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***Case Report***

# ANAESTHETIC MANAGEMENT OF A CASE OF TUBERCULAR CONSTRICTIVE-EFFUSIVE PERICARDITIS WITH BILATERAL PLEURAL EFFUSION POSTED FOR PERICARDIECTOMY

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## ABSTRACT
Perioperative management of tuberculous constrictive-effusive pericarditis (TBCEP) is always a challenge for the anaesthesiologist. Constriction develops initially, followed by effusion and may produce clinical findings typical of cardiac tamponade.
Our patient had bilateral pleural effusion along with TBCEP. He was a drug nonresponder. Many intraoperative and postoperative problems were encountered during anaesthesia for pericardiectomy. Finally, recovery was total and uneventful.

***Keywords:*** *Constrictive Effusive Pericarditis, Pleural Effusion, and Tuberculosis*

## INTRODUCTION
*Tuberculous pericarditis* occurs in 1 to 2% of patients with pulmonary tuberculosis (Brien and Pennington, 2005). The incidence has clearly declined with concomitant decline in prevalence of tuberculosis (Brien and Pennington, 2005) in developed countries but it is still common in our country. Pericardial infection occurs via extension of infection from lung, tracheobronchial tree, adjacent lymph nodes, spine or sternum or by miliary spread (Brien and Pennington, 2005). Inflammation affects both parietal and visceral pericardium. Fluid and fibrinous deposits are found in the pericardial space, eventually both layers of pericardium fuse affecting diastolic filling resulting in low cardiac output CSTnet [No Date].

## CASES
A 17 year old male patient was admitted to our hospital with progressive dyspnoea, precordial pain, bilateral pedal oedema, ascites, fever and a history of significant weight loss. He was taking isonicotinic hydrazide (INH) 300mg, rifampicin (RMP) 450mg and ethambutol (ETM) 800mg daily for the last two months for pulmonary tuberculosis diagnosed by chest x-ray and sputum examination. On examination he had pallor, bilateral pleural effusion, muffled heart sounds, hepatomegaly, ascites, pedal oedema, palpable cervical lymph nodes (bilateral – posterior triangle) and increased jugular venous pressure.
***Investigations:*** *Blood*- Haemoglobin 7gm%, neutrophil 60%, lymphocyte 35% with normal blood sugar, urea and creatinine; *Liver Function Tests*- Total protein 4.5gm/dl, albumin 3gm/dl, INR 1.66, marginally raised alanine transaminase and aspartate transaminase; *Chest x-ray*- Revealed bilateral pleural effusion and pericardial effusion as evidenced by flask shaped heart (X-ray 1); *E.C.G.*- Low voltage complex; *USG*- Bilateral pleural effusion, hepatomegaly, mild ascites. Fluid was drained by pleurocentesis – 500-800 ml/day, 1.5 liter was removed to relieve respiratory distress. Pleural fluid sent for biochemical and cytological examination – was found to be exudative with predominant lymphocytes; *Echo*- Moderate pericardial effusion. Pericardium thickened with fibrinous bands. No features of cardiac tamponade; *Lymph node (neck) biopsy and pleural biopsy*- Histopathology confirmed tuberculosis.
Treatment started with digoxin 0.25mg once daily (OD), frusemide 40 mg OD and deriphylline 100mg three times daily along with anti tuberculous drugs (ATD) which he was taking previously. He developed jaundice (total bilirubin 8 mg), two weeks later for which ATD was stopped and prednisolone 30 mg

daily was started with ciprofloxacin 500mg twice daily. *Repeat Chest x-ray*- Bilateral pleural effusion and pericardial effusion (X-ray 2); *Repeat Echo*- Thick pericardium with moderate effusion, dense fibrinous changes, and diastolic collapse of right ventricle with grade 1 mitral regurgitation.

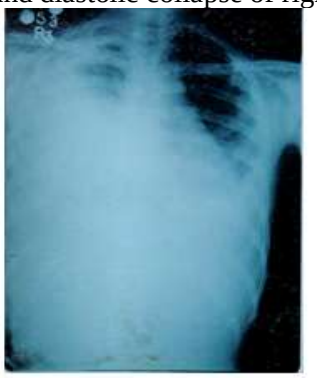

**Figure 1**

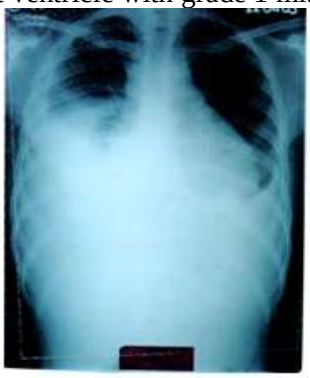

**Figure 2**

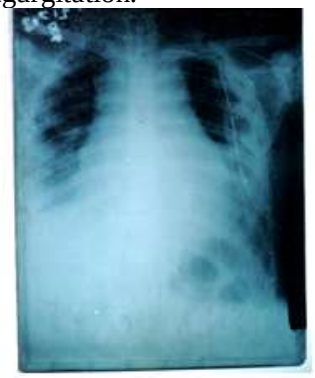

**Figure 3: Before O.T.**

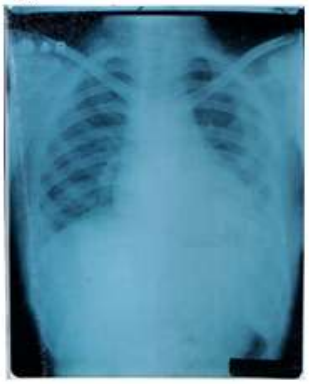

**Figure 4**

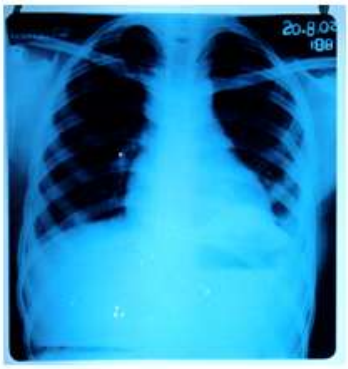

**Figure 5: During Discharge**

Patient was prepared for pericardiectomy when jaundice subsided (after four weeks, bilirubin <2mg). Preoperatively patient had Haemoglobin 10gm/dl (after 3 units of packed cell transfusion), was normotensive, pulse and urine output was normal but had grade II dyspnoea.Continuous water seal pleural drain (bilateral) was established the day before operation and 1000 ml drained. A central line was made and epidural catheter was inserted through $T_{6-7}$ interspace, position of the catheter was confirmed (lignocaine 2ml with adrenaline 1in 200,000). General anaesthesia was then administered. Induction was done with midazolam 10mg and fentanyl 100ug, IV followed by intubation with vecuronium 6mg.Anaesthesia was maintained with $O_2$ 50% + $N_2O$ 50% and incremental dose of vecuronium (total 20 mg) and IPPV. Analgesia was administered through the epidural route, initially with 0.25% bupivacaine (10ml) and 50ug fentanyl which was continued 4hrly in the postoperative period for 48 hrs.

Pericardiectomy was done. The duration of surgery was 3 hrs. Intraoperative hypotension was managed with dobutamine drip 5ug/kg/min, colloid infusion and two units of whole blood. At the end of operation the patient developed severe haemodynamic instability, dopamine (5ug/kg/min) along with dobutamine (5ug/kg/min) drip was started, and he was not extubated and transferred to ICU, was put on to ventilator (synchronized intermittent mandatory ventilation) / pressure support mode. Ceftriaxone 1gm twice daily was started and ciprofloxacin and steroid continued.

Patent developed anasarca on 4$^{th}$ postoperative day, which responded to diuretics. Nutrition was maintained both enterally and parenterally. Patient received 2 units of concentrated RBC in the immediate post operative period. With signs of improvement clinically and radiologically (X-ray3), gradually the inotropic support was withdrawn, patient was kept on spontaneous pressure support mode. On 14$^{th}$ postoperative day ventillatory support was withdrawn and patient was put on to T -piece oxygenation. On 15$^{th}$ postoperative day he was extubated and sent to the ward with bilateral pleural drain. Histopathology

of pericardium revealed tuberculous pericarditis. ATD was restarted with INH 300mg OD, ETM 800mg OD, RMP 450mg OD and PZN 1500mg OD and ciprofloxacin withdrawn. Patient was discharged after removing pleural drain on 20th postoperative day. He was advised to take ATD, frusemide and gradually tapering dose of prednisolone. At one month follow up his chest X-ray was normal (No.4).

## DISCUSSION

Till date, the prevalence of pericardial disease (PD) in patients with pulmonary tuberculosis is not common in developed countries. The only data available in the literature has reported a 1% incidence of clinically symptomatic pericardial disease in instances of pulmonary tuberculosis observed over a 10yr period (Larrieu *et al.*, 1980). Other studies have reported evidence of pulmonary lesion in patients primarily presenting with pericardial disease (Larrieu *et al.*, 1980).

Echocardiography is a proven and satisfactory investigation for assessing pericardial thickening and dense intrapericardial pathology (Schnittger *et al.*, 1978). Pericardial thickening and dense intrapericardial echoes are recognizable on M mode records from the base of heart. Echocardiography is also the earliest to differentiate responders to ATD from nonresponders (Schnittger *et al.*, 1978). The latter may go on unnoticed to develop pericardial constriction and effusion in spite of treatment, the mechanism is poorly understood (Komsuglu *et al.*, 1994; Hiroyama *et al.*, 1996). Our patient was probably an ATD nonresponder who presented with constrictive – effusive pericarditis within two months of treatment. Corticosteroid and ciprofloxacin have been used in patients with persistent pericardial effusion in ATD nonresponders (Strang, 1994; Kasper *et al.*, 2004) with good results, reducing the need for pericardiectomy. In our patient the result was unsatisfactory probably due to delay in starting therapy.

In conclusion, pericardial disease though uncommon in patients of developed world it can occur with pulmonary tuberculosis and may progress with time to a morbid situation like moderate to severe pericardial effusion and/constrictive pericarditis. Echocardiography for every drug non responder (pulmonary TB) can diagnose the pericardial disease process at the earliest so that adjunctive drugs can be instituted to avoid or to arrest the dreadful complications.

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