Case Report

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Rheumatic Pneumonia: A Rare Complication of Acute Rheumatic Fever

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Abstract

Rheumatic heart disease is still a leading cause of acquired heart disease in children, in developing countries. Rheumatic pneumonia is a well-described burt very rare manifestation of rheumatic fever, that is generally fatal. Even though necropsy confirmation is ideally required, with clinical features of consolidation and absence of features of cardiac failure one can think the possibility of rheumatic pneumonia.

Keywords: Rheumatic fever; Rheumatic pneumonia; Cardiac failure; Complication

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Introduction

Rheumatic Fever (RF) and Rheumatic Heart Disease (RHD) remain significant causes of cardiovascular diseases in the world today. Even though there is a decrease in the incidence of acute RF and RHD in developed countries during the past five decades, they remain as major public health problems in developing countries. Rheumatic fever in this century appears to be largely a disease of crowding and poverty.

ARF has extremely variable manifestations and remains a clinical syndrome for which no specific diagnostic test exists. Rheumatic pneumonia(RP) is a well-described, poorly understood, rare manifestation of rheumatic fever that is generally fatal. RP has been reported for more than a century and it has been traditionally associated with a high mortality rate[1]. We are presenting a case of RP, to increase awareness of such a forgotten entity, in order to determine optimal management.

Case Report

An 8 year old boy presented to emergency department with severe respiratory distress. On taking a detailed history, his complaints started about 8 days back with fever. Fever was of high grade and was not associated with rashes, vomiting, loose stools, seizure or pain during micturition. For initial 2 days of fever he had sore throat which relieved on its own. Child developed cough 5 days back which was productive in nature, and was progressive. For the last 2 days he was having chest pain and breathlessness. It was a central chest pain associated with breathlessness even at rest. Child preferred sitting up than lying down even at night.

On repeated questioning, mother gives a past history of fever with sore throat 6 weeks back and later, joint pain. Joint pain was noted first in the knee and later spread to ankle, and later got relieved. It was typically migratory type and disappeared with treatment from primary health centre. No history of tuberculosis or contact with

tuberculosis. Fully immunized. His growth and development were normal.

At admission, child was conscious, sick looking with severe respiratory distress having tachypnoea and retractions. Pallor present, no cyanosis/ clubbing/edema. PR-130/min, regular, high volume collapsing type,all peripheral pulses well palpable. BP-98/30 in right upper limb, 150/40 in right lower limb. RR-52/min, temp-100.5 degree F, SpO_2-96% in room air. Dancing carotids, Quincke's sign and Traube's sign were present. On cardiovascular examination: JVP raised to 4 cm, precordium was hyperdynamic. Apex in left 6th Intercostal space in the anterior axillary line. Grade I LPSH was present. S1 masked, S2 loud, S3 present. A high pitched pan-systolic murmur Grade IV/VI intensity best heard in LLSB, and a low pitched early diastolic murmur Grade III intensity heard in 3rd left sternal border. Respiratory system was normal with bilateral vesicular breath sound. GIT: 4 cm soft tender liver with no spleen/ascites.Nervous System exam-normal.

On investigation: Hb–7.9 gm%, TC-10100 cells/mm, ESR-120 mm/1st hr, RFT, LFT, S.electrolytes-all normal. CRP-positive, ASO titre-positive. Peripheral smear showed normocytic normochromic anaemia. X-ray chest: lung fields showed air bronchogram and significant cardiomegaly. ECG: sinus rhythm, HR-130/min, right axis deviation (+100 degree). Echo-mild LV dysfunction, LV dilated, moderate MR, severe AR, mild TR with mild PAH. No pericardial effusion. Child was advised complete bed rest, propped up position with oxygen. With a possibility of rheumatic fever activation, he was put on Tab. Prednisolone, Inj. CP, Inj. Frusemide and packed cell transfusion.

While admitted in the hospital, child was persistently having severe tachypnea with respiratory distress. Because of increased apprehension and restlessness he was given Inj Morphine. Later Milrinone infusion was added on day 2 of admission suspecting intractable cardiac failure (eventhough there was no oedema or basal crepitation). On next 2 days, there was a little improvement in his breathlessness.

On day 5, child became more dyspnoeic with a RR of 78/min. On auscultation air entry was reduced on right lower lung zones and tubular bronchial breathing on right interscapular and infrascapular area. All other lung areas are with good air entry with no crepitations. X-ray chest was taken and it showed right sided consolidation (Fig. 1). With this new finding we added Inj. Vancomycin also. On day 6 of admission child collapsed and succumbed to death.Since parents were not willing, we could not perform his postmortem.

In view of his past history and present admission with clinical features like extreme breathlessness, local chest findings and absence of pedal oedema and basal crepitations, we made a diagnosis rheumatic pneumonia.

Discussion

RP is a rare clinical entity first described as long ago as 1845, by Latham. He describedit as "but the heart is not the only vital organ liable to suffer inflammation in acute rheumatism. The lungs may suffer also. And the diseases which result are bronchitis, pneumonia, pleurisy".[1] In 136 cases of acute rheumatism, he observed pneumonia in 18 and noted a graver outcome in those cases. He further made the observation that pneumonia was much more common in patients with carditis than in those with the articular manifestation alone. RP was described as an entity by such prominent clinicians as Walshe[2], Cheadle[3] and Garrod[4] in the second half of the nineteenth century.

Rheumatic pneumonitis occurs in the course of acute severe rheumatic fever, usually associated with active carditis, and aggravates the clinical picture. Severe dyspnea and tachypnea, toxicity and a worsening course are the hallmarks of this process[5]. Massive consolidation of the lung without rales is the rule; radiologically, the picture often resembles pulmonary edema so that clinical differentiation may be almost impossible. The diagnosis is suggested through observation in a patient with active rheumatic carditis of an unremitting, diffuse pulmonary consolidation, marked tachypnea, and unresponsiveness to steroid and antibiotic therapy usually terminating in death.

Cheadle described six instances of pneumonia during an outbreak of ARF involving 26 cases and described hyperpnea and fever as its manifestations[3]. 1926, Rabinowitz'[5] reported a specific rheumatic pneumonia even though he failed to find Aschoff bodies in histologic sections. He observed that the pulmonary lesions could not be explained on the basis of heart failure or compression of the lung by serous effusions because these factors were not present in all his cases of rheumatic pneumonia. Many studies indicate a greater acceptance of rheumatic pneumonitis as a specific manifestation and not simply an intercurrent complication of severe rheumatic fever.[6-8]

In our case at no time werethere signs of congestive heart failure or venous congestion. In this case of ARF with carditis a severe, fulminant pneumonia of extensive proportions caused death at atime when cardiac failure was not apparent. Intense dyspnea and striking tachypnea were the outstanding features. Treatment directed at amelioration of cardiac failure and ARF were ineffective.

Even though necropsy confirmationis lacking in this case, rheumatic pneumonitis appears as a possibility. RP possesses no pathognomonic clinical features, this diagnosis and its differentiation from bacterial and viral processes remain speculative. This unusual complications, such as RP, are a challenging diagnosis for clinicians without experience of this illness.[9]

So the association of pulmonary consolidation, tachypnea, the resistance to steroid therapy and the fatal outcome in a patient with known active rheumatic carditis. support of our diagnosis of RP.

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