A 55 year old, married waiter, residing in slums, was admitted with history of precordial chest pain and dry cough for 8 days. On inquiry he had breathlessness on lying down and low grade fever with evening rise of temperature. He was chronic alcoholic and non smoker. On examination he was afebrile, pulse was 88/min, low volume with pulsus paradoxicus, BP 84/60 mmHg, RR 26/min, JVP was 10 cm H2O. There was no pallor, clubbing, cyanosis or lymphadenopathy. Cardiac examination revealed cardiomegaly, non palpable apex with muffled heart sounds. Respiratory system examination was unremarkable. His investigations were- Hb 12.4 g%, WBC 8600/cmm, P 74%, L 22%, E 1%, ESR 70mm/h. Other biochemical examinations (RFT, LFT, serum amylase) were unremarkable except low serum albumin (2.3g%). Blood glucose and urine examination were normal. Serological test for HIV was negative. His X-ray chest (Figure 1) showed hydropneumopericardium. ECG showed ST elevation with upward concavity in all the leads (Figure 2). He underwent urgent pericardial tapping with removal of 120 ml of purulent fluid and air. He was started on intravenous ceftriaxone and vancomycin. Pericardial fluid showed WBCs 4,80,000/cmm (P 93%), RBCs 32,000/cmm, proteins 3.5G%,
glucose absent, and fluid adenosine deaminase (ADA) level was 92 IU/L. Subsequently culture grew streptococcus pneumoniae. Hemaquet drainage tube was placed in pericardial fluid and daily 50-60 ml of purulent fluid was drained for next 4 days. His CT scan Chest done on day 3 showed (Figure 3) moderate hydropneumopericardium with irregular pericardial wall thickening, patchy consolidation with few confluent nodules in posterior basal segment of right lower lobe with mild bilateral pleural effusion. Enlarged lymph nodes were noted in right paratracheal, pretracheal and posterior mediastinal region, the largest measuring 2.3 x 1 cm in size. Few air specks were noted in retro-cardial, para aortic and anterior mediastinal region, suggesting mediastinal emphysema. Upper GI scopy was done to rule out oesophageal pathology. Anti-TB treatment (ATT) was started with prednisolone (1 mg/kg). There was no evidence of constrictive pericarditis on 2 D Echocardiography and patient was discharged after 14 days. He was well on follow up, steroids were stopped after 2 months and ATT was continued.

The mechanisms by which patients develop purulent pericarditis include: direct spread from an intrathoracic focus of infection, haematogenous spread, extension from a myocardial focus, direct contamination from trauma or thoracic surgery, and extension from a subdiaphragmatic suppurative focus. Staphylococcus aureus is the most common cause of purulent pericarditis. Other important pathogens include Streptococcus pneumoniae, Salmonella, Candida, and tuberculosis. Polymicrobial infections are uncommon. Spontaneous pneumopericardium is a relatively rare event, several causes have been postulated. These include trauma, infectious-secondary to gas-producing bacilli in the pericardial fluid, fistula formation-secondary to perforation of a neighbouring viscus such as oesophagus, stomach, liver abscess or bronchus and iatrogenic-secondary to pericardiectomy, and assisted positive pressure ventilation. Infection spreading to the pericardium following oesophageal perforation is usually devastating, with a survival rate of only 17 percent in one review of 60 such patients. Spontaneous pyopneumopericardium is reported following bacterial infection also but in present patient, tuberculous lymphadenopathy with secondary bacterial infection was the probable cause of pyopneumopericardium in view of absence of high grade fever or leucocytosis and presence of mediastinal lymph nodes.

References