

Chylous ascites and chylothorax due to constrictive pericarditis in a patient undergoing haemodialysis

M. Riza Altiparmak^{1*}, S. Avsar¹, S. Yanik²

¹Department of Internal Medicine and ²Microbiology, Istanbul University, Cerrahpasa Medical Faculty, Istanbul, Turkey, tel.: +90 216-410 63 29, fax: +90 216-410 63 29, e-mail: burakuzel@netscape.net, * corresponding author

ABSTRACT

Chylous ascites and chylothorax are rare clinical entities and usually caused by neoplasms, particularly lymphomas, liver cirrhosis, superior vena cava thrombosis, nephrotic syndrome, and some cardiac events such as dilated cardiomyopathy or right heart failure. Constrictive pericarditis is an extremely rare cause of this clinical state. We report a 41-year-old male patient undergoing haemodialysis who presented with chylous ascites and chylothorax. Echocardiography and heart catheterisation revealed constrictive pericarditis. He underwent pericardiectomy and after the operation the ascites and pleural effusion resolved rapidly. We suggest that constrictive pericarditis should be considered in the differential diagnosis of chylous ascites and chylothorax.

INTRODUCTION

Chylous ascites and chylothorax are rare clinical findings. Constrictive pericarditis rarely causes chylous ascites and chylothorax. To our knowledge there are only five cases in the literature presenting with chylous ascites due to constrictive pericarditis,¹⁻⁵ and only one presenting with chylothorax and recent-onset chylous ascites following thoracic band ligation.⁶ We report a patient undergoing haemodialysis who developed chylous ascites and chylothorax, secondary to constrictive pericarditis.

CASE REPORT

A 41-year-old male patient was admitted to our clinic with the symptoms of abdominal distension and dyspnoea continuing for seven months. He had been undergoing haemodialysis three times a week for two years. The cause of his chronic renal failure was chronic glomerulonephritis. Six months ago he was admitted to another clinic because of these symptoms and at that time chylous ascites and chylothorax were detected. He was given empirical anti-tuberculosis therapy for six months without any response. On physical examination, his blood pressure was 75/40 mmHg, pulse 96 beats/min, and temperature 37°C. Jugular venous pressure was high. On pulmonary auscultation, respiratory sounds were decreased in bilateral lower lung fields. Heart sounds were also difficult to hear. Ascites was detected in the abdomen. Laboratory findings were as follows: Hb 8.5 g/dl, htc 25.4%, WBC 5000/mm³, platelets 207,000/mm³, C-reactive protein 0.96 mg/dl (N: 0-0.81), erythrocyte sedimentation rate 72 mm/h, urea 111 mg/dl, creatinine 6.50 mg/dl, AST 25 U/l, ALT 32 U/l, lactate dehydrogenase (LDH) 265 U/l (N 225-450), glucose 102 mg/dl, total protein 7.4 g/dl, albumin 3.6 g/dl, amylase 75 U/l (N 25-125), cholesterol 176 mg/dl and triglycerides 83 mg/dl. The other laboratory findings were in normal ranges. Abdominal ultrasonography (USG) and computerised tomography (CT) showed bilateral atrophic kidneys and massive intraperitoneal effusion. Thorax CT revealed bilateral pleural effusions, thickness of the pericardium with calcification (*figure 1*). No lymphadenopathy or mass was visible on abdominal and thorax CT.



Figure 1

A thorax computerised tomography imagination revealing bilateral pleural effusion, localised pleural effusion at the right paracardiac region, pericardial thickness and calcification (arrow), costal pleural thickness

Paracentesis yielded a milky fluid with the following biochemical composition: triglycerides 405 mg/dl, cholesterol 85 mg/dl, total protein 4.7 g/dl, albumin 2.7 g/dl, glucose 72 mg/dl and LDH 121 U/l. Cell count of the fluid was 600/mm³. Thoracentesis also disclosed a milky fluid and laboratory studies were as follows: triglycerides 395mg/dl, cholesterol 95mg/dl, LDH 185 U/l, total protein 6.4 g/dl, albumin 2.56 g/dl, glucose 65 mg/dl and a count of cell 550/mm³. Cytology and cultures, including mycobacterial, from peritoneal and pleural fluid were negative. Transoesophageal echocardiography showed the thickness of the pericardium, pericardial effusion localised behind the right atrium and spontaneous echo contrast in the right atrium and ventricle. Ejection fraction was 60%. Right and left heart catheterisation revealed normal coronary arteries and left ventricular function. Haemodynamic findings were consistent with constrictive pericarditis. The patient underwent pericardiectomy. Postoperatively ascites and pleural effusion gradually resolved over a three-week period, and his blood pressure returned to normal ranges. He felt well and no longer complained of dyspnoea and abdominal distension. The histopathological examination of the pericardial material revealed exudation of fibrin, lipomatosis, hyalinisation and calcification which shows chronic nonspecific inflammation. There were no granulomas or malignant infiltration. Cultures of pericardial fluid for mycobacterium and other agents were sterile. A specific cause for pericarditis could not be documented.

DISCUSSION

Chylothorax and chylous ascites are rare clinical findings and characterised by milky peritoneal and pleural fluid

from elevated triglycerides, which are most commonly caused by obstruction and disruption of the thoracic duct or one of its major divisions as a result of a malignant tumour, trauma or inflammation.^{1,2,6} A milky appearance and a triglyceride level of more than 110 mg/dl generally confirm the diagnosis.^{1,7} Neoplasms, particularly lymphoma, are the most common causes.^{1,2} Liver cirrhosis, superior vena cava thrombosis, nephrotic syndrome and Behcet's disease have also been reported as the causes of chylous ascites.¹ Any cardiac cause of elevated right-sided venous pressure such as dilated cardiomyopathy, severe tricuspid regurgitation, constrictive pericarditis and right heart failure may also cause chylous ascites.^{2,8} Abdominal and thoracic CT and fluid examination must be carried out to exclude a malignancy or inflammation in the fluid. Also a lymphangiogram can be done to demonstrate the site of obstruction of lymphatic flow.¹

Constrictive pericarditis can rarely cause chylous ascites and chylothorax. To our knowledge, there are only five cases in the literature presenting with chylous ascites.^{1,5} One of these cases also had liver cirrhosis.⁴ And as we know, it is possible to develop chylous ascites and chylothorax with cirrhosis without constrictive pericarditis. In the English literature there is only one case of constrictive pericarditis presenting with chylothorax and chylous ascites following thoracic band ligation.⁶ In this case, it is unclear whether the cause of the chylous ascites was thoracic duct ligation or constrictive pericarditis.⁶

The potential mechanisms for the development of chylous ascites and chylothorax resulting from constrictive pericarditis are the increasing effective capillary filtration secondary to central venous hypertension and reduced lymphatic drainage due to the high pressure in the left subclavian vein. Increased capillary filtration may result in excessive lymph formation.^{1,3}

In our case, the patient presented with both chylous ascites and chylothorax and we detected constrictive pericarditis by thorax CT and echocardiography. The diagnosis was confirmed by left and right heart catheterisation. We could not show any other cause (malignancy, cirrhosis, thrombosis of superior or inferior vena cava, dilated cardiomyopathy and right heart failure) of the chylous ascites and chylothorax. Finally, following the pericardiectomy the rapid resolve of the ascites and pleural effusion made us conclude that the chylothorax and chylous ascites were secondary to constrictive pericarditis. Since the fluid resolved after the operation and the CT scans of abdomen and thorax did not reveal any masses that may cause lymphatic obstruction, a lymphatic scintigraphy and/or lymphangiography were not performed.

Constrictive pericarditis may occur when the healing of an acute fibrinous or serofibrinous pericarditis or a chronic pericardial effusion is followed by obliteration of the pericardial cavity with the formation of granulation

tissue.⁹ The aetiology of constrictive pericarditis is usually unclear. Marta *et al.* reported this ratio as 42%.¹⁰

Tuberculosis is the most common known cause of this disorder. Also purulent infections, trauma, cardiac operation, mediastinal irradiation, histoplasmosis, neoplastic disease, acute viral or idiopathic pericarditis, rheumatoid arthritis, SLE, and chronic renal failure treated by chronic dialysis may result in constrictive pericarditis.⁹

The aetiology of constrictive pericarditis is unclear in patients with chylous ascites, except for the case reported by England *et al.*² In that patient, the author reported that pericarditis developed after cardiac surgery.

In our case the patient was first treated with antitubercular drugs for six months, but he did not respond to the therapy. Also histopathological examination of the pericardium revealed no granulomas. So, we could exclude tuberculosis as the cause of the pericarditis. The patient also had chronic renal failure and we know that it may be the main cause of this condition. Pericardial involvement in end-stage renal failure commonly manifests as an acute uraemic or dialysis pericarditis and less commonly as chronic constrictive pericarditis.¹¹ The clinical presentation of constrictive pericarditis in uraemic patients is similar to those observed in nonuraemic patients with less frequent chest pain in uraemics than nonuraemics.¹¹

So, constrictive pericarditis should be considered in the differential diagnosis of chylous ascites and/or chylothorax.

NOTE

In memory of a beloved friend and a perfect doctor,
Sinan Auşar.

REFERENCES

1. Guneri S, Nazli C, Kinay O, Kirimli O, Mermut C, Hazan H. Chylous ascites due to constrictive pericarditis. *Int J Card Imaging* 2000;16:49-54.
2. England RW, Grathwohl KW, Powell GE. Constrictive pericarditis presenting as chylous ascites. *J Clin Gastroenterol* 2002;35:104-5.
3. Savage MP, Munoz SJ, Herman WM, Kusiak VM. Chylous ascites caused by constrictive pericarditis. *Am J Gastroenterol* 1987;82:1088-90.
4. Williams AT, Schneider RP. Chylous ascites should suggest constrictive pericarditis even in a patient with cirrhosis. *J Clin Gastroenterol* 1990;12:581-4.
5. Marshall JB, Pola L. Chylous ascites secondary to constrictive pericarditis. A discussion of pathophysiologic mechanism. *Dig Dis Sci* 1982;27:84-7.
6. Motta G, Vianello F, Pietrogrande F. Chylothorax as the first manifestation of constrictive pericarditis. *Am J Med* 2003;114:772-3.
7. Romero S. Nontraumatic chylothorax. *Curr Opin Pulm Med* 2000;6:287-91.
8. Villena V, Pablo A de, Martin-Escribano P. Chylothorax and chylous ascites due to heart failure. *Eur Respir J* 1995;8:1235-6.
9. Braunwald E. Pericardial disease. In: Fauci AS, Braunwald E, Isselbacher KJ, et al (eds). *Harrison's Principles of Internal Medicine*. International Edition. Mc Graw-Hill, 1998;1:1334-40.
10. Marta MJ, Oliviera A, Varela MG, Saavedra JA, Ravara L. Constrictive tuberculous pericarditis: case report and review of the literature. *Rev Port Cardiol* 2003;22:391-405.
11. Alpert MA, Ravenscraft MD. Pericardial involvement in end-stage renal disease. *Am J Med Sci* 2003;325(4):228-36.