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CASE REPORT
Cardiac imaging

Complete reversal of constriction physiology with antitubercular treatment without steroid: a case report

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Background Constrictive pericarditis is a chronic inflammation of the pericardium leading to the thickening of the pericardium that restricts cardiac filling. Globally tuberculosis is the commonest aetiology of constrictive pericarditis. Though normally considered to be an irreversible pathology; which requires surgical pericardiectomy, in early stage of the disease antitubercular therapy (ATT) along with steroids and other anti-inflammatory therapy can reverse the pathology. But, complete reversal of constrictive physiology with ATT without any anti-inflammatory drugs is not documented.

Case summary Here, we describe a case where a 54-year-old lady presented with progressive dyspnoea and pedal oedema for 2 years along with anorexia and weight loss for two months. Two-dimensional echocardiography and computed tomography scan was suggestive of constrictive pericarditis. In view of systemic features and high acute phase reactants, patient was started on oral ATT without any steroids. After 4 months, constriction physiology was completely reversed.

Discussion Constriction physiology if treated timely can be largely reversed only with ATT without adjuvant anti-inflammatory therapy. Further studies are required to find out the specific indications of anti-inflammatory therapy in tubercular constrictive pericarditis.

Keywords Case report • Constriction • Tuberculosis • Antitubercular therapy • Steroids • Echocardiography • Computed tomography

Learning points

- Tuberculosis is the commonest aetiology of chronic constrictive pericarditis (CCP) in the Indian subcontinent.
- Tubercular CCP can be reversed with antitubercular therapy if started well on time and continued properly.
- The indication of adjuvant steroid therapy needs to be properly defined, may not be indicated in all cases.

Introduction

Constrictive pericarditis (CP) is a form of diastolic heart failure that arises because pericardium becomes thick and inelastic and thus inhibits cardiac filling.[1] Tuberculosis is the commonest cause of CP worldwide. Case series from China, Iran, and South Africa have reported tuberculosis CP as the cause of CP in 22.2–91% of cases.[2–4] In contrast, European and North American series have reported much lower rates of tuberculosis CP (5.6% or less); most cases in

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these areas of the world are idiopathic or related to prior cardiac surgery or chest irradiation.[5–9]

The treatment of tubercular constrictive pericarditis includes anti-tubercular therapy (ATT) and anti-inflammatory therapy and in cases where symptoms do not improve one ultimately requires surgical pericardiectomy.[10] Although historically patients with tubercular constrictive pericarditis have been treated with both ATT and steroids,[11,12] steroid usage in this cohort has been mostly empirical as the evidence for anti-inflammatory therapy is weak.[13]

Timeline

A 54-year-old lady	Presented with New York Heart Association II dyspnoea for 2 years with intermittent chest pain
Transthoracic echo and contrast enhanced computed tomography thorax	Made the diagnosis of constrictive pericarditis
Patient started on oral anti-tubercular therapy without steroids	Continued for 6 months
After 6 months	There was a complete reversal of constriction physiology

Case presentation

A 54-year-old woman presented with a 2-year history of progressive dyspnoea on exertion. At the time of presentation, she had intermittent (angina) chest pain, pedal oedema, and dyspnoea at minimal exertion. For the last 2 months, she had anorexia and weight loss (4 kg in 1 month). There were no documented episodes of fever. Her past medical history was significant for hypothyroidism, well-controlled on levothyroxine supplementation.

On evaluation, the patient was normotensive, pulse regular, and afebrile. Systemic examination was unremarkable except for mild bilateral pedal oedema. Electrocardiogram showed normal sinus rhythm and was otherwise unremarkable. Her erythrocyte sedimentation rate (ESR) was 56 and C-reactive protein (CRP) 22.4 mg/L; normal <7.5 mg/L. Her N-terminal prohormone of brain natriuretic peptide level was 74.6 pg/mL. HIV antibodies were negative by enzyme-linked immunosorbent assay. Two-dimensional echocardiography (*Figure 1*) revealed mild pericardial effusion with pericardial thickness of 0.58 cm. Inferior vena cava was dilated (2.12 cm with no respiratory variation) and significant trans-mitral and trans tricuspid respiratory flow variation suggestive of effusive constrictive pericarditis. Though annulus reversus could not be documented (e'^{l} = 8.70 cm/s and e's = 7.72 cm/s), e'^{l} and e'^{s} were almost equal and e'^{l} was reduced. *E*/e' were 8.48 for septal and 7.53 for lateral annulus. Contrast-enhanced tomography of thorax (*Figure 2*) revealed mild pericardial effusion with thickened pericardium [along right ventricular (RV) free wall 6.9 mm and along left ventricular (LV) free wall 7.5 mm] with biatrial enlargement; it also revealed few homogenous subcentimetric prevascular, pre-, and para- tracheal lymph nodes without any necrosis. There was left-sided pleural effusion along with atelectatic bands in bilateral lung bases, left lingula, and bilateral upper lobes and traction bronchiectasis along medial segment of right middle lobe. Overall the computed tomography (CT) images were suggestive of chronic infective aetiology.

Cardiac catheterization revealed equalization of diastolic pressures with significant respiratory variation in LV and RV pressures. Coronary angiography revealed normal epicardial coronaries. These findings strongly suggested a constrictive physiology.

As tuberculosis is the commonest aetiology of constrictive pericarditis in the Indian subcontinent, and as there was evidence of lung involvement, the patient was started on ATT (isoniazid + rifampicin + pyrazinamide + ethambutol for 2 months followed by isoniazid and rifampicin for 4 months). The patient improved over the next 4 months, there was return of appetite with weight gain; and the repeat echocardiography (*Figure 3*) was normal with no features of either constriction or effusion. Repeat ESR was 31 and CRP came down to 8 mg/L. Repeat CT also revealed normalization of pericardial thickening and enhancement when compared with the earlier. It also revealed clearance of pleural effusion, lymphadenopathy, and lung findings when compared with the earlier CT. To evaluate the symptoms and to document the functional capacity of the patient, treadmill test was done which was normal with good functional capacity.

Discussion

Tubercular constrictive pericarditis is normally treated with ATT and adjuvant steroids. According to standard literature 75% of patients improve on this treatment.[11] However, there is an entity termed transient pericardial constriction, which have been described as spontaneous and permanent resolution of constrictive physiology on serial echocardiograms.[14] Most cases of transient constrictive pericarditis are idiopathic, mostly secondary to viral pericarditis. Typically, transient CP is managed with anti-inflammatory pharmacological therapy, which includes non-steroidal anti-inflammatory drugs, colchicine, or steroids in refractory cases. The typical clinical course implies the presence of acute inflammatory pericarditis with constriction due to inflammation, which resolves once the inflammatory process is treated.[15]

Alraies *et al.* have analysed the factors could predict reversibility of constriction and found that septal bounce, increased pericardial thickness, and normal inflammatory markers are predictors of non-reversibility of constrictive physiology, which represent less inflammation and more fibrosis as pathology of pericardial constriction, while higher inflammatory markers and mitral valve annular velocity are predictors of reversibility and potential to response to medical therapy.[16] Using tissue Doppler, septal and lateral *E*/e' were higher among those who had reversible disease (11.36 ± 4.8 vs. 7.25 ± 1.9 $P = 0.001$ and 9.8 ± 3.9 vs. 5.9 ± 2.4, $P = 0.001$), respectively. Our patient had a CRP of 22.4 mg/L, and her initial *E*/e' were 8.48 for septal and 7.53 for lateral annulus, which was significantly high, and these indicate that our patient was in an inflammatory state, which could be

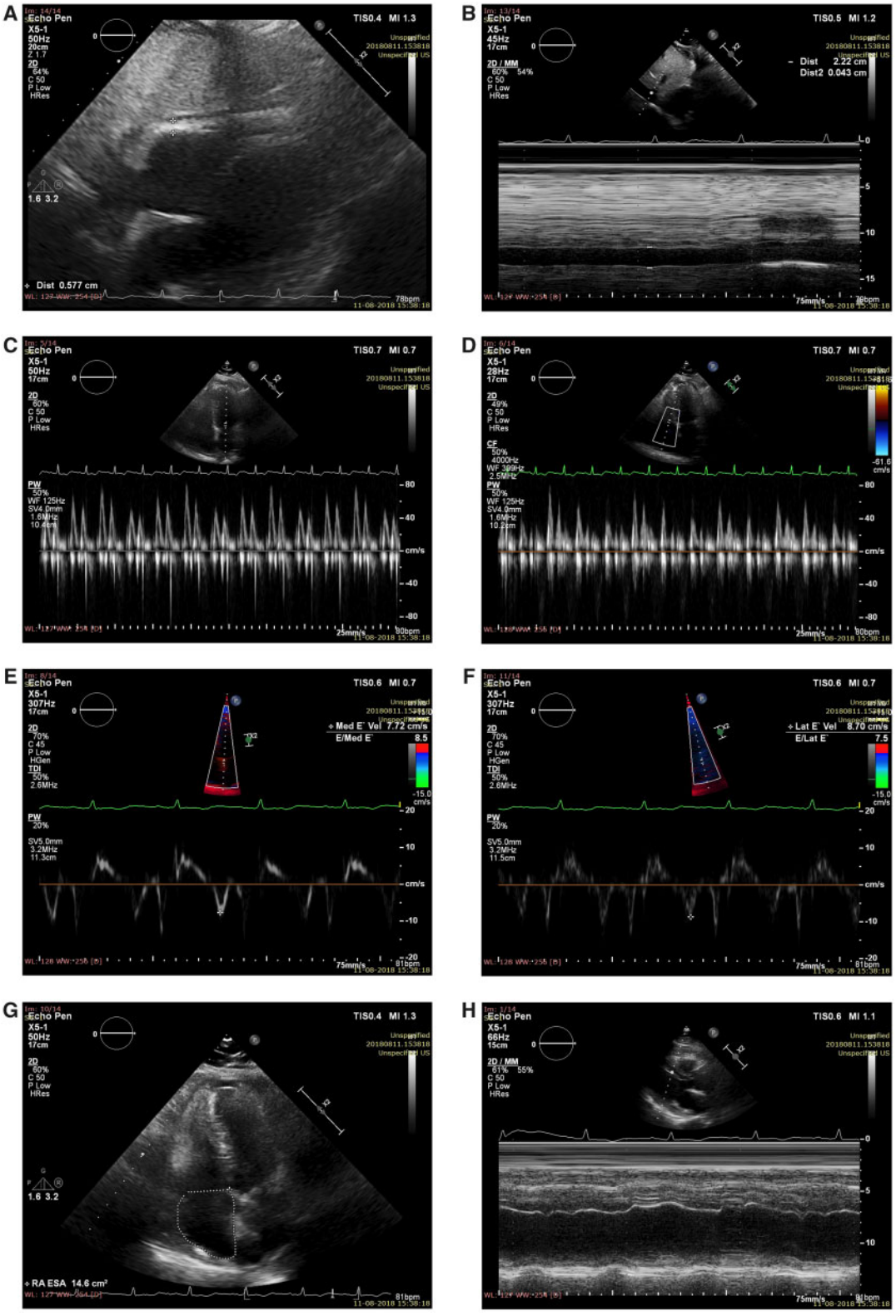

Figure 1 Echocardiographic findings in our patient: (*A*) subcostal view showing thickened pericardium, (*B*) inferior vena cava showing dilatation with no respiratory variation, (*C*) pulse wave Doppler across mitral valve showing significant respiratory variation, (*D*) pulse wave Doppler across tricuspid valve showing significant respiratory variation, (*E*) pulse wave tissue Doppler along medial annulus of mitral valve, (*F*) pulse wave tissue Doppler along lateral annulus of mitral valve, (*G*) apical four-chamber view showing normal-sized ventricles with dilatation of right and left atria (dotted area is right atrium, which is calculated to be 14.6 cm^2), and (*H*) m mode echocardiography findings along parasternal left-axis view showing good left ventricular function.

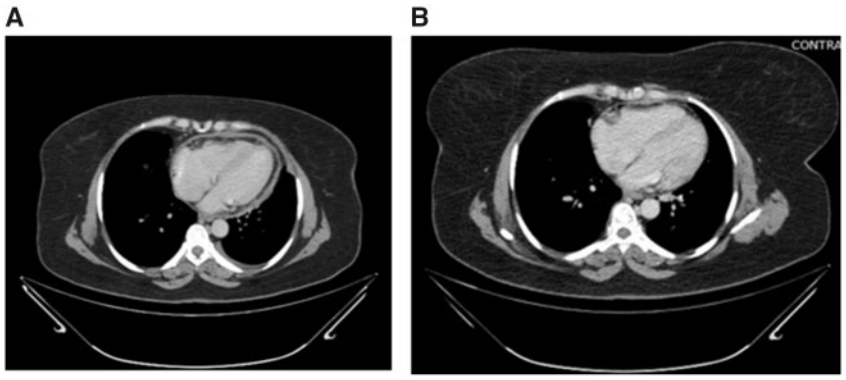

Figure 2 (*A*) Contrast-enhanced computed tomogram of thorax showing thickened and enhancing pericardium suggestive of constrictive pericarditis. (*B*) Repeat computed tomography showing almost normalization of pericardial thickness and enhancement.

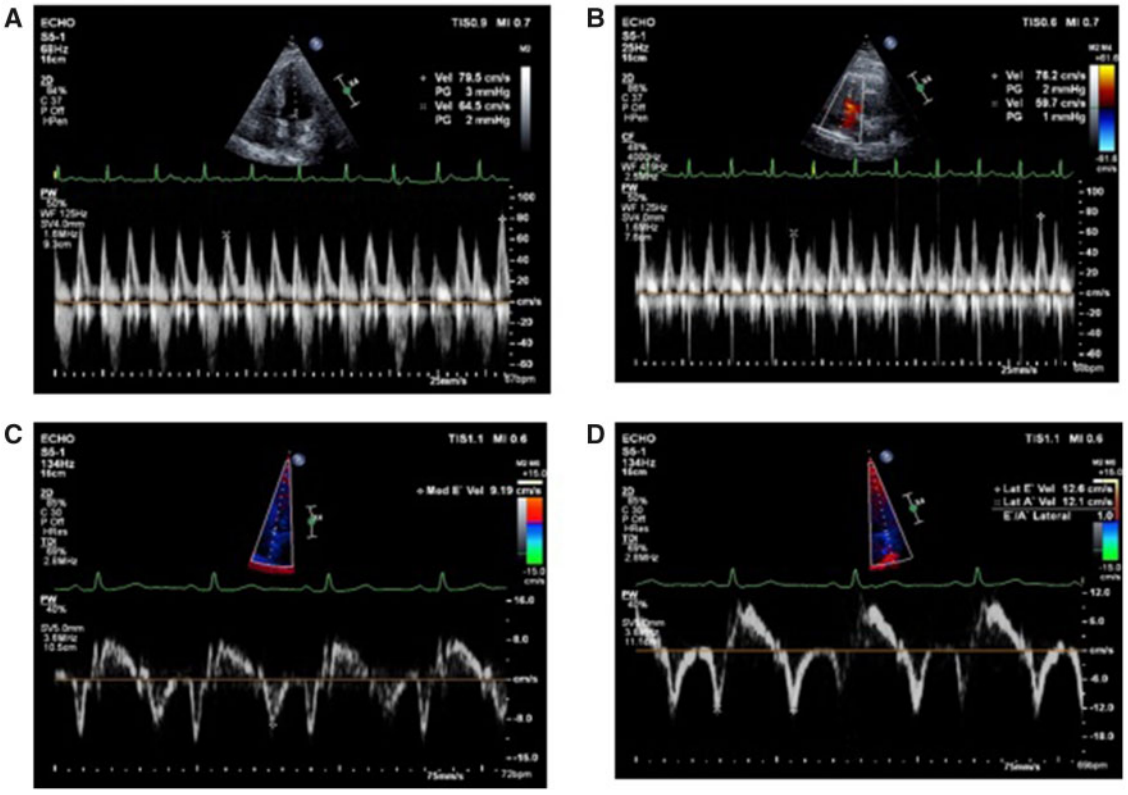

Figure 3 Echocardiographic findings in follow-up (*A*) pulse wave Doppler across mitral valve showing no significant respiratory variation, (*B*) pulse wave Doppler across tricuspid valve showing no significant respiratory variation, (*C*) pulse wave tissue Doppler along medial annulus of mitral valve, and (*D*) pulse wave tissue Doppler along lateral annulus of mitral valve.

the reason behind its reversibility. However, our patient received no steroids or other anti-inflammatory drugs, yet the constrictive physiology got completely reversed. In fact, the European guidelines give steroid a Class IIb indication in HIV negative constrictive pericarditis.[13] In our case, we were not sure with the aetiology; but in the Indian subcontinent, as tuberculosis is the commonest aetiology, we started ATT and we thought we should follow-up the patient and if required other anti-inflammatory therapy may be started. As our patient showed improvement in repeat echocardiography, we thought we should continue ATT and not give steroids. To our surprise, our patient improved completely on follow-up with ATT alone.

Hence, there is a need for specific guidelines for use of adjuvant steroids in tubercular constrictive pericarditis; further studies are required to find out specific indications.

Lead author biography

Dr Avishek Bagchi obtained his MBBS degree from Calcutta Medical College in 2010, MD degree in general medicine from Maula Azad Medical College, New Delhi in 2014, and his DM degree in cardiology in 2018 from Gobind Ballabh Pant Hospital, New Delhi. Currently, he is an associate consultant at Max Super Speciality Hospital in Saket, New Delhi.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

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