Case Report

Pyopneumopericardium in a child with acute leukemia

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ABSTRACT

Pyopneumopericardium is a rare condition with high mortality. Most cases are the result of a fistulous communication between the pericardial cavity and the gastrointestinal or the respiratory tract. Rarely, the condition may arise spontaneously. We describe a 7-year-old boy, a diagnosed case of acute lymphoblastic leukemia, on chemotherapy who developed pyopneumopericardium without pneumothorax and responded well to conservative management.

Key words: Acute leukemia, pediatric, pyopneumopericardium

INTRODUCTION

Pyopneumopericardium is an unusual condition. The known etiologies are chest trauma, complication of surgical procedures, fistulous communication between pericardial space and adjacent structures such as pleural space, bronchial tree or upper gastrointestinal tract, and continuous spread of infection from bacterial pneumonia or empyema.[1-3] Rarely pyopneumopericardium may arise spontaneously from gas-forming infection within the pericardium.[1-3] The reported bacteria that cause spontaneous pyopneumopericardium include *Peptostreptococcus*, *Bacteroides*, *Staphylococcus aureus*, *Escherichia coli*, and *Klebsiella pneumoniae*.[2-5] The diagnosis is often delayed because of lack of specific signs and symptoms. In some cases, the resulting tension pneumopericardium with tamponade can be fatal.[6] However, despite gross pyopneumopericardium, tamponade may not develop in some patients.[2,3] Here, we report a child with acute lymphoblastic leukemia who developed spontaneous pyopneumopericardium.

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CASE REPORT

A 7-year-old boy presented with high-grade continuous fever for the past 4 days. There were no other associated complaints. He was diagnosed as B-cell acute lymphoblastic leukemia (bone marrow biopsy showing 70% myeloperoxidase negative blasts positive for CD19, CD45, cCD79a on flow cytometry) and was on chemotherapy for last 3 months. He was documented to be in remission after induction chemotherapy and was presently on repeat induction cycle (INCTR protocol) for past 2 weeks. On examination, temperature was 100°F, heart rate was 128/min, respiratory rate was 22/min and blood pressure was 116/72 mmHg (without pulsus paradoxicus). Mild pallor was present. There was no pedal edema. Jugular venous pressure was not raised. Heart sounds were normally heard. Rest of the general physical and systemic examination was unremarkable.

In investigations, Hb was 8.7 g%, total leukocyte count (TLC) was 7800/mm^3 with 77% polymorphs and 21% lymphocytes. Renal function tests and liver function tests were in normal range. Blood and urine cultures were sterile. Chest radiograph was suggestive of large pneumopericardium.

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Bilaterally lung fields and costophrenic angles were clear [Figure 1]. Twelve-lead electrocardiogram revealed sinus tachycardia with rest of the parameters being normal. The patient was started on intravenous ceftriaxone, amikacin, metronidazole after sending microbiological investigations. Echocardiography was performed on day 2 of admission revealed spontaneous dense contrast echoes in pericardium with pericardial effusion suggestive of hydropneumopericardium. There was no evidence of pericardial tamponade. Conservative management was continued. There was no pulsus paradoxicus. The patient continued to have high-grade fever with persistent tachycardia. Antibiotics were upgraded to vancomycin and piperacillin-tazobactam. Repeat investigations done revealed Hb of 7.4 g% and TLC of 2050/mm^3 (Polymorph 70 Lymphocyte 30 [P 70, L 30]). Blood culture and fungal work up was not contributory. Repeat chest radiograph showed a decrease in the size of the pneumopericardium with the presence of air-fluid levels in the pericardium. Subsequently, TLC gradually improved, fever subsided on day 14 of admission and serial chest radiographs showed decrease in size of pneumopericardium with day 27 radiograph showing very minimal pneumopericardium. Intravenous antibiotics were stopped on day 31 (4 weeks after change of antibiotics), and the patient was discharged on oral amoxicillin-clavulanic acid combination and chemotherapy was restarted. Child is doing well on follow-up visits.

DISCUSSION

Spontaneous pyopneumopericardium is a rare entity with very few case reports. In immune compromised patients, fungal etiologies are frequently reported. Bonanthaya *et al*. reported a similar case with spontaneous pneumopericardium in a 5-year-old child with acute leukemia.[7] Like in our case, the cause of the spontaneous pneumopericardium could not be found. Bronchoalveolar lavage was negative for fungal growth, pneumocystis, or mycobacteria. However, the blood culture showed growth of *S. aureus* which was sterile in our case. Our patient did not require antifungal as neutropenia and clinical condition improved with broad spectrum antibiotics and presence of pus was documented initially.

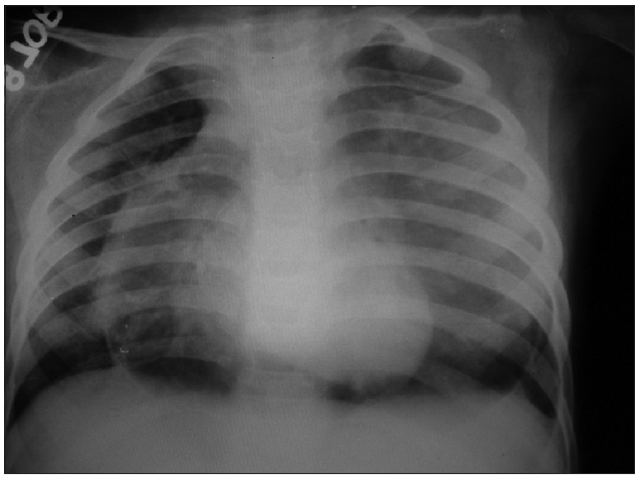

Figure 1: Chest radiograph of child with acute leukemia on chemotherapy showing large pneumopericardium

The main causes of pneumopericardium and tension pneumopericardium described in literature are chest trauma and positive pressure ventilation. The main mechanisms leading to pneumopericardium are macro perforation of the pericardium with respiratory or gastrointestinal tracts communication or pleuropericardial connection in the presence of a pneumothorax or pulmonary volutrauma with tracking of alveolar air into the pericardium. Among the reported case was a 42-year-old previously healthy man who presented with progressive weakness, lightheadedness, nausea, and pedal edema. On examination, features of pericardial tamponade were present. His chest roentgenogram revealed pericardial air-fluid level with pericardial thickening, enlarged cardiac silhouette, enlarged superior vena cava and azygous silhouettes, small bilateral pleural effusions, and bibasilar air space diseases. The diagnosis was confirmed by echocardiography. He underwent pericardiocentesis but later succumbed. His pericardial fluid and other cultures grew *S. aureus*.[5] In another report, a 77-year-old woman with poorly controlled diabetes mellitus developed spontaneous pyopneumopericardium with bilateral pneumonia. There was no tamponade. She received pericardiectomy, and her cultures yielded *K. pneumoniae*. She had an uneventful clinical course.[3] Merino *et al*. described a case of invasive aspergillosis with pneumothorax and pneumopericardium in a child with acute lymphoblastic leukemia.[8]

Sener *et al*. reported a case of spontaneous pneumopericardium in an adult with extranodal T-cell lymphoma.[9]

The diagnosis of pyopneumopericardium may be unexpected and picked on chest radiography as in our patient. The physical findings that may help in diagnosis are resonance to percussion over the precordium and mechanical splashing sound, bruit de rave hydrouligue heard over the precordium.[2,10] Pertinent roentogenographic signs include cardiomegaly, pericardial air-fluid levels, pericardial thickening, overlay of the hila by cardiac silhouette, pericardial stripes buried deep in the cardiac silhouette and pleural effusions.[4] Signs of tamponade include enlargement of superior vena cava and azygous vein shadows.[4] Ultrasonography and computed tomography scan can assist in the diagnosis, and two-dimensional echocardiography is the diagnostic procedure of choice for detecting pericardial effusions.[4] The pathognomonic

finding of pyopneumopericardium on echocardiography is the presence of hyperechoic floating particles in the pericardial effusion, which are called intrapericardial spontaneous contrast echoes.[3]

Pyopneumopericardium is a potentially fatal disease.[1,2,4] Survival depends on early diagnosis and aggressive antibiotic therapy to cover Gram-positive, Gram-negative, and anaerobic organisms. Once tamponade is recognized, immediate pericardiocentesis is suggested. With or without tamponade, early surgical intervention in the form of pericardiectomy and drainage is recommended.[2-5,7]

Our patient was among the very few pediatric cases in literature to have developed spontaneous pyopneumopericardium. Moreover, the patient responded well to conservative management alone. Immunosuppressed children are at increased risk for development of such complication due to unchecked growth of virulent and nonvirulent organism which can result in necrosis and resultant leakage of air from alveoli into pericardium.

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Conflicts of interest
There are no conflicts of interest.

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