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Long-term echocardiographic follow-up of a patient with constrictive pericarditis treated with antituberculosis drugs and pericardiectomy

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SUMMARY

A middle-aged man presented to the Department of Medicine of our hospital due to exertional dyspnoea, ascites and peripheral oedema. He was later transferred to the Department of Heart Disease as his echocardiography indicated constrictive pericarditis, confirmed by cardiac MRI and cardiac catheterisation. After a thorough investigation, his constrictive pericarditis was assumed to be caused by tuberculosis. He was treated with antituberculosis therapy followed by successful surgical subtotal pericardiectomy, leading to immediate improvement of haemodynamics, regression of symptoms and recovery of cardiac function. The patient remained stable at 5-year echocardiographic follow-up with no evidence of diastolic dysfunction.

BACKGROUND

Constrictive pericarditis (CP) is a severe cardiac disorder which often manifests as a long-term sequela following acute or chronic pericarditis due to infections. A number of cardiotropic viruses, bacterial, fungal and parasitic infections can cause CP. While rare in Western countries, tuberculosis is the most common cause of pericarditis in countries endemic to tuberculosis and HIV.[1] Other important causes of CP are malignancy, rheumatological diseases, uraemia, radiotherapy and cardiac surgery,[2–4] and in some cases the cause may be unknown (idiopathic). Chronic effusive pericarditis and inflammation in any of these conditions may lead to formation of granulomatous tissue, fibrosis, calcification and thickening of the pericardium with adhesion to the heart. Typically, CP results in impaired cardiac function, most often associated with diastolic heart failure,[5] and symptoms such as dyspnoea, fatigue, increased jugular venous pressure, ascites, pleural effusion and peripheral oedema. Although standard transthoracic echocardiography is the cornerstone, a multimodality imaging-based approach is often required to timely diagnose the condition and guide further therapeutic options.

CASE PRESENTATION

A middle-aged male patient presented to the Department of Medicine with shortness of breath and oedema in both legs for a period of 3 weeks. Three months earlier, he was diagnosed with mild liver cirrhosis, which was believed to be induced by excessive alcohol consumption, and was treated with a diuretic. He was a previous smoker, but had no medical history of hypertension, diabetes or coronary artery disease. Following a road traffic accident he suffered as a teenager, he had splenic rupture which required urgent splenectomy and a trauma to the chest causing pneumothorax, which was successfully treated. No acute or late cardiovascular complications were reported. In his work life he travelled extensively on international assignments, but the last year prior to admission he had been on extended sick leave due to back pain. He lived part-time in South-East Asia and part-time in Norway. He was previously treated for hepatitis B and was cured, but had no history of pulmonary tuberculosis or any acquired immune deficiency. He did not report fever, chills or fatigue. Despite a poor appetite, he had gained weight over the past few months.

Physical examination

On admission, the patient's temperature was 37°C, heart rate was 89 beats per minute and blood pressure was 97/64 mm Hg. Oxygen saturation was 98% in room air. His body mass index (weight in kilograms (98 kg) divided by the square of height in metres (1.93 m)) was 26.3 kg/m^2. On auscultation of the heart, there were no murmurs or pericardial friction rub. On chest auscultation, breath sounds were slightly reduced at the lung bases. There were also peripheral signs of right-sided heart failure: increased jugular venous pressure, hepatomegaly and oedema in both legs. No enlarged lymph nodes were palpable. The abdomen was distended and clinically suggestive of ascites, which was confirmed by abdominal ultrasound on admission. Owing to persistent respiratory distress, he underwent an urgent paracentesis to relieve symptoms from severe ascites.

INVESTIGATIONS

The patient's baseline ECG showed sinus tachycardia, 93 beats per minute, and low voltage QRS complex with T-wave abnormalities throughout (figure 1A,B).

His chest X-ray showed bilateral pleural effusion and an increased cardiac shadow suggestive of pericardial effusion (figure 1C,D).

Echocardiogram revealed thickened pericardial layers with increased reflectiveness and pericardial effusion (figure 1E), mild mitral and tricuspid regurgitation, and normal systolic left ventricular

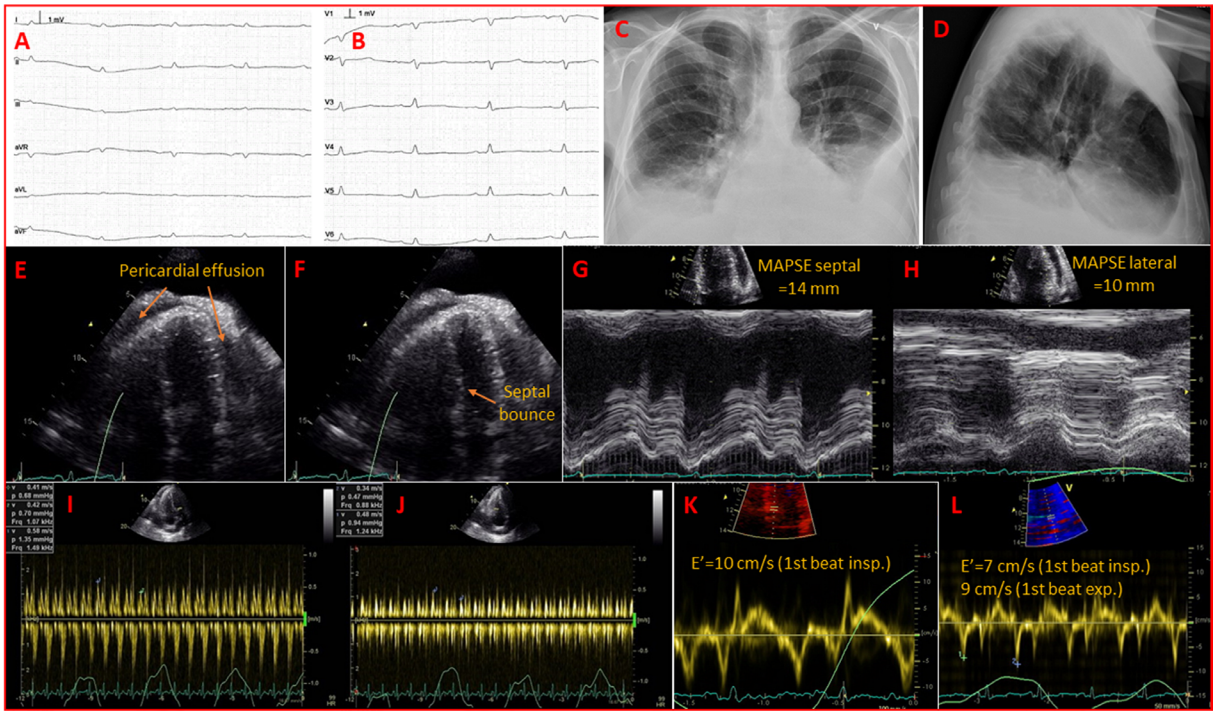
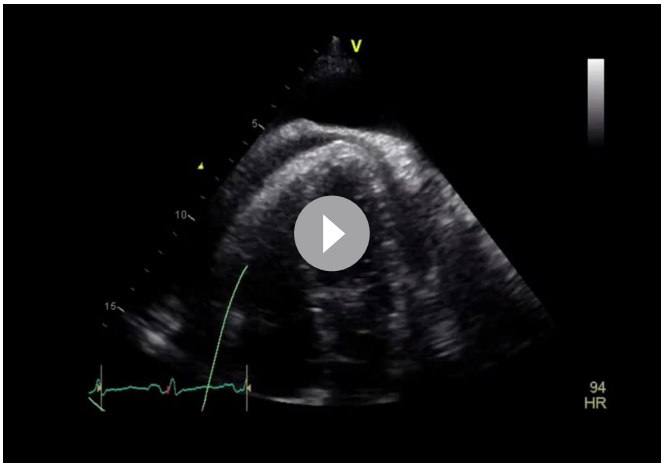


Figure 1 (A) Standard leads and (B) precordial leads of the ECG at admission. (C) Front and (D) lateral views of the chest X-ray showing bilateral pleural effusion. (E) Echocardiographic apical four-chamber view showing pericardial effusion (arrows). (F) Apical four-chamber view and typical septal bounce (arrow). (G) MAPSE (mitral annular plane systolic excursion) at the septal ring of 14 mm and (H) MAPSE at the lateral annulus of 10 mm. (I) Trans-mitral respiratory flow variation (41 cm/s and 58 cm/s) and (J) trans-tricuspid respiratory flow variation (34 cm/s and 48 cm/s). (K, L) Tissue Doppler images and diastolic velocities (E'). Septal E' (K) is higher (10 cm/s) than lateral E' (L) (7 cm/s) (annulus paradoxus).

(LV) ejection fraction (LVEF) of 55%, but mildly reduced right ventricular (RV) long-axis function (tricuspid annular plane systolic excursion 1.5 cm). There was a clear septal bounce or inspiratory shift towards LV (figure 1F, video 1). The inferior vena cava was dilated with little respiratory diameter variation (figure 2A). The echocardiographic findings were strongly suggestive of CP (figure 1E–L).

Cardiac magnetic resonance (CMR) showed pericardial effusion of 14 mm at the RV apex and 11 mm at the LV apex towards the inferior wall and a moderately thickened pericardium of 3 mm, with signs of oedema and pericardial late gadolinium contrast enhancement (LGE) (figure 2B–D). LV end-diastolic volume of 84 mL and end-systolic volume of 39 mL with a stroke volume of 45 mL, cardiac output of 4 L/min and LV ejection fraction of 54% were also noted, as well as slightly dilated right atrium and RV with atrial septal deviation towards the left atrium, suggesting increased filling pressure in the right heart. A dys-synchronous septal motion, particularly in the distal/apical part, was noted.



Video 1 Echocardiographic 4-chamber view at admission showing septal bounce or inspiratory shift towards LV.

Prior to cardiac catheterisation, the patient was restless and had significant shortness of breath. A therapeutic pericardiocentesis was performed; 200 mL of bloody pericardial fluid were drained, which led to some improvement in his breathlessness. Coronary angiography did not show any signs of atherosclerosis in the left main stem, circumflex and right coronary arteries (data not shown). Only a minor atherosclerotic plaque was detected in the proximal left anterior descending artery. The proximal parts of the epicardial coronary arteries showed rapid filling, while the most distant branches showed a delayed flow pattern, suggesting poor microvascular circulation. Overall, there were no flow-limiting coronary stenoses which could explain the patient's symptoms of heart failure. Similarly, there were no significant pressure gradients across the aortic valve. End-diastolic pressure in the LV was 26 mm Hg (figure 2E). On right heart catheterisation, mean pressure in the right atrium was 26 mm Hg, systolic pressure in the RV 37 mm Hg, RV end-diastolic pressure (RVEDP) 27 mm Hg (figure 2F), systolic/diastolic pressure in pulmonary artery 30/22 mm Hg, mean pressure 26 mm Hg and mean pulmonary capillary wedge pressure (PCWP) 29 mm Hg (figure 2G). Both systolic and end-diastolic pressures in the RV were significantly increased, yielding a narrow pulse pressure, where most filling occurred in early diastole. The pressure curves showed a typical 'dip-and-plateau' pattern (square root sign) (figure 2F), consistent with CP.

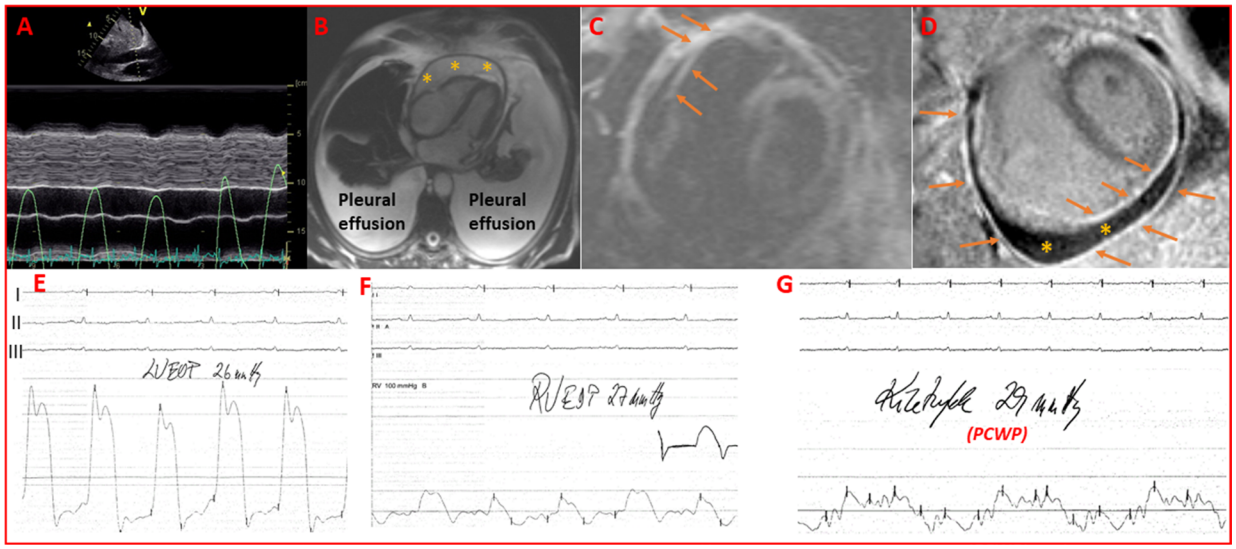


Figure 2 (A) A wide inferior vena cava with restricted respiratory diameter variation. (B) MRI of the heart showing pericardial effusion (asterisks) and bilateral pleural effusions. (C, D) MRI displaying thickened and oedematous pericardium (arrows) enclosing the heart and the pericardial effusion (asterisks). (C) A short-axis T2-weighted short-tau inversion recovery (STIR) image and (D) a corresponding late gadolinium enhancement image. (E–G) Invasively measured pressure curves: (E) left ventricular end-diastolic pressure of 26 mm Hg, (F) right ventricular end-diastolic pressure of 27 mm Hg and (G) pulmonary capillary wedge pressure (PCWP) of 29 mm Hg. End-diastolic pressures are equally elevated in all cardiac chambers.

Laboratory tests

Laboratory tests at admission showed normal haemoglobin of 13.6 g/dL (reference range 11.7–15.3 g/dL), white cell count of 7.1×10^9/L ($3.5–11.0\times10^9$/L) and C reactive protein of 42–38 mg/L (reference range <5 mg/L). Erythrocyte sedimentation rate was 17 mm/hour (reference range 1–15 mm/hour). Serum creatinine level was 95 μmol/L at admission and 54 μmol/L on discharge (reference range 45–90 μmol/L), and estimated glomerular filtration rate (eGFR) was >60 mL/min/1.73 m^2. Serum uric acid was 499 μmol/L (reference range 230–480 μmol/L), sodium 119–121 mmol/L (reference range 137–145 mmol/L), potassium 5.6–4.2 mmol/L (reference range 3.5–5.0 mmol/L), chloride 79–84 mmol/L (reference range 98–106 mmol/L) and albumin 25-30-29 g/L. Liver function tests were mainly normal, with alanine transaminase of 31 IU/L, aspartate aminotransferase 30 IU/L, alkaline phosphatase 105–120 IU/L and bilirubin 30 μmol/L (reference range <19 μmol/L). Troponin T was at 17–19 ng/L (reference range <15 ng/L) and pro-brain natriuretic peptide (pro-BNP) was at 135 pmol/L (reference range <25 pmol/L).

Rheumatological examination ruled out rheumatoid arthritis, systemic lupus erythematosus, scleroderma and sarcoidosis.

We did a comprehensive work-up to identify any infectious aetiology. He had serological evidence of prior infections with Epstein-Barr virus (EBV), measles, mumps, varicella zoster virus (VZV) and hepatitis B (HBV; positive core and surface antibodies, negative PCR). He had hepatitis A IgG antibodies, compatible with prior infection or vaccination. Serological testing was negative for cytomegalovirus (CMV), hepatitis C (HCV) and HIV. PCR on blood was negative for EBV, CMV, HBV and HCV. Serological tests were negative for a number of bacterial infections such as borreliosis, brucellosis, leptospirosis, *Coxiella burnetii* (Q fever), syphilis, *Mycoplasma pneumoniae* and respiratory *Chlamydia* species. He had non-specific borderline levels of *Bordetella pertussis* IgG antibodies. Urine culture and PCR excluded *Neisseria gonorrhoeae* and *Chlamydia trachomatis* infections.

Fungal infections such as aspergillosis, candidiasis, coccidiosis and *Nocardia* were negative by culture and PCR. Serological testing for parasitic infection was negative for toxoplasmosis (IgM and IgG), schistosomiasis, *Strongyloides stercoralis* and *Toxocara canis*.

The pericardial fluid was blood-stained, contained an elevated level of leucocytes (2.6×10^9/L, upper limit 0.2×10^9/L), and was an exudate according to Light's criteria (ratio of protein in the pericardial fluid and serum ≥0.5 (40 g/L: 74 g/L); ratio of lactate dehydrogenase (LDH) in the pericardial fluid and serum higher than 0.6 (281 U/L:183 U/L); and pericardial fluid LDH higher than two-thirds of the upper limit of LDH for serum (281 U/L, upper limit 205 U/L). Adenosine deaminase enzyme testing was not available at the time, and interferon gamma release assay (IGRA) testing was not performed on the pleural fluid. PCR on pericardial fluid was negative for enteroviruses, herpes simplex virus (HSV) 1 and 2, VZV and CMV, but positive on EBV DNA, which was concluded to represent prior EBV infection, consistent with serological test results.

Multiple pericardial fluid samples were negative for bacterial, mycobacterial and fungal culture.

Histological examination of a pericardial biopsy showed fibrinous haemorrhagic pericarditis, with lymphoplasmocytic infiltration, but no evidence of malignancy or granulomas. Pericardial biopsy PCR for 16sDNA showed no bacterial DNA, and specific PCRs were negative for tuberculosis, HSV 1 and 2 and VZV. There was no growth in culture for mycobacteria and anaerobic or aerobic bacteria. One sample yielded growth of a single colony of *Staphylococcus warneri*, considered to be a probable contaminant.

Multiple sputum samples were negative on microscopy of Ziehl-Neelsen stained smears, on PCR and on mycobacterial culture. IGRA (QuantiFERON) was negative. Chest X-ray showed bilateral pleural effusions, but no infiltrates or other signs suspicious for tuberculosis. CT scan of the thorax and abdomen confirmed thickened/calcified pericardium with effusion, abundant pleural fluid and compression atelectasis and

ascites, and revealed enlarged glands in the mediastinum (longest diameter 1.2 cm) and retroperitoneally and intraperitoneally.

DIFFERENTIAL DIAGNOSIS

Pericardial and pleural effusion is rarely seen in mild forms of liver cirrhosis, and the patient did not show evidence of failure of liver protein synthesis. Furthermore, absence of proteinuria ruled out nephrotic syndrome. An emergency echocardiogram showed an advanced stage of effusive-constrictive pericarditis. This was further confirmed by CMR scan and cardiac catheterisation. He did not have any previous cardiac surgery or radiation to the mediastinal region. Hence, the CP was by definition primary. The patient repeatedly underwent drainage of pleural effusion, ascites and pericardial effusion with cytological investigation, and no malignant cells in the pericardial fluid were found. Histological examination of a pericardial biopsy did not reveal the aetiology and importantly did not find evidence of malignancy. Similarly, extensive evaluation for a wide number of infectious causes including cardiotropic viruses, bacteria, mycobacteria, parasites and fungi did not provide conclusive results. As he had worked in several tropical countries and lived in tuberculosis-endemic settings in South-East Asia over time, tuberculous pericarditis was suspected. In such a setting, constrictive, calcified pericarditis is highly suspicious for tuberculous aetiology. The finding of borderline enlarged mediastinal and abdominal glands was also compatible with tuberculosis. Although comprehensive work-up did not provide aetiological proof of tuberculosis, we concluded with a diagnosis of presumptive tuberculous pericarditis based on history of likely exposure in an endemic setting, compatible clinical features and lack of an alternative explanation. We treated him ex juvantibus with standard antituberculous treatment followed by surgery.

TREATMENT

During the first 3 weeks after admission, the patient underwent an extensive work-up to find the aetiology of his CP and received mainly supportive treatment. He had hyponatraemia at admission probably caused by spironolactone and the medication was stopped until the serum sodium chloride level normalised. He also received sodium chloride tablets during hospitalisation. Tuberculosis was thought to be the most likely aetiology for his chronic CP, although cytological and serological tests including PCR were negative for tuberculosis. The patient was started on empirical treatment according to guidelines, receiving rifampicin 750 mg once daily, isoniazid 375 mg once daily, pyrazinamide 2000 mg once daily and ethambutol 1375 mg once daily, with the addition of pyridoxine 40 mg once daily and prednisolone 60 mg once daily. Due to pre-existing mild degree of liver cirrhosis and potential hepatotoxic effects of antituberculous drugs, liver function tests were carefully monitored. After 1 week ethambutol was discontinued due to visual impairment. The following week the three remaining antituberculous drugs were temporarily stopped due to rise in serum bilirubin level. Rifampicin 450 mg once daily and isoniazid 300 mg once daily were reintroduced, while pyrazinamide was replaced with moxifloxacin 400 mg once daily. After 6 weeks of treatment with antituberculous drugs, the patient still suffered breathing difficulty, turning into the New York Heart Association functional class IV. Hence, following discussion at the multidisciplinary heart team, he underwent a successful subtotal pericardiectomy on the beating heart through a median sternotomy and with the use of extracorporeal cardiopulmonary bypass circulation. The whole heart was completely adherent with the adjacent calcified/thickened pericardium. The heart was successfully released from the pericardial sack. This immediately improved haemodynamics as reflected by invasive pressures, as well as clinical improvement and relief of shortness of breath. A week later, a final pericardiocentesis was performed causing gradual clinical improvement. The pericardial, pleural and peritoneal effusions regressed with no further accumulation of fluids in the chest or abdomen. He had an uneventful postoperative recovery and was able to walk on his own without the need for oxygen supplementation. After a total of 8 weeks stay in the hospital, he was discharged to a rehabilitation institution. He continued antituberculous treatment with moxifloxacin (Avelox) 400 mg once daily, isoniazid 300 mg once daily and rifampicin (Rimactan) 450 mg once daily, as well as pyridoxine 40 mg once daily, cholecalciferol/calcium carbonate (Calcigran Forte) 1000/800 mg once daily, spironolactone 50 mg once daily and bumetanide (Burinex) 1 mg two times per day. Prednisolone 5 mg once daily was tapered in 1 week. Sodium chloride tablets were stopped at discharge.

OUTCOME AND FOLLOW-UP

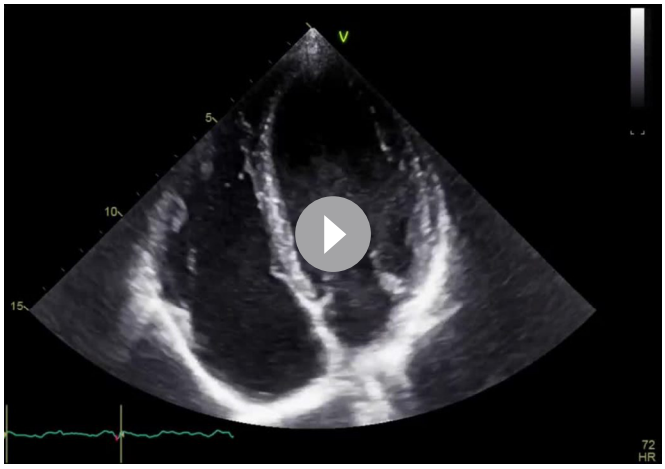
Antituberculous drugs were stopped after 6 months of treatment. The patient felt well, had no longer breathing difficulties and gained a stable weight of 76 kg. His blood pressure was 110/65 mm Hg and his heart rate was 80 beats per minute. There were no signs of elevated jugular venous pressure, ascites or peripheral oedema. Auscultation of the heart and lungs was unremarkable.

C reactive protein was 6 mg/L, haemoglobin 12.6 g/dL and alanine transaminase 32 IU/L. Troponin T was 18 ng/L and pro-BNP was 31 pmol/L. Echocardiography showed normal LV and RV systolic function. There were no signs of pericardial effusion and constrictive physiology on follow-up visits.

Repeated echocardiograms at 1, 2, 3 and 5 years of follow-up showed completely normal systolic and diastolic function and no signs of constrictive physiology or pericardial effusion (video 2 and figure 3). The patient also remained symptom-free in all these follow-up visits. A repeated CMR was not performed during follow-up to assess whether the LGE persisted or if it was due to inflammation.

DISCUSSION

There are three important learning points to this case study: (1) echocardiography at presentation showed almost all typical



Video 2 Echocardiographic apical 4-chamber view at 5-years follow-up showing normal systolic and diastolic function and no signs of constrictive physiology or pericardial effusion.

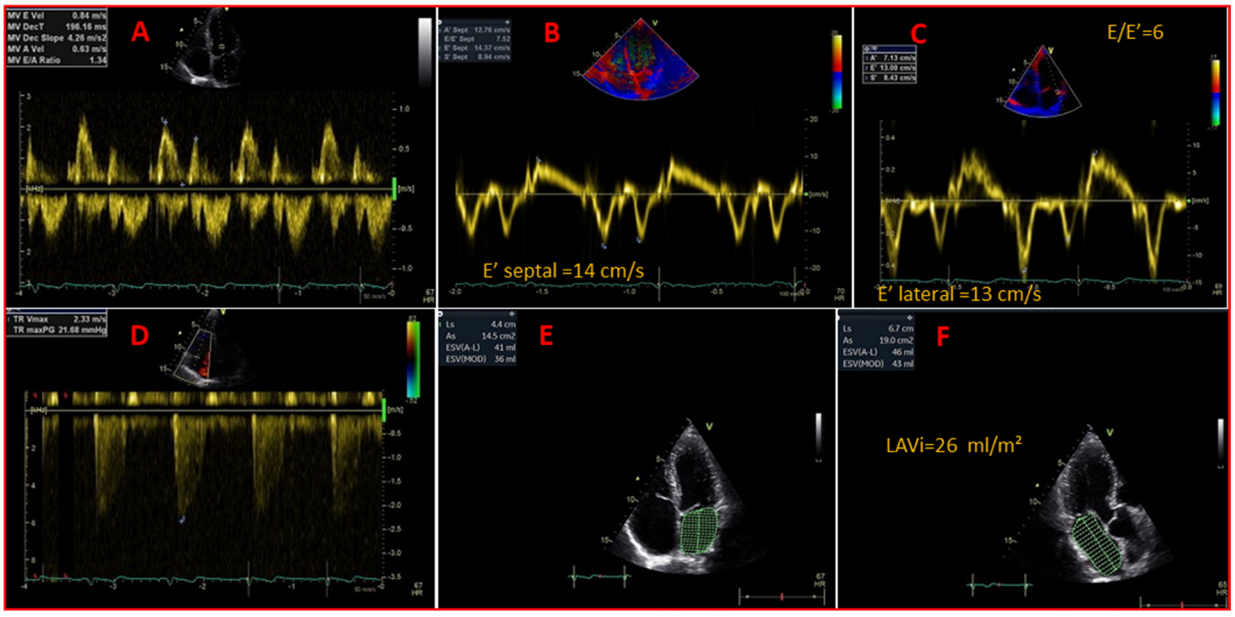


Figure 3 Normal diastolic function according to the current European guidelines. (A) Trans-mitral E velocity of 0.84 m/s and (B, C) tissue Doppler velocities of E′ septal of 14 cm (B) and E′ lateral of 13 cm/s (C), yielding an E/E′ ratio of 6.2. (D) The peak velocity of tricuspid regurgitant jet (TR Vmax) of 2.3 m/s and (E, F) left atrial index (LAVi) of 26 mL/m^2, which are all normal.

features of CP, which was further confirmed by CMR and cardiac catheterisation; (2) cardiac surgery with subtotal pericardiectomy resulted in immediate improvement of haemodynamics, symptoms and restoration of cardiac function, which remained stable even at 5-year echocardiographic follow-up; and (3) tuberculosis is a frequent cause of CP globally and is difficult to diagnose and must be kept in mind as a differential diagnosis even in a low-endemic setting such as Northern Europe.

Constrictive physiology

In CP, the thickened and rigid pericardium and adhesions with the heart limit the expansion of the heart as well as inhibit the transmission of physiological intrathoracic pressure changes.[2] This results in an elevation and equalisation of diastolic pressures in all cardiac chambers and fixed cardiac volumes. Patients often have sinus tachycardia, or rapid supraventricular arrhythmias, most often atrial fibrillation, and a biatrial enlargement.[3] The pro-BNP level may be normal or slightly elevated. Echocardiography is often the first-line diagnostic modality and prompts accurate diagnosis. In our patient, an echocardiography at admission indicated CP. The pericardial layers were thickened and hyper-reflective, and there was a clear exaggerated abnormal motion of the interventricular septum with bowing into the LV during inspiration and normalisation during expiration (video 1). The basic physiological explanation for this phenomenon, also called septal bounce, is the flow increase to the right atrium and RV and decrease to the left atrium and LV during inspiration, with the opposite effect during expiration. The reduced venous return to the left heart during inspiration results in septal shift towards LV. The other echocardiographic criteria for CP, as recommended by the 2015 European Society of Cardiology guidelines for the diagnosis and management of pericardial diseases and by Mayo Clinic (box 1), were also present in our patient.[4 6]

Furthermore, serial echocardiograms after the completion of antituberculosis treatment and pericardiectomy showed sustained normal cardiac function (LVEF) without any sign of constrictive physiology for several years of follow-up. Of note, at 5-year echocardiographic follow-up, there were no signs of diastolic dysfunction (figure 3) as defined by the current European guidelines.[7] In some cases of transient CP caused by tuberculosis, LVEF may be within the normal range, but regional or global longitudinal strain by speckle tracking echocardiography may be reduced, reflecting subclinical involvement of the myocardium.[8] This highlights the importance of monitoring myocardial function by strain imaging as supplement to routine LVEF on serial echocardiograms.

In CP, pericardiectomy may be required in some cases to normalise end-diastolic pressures in cardiac chambers and cardiac output. In our patient, an early cardiac surgery with removal of adherences and release of the heart from the surrounding calcified/thickened pericardium led to immediate improvement of haemodynamics and symptoms and cardiac function recovery, with no evidence of residual constriction during long-term echocardiographic follow-up. However, it is also important to note

Box 1 Echocardiographic criteria for constrictive pericarditis (CP) recommended by the 2015 European Society of Cardiology guidelines for the diagnosis and management of pericardial diseases and by Mayo Clinic[4 6]

Parameters

- Thickened/calcified pericardium (≥4 mm)*.
- Septal bounce (respiration-related ventricular septal shift).
- Respiratory variation of the mitral inflow peak E velocity of >25%.
- Medial mitral annular e′ velocity ≥8 cm/s (annulus paradoxus; e′ septal > e′ lateral).
- Inferior vena cava diameter >21 mm or <50% change and dilated hepatic veins.
- Decreased expiratory forward diastolic hepatic vein velocities with large reversals (expiratory diastolic reversal/forward velocity ratio ≥0.79).

*Approximately 20% of patients with CP may have normal pericardium.[2]

that in a chronic effusive pericarditis, a residual constriction and chronic irreversible myocardial dysfunction may last even following a successful pericardiectomy, which highlights the importance of knowing the underlying aetiology.

Pericarditis is a rare extrapulmonary manifestation of tuberculosis, evident in 1% of cases of tuberculosis at autopsy. Although tuberculosis accounts for only 4% of pericarditis cases in high-income countries, it is by far the most common cause of CP in endemic areas in Africa and Asia, particularly where there is high prevalence of HIV infection.[1] In Sub-Saharan Africa, tuberculosis accounts for up to 70% of pericarditis cases in the general population and more than 90% of cases among people living with HIV.[9]

Unfortunately, tuberculous pericarditis is difficult to confirm microbiologically, with low sensitivity of pleural fluid examination with commonly available methods such as microscopy of Ziehl-Neelsen stained fluid (sensitivity 0%–42%), PCR (50%) and mycobacterial culture (sensitivity 53%–75%),[1 10] particularly in later stages of the disease. Indirect tests such as IGRA (QuantiFERON) can be negative in 15%–20%, and Mantoux test is difficult to interpret in persons who have received BCG vaccine, such as our patient.

Since untreated tuberculous pericarditis is lethal in 80%–90% of cases and appropriate treatment can reduce mortality to 8%–17%, treatment must often be started ex juvantibus before the results of diagnostic work-up is ready.[1] In retrospect, the diagnosis of tuberculous pericarditis was not ascertained unequivocally in our patient. However, considering the clinical findings of an exudative, constrictive, calcified pericarditis and enlarged mediastinal glands in a person with a history of living in an epidemic setting, and the absence of other plausible explanations, tuberculous aetiology was likely and ex juvantibus treatment warranted. The fact that he recovered and remained healthy 5 years after antituberculous treatment and pericardiectomy, rules out malignancy as a main differential diagnosis and makes tuberculous pericarditis more likely in retrospect. His history of splenectomy after a prior car accident incurs a twofold increased risk for tuberculosis.[11] On the other hand, the chest trauma could have also caused haematopericardium, which in principle could have caused pericarditis, although it is unlikely for this to manifest clinically only several decades later.

Up to half of tuberculous pericarditis cases get permanent sequela in the form of CP, commonly referred to as 'Panzerherz' (armoured heart).[1] Prednisolone is used to hasten improvement and reduce the incidence of CP,[12 13] particularly in patients with large effusions and high risk. Current guidelines recommend pericardiectomy if the patient's condition is not improving or is deteriorating after 8 weeks of antituberculosis drugs,[4] which was also the case in our patient. Hence, early intervention with pericardiectomy should be considered in patients who fail to respond to conservative treatment with antituberculosis drugs and other supportive measures.[14] Pericardiectomy for CP is associated with low mortality rates and good early and late outcome.

To conclude, CP is associated with a diastolic heart failure syndrome and persistent symptoms. A careful multimodality imaging-based approach is required to timely diagnose the condition and guide further therapeutic options. Despite a thorough investigation, it is not always possible to determine the exact aetiology of CP. In unresolved cases of pericarditis in patients living in endemic countries, tuberculous aetiology should not be overlooked and ex juvantibus treatment is justified. For patients who fail to respond to conservative treatment, an urgent surgical solution with pericardiectomy may improve haemodynamics and symptoms and restore normal cardiac function, with rapid recovery and good clinical outcome. Serial echocardiograms can provide important information on short-term and long-term therapeutic success following a successful pericardiectomy.

Learning points

- Transthoracic echocardiography is the first-hand imaging modality for assessment of constrictive pericarditis (CP); however, a multimodality imaging-based approach is often required to timely diagnose the condition and guide further therapeutic decisions.
- Tuberculosis is a frequent cause of CP worldwide, is difficult to diagnose and must be kept in mind as a differential diagnosis even in a low prevalence setting such as Northern Europe.
- Cardiac surgery with pericardiectomy is associated with immediate improvement of haemodynamics and symptoms and leads to restoration of cardiac function, which may remain stable for many years.

Contributors All authors fulfil the authorship criteria. SS, ØB and TL contributed to the conception of the case report, acquisition and interpretation of data, and drafting the paper. ØB, BB and RH treated the patient and critically revised the manuscript for important scientific content. All authors approved the final submission.

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