Reminder of important clinical lesson

Ascites as a manifestation of effusive-constrictive pericarditis in an African patient

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Summary

A 34-year-old man from Sao Tome and Principe, Africa, with a history of tuberculosis and alcohol ingestion greater than 100 g/day presented with progressively worsening dyspnoea, right upper quadrant pain and progressively increasing abdominal volume. Physical examination revealed distended jugular veins, tender hepatomegaly, moderate ascites and oedema of lower limbs. MRI revealed collection of pericardial fluid compressing the right ventricle and a thickened pericardium, consistent with an effusive-constrictive pericarditis. Cultures and biopsies were undertaken. Antituberculosis drugs and prednisolone were initiated and drainage of the pericardial collection and pericardiectomy were performed. He recovered with complete remission of signs and symptoms.

BACKGROUND

A cardiac aetiology is found in about 3% of all cases of ascites. In industrialised countries, idiopathic, cardiac surgery and mediastinal irradiation are the main causes of constrictive pericarditis. In developing countries and in immunocompromised individuals tuberculous aetiology predominates.

This case reports a rare cause of ascites in Europe and warns for a high degree of suspicion needed for pericarditis diagnosis, which may be life-threatening and is treatable. This case is well illustrated and brings together gastroenterology, cardiology and cardiothoracic surgery.

CASE PRESENTATION

We report a 34-year-old African-American man from Sao Tome and Principe, Africa. He referred to a history of tuberculosis, with irregular treatment for 4 months, and malaria. Over the past 6 months he had been hospitalised several times and later evacuated to Portugal. He mentioned an alcohol ingestion greater than 100 g/day. He has denied blood transfusions, drug addiction or other risky behaviours.

He presented with worsening dyspnoea, lower-limb swelling, right upper quadrant pain and progressively increasing abdominal volume evolving over 1 year.

Physical examination revealed distended jugular veins, tender hepatomegaly, moderate ascites and oedema of lower limbs. An early third heart sound was present on cardiac auscultation and vesicular breath sounds were absent in the lower two-thirds of the right hemithorax.

INVESTIGATIONS

Laboratory tests ruled out causes of chronic liver disease like hepatitis B and C, autoimmune liver disease, Wilson disease, haemochromatosis and α 1-antitrypsin deficiency. There was a slight change in liver tests (aspartate aminotransferase 58 (normal <34 IU/l), alanine aminotransferase 56 (normal <49 IU/l), γ -glutamyl transpeptidase 168 (normal <73 IU/l) and normal alkaline phosphatase) with

prolonged prothrombin time (INR—international normalised ratio 1.7); C reactive protein was normal, N-terminal B-natriuretic peptide (NT-proBNP) was 560 pg/ml (N<125 pg/ml) and HIV 1 and 2 were both negative. Tuberculin test was highly positive (20 mm induration), not excluding tuberculosis reactivation, despite its low specificity in endemic areas of tuberculosis like Sao Tome.

Chest radiograph showed right pleural effusion, without cardiac calcifications. Abdominal ultrasound revealed ascites and hepatic veins dilatation in 'goose foot' typical appearance and increased liver size with regular borders. Hepatic transitory elastography has shown very high values suggestive of an intense fibrosis/cirrhosis (33.6 kPa).

The serum-ascites-albumin gradient was greater than 1.1, consistent with portal hypertension. Pleural effusion fluid had exudates' characteristics. Pleural biopsy showed no significant changes and Ziehl-Neelsen and culture for acid-alcohol resistant-bacilli were both negative.

Echocardiogram revealed a cystic image attached to the right ventricle. MRI revealed pericardial fluid collection compressing the right ventricle and a thickened pericardium (figure 1), consistent with an effusive-constrictive pericarditis.

Fungal, bacterial and micobacterial cultures of sputum, gastric juice, pleural and ascitic fluid, pericardial abscess and pericardial biopsy were all negative.

DIFFERENTIAL DIAGNOSIS

Marked alcohol consumption and origin from a region with high prevalence of viral hepatitis in a patient with ascites might seem consistent with a cirrhotic aetiology. The high degree of clinical suspicion, cardiac auscultation and distended jugular veins on physical examination suggested a cardiac aetiology.

TREATMENT

Antituberculosis drugs (isoniazid, pyrazinamide and ethambutol) and prednisolone were initiated. The patient

Figure 1 Cardiac MRI showing an abscess (large arrow) compressing the right ventricle and thickened pericardium (small arrow).

was sent to the department of cardiothoracic surgery and underwent drainage of the pericardial collection and pericardiectomy (figure 2).

OUTCOME AND FOLLOW-UP

Postop was uneventful. After the initiation of surgery and therapy he had shown no dyspnoea, ascites and oedema had

progressively disappeared and the INR normalised over the first week. The value for hepatic elastography decreased to 15.3 kPa on the 13th day after surgery.

DISCUSSION

Liver cirrhosis (mostly viral hepatitis B or C and alcoholic) is the main cause of ascites, accounting for over 75% of all

Figure 2 Pericardiectomy picture showing fibrosis (large arrow) and thickened pericardium (small arrow).

cases.¹ Cardiac aetiology represents about 3% of the cases of ascites.²

Effusive-constrictive pericarditis is a rare entity consisting of a constrictive component exerted by visceral pericardium, accompanied by pericardial effusion. Tuberculosis is a very common but not pathognomonic cause of effusive-constrictive pericarditis in regions where this infection is endemic.³ Tuberculosis is now a rare cause of subacute pericarditis in industrialised countries (4–6%), remaining the main cause in developing countries and in patients with HIV infection.⁴

The epidemiological context is thus particularly important in the investigation of rare causes of ascites.

Signs and symptoms arise from volume overload with peripheral oedema, pleural effusion, hepatomegaly and ascites, increased jugular venous pressure (93% of all patients)⁵ and decreased cardiac output, with fatigue, palpitations and dyspnoea. The pericardial knock described as early diastolic sound is caused by the sudden distension of the left ventricle against the thickened pericardium.⁶ Cachexia and hepatic dysfunction with ascites may occur in the later stages. Jugular engorgement that remains after ascitic fluid drainage indicates a cardiac aetiology, since in patients with non-cardiac cirrhosis jugular venous pressure falls off rapidly with ascitic fluid drainage.⁷

NT-proBNP plasma levels can be only slightly increased in constrictive pericarditis.⁸

Calcifications on chest radiography are best seen on lateral or anterior oblique projections. It may be present in less than 50% of all cases⁹ and are more often associated with idiopathic or tuberculous aetiology.¹⁰ Pericardial calcification is an independent factor associated with increased perioperatively mortality.¹⁰

We should recall that echocardiography is an initial complementary procedure on imaging and haemodynamic evaluation of patients with suspected constrictive pericarditis, although it may not definitively exclude the diagnosis of constrictive pericarditis.⁹

CT is useful in demonstrating pericardial thickening (> 4 mm) and calcification, although about 20% of patients with surgically proven constrictive pericarditis, pericardial thickening is not present in cardiac CT.⁹ Therefore the absence of pericardial thickening in the CT scan does not rule out the diagnosis. MRI is defined in some studies as a diagnostic procedure of choice in constrictive pericarditis approach and is superior in differentiating small pericardial effusions from thickened pericardial membrane.^{11–12} Invasive haemodynamic evaluation is used occasionally. Even after exhaustive study with these complementary diagnostic methods, diagnosis may remain unclear.¹³

Although the tuberculin test is less specific in the most affected areas such as Sao Tome and Principe, the result in our patient does not exclude tuberculosis. Negative results of pleural and pericardial biopsy and culture of pleural fluid, abscess, sputum and gastric juice does not rule out tuberculous aetiology because extrapulmonary forms of tuberculosis are usually paucibacillary which decreases sensitivity and specificity of direct examination and culture, especially in the absence of concomitant

pulmonary tuberculosis. Since it is not always possible to isolate *Mycobacterium tuberculosis*, the diagnosis and therapeutic decision often relies on epidemiological, clinical, imaging and laboratory data.¹⁴

Antituberculous and steroids therapy should be initiated and had shown to decrease mortality and need for pericardiectomy.¹⁵ Pericardiectomy is the accepted standard treatment in patients with chronic constrictive pericarditis, noting the high mortality (more than 6%) reported by most experienced centres.¹⁵

Decline on hepatic transitory elastography values after surgery (33.6–15.3 kPa), confirms that the first examination was overestimated by hepatic congestion.¹⁶

Learning points

- ▶ Consider epidemiological context in the investigation of rare causes of ascites.
- ▶ Echocardiography may not definitively exclude the diagnosis of pericarditis.
- ▶ Remember MRI as diagnostic procedure of choice in the constrictive pericarditis approach.
- ▶ Combined treatment (surgical and medical) can be lifesaving, despite the significant operative mortality.

Competing interests None.

Patient consent Obtained.

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