştır. Sezeryan hattında endometrioma genelde 5 yıldan sonra ortaya çıkmasa da sezerysan sayısının artışına da bağlı olarak bu vakada olduğu gibi geç tespit edilen olgular da mevcuttur.(6)

Fizik muayene , anamnez , TV USG,MR,BT ile desteklenen tanı, kesin olarak patoloji ile konulur ve tedavisi eksiyondur. Bu olgudaki hastamıza da tedavi amaçlıeksizyon önerilmiştir.Biyopsi sonrası şikayetleri hafiflemesi üzerine eksizyon işlemi hasta ile bilgi paylaşımı yapılarak ertlenmiş ve hasta progestogen başlanarak takibe alınmıştır.

SONUÇ

İstmosel hattında endometrioma, doğru tanı ve uygun tedavi yaklaşımları ile başarılı bir şekilde yönetilebilecek bir klinik durumdur. En etkili tedavi yöntemi cerrahi eksizyondur. Bu patolojinin tamamıyla aydınlığa kavuşabilmesi için gelecekte daha çok çalışmaya ihtiyaç vardır.

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VAKA TAKDİMİ CASE REPORT

CEREBROVASCULAR DISEASE DUE TO APICAL THROMBUS SEEN AFTER PERIPARTUM CARDIOMYOPATHY
PERİPARTUM KARDİYOMİYOPATİ SONRASI GÖRÜLEN APİKAL TROMBÜSE BAĞLI SEREBROVASKÜLER HASTALIK

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ABSTRACT

Peripartum cardiomyopathy (PPCM) is a rare type of cardiomyopathy. Its mechanism has not been fully elucidated, and the earliest symptom is shortness of breath due to systolic dysfunction seen near or after giving birth. Arterial and venous thromboembolic conditions may be encountered in these patients.We aimed to present a case of acute cerebrovascular embolism due to thrombus developing in the left ventricular apex as a result of PPCM in a 32-year-old postpartum patient.

Keywords: *cardiomyopathy, peripartum cardiomyopathy, pregnancy, stroke, thromboembolism*

ÖZET

Peripartum kardiyomiyopati (PPKM) nadir görülen bir kardiyomiyopati türüdür. Mekanizması tam olarak aydınlatılamamıştır ve en erken semptomu doğuma yakın veya doğum sonrasında görülen sistolik fonksiyon bozukluğuna bağlı nefes darlığıdır. Bu hastalarda arteriyel ve venöz tromboembolik durumlarla karşılaşılabilir. Bu bildiride, 32 yaşında bir hastada doğum sonrası gelişen PPKM'ye bağlı sol ventrikül apeksinde gelişen trombüsün neden olduğu akut serebrovasküler emboli olgusunu sunmayı amaçladık.

Anahtar Kelimeler: *kardiyomiyopati, peripartum kardiyomiyopati, gebelik, strok, tromboemboli*

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INTRODUCTION

Peripartum cardiomyopathy (PPCM) is a rare type of heart disease that occurs in women during the last month of pregnancy or the first 5 months after giving birth. It is characterized by the onset of heart failure in these women, without any other identifiable cause of heart failure (1). The variables contributing to the higher prevalence of PPCM include, older maternal age, preeclampsia, the presence of cardiovascular risk factors during pregnancy and improved diagnostic capabilities resulting from increased knowledge about this condition (2). Women who develop peripartum cardiomyopathy (PPCM) are more prone to experiencing arterial and venous thromboembolic events. Apical thrombus formation in the left ventricle is a frequent issue in patients with PPCM. Patients may experience acute cerebral embolism as a result of that cardiac thrombus (3).

CASE REPORT

A 32-year-old female her first pregnancy at 27 weeks and 2 days' gestation week is admitted to the gynecology and obstetrics emergency department due to vaginal bleeding. During the examination, it was determined that the fetus did not show any cardiac activity and there was placental abruption, and an emergency cesarean section was planned. After the procedure, the fetus was accepted as exitus. The patient had complaints of shortness of breath and palpitations that started on the second day after giving birth in the ward. The patient's vital signs were as follows: blood pressure was 135/72 mmHg, pulse rate was 108 beats per minute, and the heart rhythm was sinus. The oxygen saturation in the peripheral blood when breathing room air was 79%. Diffuse rales were detected bilaterally up to the upper zones during the physical examination of the lungs. The chest X-ray of the patient showed bilateral signs of congestion (Figure-1).

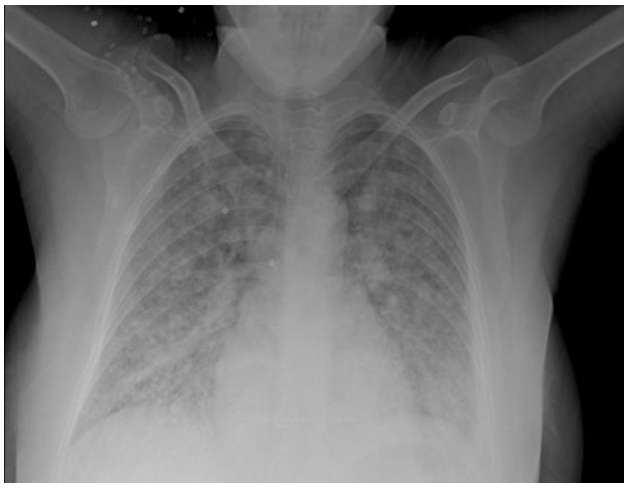

Figure 1. The PA chest X-ray of the patient showed bilateral signs of congestion

Electrocardiography (ECG) showed a significant presence of wide T wave negativity and loss of R waves in the anterior leads (Figure 2). The laboratory results showed that the high sensitivity troponin (hsTnI) level was 333.0 ng/l (normal range: 0-39), the D-dimer level was 1.93 mg/l (normal range: 0-0.55), the creatine level was 0.37 mg/dl (0.5-1.1mg/dl) and the hemoglobin level was 12.1 g/dl (12.5-15.1 g/dl) The patient was consulted to the cardiology department due to acute dyspnea. The cardiologist conducted a transthoracic echocardiogram (TTE) and determined that the patient's left ventricular ejection fraction was 30% as a result of significant global hypokinesia. The patient was transferred to the coronary intensive care unit after being diagnosed with PPCM. The administration of bromocriptine was not initiated due to its unavailability. The patient was started on intravenous (IV) diuretic . Sodium glucose cotransporter 2 inhibitor (dapagliflozin), angiotensin converting enzyme inhibitor (zofenopril), mineralocorticoid receptor antagonist (aldosterone) were added as oral therapy. Dyspnea improved during the follow-up period. At the 54th hour of observation, the patient experienced left-sided hemiplegia and was referred to the neurology department for consultation. On physical examination, facial wrinkles and nasolabial folds disappeared. The upper and lower extremities on the left side appeared paralyzed. TTE was performed with suspicion of cardiac thromboembolism. During the TTE procedure, a 15x18 mm thrombus was found in the apex of the left ventricle (Figure 3).

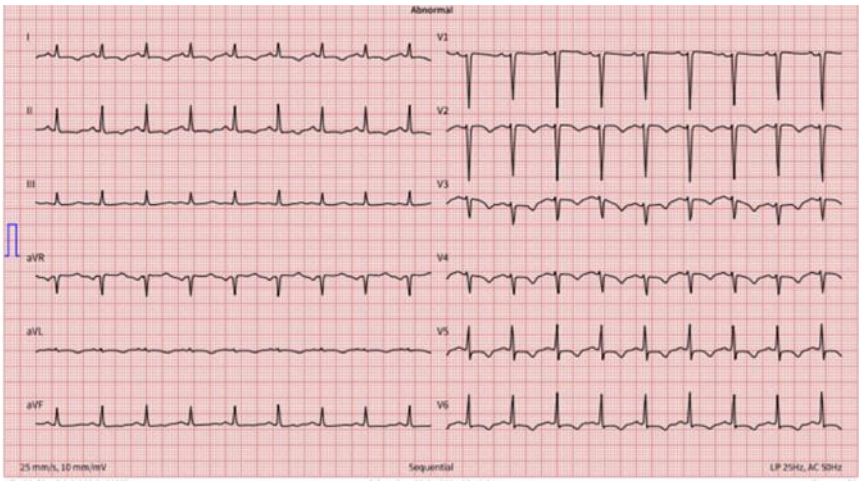

Figure 2. Electrocardiography (ECG) showed a significant presence of wide T wave negativity and loss of R waves in the anterior leads. It is indicated with black arrows.

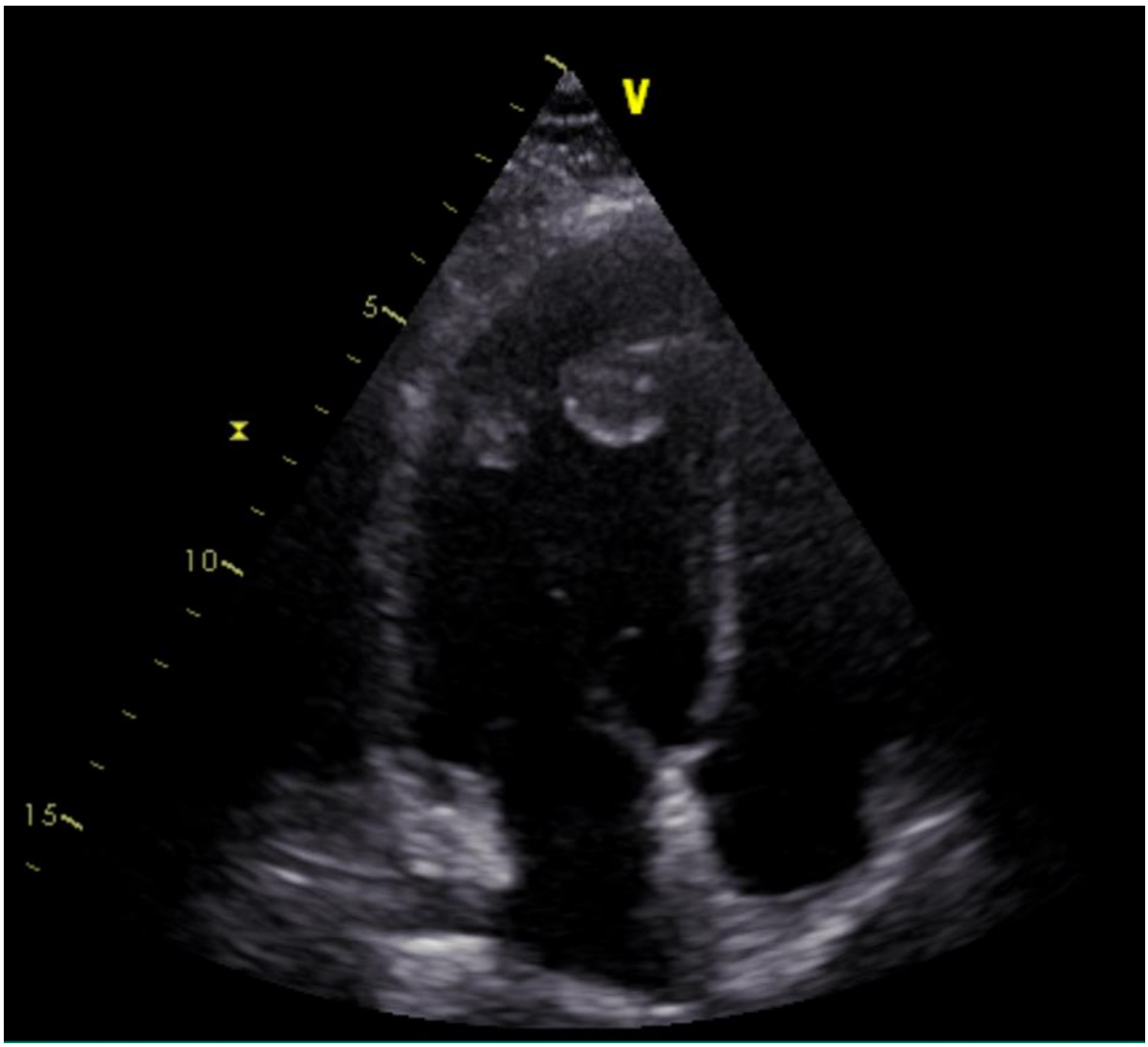

Figure 3. A thrombus of 15x18 milimeters was found at the left ventricular apex in the TTE procedure. It is indicated with black arrows

Then, the patient underwent computed tomography and diffusion MRI imaging. Afterwards, the patient's physical examination findings improved. Upon detection of normal findings in the imaging, the patient was evaluated as having a transischemic attack (TIA). Intravenous heparin and warfarin were started for thrombus treatment in the patient who had not previously received anticoagulant treatment. Later, when the INR values were regulated between 2.5-3.0, IV heparin treatment was discontinued. The last TTE performed before the patient was discharged determined that the thrombus had decreased in size and the left ventricular ejection fraction had increased to 45%. The patient was referred to the hematology department due to fetal death in uterus and the presence of a blood clot at the apex of the heart. Examinations confirmed that there were no coagulation problems. The patient was excluded from the diagnosis of anti-phospholipid antibodies.

He was discharged with warfarin treatment in addition to his current heart failure treatment and was called for outpatient clinic follow-up examinations. Written informed consent was obtained from the patient.

Intravenous heparin and varfarin were started for thrombus treatment in the patient who had not received anticoagulant therapy before. Later, IV heparin treatment was discontinued when INR values were regulated between 2,5-3,0. In the last TTE performed before the patient's discharge, it was determined that the thrombus size decreased and the left ventricular ejection fraction increased to 45%.

The patient was referred to the hematology department because of fetal demise within the uterus and the presence of a blood clot in the apex of the heart. The examinations confirmed the absence of a coagulation issues. Anti-phospholipid antibody diagnosis was excluded in the patient. She was discharged with varfarin treatment in addition to her current heart failure treatment and was called for outpatient clinic control examinations. Written informed consent was obtained from the patient.

DISCUSSION

Peripartum cardiomyopathy is defined as the development of left ventricular (LV) systolic dysfunction (EF < 45%) and heart failure symptoms in the last month of pregnancy or the first five months after giving birth in women with no heart disease history. Etiology has not been fully elucidated. Some studies have suggested that myocarditis due to viral infections may be among the causes of PPCM. Another possible mechanism is that it may be an autoimmune process that occurs in the maternal myocardium when fetal antigen mixes with the maternal blood (4). Risk factors of PPCM include advanced maternal age, numerous pregnancies, obesity, low socioeconomic status, cardiovascular risk factors, and preeclampsia (5). Studies have shown that the anti-angiogenic subfraction of prolactin and oxidative stress, the 16kDa prolactin, supports the treatment of PPCM, causing profound endothelial damage and subsequent cardiomyocyte dysfunction, whereas bromocriptine, a dopamine-D2-receptor agonist and blocker of prolactin release, is unable to block it. (6)The condition may deteriorate rapidly and can resolve spontaneously. In half of the patients with PPCM, left ventricular systolic function improves, but no improvement is observed in the other half.(7). Bromocriptine is used to reverse prolactin's unfavourable effects, dopamine suppresses prolactin secretion via dopamine D2 receptors. Therefore, dopamine generally has a prolactin secretion-reducing effect. Bromocriptine is a drug that works as a dopamine D2 receptor agonist and is widely used to suppress prolactin secretion (8).

Patients with PPCM are at risk of thrombus formation and thromboembolism due to both the hypercoagulable state of pregnancy and the immobility of blood due to severe left ventricular sys-

tolic dysfunction .Two-dimensional TTE is the most widely used technique for non-invasive diagnosis and follow-up of left ventricular thrombi .The presence of a genetic tendency towards thrombus, along with the enlargement of the left ventricle and weakened pumping ability of the heart, increases the likelihood of thrombus formation within the heart . A thrombus originating in the heart can migrate to the systemic circulation, leading to the development of many clinical disorders, such as; myocardial infarction, cerebral embolism, and pulmonary embolism .(5)

There is no specific guideline for the treatment of ventricular thrombus due to PPCM. Anticoagulation therapy has been reported to be effective in the majority of cases . The worsening of ventricular thrombus in the early stage of the disease weighs against the possibility of embolization with resolution of the ventricular wall movement defisit . In our case, when cerebral embolization occurred, improvement in ventricular wall movement was observed. The patient with a brain embolism was promptly taken to the angiography unit, for interventional cerebral arterectomy. It is recommended to consider thrombolytic treatment as an alternative, while carefully assessing the balance between the potential benefits and risks, if cerebral arterectomy is not available.

Different surgical thrombectomy techniques have been described in the literature, such as robotic and/or video-assisted surgery with transmitral or apical Access . However, clinical management of patients with PPCM complicated by an intraventricular thrombus remains a challenge due to a lack of evidence to inform therapeutic decision making (9).

CONCLUSION

The management of clinical treatment of cerebral embolism of left ventricular thrombus should be organized by a team consisting of cardiologists, neuroradiologists and cardiac surgeons. Echocardiographic examination is important for left ventricular thrombus in patients with PPCM. While anticoagulant treatment is effective in preventing ventricular thrombosis, it is important to consider surgical thrombectomy as an alternative option for treating left ventricular thrombus with apical morphology if anticoagulant treatment leads to adverse effects.

It is crucial for clinicians to have knowledgeable about PPCM and take it into mind to begin treatment as soon as possible for a potentially fatal condition while the diagnosis of a dyspneic woman during pregnancy or postpartum period.

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VAKA TAKDİMİ CASE REPORT

AORTIC DISSECTION PRESENTING WITH ISOLATED RIGHT ARM PAIN THE CRITICAL ROLE OF BEDSIDE ULTRASOUND IN THE EMERGENCY DEPARTMENT
İZOLE SAĞ KOL AĞRISI İLE BAŞVURAN AORT DİSEKSİYONU ACİL SERVİSTE YATAK BAŞI ULTRASONUN KRİTİK ROLÜ

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ABSTRACT

Aortic dissection (AD) is a life-threatening condition that typically presents with a sudden onset of severe chest or back pain. However, atypical presentations can lead to delays in diagnosis and an increased mortality rate. This case report discusses a 64-year-old male patient who presented to the emergency department with symptoms of distress and isolated right arm pain. Through bedside ultrasonography (USG) within the Point-of-Care Ultrasound (POCUS) protocol, a free flap was identified, prompting further investigation via CT angiography for definitive diagnosis. The patient was subsequently diagnosed with aortic dissection. This case underscores the critical role of bedside POCUS in the early diagnosis of life-threatening conditions such as AD, particularly in patients with atypical symptoms presenting to the emergency department.

Keywords: Aortic dissection, atypical presentation, bedside ultrasonography, emergency medicine, Point-of-Care Ultrasound (POCUS)

ÖZET

Aort diseksiyonu (AD), genellikle ani başlangıçlı ve şiddetli göğüs ya da sırt ağrısıyla kendini gösteren, hayatı tehdit eden bir durumdur. Ancak, atipik prezentasyonlar tanıda gecikmelere ve mortalite oranının artmasına neden olabilir. Bu sunumda, fenalaşma ve izole sağ kol ağrısı şikâyeti ile acil servise başvuran, Point of Care Ultrasound (POCUS) protokolünde yatak başı ultrasonografi (USG) ile serbest flep tespit edilmesinin ardından kesin tanı amacıyla BT anjiyografi çekilen ve aort diseksiyonu tanısı konulan 64 yaşında bir erkek hasta ele alınmaktadır. Bu vaka, atipik semptomlarla başvuran hastalarda acil serviste yatak başı POCUS'un AD gibi hayatı tehdit eden durumların erken tanısındaki kritik rolünü vurgulamaktadır.

Anahtar kelimeler: Aort diseksiyonu, atipik prezentasyon, yatak başı ultrasonografi, acil tıp, Point of Care Ultrasound (POCUS)

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GİRİŞ

Aort diseksiyonunun klasik semptomları "yırtılır tarzda" göğüs /sırt ağrısı, ekstremiteler arasında nabız ve tansiyon farklılıkları olsa da, olguların %10-30'u atipik bulgularla (izole ekstremite ağrısı, nörolojik defisit, karın ağrısı vb.) başvurabilir**(Şekil 1)**. Yatak başı USG, hızlı ve invaziv olmayan bir şekilde aort lümeni içindeki gerçek lümen ile yalancı lümeni ayıran intimal flepi saptayarak zamanında müdahaleye ve erken tanıya yardımcı olmaktadır.

TİPİK SEMPTOMLAR	ATİPİK SEMPTOMLAR
1. Ani ve Şiddetli Göğüs Ağrısı ➢ Genellikle "bıçak saplanır gibi" veya "yırtılma hissi" şeklinde tanımlanır. ➢ Hastaların %72-85'inde bildirilmiştir.	**1. İzole Kol veya Bacak Ağrısı** ➢ Subklavian veya iliak arter tutulumu ile ilişkili. ➢ Hastaların %5–15'inde bildirilmiştir.
2. Sırt Ağrısı ➢ İnterskapular alanda hissedilir ve sıklıkla göğüsten sırta doğru yayılır. ➢ Hastaların %50-60 bildirilmiştir.	**2. Nörolojik Semptomlar** ➢ İnme, parapleji veya bilinç değişiklikleri. ➢ Hastaların %5-10'unda bildirilmiştir.
3. Hipertansiyon ➢ Tip B diseksiyonlarda daha yaygın. ➢ Hastaların %49-70'inde bildirilmiştir.	**3. Karın Ağrısı** ➢ Abdominal aorta, mezenterik iskemi veya renal arter tutulumuna işaret edebilir ➢ Hastaların %20-30'unda bildirilmiştir.
4. Nabız Farklılıkları ➢ Subklavian, femoral veya karotid arter tutulumuna bağlı. ➢ Hastaların%15-30 oranında bildirilmiştir.	**4. Senkop** ➢ Perikardiyal tamponad veya serebral hipoperfüzyona bağlı ➢ Hastaların %5-13'ünde bildirilmiştir.

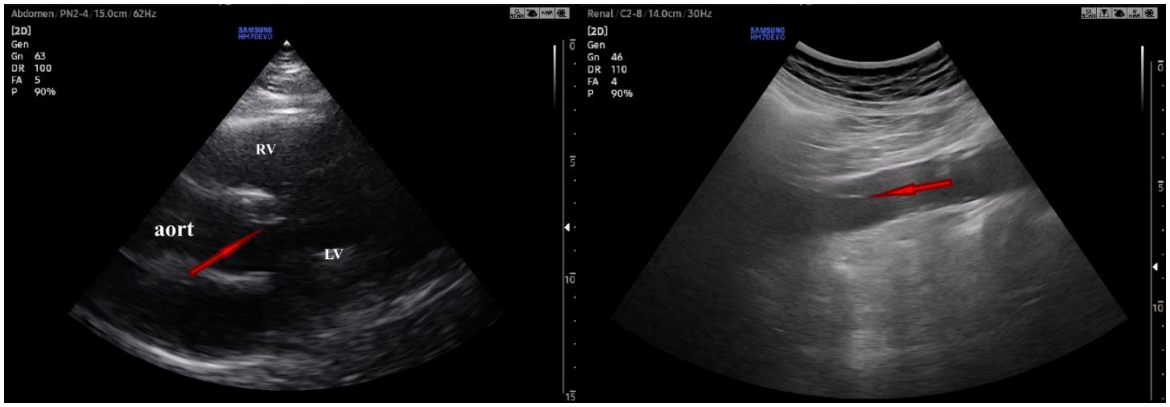
Şekil 1. Aort Diseksiyonu tipik ve atipik semptomlar

OLGU SUNUMU

64 yaşında erkek hasta, 3 saat önce aniden sağ kolunda şiddetli ağrısı olmuş ve kısa süreli fenalaşmış. İş arkadaşları ambulans çağırmış ama hasta şikâyetinin geçtiğini söyleyerek evine gitmiş. 20 dk sonra ağrısının tekrar başlaması üzerine çağırılan ambulans ekibi tarafından hasta acil servise getirildi. Hipertansiyon ve diyabetes mellitus hikâyesi olmayan hasta sağ kolda çok şiddetli ağrı tariflemekteydi. Ağrısının omuzdan koluna yayılan "keskin ve dayanılmaz bir ağrı olduğunu ifade eden hasta ağrısı başlamadan önce herhangi bir darbeye maruz kalmadığını şuan göğsünde ya da sırtında ağrı olmadığını belirtti.

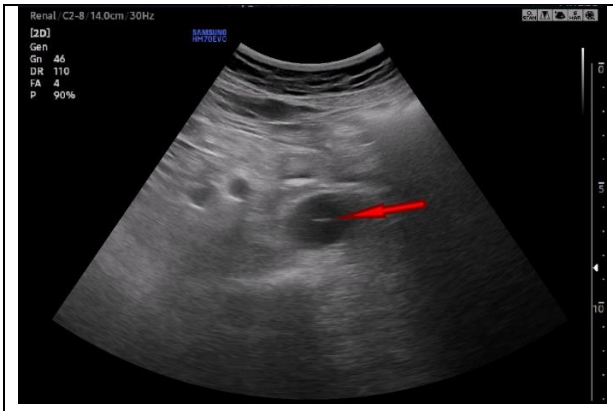
Hastanın fizik muayenesinde sol radial nabız 110/dk, tansiyon arterieyel sol koldan 170/100mmHg ölçülmesine rağmen sağ koldan tansiyon ölçülemedi, nabız alınamadı. sPO_2:98, Hastanın nörolojik ve kas-iskelet sistemi muayenesi normaldi. EKG sinüs taşikardisiydi.

Hastaya yapılan POCUS protokolünde yatak başı USG'de çıkan aortta girişinde şüpheli serbest hareketli intimal flep**(Şekil 2)** tespit edilmesi üzerine arkus aorta incelendi hasta uyumsuzluğu nedeniyle değerlendirilemedi, abdominal aorta da serbest hareketli intimal flep tespit edildi **(Şekil 3,4)**. Ayrıca sağ karotiste lümen içinde trombüs mevcuttu ve akım azalmıştı **(Şekil 5)**. Hastaya **Aort Diseksiyonu** ve **Perieferik Tutulum** şüphesi ile ivedilikle aort ve boyun BT Anjiografi çekildi.

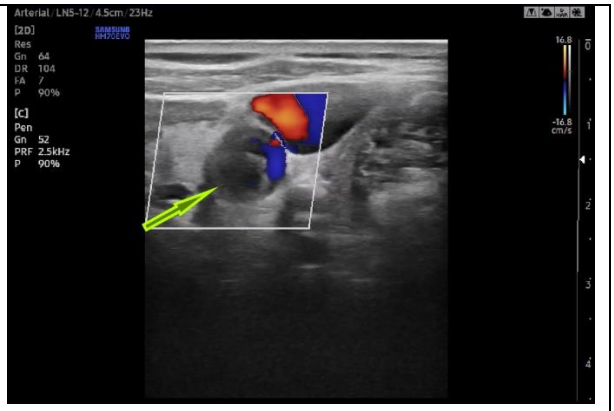


Şekil 2: Aort kökünde diseksiyon flebi(kırmızı ok)

Şekil 3: Abdominal aorta uzun akstan diseksiyon flebi(Kırmızı ok)

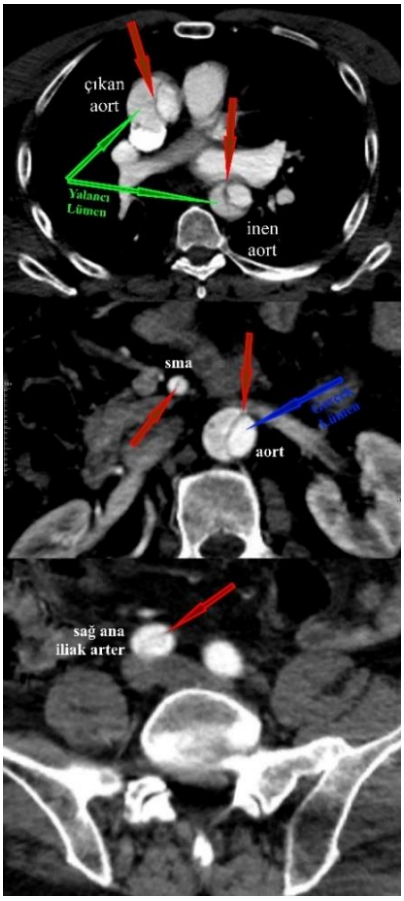


Şekil 4: Abdominal aorta kısa akstan diseksiyon flebi(Kırmızı ok)

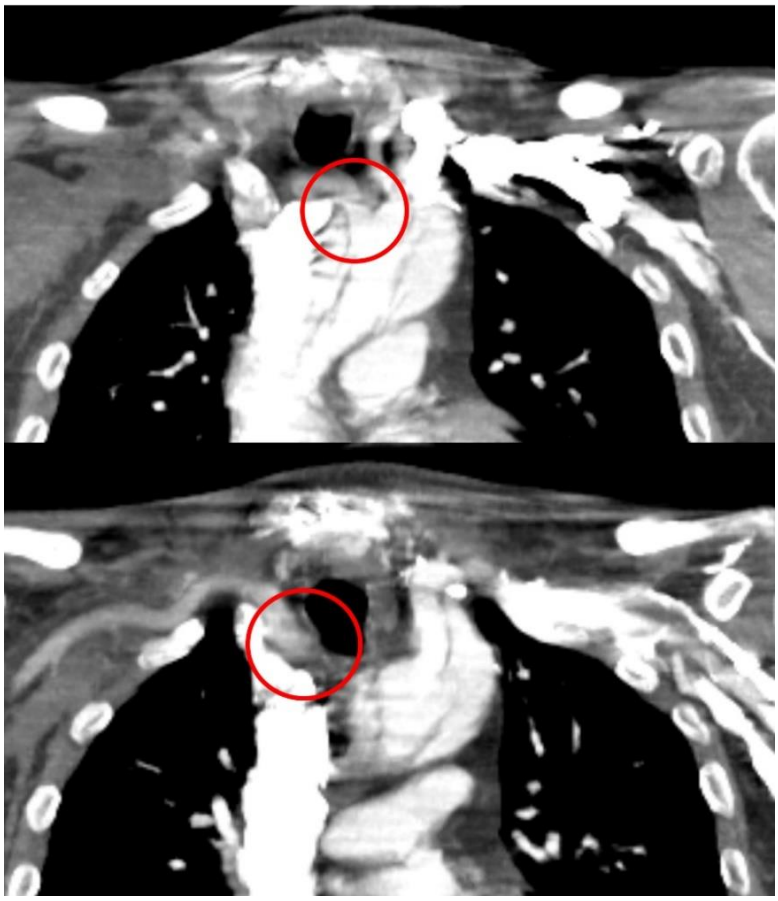


Şekil 5: Sağ karotis içinde trombüs(yeşil ok)

Yapılan BT Anjiyografide, aort kökünden sağ eksternal iliak artere kadar uzanan diseksiyon tespit edilmiştir. Bunun yanı sıra, sağ subklavian arter, sağ internal karotis arter ve süperior mezenterik arterin de diseksiyon sürecinden etkilendiği belirlenmiştir **(Şekil 6, 7)**. Stanford Tip A Aort Diseksiyonu tanısı konulan hasta, acilen kardiyovasküler cerrahi müdahalesi amacıyla bir üst merkeze sevk edilmiştir.



Şekil 6: Çıkan aorta, inen aorta, abdominal aorta, sma ve sol iliak arterde; diseksiyon filebi(Kırmızı oklar), yalancı lümen (Yeşil oklar) ve gerçek lümen (Mav ok) görünümü.



Şekil 7: Aort disksiyonu sağ subklavian arter tutulumu ve buna bağlı dolum defekti (Kırmızı daire)

TARTIŞMA

Bu vaka, iki kritik noktayı vurgulamaktadır:

Aort Diseksiyonunda Atipik Prezentasyonlar: İzole şiddetli sağ kol ağrısı, diseksiyonun sağ subklavian arteri tutmasına bağlı gelişebildiği gibi literatürde AD'nin diğer periferik tutulumlarına ve rüptürüne bağlı inme, senkop veya akut karın ile karışabileceği bildirilmiştir, bu nedenle klinik şüphe ve ayrıntılı muayene esastır.

Acil Serviste Yatak Başı Ultrasonografinin Rolü: Travma dışı kritik hastalarda POCUS protokolü kapsamında yatak başı USG ile intimal flep tespit edilmesi, tanı ve müdahale süreçlerini hızlandırmaktadır. Deneyimli hekimlerin uyguladığı yatak başı USG'nin, AD tanısında duyarlılığı %73–100 arasında değişmektedir ve özellikle nonspesifik semptomlarla başvuran hastalarda hayat kurtarıcı bir rol oynamaktadır. Ancak, yatak başı USG'nin hekim deneyimine olan bağımlılığı ve desenden aortun sınırlı görüntülenebilirliği bu yöntemin kısıtlılıkları arasında yer almaktadır. Bununla birlikte, hizmet içi eğitimlerin yaygınlaştırılması ve USG'nin acil servislerde rutin bir uygulama haline getirilmesi, hekim deneyimini artırmakta ve klinik şüphenin doğru yönlendirilmesi ile erken müdahale şansını önemli ölçüde yükseltmektedir.

SONUÇ

Aort diseksiyonu, özellikle yüksek risk taşıyan hastalarda açıklanamayan ekstremite ağrısı gibi semptomlar mevcut olduğunda ayırıcı tanıda mutlaka düşünülmelidir. Yatak başı USG, acil serviste tanısal süreci hızlandıran, erişilebilir ve etkili bir tanı aracıdır. Acil hekimleri, klasik semptomlar gözlenmese dahi, klinik şüphe durumunda aort patolojilerini değerlendirmek için yatak başı USG'yi etkin bir şekilde kullanmalıdır.

Bu olgu, atipik aort diseksiyonu (AD) prezentasyonlarında klinik şüphenin kritik önemini ve yatak başı USG'nin acil serviste erken tanı sürecindeki vazgeçilmez rolünü vurgulamaktadır.

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