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INTRODUCTION
Diabetes mellitus is a chronic disease that results from; relative or absolute insulin deficiency, insulin resistance, or both and could be classified according to the etiology of the disease (1). Genetic and environmental factors could contribute to diabetes mellitus and its complications. The diabetes mellitus prevalence in the world in 2019 is estimated to be 9.3% (463 million people), increasing to 10.2% (578 million) by 2030 and 10.9% (700 million) by 2045 (2). Genetic and environmental factors can be the cause of this chronic metabolic disorder (1). Diabetes mellitus causes acute and chronic complications, which affect many organ systems. If left untreated, as a result of long-term hyperglycemia, these complications result in morbidity and mortality. Long-term hyperglycemia is known to be related to oxidative stress and chronic inflammation (3-5). Chronic complications of diabetes mellitus are classified as vascular and non-vascular complications. Types of vascular complications are also classified as microvascular and macrovascular (1,6). Microvascular complications of this metabolic disorder are retinopathy, neuropathy, nephropathy. Macrovascular complications are coronary heart disease, peripheral arterial disease, cerebrovascular disease. In this review, we aim to discuss chronic complications of diabetes mellitus and the pathogenesis of these complications.

Pathogenesis of Diabetes Mellitus Complications
Genetic factors, molecular mechanisms, or both may lead to chronic complications of diabetes mellitus (1). Complications are related to the thought which this disease is a process of chronic inflammation and oxidative stress. Functions of endothelial capillary cells are impaired by-products of this inflammation (7). Oxidative stress disproportionates oxidant and antioxidant systems, elevated free radicals, and reactive oxygen species. Chronic hyperglycemia is the leading cause as it triggers the molecular mechanisms that cause complications; however, lipid catabolism defects and overproduction of reactive oxygen species are also causes of chronic complications (3). These molecular mechanisms fall into four main groups:

- Increased polyol pathway
- Increased hexosamine pathway
- The increased intracellular advanced glycation end product (AGE)
- Activation of protein kinase C

In addition to these molecular mechanisms, increased immune cell...
activations are thought to be one of the reasons for complications (5).

**Increased Polyol Pathway**

The polyol pathway is the cascade in which glucose is converted to sorbitol by aldose reductase, and sorbitol is converted to fructose by sorbitol dehydrogenase (3). This pathway could also be named the sorbitol-aldose pathway. This pathway is activated when glucose quantity is increased. Hyperglycemia in the blood raises aldose reductase activity; as a result of this, the conversion of glucose to sorbitol increases. Increased quantity of sorbitol in the intracellular region causes osmotic detrimet and oxidative damage (3). Henceforth, sorbitol is converted to fructose by sorbitol dehydrogenase. Accelerated activation of these enzymes decreases NADPH essential for antioxidant function in the body; therefore, oxidative damage occurs (1). In addition to decreased NADPH, increased sorbitol leads to hydration and membrane damage by generating osmotic stress. Also, fructose is a reason for glycation stress (Glicative stress).

**Increased Hexosamine Pathway**

Normal glycolysis pathway occurs in the body as long as there are normal glucose levels in the blood (3). However, if there is an increased glucose level in the blood, this normal pathway turns into the hexosamine pathway by supplying more fructose - 6 - phosphate. This alteration increases oxidative stress because of the overproduction of uridine diphosphate-N-acetyl glucosamine, known as UDP-GlcNac. N-acetylglucosamine, produced by splitting to serine and threonine, induces the factors that promote complications such as PAI-1 or TGF-β (1). Increased levels of PAI-1 and TGF-β induce fibrosis, vascular atherosclerosis, and mesangial cell injury. In addition to these mechanisms, N-acetyl glucosamine leads to elevated hydrogen peroxide levels, impairs normal glucose metabolism, and could affect gene expressions of glucose metabolism pathway genes (3).

**Increased Intracellular Advanced Glycation-end Product (AGE)**

Glucose undergoes autooxidation in the cell when it maintains chronically elevated levels (3). By this oxidation, generating of dicarbonyls is increased. Dicarbonyl species, called advanced glycation end products (AGE), are methylglyoxal, glyoxal, 3-deoxyglucosone. AGEs transmute normal metabolic functions of cells and gene expression of DNA. AGEs also lead to the alteration of extracellular matrix proteins, and by this alteration, AGEs damage cellular signaling mechanisms. These altered proteins induce the expression of IL-1, IGF-1, TNF-α, TGF-β, VCAM-1, VEGF by binding to receptors of endothelial cells and macrophages (1).

**Activation of Protein Kinase C**

Diacylglycerol induced by hyperglycemia activates the protein kinase c pathway. Activation of the protein kinase c pathway results in activation of protein kinase c isoforms such as β and δ. These isoforms increase diabetic complications by stimulating the production of VEGF, TGF-β, PAI-1, NF-κβ, MGO(methylglyoxal), NADPH oxidases (1).

All of these mechanisms stimulate increased production of superoxide, and they are causes of diabetic complications. In addition and related to these mechanisms, hyperglycemia induces overproduction of reactive oxygen species known as ROS. Some specimens of ROS are hydroxyl(•OH), hydrogen peroxide (H₂O₂), superoxide anion(O₂•⁻) and of these specimens, for oxidative radicals, superoxide anion is the primary molecule because of is convertible to other oxidative products. Reactive oxygen products are products of aerobic metabolism pathways of mitochondria, peroxisomes, endoplasmic reticulum. Oxidative injury occurs when these molecules are produced at high levels or removed in small quantities. Reactive oxygen species lead to inflammation in kidneys, pancreas, liver, endothelial cells, nerves, and eyes (4).

**Genetic and Epigenetic Factors for Diabetes Mellitus and Complications of Diabetes Mellitus**

Insulin genes settle on the short arm of chromosome 11 (1). Underlying genetic and epigenetic factors of diabetes mellitus are topics of interest and research (7). Especially Type 2 Diabetes Mellitus has an intense genetic background, but this genetic background generally does not correspond with the Mendelian pattern; also, this disease fits the heterogeneous pattern (1). Diabetes Mellitus may be monogenic or polygenic. For example, TCF7L2, a transcription factor in Wnt signaling, and variants of the gene encoding TCFL2, is related to an increased risk for diabetes mellitus. Some autosomal dominant genetic defects of pancreatic β cells are related with defects or various mutations of these genes; HNF4A, GCK, HNF1A, PDX1, HNF1B, NEUROD1, KLF11, CEL, PAX4, BLK, APPL1, GATA4, GATA6, INS, KCNJ11, ABBC8. Some autosomal recessive genetic defects of pancreatic β cells are related with defects or various mutations of these genes; GCK, PDX1, PTF1A, NEUROG3, RFX6, GLIS3, NKX2-2, MNX1, EIF2AK3, IER3IP1, WFS1, SLC19A2 (1). In addition to these genetic factors, micro RNA abnormalities may be responsible for the diabetes mellitus process. MicroRNA, known as miRNA, are the molecules that affect their target genes in a post-transcriptional stage (7). In diabetes mellitus duration, they lead to endothelial dysfunction, impaired lipid metabolism, and inflammation process. In the future, diagnostic studies and treatment modalities for diabetes, and complications of diabetes, would be more beneficial with the increase of research and knowledge about the genetic background of diabetes mellitus.

Chronic Complications of Diabetes Mellitus Complications of diabetes can be divided into acute and chronic. In this review, our focus was chronic microvascular complications of diabetes. Diabetes leads to complications in vascular tissue, nerves, lens, skin. These complications are divided into two main groups: microvascular and macrovascular (1). These complications are related to increased morbidity and mortality, decreased quality of life, extremity loss, vision loss, organ failure, or loss. Because of
diabetes, personal and social life worsening results, diabetes and its complications are significant.

**Microvascular Complications**

Microvascular complications are retinopathy, nephropathy, neuropathy and occur by capillary basement membrane injury, small vessel disease, defect of vascular permeability, high water retention, injury of angiogenic cells, generalized edema, impairment of tissue neogenesis. Microvascular complications of diabetes are related to decreasing or loss of visual functions, kidney failure, sexual dysfunction, and peripheral neuropathy.

**Diabetic Retinopathy**

Diabetic patients, especially ones who have been diabetic for more than five years, should be referred to an ophthalmologist in their follow-up process (6). There can be macular edema in every diabetic patient, but diabetic retinopathy divides into two main categories: non-proliferative and proliferative retinopathy (6). By activation of the protein kinase c pathway, diabetic vascular complications increase because of elevated cellular permeability (3). In addition to this mechanism, hyperglycemia and oxidative stress accelerate diabetic retinopathy.

**Diabetic Nephropathy**

Because of diabetes mellitus, diabetic nephropathy is responsible for 4000 cases of end-stage renal disease in the United States, and renal failure occurs in one-third of all patients (1). The initial clinical finding of diabetic nephropathy is proteinuria, and albumin excretion could be the first sign for diabetic nephropathy (6). Nodular intercapillary glomerulosclerosis, known as Kimmelstiel-Wilson lesions, is less common than diffuse glomerulosclerosis (1).

In diabetic patients, glomerular endothelial cell barrier and endothelial basement membrane injury occur because of oxidative stress; therefore, protein filtration increases. Angiotensin gene expression is affected by oxidative stress (3). AGEs lead to stimulation of cytokines; by this mechanism, diabetic nephropathy could worsen.

**Diabetic Neuropathy**

Peripheral and autonomic neuropathies occur in chronic hyperglycemia and manifest as gastroparesis, incontinence, sexual dysfunction, and distal symmetric polyneuropathy. In autonomic neuropathy, digestive systems functions are impaired; therefore, it manifests as nausea, vomiting, abdominal pain, heartburn, slowed stomach emptying, constipation, diarrhea, and gastrointestinal tract infections. Because neuropathy leads to loss of sensation, diabetic foot disease occurs in peripheral diabetic neuropathy. Chronic hyperglycemia in the blood leads to an activated polyol pathway, and therefore as addressed before, this pathway diminishes NADPH. In addition to these mechanisms, the sorbitol pathway is also responsible for nerve cell damage (3). Increased free radicals worsen erectile dysfunction and impair ejaculation.

**Macrovascular Complications**

Macrovascular complications are coronary arterial diseases and peripheral arterial diseases; complications manifest as myocardial infarction, stroke, peripheral vessel disease; and they occur by extensive vessel injury, accelerated atherosclerosis, and impaired lipid metabolism. This complication occurs by over-production of ROS, vasoconstriction, and impairment of lipid metabolism. In diabetic patients, microangiopathy occurs and leads to cardiomyopathies.

**Other Complications of Diabetes Mellitus**

Skin pathologies occur in diabetes because of poor glycemic control and manifest as candidal infections, vulvovaginitis, necrobiosis lipoidica diabeticorum. These are complications caused by increased glucose delivery to the skin and dehydration of the skin.

Bone and joint complications occur by over-production of glycation-end products and manifest as Dupuytren contractures, Carpal tunnel syndrome, diabetic cheiroarthropathy (Diabetic stiff hand syndrome or limited joint mobility syndrome), diffuse idiopathic skeletal hyperostosis, bursitis, decreased bone mineral density (1).

**Conflict of interests**

The authors declare that they have no competing interests.

**Financial Disclosure**

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**REFERENCES**

INTRODUCTION

Familial Mediterranean Fever (FMF) primarily affects a population that originates from around the Mediterranean Sea and is characterized by peritonitis, pleuritis, arthritis, or erysipelas-like skin disease accompanied with recurrent episodes (attacks) of fever (1). Diagnosis of FMF is made based on clinical symptoms, taking into account the physical examination and laboratory findings with a careful anamnesis and the support of ethnicity and family history (2). The cause of abdominal pain seen in Familial Mediterranean Fever is inflammation in the peritoneum; therefore, patients may have peritonitis signs, and with these findings, the disease may be confused with other acute abdominal conditions.

CASE REPORT

A 21-years old male patient with known thalassemia minor and with no other chronic diseases, was admitted to our hospital with abdominal pain and fever complaints. The patient stated that the abdominal pain started the day before and gradually increased; it was not belt-like, did not hit the back, and did not change with movement. The patient did not describe nausea, vomiting, diarrhea, or constipation. The patient had no history of gallstones, alcohol use, medical drug use, and herbal or synthetic substance use. On physical examination, the patient's vitals were stable, conscious, oriented, and cooperative. In his abdominal examination, there was tenderness on superficial palpation in the epigastric region of the abdomen and tenderness on deep palpation in all quadrants of the abdomen, there was no palpable mass in the abdominal examination, and there was no guarding or rebound, palpable hepatomegaly and splenomegaly were not detected. There was no pathology on the respiratory system, cardiovascular system, neurological and musculoskeletal system. No rash and no palpable lymphadenopathy were detected on the patient’s body.

The blood tests results of the patient were as follows; (WBC): 14.000/ml, neutrophils: 9700/ml, lymphocytes: 2000/ml, monocytes: 2000/ml, erythrocyte count (RBC): 6.39 million/μL, hemoglobin: 11.8 g/dL, hematocrit: %36, MCV (mean cell volume): 56 fL, c-reactive protein (CRP): 117 mg/L, amylase: 290 U/L, lipase: 255 U/L, glucose: 106 mg/L, urea: 19 mg/L, creatinine: 0.86 mg/dL, ALT (Alanine Aminotransferase): 14 U/L, AST (Aspartate Aminotransferase): 21 U/L, LDH (Lactate Dehydrogenase): 236 U/L. In Complete Urinalysis hemoglobin: (1+), leukocyte esterase (-), nitrite (-), protein (-), ketone (-), were detected. Acute pancreatitis was considered in the preliminary diagnosis of the patient who presented with...
epigastric abdominal pain, and had elevated amylase and lipase levels. However, the diagnosis of acute pancreatitis was later dismissed because the patient's abdominal pain was not typical, and there were no findings in favor of pancreatitis or other abdominal pathologies in the Abdominal Computed Tomography (CT) imaging. Blood cultures and urine cultures were obtained in terms of a possible infection focus that may be present in the patient, and ceftriaxone antibiotic therapy was started empirically. A detailed physical examination of the patient has performed again, but no focus was observed to indicate infection. No infiltration was also detected in the thorax CT imaging.

Leukocyte esterase and other infection markers were found to be negative when the complete urinalysis was repeated. Infection disease department's consultation about the patient was also taken, and HIV (Human Immunodeficiency Virus), Hepatitis, CMV (Cytomegalovirus), Herpes, Rubella, EBV (Epstein Barre Virus), Mumps and Brucella tests were sent from the patient. Peripheral smear test of the patient was examined, erythrocytes were hypochromic microcytic, abundant rod cells were observed, platelets were sufficient, no atypical cells were observed. When the patient was questioned again in detail on the second day of his hospitalization, it was learned that the patient had similar complaints before, he had attacks in the form of abdominal pain and fever, and these attacks lasted for a few days. When the patient's family history was questioned, it was learned that his cousin had similar complaints. Thereupon, the diagnosis of Familial Mediterranean Fever (FMF) was considered in the patient. In addition to previous tests, ESR (Erythrocyte sedimentation rate) and fibrinogen tests were requested from the patient: ESR 29 mm/hr and fibrinogen 659 mg/dL. Thus, the patient were diagnosed with FMF regarding his abdominal pain, fever of 39 degrees, elevated white blood cell count, elevations in sedimentation, CRP, and fibrinogen levels and considering his abdominal pain and fever histories in the form of attacks, and colchicine treatment has started. After colchicine treatment, the patient's complaints regressed, and the inflammatory parameters returned to normal. Blood and urine cultures remained negative in the follow-up. Viral tests were negative, and brucella was negative, Eliza panel was negative. In the abdominal USG of the patient, bilateral kidney size, parenchymal thickness, and parenchymal echogenicity were reported as normal. In addition, FMF/MEFV gene target region/mutation analysis was requested from the patient for genetic counseling. The patient, whose complaints regressed and laboratory findings returned to normal, was discharged and called for a follow-up due to colchicine treatment has started. After colchicine treatment, the patient's pain was questioned in detail, it was not in the form of typical pancreatitis pain. Also, despite the patient's blood tests showed elevated amylase and lipase levels, the amylase and lipase elevations were not more than three times the reference upper limit, and no finding in favor of pancreatitis was reported in the abdominal CT (computed tomography) imaging. So, in the patient who was thought to have acute pancreatitis in the initial evaluation, the diagnosis of acute pancreatitis was dismissed.

Familial Mediterranean Fever (FMF) is an inherent disease characterized by signs of serosal inflammation such as abdominal pain, pleuritis, arthritis, and erysipelas-like skin lesions accompanied by recurrent episodes of fever (attacks). Familial Mediterranean fever (FMF) is generally considered an autosomal recessive disease, and affected individuals have biallelic pathogenic mutations in the MEFV gene located on the short arm of chromosome 16 (5,6). The disease is an inherited disease that usually occurs in people of Mediterranean descent, but it can affect people of any ethnic group (7). FMF diagnosis is made by careful anamnesis, clinical findings, family history, biochemical data, response to treatment, and exclusion of other familial periodic fever syndromes and supported by family history and ethnicity. The genetic tests for FMF are used to support diagnosis and consultation for family members in a patient who has clinic criteria of FMF (2).

The diagnosis of familial Mediterranean fever (FMF) is based on Tel-Hashomer clinical criteria, and the diagnosis of FMF is made in a patient with typical attacks using a combination of the following criteria: ≥1 major criterion or ≥2 minor criteria or 1 minor plus 5 supporting criteria or 1 minor criterion plus ≥4 of the first 5 supporting criteria (8).

Typical attacks are defined as episodes of pain associated with serositis, the presence of recurrent episodes (≥3 of the same type), the presence of fever (rectally measured fever of 38°C or higher), and short duration (12 hours to 3 days). Solitary episodes of fever can be considered a typical episode if they are recurrent, short-lived, and have no other definable cause (8).The major criteria used in the clinical diagnosis of FMF are peritonitis (generalized), pleuritis (unilateral) or pericarditis, monoaorthritis (hip, knee, ankle), or fever alone, accompanying a typical attack. The minor criteria used in the clinical diagnosis of FMF are abdominal pain, chest pain, monoarthritis, exercise-related leg pain, and favorable response to colchicine. The supportive criteria used in the clinical diagnosis of FMF are family history of FMF, appropriate ethnic origin, age <20 years at onset of disease, an attack that is severe and requiring bed rest, attack's spontaneous remission, the symptom-free interval between
attacks, increase in inflammatory markers such as white blood cell count, sedimentation, fibrinogen, serum amyloid A within attacks, episodic proteinuria/hematuria, history of negative laparotomy or a removal of the normal appendix, a history of consanguineous marriage (8).

In the case that we presented, our patient's attacks were typical. In addition, 1 major criterion was; peritonitis findings in our patient, 1 minor criterion was; favorable response to colchicine, 6 supportive criteria were; family history of FMF, Mediterranean origin, an attack that required bed rest, attack's spontaneous remission, the symptom-free interval between attacks and increase in inflammatory markers within attacks, Thus, FMF was diagnosed according to Tel-Hashomer clinical criteria.

As a result, the patient who applied with the complaint of epigastric abdominal pain and had high levels of amylase and lipase got diagnosed with FMF after deepening the patient's anamnesis and further examinations, although it was thought to might be acute pancreatitis in the first evaluation. In this case report, we wanted to draw attention to the fact that FMF can mimic other acute abdominal conditions such as acute pancreatitis clinically and as per laboratorial results, so the diagnosis of FMF should be kept in mind in young patients who presents with abdominal pain and do not have a clear etiology after the initial clinical, laboratory and radiological evaluations.

**Conflict of interests**
The authors declare that they have no competing interests.

**Financial Disclosure**
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**REFERENCES**

INTRODUCTION

Normal pericardium surrounds the pericardial cavity and contains an ultrafiltrate plasma. Usually, pericardial effusion is mentioned when this fluid exceeds the required amount of 15-50 cc (1). The cause of pericardial effusion and disease varies depending on the epidemiology and patient population. Most of the cases, which are difficult to diagnose and treat, are considered idiopathic. At this stage, the definitive diagnosis is significant in terms of the prognosis of the patients.

Pericardial effusions can occur acute, subacute, or chronic. The normal pericardium may stretch to accommodate volume increases, depending on how rapidly the effusion develops. Slow-growing effusions are more flexible than rapidly growing effusions. Pericardial effusion and diseases may occur due to an isolated disease affecting the heart or systemic disease manifestation. Differential diagnosis of the disease and multidisciplinary, contemporary treatment approaches are essential to reveal treatable causes. Effusion can be detected incidentally by diagnostic procedures in symptomatic patients or by echocardiography in asymptomatic patients. The first thing to do after the effusion is detected; is to determine the amount of effusion, evaluate the patient's hemodynamic status, and determine the associated diseases that may lead to this.

CASE REPORT

A 66-year-old female patient with a known Essential Hypertension had admitted to our clinic with complaints of fatigue, constant sleepiness, headaches, and chest pains that last for about a week. The patient stated that the chest pain was intermittent, increased significantly in the lying position, was a needle-prickling style, and decreased when sitting. She stated that her headache complaint showed no specificity and continued intermittently. She had a history of cholecystectomy 30 years ago, no history of smoking, alcohol, or herbal medicine use, and she had a history of 4 mg Benidipine and 50 mg Losartan potassium antihypertensive drug use for hypertension. There was no acute pathological finding in the systemic physical examination of the patient. Biochemical test results were as normocytic anemia, acute renal failure, increased sedimentation, and in the complete urinalysis, pyuria was found [Table 1]. Patient consent was obtained to share the biochemistry and imaging results.

A posteroanterior chest X-ray was performed on the patient who complained of chest pain. Pleural effusion and cardiothoracic index were increased in the imaging of the patient [Figure 1].
Figure 1. Posteroanterior chest radiograph of the case

Due to the current pandemic period (COVID-19), scanning with thorax computed tomography was planned for the patient whose clinical status and laboratory results were evaluated and whose infection parameters were found to be high. Echocardiographic evaluation was planned for the patient who had pleural effusion in the bilateral hemithorax and effusions in the pericardial area, whose possible covid-19 pneumonia diagnosis was excluded in the computed tomography scan and who had no known cardiac disease or heart failure diagnosis [Figure 2]. Ertapenem 1 gram/day parenteral antibiotic therapy was started for the patient who had pyuria and high infection parameters in the complete urinalysis. In the echocardiographic evaluation of the patient, pericardial effusion and fibrin bands of 2 cm were detected in the deepest part. Pulmonary arterial pressure was measured as 35-45 mmHg. Oral treatment of 2*0.5 mg colchicine was started in the patient with pericardial effusion. Non-steroidal anti-inflammatory treatment was not started in the patient because of acute renal failure. It was planned to evaluate rheumatological autoantibodies for connective tissue diseases that may cause pericardial effusion. It was planned to investigate the possible causes and determine the etiology of the patient, whose pleural and pericardial effusions to explain chest pain complaints.

Due to the development of acute respiratory distress during the patient's follow-up in the service, control echocardiography was planned with the preliminary diagnosis of cardiac tamponade. It was observed that there was no increase in the amount of pericardial effusion, and there was no cardiac tamponade. Pulmonary CT angiography scanning was planned for the preliminary diagnosis of an acute pulmonary embolism due to evaluated arterial blood gas results; hypoxia, hypocarbia, and respiratory alkalosis. In the computerized tomography imaging of the patient, the preliminary diagnosis of pulmonary embolism was excluded, and the amount of pleural effusion was found to be increased. Parenteral diuretic therapy and parenteral steroid therapy at a dose of 1 mg/kg were started in the patient for acute pulmonary edema. After the current treatment was administered for three consecutive days, treatment continued with oral maintenance dose steroid. With these treatments, it was observed that the patient's complaints regressed, there was a significant improvement in her clinical condition. During the follow-up, the patient was scheduled for diagnostic pericardiocentesis with control echocardiography four days later. After diuretic, steroid, and colchicine treatments, the patient's pleural and pericardial effusions regressed significantly. However, due to insufficient effusion diagnostic, interventional procedures could not be performed.

Pre-diagnosis of malignant neoplasm of the breast was excluded with breast ultrasonography and mammography scanning of the female patient, who was over 50 years old. No thoracoabdominal mass or gynecological pathology was detected in the thoracic and abdominal scanning of the patient who had no smoking history. Rheumatological parameters evaluated for connective tissue diseases were negative. The preliminary lymphoma diagnosis was excluded in the patient who did not have symptoms of fever, sweating, weight loss, pathological lymphadenopathy in the systemic physical examination and superficial tissue ultrasonography, and atypical cells did not detect in the peripheral smear evaluation. Serum Adenosine deaminase enzyme level results were average in the patient. The patient had no previous tuberculosis, history of contact, and had no cavitary lesion or fibrosis in lung scanning. The submitted Quantiferon test was indeterminate, and the test was repeated. The pre-diagnosis of tuberculosis infection was excluded in the patient whose test result was negative. All pre-diagnoses reviewed in the differential diagnosis were excluded, C-reactive protein and sediment levels in the control blood biochemistry, active chest pain, and effusions regressed. The patient was evaluated as an idiopathic pericardial effusion case and was discharged with the recommendations for outpatient control.

<table>
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pleural effusion with pericardial effusion, we also supported our clinical suspicion of pericardial effusion. In our case, who had needed in non-diagnostic echocardiographic findings with a high computed tomography or magnetic resonance imaging may be evaluated as idiopathic. Additional imaging modalities such as algorithmic approaches until the last stage, and the case was treatable causes. In our case, differential diagnoses were made in standardized practice guidelines. Thus, it aimed to reduce the number of idiopathic cases and identify treatable causes. In our case, differential diagnoses were made with algorithmic approaches until the last stage, and the case was evaluated as idiopathic. Additional imaging modalities such as computed tomography or magnetic resonance imaging may be needed in non-diagnostic echocardiographic findings with a high clinical suspicion of pericardial effusion. In our case, who had pleural effusion with pericardial effusion, we also supported our diagnosis with thoracic computed tomography imaging. After detecting pericardial effusion, the first thing to do is to evaluate the hemodynamic status of the patient. The clinical situation varies according to the amount of pericardial fluid and the rate of accumulation. If pericardial fluid accumulates rapidly, it can cause cardiac tamponade within minutes. If it accumulates slowly, it can reach large amounts without causing signs and symptoms (5,6). Classic symptoms include orthopnea, dyspnea after exertion, and chest pain. Additional symptoms due to local compression; nausea (diaphragm), dysphagia (esophagus), hoarseness (laryngeal nerve), and hiccups (phrenic nerve) may develop. Nonspecific symptoms include cough, weakness, fatigue, anorexia, and palpitations. Hypotension and sinus tachycardia indicates the compressive effect of pericardial fluid on adjacent anatomical structures. Our case also had complaints of fatigue and chest pain at the time of admission. A patient's fever is a nonspecific sign that may be associated with infectious or immune-mediated pericarditis (7-9).

The development of cardiac tamponade without inflammatory manifestations is associated with a higher risk of neoplastic etiology, while cardiac tamponade and a severe effusion without inflammatory manifestations are usually associated with a chronic idiopathic etiology (10,11).

Pericardial effusion management aims to treat the underlying disease. In approximately 60% of cases, the effusion is associated with a known disease. When the cases with pericardial effusion are symptomatic, and when medical treatments fail, patients with impaired hemodynamic stabilization should be considered for surgical drainage of effusion (2,8,12). In idiopathic chronic effusions, non-steroidal anti-inflammatory drugs, colchicine, and corticosteroids can be administered, but there is no proven study on these treatments (13-15). Invasive treatment options should be considered in large amounts of effusions unresponsive to medical treatment.

CONCLUSION

Pericardial effusion prognosis is related to the etiology of the disease. In the studies performed, infectious and idiopathic cases were found to have a good prognosis (16). Although the etiology of most cases is unknown, etiologies can be revealed with long-term follow-up. At this point, the important thing is to continue the follow-up of the cases that we consider as idiopathic, taking into account the hemodynamic status of the patient, the amount of effusion, and the rate of development, in order not to miss the treatable causes of pericardial effusion. In the long-term follow-ups, it has been revealed that the cases may have tuberculosis infection or malignancy (10). However, the optimal follow-up period has not been defined for the cases. In a study conducted by Imazio et al. in three Italian centers between 2000 and 2015, it was observed that cardiac tamponade could develop in a small number of cases, while the amount of effusion regressed in some (11). Therefore, it should be kept in mind that asymptomatic patients may experience deterioration in their hemodynamic
status during their follow-up. Our pericardial effusion case with idiopathic etiology benefited from oral maintenance dose steroid and colchicine treatments; the amount of effusion regressed and was followed up for a long time.

Conflict of interests
The authors declare that they have no competing interests.

Financial Disclosure
All authors declare no financial support.

Informed Consent
Written consent was obtained from the patient and his parents.

REFERENCES
A Case report: A Case of acute pancreatitis due to external compression of diffuse large B-CELL lymphoma mass to the pancreas

INTRODUCTION

Acute pancreatitis (AP); It is a clinical issue characterized by damage caused by enzymes in the acinar cells of the exocrine pancreas due to a pathological cause and development of local and systemic inflammation secondary to damage. Gallstones and alcohol are the most common causes of acute pancreatitis in our country and around the world. In addition; many conditions such as drugs and toxins, some viral or bacterial infections, metabolic conditions such as hypertriglyceridemia/hypercalcemia, endoscopic retrograde cholangiopancreatography (ERCP) procedure, abdominal traumas, peripancreatic or extra pancreatic malignancies, autoimmunity, genetic/hereditary causes can also cause acute pancreatitis. Diffuse large B-cell lymphoma (DLBCL) is the most common histological subtype of non-Hodgkin lymphoma (NHL). Patients with DLBCL typically present with a rapidly growing symptomatic mass. The mass is usually a nodal growth in the neck or abdomen or in the mediastinum in the case of primary mediastinal large B-cell lymphoma. However, the mass lesion due to DLBCL can be found anywhere in the body.

CASE REPORT

An 85 years old male patient who is diagnosed with hypertension and chronic kidney disease was admitted because of oral intake disorder (decreased oral intake) and pain; he stated that the pain started a week ago and happened after eating; it spreads to the back in a belt-like manner from below the sternum, the pain happens after eating. In the examinations of the patient in the emergency room, the first results followed were as: amylase: 782 u/l, lipase: 1054 u/l, urea: 163 mg/dl, creatinine: 4.83 mg/dl (the patient's baseline creatinine value was 3 mg/dl). With typical abdominal pain and pancreatic enzyme elevations, the patient was hospitalized with the diagnosis of acute pancreatitis and pre-renal acute renal failure on the basis of chronic kidney disease. The patient had no history of gallstones, alcohol use, viral and bacterial infection, trauma, previous pancreatitis attack. It was learned that chronic kidney disease in the patient developed due to polycystic kidney disease and had been present for a long time. On physical examination of the patient, results were; blood pressure 140/80 mm/hg, heart rate: 82/min, fever: 36.0 °C and respiratory rate was 14/min. The patient was conscious,
oriented and cooperative. The examination of the respiratory system and cardiovascular system was normal. In his abdominal examination, there was tenderness on deep palpation in the right upper quadrant of the abdomen and epigastric region; there was no defense and rebound, hepatomegaly and splenomegaly were not detected. Neurological examination was normal. In the patient's physical examination, there was no palpable lymphadenopathy in cervical, axillary, inguinal regions. The patient had urine output. In the patient's blood test, results were; hemoglobin: 9.7 g/dl, hematocrit: 30.8%, MCV (mean erythrocyte volume): 91 fL, platelets: 380,000, urea: 163 mg/dl, creatinine: 4.83 mg/dl, Na: 141 mmol/L, K: 5.6 mmol/L, sedimentation: 52 mm/h, CRP (C-reactive protein): 1.0 mg/L, ALT (Alanine aminotransferase): 8 U/L, AST (Aspartate Aminotransferase): 17, LDH (lactate dehydrogenase): 533 U/L, albumin: 30.6 g/L, calcium: 8.8 mg/dl, triglyceride: 102 mg/dl, pH in arterial blood gas: 7.28, pCO2: 31 HCO3: 15.8, lactate: 11 mg/dl.

In the patient's abdominal ultrasonography (USG); An anechoic cyst of approximately 15 mm in diameter in the left lobe of the liver, and a mass lesion of approximately 5x5 cm in diameter in the hepatic hilum, with hyperechoic lobulated contours, compressing the vena cava were observed. The thickness of the gallbladder wall increased diffusely and bile sludge and millimetric stones were observed in the lumen of the gallbladder, but the common bile duct was in normal width and the pancreas was also normal. Spleen sizes were natural. In the abdominal computed tomography (CT) imaging of the patient, a 2 cm hypodense lesion was observed in the left lobe of the liver (Figure 1, Figure 2).

Thereupon, interventional radiology was consulted for biopsy of the detected mass in the patient, and dynamic-diffusion magnetic resonance imaging (MRI) of the abdomen was performed for detailed evaluation before the biopsy procedure. In MR imaging, the liver was in an average size and its contours showed an undulating course. A mass lesion that is located adjacent to the liver at the level of segment 4B in the left lobe of the liver was observed in the medial part, which slightly pushed the celiac trunk to the left lateral, enveloping the inferior vena cava anteriorly, left laterally and posteriorly, and without any interphase with the liver parenchyma. The defined mass lesion reached the dimensions of approximately 7.2x5 cm in the axial plane, and the long axis of the lesion reached 8 cm. Although the intra/extra-axial distinction was made well for the defined lesion, the findings suggested that it was primarily located extra axially. In dynamic series, when contrast agent was applied, intense homogeneous contrast enhancement was observed in the lesion, which was considered to be conglomerated lymphadenopathy, significant diffusion restriction in favor of malignancy was observed. Although the pancreas was slightly pushed to the left, no focal lesions were observed in the pancreatic head, body and tail parenchyma (Figure 3).

In the light of current imaging studies, a thick needle biopsy (trucut biopsy) was performed from the mass detected in the patient. The biopsy result was reported as non-Hodgkin lymphoma consistent with diffuse large B cell. As a result; The patient, who was admitted to the internal medicine service with the diagnosis of acute pancreatitis, was diagnosed with diffuse large B-cell lymphoma tissue by biopsy from the mass detected in his imaging, and the patient was referred to the oncology clinic.
DISCUSSION

Acute pancreatitis (AP) is a clinical issue that can have many different etiologies and progresses with local and/or systemic symptoms and signs due to a pathological cause and local and systemic inflammation develops secondary to this. Patients with AP typically present with middle epigastric and/or right upper quadrant pain; The character of the pain is generally continuous, knife-like and spreading to the back or sides. The diagnosis of AP is made by the presence of at least two of the following criteria: stereotypical abdominal pain, serum amylase and/or lipase values greater than three times the upper limit of normal, characteristic findings on abdominal imaging (1). Although there were no signs of pancreatic inflammation in our patient's imaging, he was diagnosed with AP because he had typical abdominal pain and amylase and lipase elevations more than three times the upper limit of normal.

In addition to the diagnosis of AP, the patient with pre-renal acute renal failure, which had elevated urea-creatinine and developed on the basis of chronic kidney disease, was treated and followed up. During the patient's follow-up, his clinic improved, amylase and lipase levels decreased, and baseline values of urea creatinine levels decreased.

Gallstones and alcohol are the most common causes of acute pancreatitis in our country and the world. Therefore, transabdominal ultrasonography (TAUS) and alcohol use history should be questioned in all patients with AP (2,3). When the patient in our case was questioned, there was no history of gallstones and alcohol. Although the thickness of the gallbladder wall increased diffusely and bile sludge and millimetric stones were observed in the lumen of the gallbladder in the transabdominal USG of our patient, due to the average width of the common bile duct, the normal appearance of the pancreas and the mass described in the liver hilum, gallstones were not considered in the foreground in the etiology, and the mass described in the patient became the primary aspect for diagnosis. Although rare, a pancreatic tumor or cystic neoplasm should be considered as a cause of AP in patients over 40 years of age without an obvious etiology (4,5). Because the patient in our case was of advanced age and normochromic normocytic anemia and increased sedimentation were found in blood tests, it was thought that the mass in the patient's scanning that pushed the pancreas slightly to the left might be malignant.

Diffuse large B-cell lymphoma (DLBCL) is the most common histological subtype of non-Hodgkin lymphoma (NHL), accounting for approximately 25% of NHL cases (6,7). Patients with DLBCL typically present with a rapidly growing symptomatic mass. The mass is usually a nodal growth in the neck or abdomen, or in the mediastinum in the case of primary mediastinal large B-cell lymphoma. However, the mass lesion due to DLBCL can be found anywhere in the body.

The fact that the mass detected in our patient had an image similar to conglomerate lymphadenopathy and no focal lesion was observed in the pancreatic trunk and tail parenchyma, suggested that the mass may not be a malignancy of pancreatic or liver origin but may be an NHL presenting with lymph node enlargement in the abdominal region. For tissue diagnosis of DLBCL, an excisional or incisional biopsy of lymph nodes, trucut biopsy, or fine-needle aspiration (FNA) can be performed. Excisional biopsy should always be preferred to trucut biopsy for diagnosis (8). Although we suspected a possible lymphoma when the biopsy was planned to make a definitive diagnosis in our patient, trucut biopsy sampling was performed from the mass, as the morphology and localization of the mass in the patient were not suitable for excisional biopsy. The biopsy result of the patient was reported as non-Hodgkin lymphoma consistent with diffuse large B cell.

As a result; In our case, an 86-year-old male patient who presented with epigastric abdominal pain was hospitalized with the diagnosis of acute pancreatitis and pre-renal acute renal failure, and he was diagnosed with diffuse large B-cell lymphoma after a biopsy from the mass that found in the imaging studies. It was considered that acute pancreatitis developed due to the compression of the pancreas and local irritation caused by the mass. In this case report, we wanted to draw attention to the fact that acute pancreatitis should be kept in mind, which may develop due to a malignancy itself or compression of the pancreas, in advanced age patients, whose medical history and laboratory examinations do not have a clear cause that may lead to acute pancreatitis.

Conflict of interests
The authors declare that they have no competing interests.

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Informed Consent
Written consent was obtained from the patient and his parents.

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INTRODUCTION

Traumatic bone cysts are non-epithelial cavities of the jawbones. The absence of true epithelium in the cavity prevents it from being identified as a true cyst (1).

It has been referred to by many names in the literature, including solitary bone cyst (2), hemorrhagic bone cyst (3), extravasation cyst (4), progressive bone cavity (5), and simple bone cyst (6). Many nomenclature efforts show that the etiology of the defect is still not totally understood. The definition of traumatic bone cyst is currently the most valid definition for this lesion (7). It is usually seen in the second decade (3,8-10). The most common site is the mandible corpus region. The second most common site is the mandible symphysis region. It has also been reported to occur in the mandible ramus, condyle, and maxilla anterior segment. It is mostly noticed during routine radiological examination (8,10,11). Pain is not often seen. Less common symptoms are tooth sensitivity, fistulas, paresthesia, delayed eruption, inferior alveolar canal displacement, and pathological mandible fracture.

Expansion of the lesion towards the buccal cortical bone may cause intraoral and extraoral swelling. Adjacent teeth are generally vital, not displaced, and resorption is not seen in the roots of adjacent teeth (3,5,9,12,13).

The absence of histological material in the cavity prevents the histological diagnosis (13,14). Generally, it is recommended to open the cavity surgically and curette all the walls in the treatment. Thus, the clot formed in the cavity as a result of bleeding results in bone formation in the progressive process (15,16).

CASE REPORT

A 14-year-old female patient was referred to Hatay Mustafa Kemal University, Faculty of Dentistry, Department of Maxillofacial Surgery due to a lesion that was noticed as a result of a routine dental examination. A unilocular radiolucent lesion of 5-6 cm in diameter was observed in the right mandible ramus, condyle, and corpus regions. Finding the lesion around the existing impacted wisdom tooth (third molar) suggested the possibility of a dentigerous cyst, keratocystic odontogenic tumor, or unicystic ameloblastoma. However, there was no inferior alveolar canal displacement in this case [Figure 1].

Primarily, marsupialization treatment was considered to prevent possible pathological bone fracture or inferior alveolar nerve damage that may occur during the operation. For this purpose, no cyst epithelium or pathological contents were observed after a surgical incision was made under local anesthesia. Considering that it was a traumatic bone cyst, the cavity walls were curetted and bled as much as possible. The cavity was then sutured to
leave it open.

The patient was instructed to clean the cavity by irrigating with serum physiological at least four times a day. The patient was called for routine controls at the end of the post-op 1st week, 2nd week, 1st month, 3rd month, 6th month, and 1st year. At the end of 1 year, the cavity left open was sutured to close it.

CONCLUSION

As a result of the patient's routine controls, it was observed that the cavity in the bone shrank. Radiographs were taken at the end of the 1st month, 3rd month, 6th month, and 1st year showed that the cavity was almost completely closed [Figure 2,3,4].

DISCUSSION

In this case, as in other cases reported in the literature, the lesion in the bone was healed as a result of surgical curettage and hemorrhage (15,16). Pommer suggested that the hematoma formed as a result of trauma to the bone is effective in this process. He thought that after the hematoma, the enzymatic activity of the blood clot resorbs the adjacent bone (17). Blum and Thoma also think that this lesion occurred due to a previous traumatic process in the jaws. They suggested that subperiosteal hematoma increases osteoclastic bone activity (18,19).

It is observed that the time between the discovery of the traumatic bone cyst in the jaw and the previous trauma history varies between 1 week and 20 years (4,8,13).

Although the pathogenesis and etiology are not fully understood in general, it has been understood that it is largely caused by trauma. In our case, although it is thought that a traumatic bone cyst occurred as a result of iatrogenic trauma, definitive evidence is lacking.

Conflict of interests
The authors declare that they have no competing interests.

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Informed Consent
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