

Tuberculous constrictive pericarditis

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DESCRIPTION

A previously healthy 53-year-old Brazilian man presented with a 3-month history of anasarca, dyspnoea on minimal exertion, orthopnoea, paroxysmal nocturnal dyspnoea, low-grade fever and an unintentional 12 kg weight loss. No prior medical condition was related. No alcohol, tobacco or illicit drug use. He lived in an area that was endemic for tuberculosis, but denied sick contacts. Vital signs at admission were: blood pressure 100/60 mm Hg, heart rate 110 bpm, respiratory rate 26 bpm and oxygen saturation of 98%. Physical findings included jugular venous distension, pulsus paradoxus of 30 mm Hg, severe lower extremities oedema, bilateral rales and reduced lung sounds in both bases. Laboratories parameters including complete blood count, renal and hepatic function were within normal limits, brain natriuretic peptide 105 pg/mL (normal range (NR) <100 pg/mL), serum total protein 4.5 g/dL (NR=6–8 g/dL), Lactic acid dehydrogenase 500 U/L (NR=140–280) and HIV negative. There was a clear pericardial rub. An ECG showed sinus bradycardia and left bundle branch block. An echocardiogram revealed left ventricle ejection fraction of 64%, dilated inferior vena cava, mild concentric hypertrophy, early diastolic Doppler tissue velocity at the mitral annulus (E') of 9 cm/sec, E/e' ratio of 1.3, E/A wave ratio of 1.2, hepatic vein Doppler showed reversal of forward flow during expiration and increased pericardial thickness. A chest CT is shown below (figure 1A). A diagnostic thoracentesis was done and the analysis showed: total protein 3.5 g/dL, amylase 25 U/L (NR 20–104), lactic acid dehydrogenase 408 U/L, normal adenosine deaminase, negative acid-fast bacillus, 145 white blood cells (89% lymphocytes) and negative culture. The analysis was compatible with an exudate according to Light's criteria: three out three criteria met. The patient was treated with diuretics with a minimal relief. A pericardiectomy was performed showing a thickened pericardium (figure 1B).

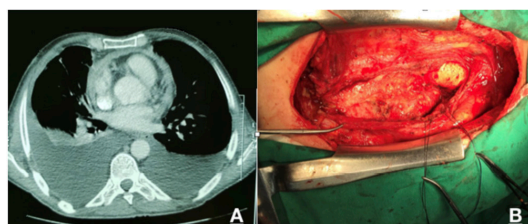


Figure 1 (A) Chest CT—bilateral pleural effusion, moderate pericardial effusion and significant pericardial thickening. (B) Surgical findings—pericardial thickening.

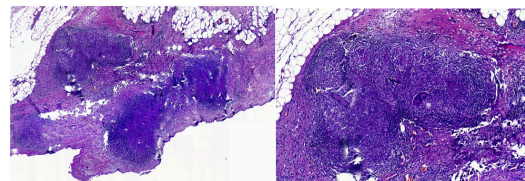


Figure 2 Photomicrograph from a biopsy specimen of the pericardium shows chronic granulomatous pericarditis with caseous necrosis suggestive of tuberculous pericarditis (H&E).

Histopathological studies were suggestive of tuberculosis infection (figure 2). A patient was treated with rifampicin 600 mg/day, isoniazid 300 mg/day, pyrazinamide 2 g/day, ethambutol 1.6 g/day for 2 months followed by more 4 months of rifampicin and isoniazid in same doses. No postsurgical complication was reported. A patient has been followed monthly for one year after surgery and he has been asymptomatic so far. He was diagnosed with hypertension that may explain prior left bundle branch block and concentric hypertrophy. Follow-up echocardiograms at 6 and 12 months did not show relapse.

Constrictive pericarditis is an uncommon disease that often mimics other pathologies. There is fibrous thickening of the pericardium, which impedes normal diastolic filling of the heart. This leads to a reduction in stroke volume by reducing venous return. This commonly results in symptoms of exertional dyspnoea, fatigue and can cause an elevated jugular venous pressure.¹ It has been proposed that the rubbing together of the inflamed, adjacent visceral and parietal pericardial surfaces is responsible for pericardial friction rubs. However, they may be heard even when

Learning points

- ▶ Constrictive pericarditis is uncommon and sometimes mimics other pathologies and should be in the differential for patients with chronic heart failure with preserved ejection fraction.
- ▶ It is important to have a high clinical suspicion, however, echocardiogram, haemodynamic evaluation, CT and MRI are essential in the diagnosis of constrictive pericarditis.
- ▶ Tuberculosis is the most common infectious aetiology in patients with constrictive pericarditis. The treatment is pericardiectomy and pharmacological therapy with rifampin, ethambutol, isoniazid and pyrazinamide for 6 months.



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a large effusion separates the two surfaces, and once the effusion is evacuated the rub often disappears.²

Tuberculous pericarditis is found in less than 1%–5.6% of the constrictive pericarditis patients in North America and Europe, but can happen in more than 30% in endemic countries.³ Progression to constrictive pericarditis, even with optimal antituberculosis therapy (without corticosteroid therapy), is reported in up to 30% of cases.⁴

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