Development of chylothorax and chylous ascites in a patient with congestive heart failure

Konjestif kalp yetersizliği olan bir hastada şilotoraks ve şilöz asit gelişimi

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Summary – Chylothorax and chylous ascites are very rare clinical entities generally caused by obstruction and disruption of the thoracic duct. A 60-year-old man presented with exertional dyspnea, fatigue, and chest discomfort of 18-month history. Physical examination revealed S_4, bilateral pretibial edema, and moderate amount of ascites. Computed tomography and X-ray of the thorax showed left-sided pleural effusion. Abdominal imaging showed normal liver and spleen structure with intraperitoneal effusion and periportal edema. Thoracentesis and paracentesis yielded a milky, lipemic fluid of exudative nature. Biochemical analysis of the fluids showed a high triglyceride content and elevated lymphocyte count, typical of chylous fluid. All laboratory analyses for possible etiologies including neoplasms, tuberculosis, and cirrhosis were negative. Positron-emission tomography did not show any pathological uptake. Transthoracic echocardiographic examination showed bilateral atrial enlargement, left ventricular hypertrophy, anteroseptal hypokinesia and akinesia, and moderate mitral and tricuspid regurgitation, with an ejection fraction of 25%. Coronary arteries were normal on angiography. The patient was diagnosed with severe congestive heart failure accompanied by chylothorax and chylous ascites. Despite appropriate treatment, there was little change in congestion and no change in symptoms. He died during ultrafiltration therapy due to hemodynamic collapse and asystole.

Özet – Şilotoraks ve şilöz asit nadir görülen patolojik durumlardır; genellikle göğüs kanalının tıkanması veya hasarı sonucu oluşur. Altmış yaşında erkek hasta, 18 aydır var olan efor dispnesi, halsizlik, göğüste sıkıntı yakınmalarıyla kliniğimize başvurdu. Fizik muayenede S_4, pretibial ödem ve orta derecede asit saptandı. Göğüs bilgisayarlı tomografisi ve akciğer grafisinde sol taraflı plevral efüzyon görülürken; batın incelemelerinde karaciğer ve dalak yapısı normal bulundu, intraperitoneal efüzyon ve periportal ödem izlendi. Yapılan torasentez ve parasentezde süt kıvamında, lipemik, eksüda özelliğinde sıvı geldi. Bu sıvıların biyokimyasal incelemesinde, şilöz sıvı ile uyumlu olarak, trigliserit düzeyi yüksek ve lenfosit sayısı artmış bulundu. Neoplazi, tüberküloz, siroz gibi olası etyolojilere yönelik tüm laboratuvar incelemeleri negatif sonuç verdi. Pozitron emisyon tomografi incelemesinde patolojik tutulum artışı görülmedi. Transtorasik ekokardiyografide atriyumlarda genişleme, sol ventrikül hipertrofisi, anteroseptal hipokinezi ve akinezi ve orta derecede mitral ve triküspit yetersizliği saptandı, sol ventrikül ejeksiyon fraksiyonu %25 idi. Koroner anjiyografide ise koroner arterler normal bulundu. Hastaya, şilotoraks ve şiloz asidin eşlik ettiği, ağır konjestif kalp yetersizliği tanısı kondu. Uygun tedaviye rağmen, hastanın konjestiyon bulguları ve semptomlarında düzelme olmadı. Ultrafiltrasyon tedavisi gördüğü sırada hasta hemodinamik çöküş ve asistole bağlı olarak yaşamını yitirdi.

Chylothorax and chylous ascites are very rare clinical entities. The most common etiologies are neoplasms and trauma. Congestive heart failure,[1] constrictive pericarditis,[2-4] ischemic heart disease,[5] cirrhosis, superior vena cava thrombosis, nephrotic syndrome, dilated cardiomyopathy,[4] and rheumatic mitral stenosis[6] may be associated with chylothorax and chylous ascites. There are only few cases in the literature that presented with both chylothorax and chylous ascites due to nonischemic congestive heart failure. We report on a patient who developed chylothorax and chylous ascites secondary to nonischemic congestive heart failure.

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CASE REPORT

A 60-year-old male patient was admitted to our clinic with exertional dyspnea, fatigue, and chest discomfort of 18-month history. His past medical history was unremarkable. On physical examination, his body temperature was 36.8 °C, blood pressure was 90/60 mmHg, and pulse rate was 76 beats/min. Jugular veins were distended and respiratory sounds were inaudible in bilateral lower lung fields. Heart auscultation was normal except for the presence of S_4. Bilateral pretibial edema and moderate amount of ascites were present. Electrocardiography showed low QRS voltage with normal sinus rhythm. Chest radiography and computed tomography of the thorax showed left-sided pleural effusion (Fig. 1). Abdominal ultrasonography and computed tomography showed normal liver and spleen structure, intraperitoneal effusion, and periportal edema without lymphadenopathy. Laboratory findings showed a normal polymorphonuclear leukocyte count (4,500/mm^3), but thoracentesis yielded a milky, lipemic fluid. The pleural fluid was 650 ml and of exudative nature in comparison with the biochemical features of the plasma which was taken simultaneously during thoracentesis. Paracentesis also revealed a milky fluid mostly consisting of polymorphonuclear leukocytes. Peritoneal fluid was also exudative in nature (Table 1). Cytology and cultures including mycobacterial PCR testing from peritoneal and pleural fluids were negative. Tumor markers including carcinoembryonic antigen and CA19-9 were all within normal ranges. Rheumatologic serology tests including sedimentation rate, C-reactive protein (1.8 mg/l), antinuclear antigen, anti-dsDNA, and rheumatoid factor did not show any significant abnormalities. Evaluation with positron-emission tomography did not show any pathological uptake suggestive of malignancy or lymphadenopathy.

Table 1. Biochemical analyses of blood, peritoneal fluid and pleural fluid

	Blood	Peritoneal fluid	Pleural fluid
Glucose (mg/dl)	91	136	120
Total protein (g/dl)	4.3	5.4	5.9
Albumin (g/dl)	2.3	3.8	3.5
Lactate dehydrogenase (UI/l)	105	178	220
Triglyceride (mg/dl)	273	873	458
Total cholesterol (mg/dl)	132	111	102
Leukocyte count (/mm^3)	4500	530	470

Echocardiography showed bilateral atrial enlargement, left ventricular hypertrophy, anteroseptal hypokinesia and akinesia, and moderate mitral and tricuspid regurgitation. Estimated pulmonary artery systolic pressure was 35 mmHg. Ejection fraction was 25%. Coronary angiography showed normal coronary arteries. Left ventricular pressure tracings did not show a square root sign or elevated left ventricular end-diastolic diameter pressure, ruling out constrictive pericarditis. Constrictive cardiomyopathy accompanied by severe left ventricular systolic dysfunction was also ruled out due to the absence of pericardial calcification or thickening on computed tomography or echocardiographic examination. The patient was diagnosed as having severe congestive heart failure accompanied by chylothorax and chylous ascites. Con-

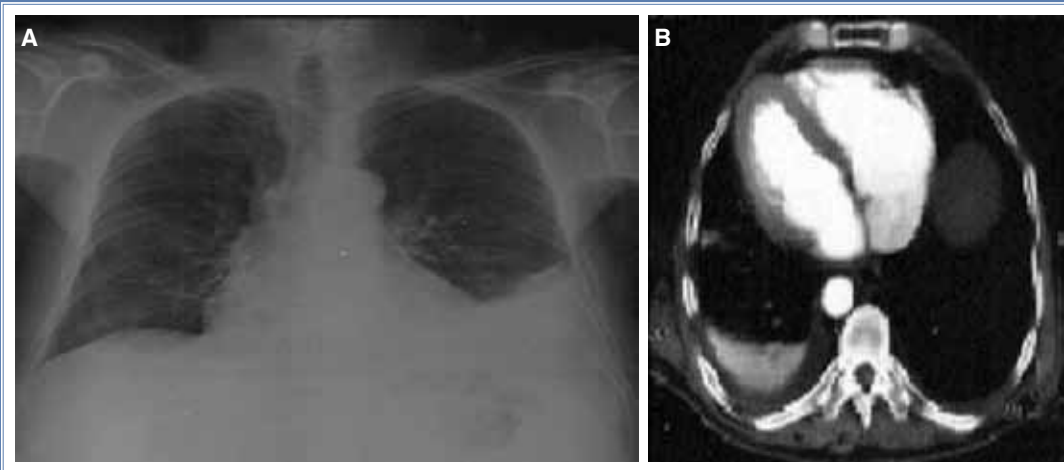

Figure 1. (A) Chest X-ray shows cardiomegaly and left-sided pleural effusion due to congestive heart failure. **(B)** Thoracic computed tomography shows moderate left-sided pleural effusion with massive cardiomegaly.

sidering the presence of symptomatic bradycardia, wide QRS complex, and NYHA class IV heart failure, a biventricular pacemaker was implanted. His dietary fat intake was restricted and he was given high-dose intravenous diuretic therapy along with levosimendan perfusion followed by intravenous inotropic agents. Control thoracentesis showed a 50% decrease in triglyceride concentration in the pleural effusion. His medical therapy was continued for 30 days due to slight changes in congestion and no change in symptoms until the patient was considered to be unresponsive to treatment; then ultrafiltration was performed. Unfortunately, he died during ultrafiltration therapy due to hemodynamic collapse and asystole.

DISCUSSION

Chylothorax is an uncommon, but clinically important disease that may lead to life-threatening conditions if not treated. Half of all chylothorax cases are right-sided, one-third is left-sided, and the remaining are bilateral. It should be noted that not all chylous pleural effusions appear milky white. Almost 50% of cases present as bloody, yellow or green, turbid, serous or serosanguineous effusions. Pleural fluid triglyceride levels of >110 mg/dl, presence of chylomicrons, low cholesterol level, and elevated lymphocyte count are diagnostic for chylothorax.[7] Problems encountered in patients with chylothorax include losses of electrolytes, vitamins, immunoglobulins, essential proteins and fats that may have critical consequences, as well as hypovolemia, hyponatremia, metabolic acidosis, and hypocalcemia.

Therapeutic modalities for chylothorax include oral medium-chain triglycerides, total parenteral nutrition, tube drainage, direct ligation of the thoracic duct, mass ligation of the supradiaphragmatic thoracic duct, pleuroperitoneal shunting, pleurectomy, and pleurodesis with glue or talc.[8,9] Although ascites is a common complication of liver disorders, only 3% of cases are associated with cardiac pathologies such as left heart failure. Chylous ascites is a rare condition (<1%) defined by the presence of high concentration of triglycerides in the ascitic fluid (>200 mg/dl).[10] Chylothorax and chylous ascites are generally caused by obstruction and disruption of the thoracic duct. While the most common causes of these pathologic events are lymphomas, tuberculosis, and trauma, other conditions may occasionally be seen including constrictive pericarditis, cirrhosis, superior vena cava thrombosis, nephrotic syndrome, dilated cardiomyopathy, and rheumatic mitral stenosis. Contrary to our case, most cases with heart failure presenting with chylothorax and chylous ascites are related to ischemic cardiomyopathy.[5,11] Rarely, congestive heart failure secondary to various causes may lead to chylothorax or chylous ascites.[4,6,11]

There are several mechanisms leading to chylothorax and chylous ascites in congestive heart failure. First, high venous pressure induces abdominal lymph production by increasing capillary filtration. The lymphatic flow of the thoracic duct may increase by up to 12-fold of the normal rate, but the rigidity of the veno-lymphatic junction in the neck counteracts the lymphatic flow.[12] Second, high pressure in the left subclavian vein decreases lymphatic drainage and in the presence of restricted lymphatic drainage of any cause, the lymphatic venous collaterals become incapable of handling normal lymphatic flow form.[11] In our case, the pleura and peritoneum contained chylous fluid and other probable causes such as occult neoplasia, trauma, and ischemic or constrictive cardiomyopathy were ruled out by imaging and catheterization techniques. However, the absence of right heart catheterization could be considered a limitation in the diagnostic work-up of our case. In our case, we think that elevation in the central venous pressure accompanied by left and right heart failure, induced by nonischemic heart disease, might have increased the lymphatic flow of the thoracic duct and reduced lymphatic drainage into the left subclavian vein, giving rise to chylothorax. On the other hand, high venous pressure induces abdominal lymph production and increases the lymphatic flow of the thoracic duct, resulting in increased capillary filtration and chylous ascites. Although it is a very common presentation, coexistence of chylous ascites and chylothorax is very rare in patients with heart failure. Thus, clinicians should be aware of these rare manifestations and complications in patients with heart failure.

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Key words: Chylothorax/etiology; chylous ascites/etiology; heart failure/complications.

Anahtar sözcükler: Şilotoraks/etyoloji; şilöz asit/etyoloji; kalp yetersizliği/komplikasyon.