size, and peristalsis with no signs of peritoneal irritation.

Laboratory tests showed the presence of high amylase, lipase, and CRP levels and triglyceride levels of 218 mg/dL, with normal bilirubin, transaminase, LDH, and alkaline phosphatase values. Electrocardiogram was normal. No changes were seen in chest and abdominal X-rays.

Antibiotic coverage and fluid therapy were started, and absolute diet was maintained.

A picture of severe abdominal pain in epigastrium and the periumbilical region, often irradiating to the back, nausea, and high serum amylase or lipase levels usually confirms diagnosis of pancreatitis. Fever and ST decreases in the electrocardiogram are not uncommon.

While the main causes of pancreatitis are stones, alcohol consumption, high triglyceride levels, drugs, etc., it should also be considered in the differential diagnosis of abdominal pain in patients with polycystic kidney disease.[4]

Ultrasonography revealed a liver consistent with liver steatosis or chronic liver disease, with multiple cystic lesions. No stones were seen in the gallbladder. The bile tract was not dilated. Several cysts up to 2.6 cm in diameter were seen in the pancreas. Bilateral nephrectomy was performed.

A CT scan of the abdomen identified a cystic mass with a multilobulated contour in the pancreatic neck region, approximately 4.7 x 3.7 cm in size, that dilated the pancreatic duct at pancreas body and tail level (fig. 1).

An echoendoscopy confirmed the presence of multiple thin-walled, anechoic cysts of various sizes with no solid contents in the pancreas head and isthmus causing a 5-mm dilation in the Wirsung's duct.

No fever or leukocytosis was found at any time. The maximum amylase, lipase, and CRP levels achieved were 628 U/L, 8806 U/L, and 70 mg/L respectively. These concentrations subsequently decreased gradually during the course, abdominal pain improved, and oral diet could be restarted at 4 days with good tolerance.

The final diagnosis was acute pancreatitis, probably obstructive in nature.

As this was the first episode of pancreatitis, and given the clinical and laboratory improvement, a continued watching attitude was decided, but if the patient should experience a new episode in the future[4] or evidence of chronic pancreatitis occurred,[5] more aggressive measures already used at hepatic level,[6] such as cyst aspiration and sclerosis, surgical or laparoscopic treatment, transplant, etc. would be considered.

We think this is an interesting case, because pancreatic extrarenal cysts are usually asymptomatic.

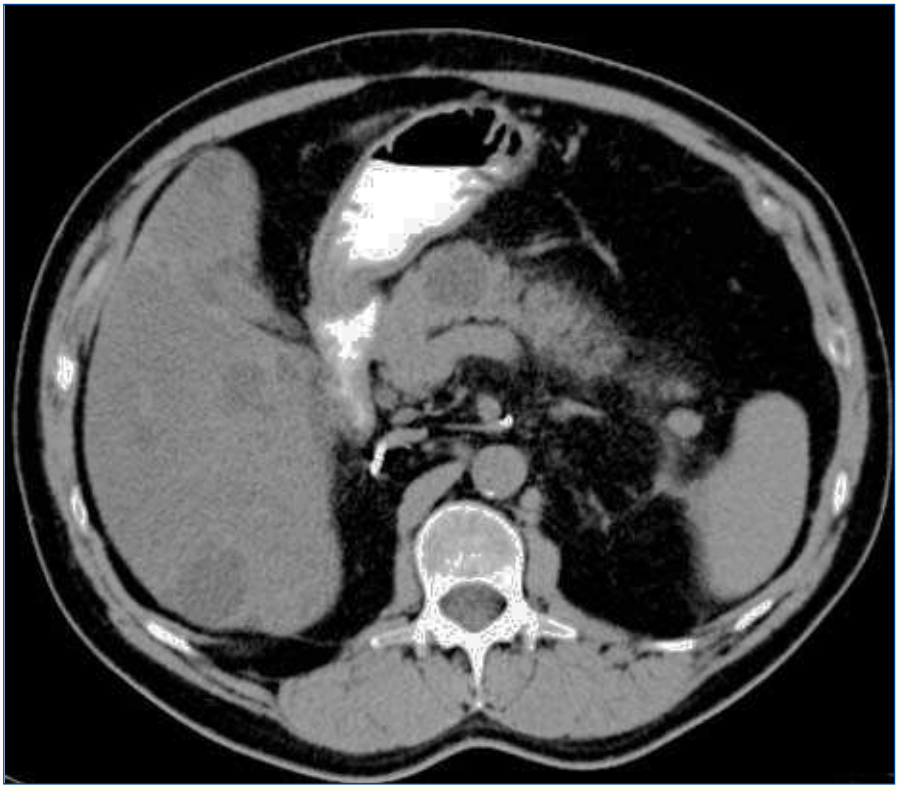

Figure 1.

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A. Sastre López, M.ª R. Bernabéu Lafuente, M.ª V. Íñigo Vanrell and J. M. Gascó Company

Servicio de Nefrología. Hospital Son Llàtzer. Palma de Mallorca.

Correspondence: *Aránzazu Sastre López. aranchasastre@hotmail.com. Hospital Huca. Avda. Fernández Ladreda, 30. 24005 León.*

Chylotorax: an uncommon cause of pleural effusion in patients on haemodialysis

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To the editor: Chylothorax is an accumulation of lymph (containing a great amount of lymphocytes, triglyce-

rides, and chylomicrons) in the pleura as a consequence of impaired drainage of the thoracic duct. Chylothorax may result from traumatic or non-traumatic causes (such as neoplasms, sarcoidosis, chest irradiation, etc.), but there are also idiopathic forms.[1,2]

The thoracic duct may be damaged during placement of central catheters or as the result of stenosis or thrombosis of the thoracic veins caused by the catheter.[3] However, it is noteworthy that very few cases have been reported in the literature despite the enormous number of catheters placed.[4]

The case of a female patient on haemodialysis who developed chylothorax secondary to a superior vena cava lesion related to the presence of a right jugular catheter is reported here.

CASE REPORT

A 72-year old female patient included in a regular haemodialysis programme in April 2004 because of chronic kidney disease secondary to diabetic nephropathy. The patient had many vascular access problems since she entered the programme. She was initially placed a tunnelled right jugular catheter. In May 2004, a brachiocephalic arteriovenous fistula was performed in the upper limb, but experienced early thrombosis. A vascular study considered a new fistula or prosthesis unfeasible. In May 2005, oral anticoagulant therapy was started due to catheter dysfunction. An angio-MRI (fig. 1) showed stenosis of the right internal jugular and superior cava veins. The superior vena cava was dilated and a tunnelled catheter was placed through the right jugular vein with the tip at inferior cava level, which allowed for continuing haemodialysis therapy.

In November 2005, the patient experienced a gradual increase in dyspnoea until she also suffered dyspnoea at rest. Chest x-rays showed a right pleural effusion with no fever or signs of infection. Laboratory test results included a WBC count of 9130/mm^3, haemoglobin 14.3 g/dL, creatinine 6 mg/dL, glucose 125 mg/dL, total protein 61.2 g/L, LDH 120 IU/L, triglycerides 237 mg/dL, and cholesterol 178 mg/dL; Kt/V was 1.7 and protein catabolic rate (PCR) 0.86. A thoracentesis yielded a milky fluid with 1620 cells/mm^3 (95% lymphocytes), glucose 128 mg/dL, protein 34 g/L, LDH 86 IU/L, triglycerides 754 mg/dL, pH 7.43, and adenosine deaminase 15 IU/L. Cytology was benign and culture was sterile.

Based on a diagnosis of chylothorax, a pleural draining tube was placed and pleurodesis with talc was attempted, but was ineffective. In March 2006, the patient was admitted for right upper lobe pneumonia that improved on antibiotic therapy. A CT scan of the chest showed an encapsulated pleural effusion, with prevented pleurodesis. Videothoracoscopy was considered but was rejected by the patient, who subsequently died from septic shock of a pulmonary origin. Necropsy was not authorised.

DISCUSSION

Patients on haemodialysis may experience pleural effusion due to highly diverse causes.[4] While exudates may occur in the setting of infections, neoplasms, uremic pleuritis, or haemothorax, transudates due to volume overload or an impaired venous drainage occur in most cases.[4-7] Occurrence of chylothorax in patients on haemodialysis is very uncommon.[4]

The cause of chylothorax in the reported patient is difficult to establish, but caval stenosis or a direct lesion to the thoracic duct may have possibly been involved in its pathogenesis.[8,9] Diagnosis was confirmed based on the milky appearance and composition of the fluid (triglyceride levels higher than 110 mg/dL and elevated lymphocyte count).

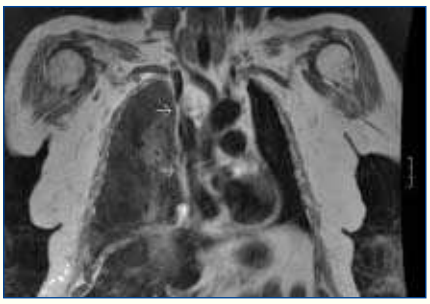

Figure 1. Angio-MRI of the chest showing severe stenosis of the superior vena cava (top arrow) and filling defects suggesting venous thrombi (bottom arrow).

Treatment of chylothorax is controversial and depends on both its cause and symptoms. The approach may range from conservative treatment to elective surgery.[10] A low fat diet with medium chain triglycerides (that are directly absorbed to blood) is advised to decrease the amount of lymph. Chemical pleurodesis using tetracycline, bleomycin, or talc, and pleuroperitoneal shunts have been shown to be useful.[10] In patients with vena cava obstruction, angioplasty may solve the problem.[11] Surgery by minimally invasive thoracoscopy with supradiaphragmatic repair or ligation of the thoracic duct may correct chylothorax.[2,10,12] In order to prevent infectious complications and malnutrition, chylothorax fluid has been successfully reinfused during haemodialysis in some patients.[4]

To sum up, chylothorax associated to central catheters for haemodialysis is uncommon and requires adequate diagnostic evaluation and a specific therapeutic approach.

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M. Torres Guinea, G. De Arriba, M.ª A. Basterrechea and J. Ocaña
Nefrología. Hospital Universitario de Guadalajara. Departamento de Medicina. Universidad de Alcalá.
Correspondence: *Gabriel de Arriba. garribad@senefro.org. Hospital Universitario de Guadalajara. Donante de sangre, s/n, 19002 Guadalajara.*

Mixed cryoglobulinemia in patients with dual HCV/HIV infection: analysis of cryoprecipitate as a therapeutic decision tool

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To the editor: Infection by the hepatitis C virus (HCV) is the main cause of mixed cryoglobulinemia.[1,2] However, the human immunodeficiency virus (HIV) also causes cryoglobulinemia.[3] In high-risk people, dual HIV/HCV infection is common. The decision to add treatment for HCV to antiretroviral medication is difficult, because it may be harmful in immunodepressed individuals. A more precise diagnosis is also required because occult infection exists, with a falsely negative antibody test.[4]

In order to contribute to solve this problem, we studied cryoprecipitates from HIV-positive patients with cryoglobulinemia and membranoproliferative glomerulonephritis (MPGN). Serologic tests were positive for HIV/HCV in one patient, and for HIV alone in two patients.

The result was markedly clear: in the patient with dual infection, the cryoprecipitate only showed anti-HCV activity, while in the two patients with HIV infection alone, the cryoprecipitate only showed anti-HIV activity. These data allowed for taking the decision that only one patient had to be receive treatment for HCV.

CASE 1

A 45-year old male with HIV and HCV infection (genotype 1a) on antiretroviral therapy and with negative HIV load. In March 2007, the patient experienced nephrotic syndrome, and was diagnosed MPGN (table I). Cryoprecipitate was positive for HCV alone, and additional treatment with alpha-interferon was started.

CASE 2

A 36-year old male with HIV infection detected two years before and diagnosed of non-Hodgkin lymphoma in May 2006. He then had nephrotic syndrome with normal kidney function and positive cryoglobulins (table I). No antibodies to hepatitis C and B viruses were detected, and type I MPGN was diagnosed. Cryoprecipitate was positive for HIV and negative for HCV. Antiretroviral and chemotherapeutic therapy was started.

CASE 3

A 35-year old male with known HIV infection for eight years who showed nephrotic proteinuria, normal kidney function, and positive cryoglobulins (table I). A type I MPGN was diagnosed. HIV was only shown to be present in the cryoprecipitate, and treatment with antiretrovirals and steroids alone was started.

In all three cases, the cryoprecipitate was separated from the supernatant by centrifugation (3,000 rpm at 4 ºC), with adhered proteins being removed by washing with warm saline. Viral RNAs were extracted using Cobas-Amplipred (Roche). HIV and HCV loads were measured by RT-PCR using Cobas-Amplicor ultrasensitive for HIV-1 (limit, 50 copies/mL) and by PCR, using Taq Man 48 for HCV (limit, 10 IU/mL).

Several studies have shown the relationship between HIV and HCV infections, and the role of cryoglobulins in causing renal damage, particularly MPGN.[5-9] It is unknown whether the higher percentage of cryoglobulinemia found in cases with dual HIV/HCV infection[2] is due to one and/or the other virus.[3] No reference has been found in the literature to the concomitant presence of both viral particles in the cryoprecipitate from patients with dual HIV/HCV infection.

Analysis of the cryoprecipitate is a useful tool for differential diagnosis of cryoglobulinemia in patients with dual HIV/HCV infection. It may also be used as a supplemental test to rule out a diagnosis in patients apparently monoinfected by HIV.

Table I. Main laboratory data from the three patients at the time of cryoprecipitate analysis

	Case 1	Case 2	Case 3
Plasma creatinine (mg/dL)	3.5	0.73	1.4
Plasma albumin (g/dL)	3	2.4	1.3
Proteinuria (g/24 hours)	9	4	5
Complement C3 (mg/dL)	109	49	111
Complement C4 (mg/dL)	47	24	24
Cryoglobulins	Positive	Positive	Positive
HIV viral load (copies/mL)	42	69,000	27,900
HCV viral load (copies/mL)	245,714	0	0
HIV viral load in cryoprecipitate (copies/mL)	0	1.200	700
HCV viral load in cryoprecipitate (copies/mL)	42,840	0	0
CD4 lymphocytes/mL	746	100	458

Table 1 shows the main laboratory data from the three patients at the time of cryoprecipitate analysis.