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RESEARCH ARTICLE

PYOPERICARDIUM IN A STRUCTURALLY NORMAL HEART

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ABSTRACT

In pyopericardium, pus accumulates in the pericardial space as a result of infection by pyogenic organisms, most common of which are Staphylococcus aureus and Mycobacterium tuberculosis. These patients are at risk of cardiac tamponade. Causes linked to pyopericardium are pneumonia, empyema, thoracic surgery and haematogenous spread through sepsis. Factors that can predispose to pericarditis include immunosuppression and previous pericardial inflammation and fibrosis. (1).We present a case of pyopericadium in a structurally normal heart which was initially suspected by cardiomegaly in the chest X ray which was taken while working up a male child with fever and cough of 2 weeks duration.

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INTRODUCTION

Pyopericardium is a rare condition with a high mortality rate in which infection propagates in the pericardial space, leading to pericardial effusion and cardiac tamponade, which may cause cardiogenic shock and death.Bacterial etiology includes Staphylococcus, Haemophilus influenzae, Streptococcus, pneumococcus,Meningococcus, Mycoplasma, tularemia, Listeria, leptospirosis, tuberculosis (TB), Q-fever, and salmonella.(1).We present a case of pyopericardium in a structurally normal heart with no primary source of infection or any predisposing factors

CASE REPORT

9 year old boy was brought to the Emergency room with complaints of high grade intermittent fever and cough of 2 weeks duration. He also complained of retrosternal chest pain since the past 3 days. He was initially treated on OPD basis in a local hospital with oral antibiotics. As symptoms persisted, blood tests were done which showed neutrophilic leukocytosis with elevated inflammatory markers. He was admitted and given IV antibiotics .Chest X ray was not taken. As fever persisted and as he developed chest pain , he was referred to our hospital for further management. There was no h/o recurrent episode of fever and cough in the past. He was born by normal vaginal delivery with birth weight of 3.2 kg and was developmentally normal and immunised for age. There was no h/o contact with TB. At admission to ER , he was febrile and was not tachypneic. Vitals were stable. Systemic examination showed apex beat in the sixth intercostal space,1 cm lateral to left mid clavicular line. There was no tracheal or mediastinal shift. Heart sounds were normal .There were no murmurs. No signs of heart failure except cardiomegaly. Counts showed neutrophilic leukocytosis with elevated inflammatory markers. (Total counts-15640, poly-54.6 %, lymph-38.6 %, Hb-10.1 g/dl, Platelet-815 K/UL, CRP-177.64 mg/L). ESR was also elevated (96 mm/hr).LFT/RFT/Serum electrolytes were within normal limits. Chest X ray showed cardiomegaly with no large infiltrates. He was started on Inj Ceftriaxone after sending samples for culture and serology for Mycoplasma. Mantoux and sputum for genXpert -AFB were negative. IgM Mycoplasma was negative. An ECHO was done in view of persistent fever and cardiomegaly which showed structurally normal heart with normal LV systolic and diastolic function. There was an organized fluid collection around the heart, predominantly around the ventricles. A MDCT aortogram done showed borderline pericardial thickening (2 -2.5mm). Moderate amount of fluid was noted within the pericardial cavity (Mean Hu 15) Figure 1. Parenchymal bands were noted in the left lower lobe.

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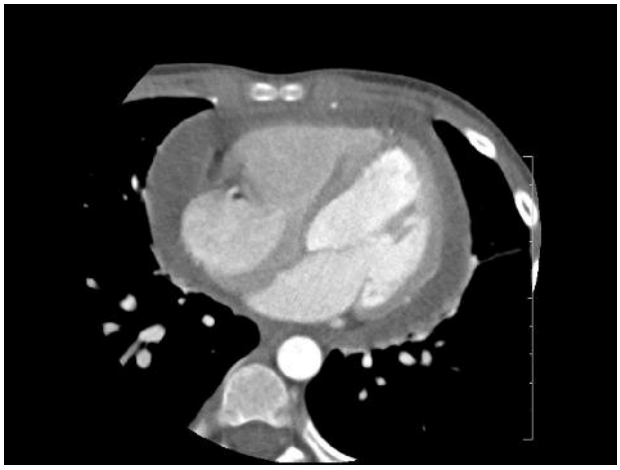

Figure 1. MDCT Angiogram showing borderline pericardial thickening (2 -2.5mm)

There was no evidence of LV/ chamber aneurysms or its rupture. Coronaries were normal. Inj Vancomycin was added. Serial monitoring with Echocardiogram done showed organization of pericardial collection predominantly anteriorly surrounding the great arteries. As fever persisted ,he underwent pericardiotomy after 12 days of admission and intra-operatively was noted to have thickened pericardium with vascular adhesions between cardiac structures and pericardium. Organised transudate was noted all over pericardial cavity. The excised tissue was sent for culture,fungal culture and MTB-GenXpert which came back as negative. Histopathological examination showed chronic inflammation with suppuration. Child improved post procedure and was discharged home on oral Linezolid after receiving IV antibiotics for 28 days. Oral Linezolid was given for a total duration of 14 days. Review ECHO done 10 weeks after procedure showed no significant heart defects. LV, RV function were normal. There was no evidence of constriction/effusion/PAH.

DISCUSSION

In the present antibiotic era, pyopericardium is uncommon. In many cases, it may lead to constrictive pericarditis with a fatal outcome. Mortality rate in treated patients is 40%, mostly due to cardiac tamponade, toxicity, and constriction. It is usually a complication of an infection originating elsewhere in the body, arising by contiguous spread or haematogenous dissemination (2). Predisposing conditions are pericardial effusion, immunosuppression, chronic diseases, cardiac surgery and chest trauma. The disease appears as an acute, fulminant infectious illness with short duration. Diagnosis can be delayed due to the initial source of infection distracting from the underlying cardiac problem. In our case,the probability of a community acquired pneumonia was initially considered. However the absence of lung infiltrates and the presence of cardiomegaly in the chest X ray with poor response to IV antibiotics were the clues to suspect an underlying cardiac pathology.

The likely reason for negative cultures would be due to prior antibiotic usage. As the fever did not subside, after trial of appropriate IV antibiotics , our patient was taken up for pericarditomy, following which he showed clinical improvement. The most common organism reported to cause pyopericardium in previous cases is Staphylococcus aureus. It has been reported that Gram-negative bacteria and fungi are becoming more frequent due to infection in immunocompromised hosts (3). Causes linked to pyopericardium are pneumonia, empyema, thoracic surgery and haematogenous spread during sepsis. More rare causes include perivalvular abscess rupture in endocarditis and spread along fascial planes from the oral cavity (4)(5)(6) In our patient the source of primary infection could not be identified. Our patient also did not have any predisposing factors or any h/o trauma or surgery. In a child with persistent fever, cough and chest pain a cardiac cause should be considered if a lung pathology could not be identified. Chest X ray followed by ECHO may help in identifying the underlying pericardial pathology. Cardiomegaly in chest X ray may give a clue to underlying pericardial disease. Although there is general agreement that surgical drainage is mandatory, the approach, methods of drainage, and the extent of drainage are variable (7) .We conclude that pericardiectomy has a definite place in the management of purulent pericarditis.

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