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ENCOUNTERING PULMONARY TUBERCULOSIS FROM HENOCH-SCHONLEIN PURPURA

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ABSTRACT

In patients with pulmonary tuberculosis, Henoch–Schönlein purpura (HSP) is an exceedingly rare disease, with just a few recorded cases. It is difficult to make a definitive diagnosis when HSP occurs as an initial manifestation in patients with pulmonary tuberculosis, relative to patients with normal clinical symptoms. In this context, a pulmonary tuberculosis case that initially showed HSP was identified and the associated literature was reviewed.

Keywords: Purpura, Vasculitis, Pulmonary Tuberculosis

INTRODUCTION

A type of vasculitis that affects joints, skin, and other organs, Henoch-Schönlein purpura (HSP) is very common in children, while it is extremely rare in adult patients. Purple rash, arthritis, gastrointestinal, and/or renal involvement are characterised by HSP. Several sets of diagnostic criteria for HSP have been proposed since 1990s **[1]**, and their diagnosis depends primarily on standard clinical characteristics, symptoms, and histopathological findings. Many infectious factors have been identified as significant

causative factors for it such as viruses and bacteria. The manifestation of HSP before pulmonary tuberculosis, has been reported in seldom **[2]**. A patient with HSP is reported in this study as an initial manifestation of pulmonary tuberculosis.

CASE REPORT

22 year old male presented to the emergency with purpura on the bilateral lower limbs and buttocks associated bilateral knee pain, diffuse abdominal pain with history of blood in stools. The patient had no history of fever, cough, expectoration, breathlessness, chest pain. He had no other comorbidities or any history of recent illness. On examination patient was moderately built conscious, oriented to person place and time. Vitals were stable, maintaining saturation on room air. Chest sound was normal, Lung fields were clear; abdomen had normal bowel sounds with no organomegaly. No focal neurological deficit was elicited. Clinically patient was diagnosed to have Henoch-Schonlein purpura and was treated with vitamin C and an antihistamine for week, patient was recovered from the manifestations. However, 4 weeks later patient presented with same manifestation, in addition with fever and chest pain which aggravated on inspiratory phase with no chills and rigor. On examination he was febrile (100degF) with tachycardia (100bpm). Routine investigations like Complete blood count, viral markers were sent which showed no significant abnormalities. The C-reactive protein was 9.5 and Erythrocyte sedimentation rate was 15mm/h. Chest X ray showed increased broncho vesicular markings with clear lung fields. In the meanwhile induced sputum was sent for geneXpert. After that patient was started on intravenous third generation cephalosporin and antipyretics. The compliance of treatment was poor, additionally CT chest was done which showed patchy shadows in left apex lobe. Following geneXpert found to be positive. Based on these reports and findings patient was diagnosed with pulmonary tuberculosis associated with Henoch-Schonlein purpura. After that patient was started on anti-tubercular drugs. In a span of 2 weeks in treatment purpura disappeared, chest pain was relieved without any adverse effects. On follow up patient had no recurrence fever, Henoch-Schonlein purpura, chest pain and patient attained full recovery after the treatment.

DISCUSSION

Tuberculosis is the worldwide public health disease which has various presentations which challenges in diagnosis and management **[3]**. The various clinical manifestations includes anemia, myelofibrosis, pancytopenia and thrombocytopenia**[4]**. Here patient presented with HSP an extrapulmonary manifestation of tuberculosis.HSP is a type of vasculitis which characterized primarily by the eruption of diffuse urticarial plaques with palpable purpura on the lower limbs, abdominal pain, joint pain and irregular urine analysis. It need

one of the manifestation for conforming the diagnosis which are diffuse abdominal pain, leukocytoclastic vasculitis with predominant IgA deposits on skin biopsy, acute arthritis, or arthralgias of any joint, and renal involvement as evidenced by proteinuria and/or hematuria **[2]**.

In this patient the presenting symptom was palpable purpura with normal platelet count, bilateral knee pain without fever, abdominal pain, bloody stools, cough, expectoration and chest pain. Following this clinical presentation patient was diagnosed as HSP. After that patient was treated with antihistamines for a week. Later patient presented with fever and recurrence of purpura and elevated hear beat, chest pain during inspiratory phase. By the inference of the CT report and geneXpert patient diagnosed as pulmonary tuberculosis. After that patient was started on anti-tubercular drugs, patient responded well and had no recurrence during the follow up.

Infections like beta-hemolytic streptococcus, Staphylococcus aureus, Helicobacter pylori, Hemophilus parainfluenza, Coxsackie virus, adenovirus, hepatitis A virus, and hepatitis B virus may present as HSP **[5]**. In tuberculosis the pathogen causes vascular damage by infiltration of vessel wall which leads to immunological reactions. Immune complex deposition is the most common cause of vascular injury as compared to the direct injury caused by the bacteria. One theory is that, increase in serum IgA following secretion of transforming growth factor beta (TGF-b) by the activated T cells. These IgA then attaches to the endothelial cells, activates the complement factors or increases the interleukin-8 levels, either way causes cell lysis. Further, IL-8 activates the polymorphonuclear neutrophils, releasing reactive oxygen species which in turn leads to additional endothelial damage. All the inflammatory mediator levels are elevated. Increase in TNF-a makes the attachment of IgA with the endothelial cells more favorable. HLA carrying a genetic risk for tuberculosis is also associated with increased risk of IgA vasculitis.

CONCLUSION

In this case, the purpuric rash occurred without the presence of specific allergens or other pathogens, and disappeared following antituberculosis treatment. All the above-mentioned theories suggest a strong relationship between HSP and tuberculosis, and also tuberculous bacilli can increase the risk for HSP. Both the pathogenic and genetic factors associated with tuberculosis can cause vascular damage by inducing inflammatory reactions. So, when a young patient with no other comorbidities present with such repetitive and persistent symptoms, it is important to suspect tuberculosis for prompt treatment and good prognosis.

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