



HENOCH-SCHONLEIN PURPURA IN A CASE OF DISSEMINATED TUBERCULOSIS - A RARE PRESENTATION

Medicine

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ABSTRACT

Henoch-schonlein purpura (HSP) is an acute, systemic, immune complex mediated, leukocytoclastic vasculitis seen more commonly in children. It presents with the clinical triad of non-thrombocytopenic palpable purpura, arthritis and abdominal pain. We present a case of disseminated tuberculosis associated with HSP in a 57 year old male patient, who developed non-pruritic, painless purpura after six days of anti-tuberculosis treatment. The skin biopsy revealed granular blood vessel wall staining with IgA, C₃ and fibrinogen suggestive of HSP. The cutaneous vasculitis in this disappeared with the continuation of anti-tubercular drugs. The association of tuberculosis and HSP is very rare.

KEYWORDS

Henoch-schonlein purpura, tuberculosis.

INTRODUCTION

Henoch-Schonlein purpura is an acute, systemic, immune complex-mediated, leukocytoclastic vasculitis which occurs more commonly in children younger than 10 years of age, but can affect adult of all ages.¹ It has a slight predominance in males and more severe in adults.² HSP is an IgA-mediated immune vasculitis. Presents with clinical triad of non-thrombocytopenic palpable purpura, arthritis, and abdominal pain. The classic purpuric skin rash is more commonly seen over the extensor surfaces of lower extremities, belt line, and buttocks.

Tuberculosis is the most common cause of death in the world due to a single infectious agent in adults and accounts for over a quarter of all avoidable deaths globally. India accounts for one-fourth of the estimated global incident TB cases.³ The association of tuberculosis with HSP is very rarely reported. Here we present a case of disseminated tuberculosis associated with Henoch-schonlein purpura in a male adult patient. HSP usually follows an infection due to an abnormal immune response though the causative factors are not known.

CASE REPORT

A 57 year old male shopkeeper by occupation presented to OPD with complains of cough, loss of appetite, loss of weight, and low grade fever since the past one month. He was newly diagnosed diabetes mellitus. Chest x ray revealed a cavity with surrounding non homogenous opacity in the left upper lung zone which was suggestive of pulmonary tuberculosis. The sputum smear for AFB was negative but sputum CBNAAT detected mycobacterium tuberculosis sensitive to rifampicin.

Patient was diagnosed as pulmonary tuberculosis with diabetes mellitus and started on anti-tubercular drugs (4 tablets of 4 FDC) and insulin. However after six days of starting the treatment patient developed multiple non pruritic, painless, palpable purpura on the extensor surface of both lower limbs with few erosions covered with scab and scarring on the dorsum of foot (Figure 1). The patient had no history of atopy or allergy to any drugs.

Blood test showed the following: Hb,10.3 ;leukocytes, 8390*1000/ul(neutrophils,58%; lymphocytes, 32%; eosinophil, 03%, monocytes,06%;basophil01%);platelet,3.5 lakhs/cu mm; haematocrit,37%; peripheral smear showed mild hypochromia, mild anisocytosis. Blood urea,25mg/dl; serum creatinine,0.7mg/dl. Liver function test was within normal limits. HbA1c was 14.9. The coagulation profile was within normal range. Rheumatoid factor, CRP and ANA negative, HbsAg negative, anti-HCV negative and anti-HIV negative. Routine urine and stool examination showed no abnormality.

USG whole abdomen showed terminal ileal loops and caecum appeared mildly edematous with few hypoechoic mesenteric lymph nodes noted in this region, largest measuring 4mm in short axis diameter suggestive of abdominal tuberculosis. The skin biopsy showed endothelial swelling with extravasation of RBC, perivascular infiltration of neutrophils and nuclear dust which was consistent with leukocytoclastic vasculitis (Figure 2). Immunofluorescence of skin biopsy revealed blood vessel wall staining with IgA, C₃ and fibrinogen suggestive of Henoch schonlein purpura (Figure 3).

The anti tuberculosis medication of the patient was withheld and was subjected to drug challenge test due to clinical suspicion of a drug induced cutaneous reaction. Patient tolerated the drugs in order of isoniazide, rifampicin, ethambutol and pyrazinamide in full dosage. There was no cutaneous manifestation during this process. Patient was again restarted on anti tubercular drugs without the recurrence of purpura and discharged. Patient was followed up in two months and six month, was found to be asymptomatic, disappearance of the cavity in chest xray and sputum was negative. He was declared cured.



Figure 1: Multiple non pruritic, painless, palpable purpura on the extensor surface of both lower limbs.

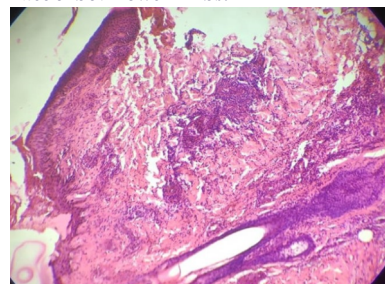


Figure 2: Skin biopsy showing endothelial swelling with extravasation of RBC, perivascular infiltration of neutrophils and nuclear dust.

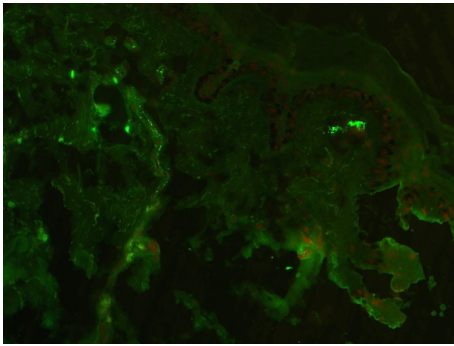


Figure 3: Immunofluorescence -blood vessel wall staining with IgA, C3 and fibrinogen.

DISCUSSION

Henoch schonlein purpura is a systemic leukocytoclastic vasculitis. There are two general types of pulmonary tuberculosis-related vasculitis: leukocytoclastic vasculitis which is a manifestation of pulmonary tuberculosis and anti-tuberculosis medication-associated vasculitis (particularly with rifampicin therapy).⁴ Factors such as chemicals, infections, toxins, trauma and malignancy has also been implicated to precipitate HSP. Hypersensitivity vasculitis, usually results from the deposition of immune complexes formed by antibodies against *Mycobacterium tuberculosis* proteins. The circulating immune complexes has been demonstrated in 56% of patients with active tuberculosis.⁵

Some studies have demonstrated a relationship between rifampicin use and onset of vasculitis.⁶ Upon withdrawal of rifampicin medication the skin lesions of rifampicin induced vasculitis improves. There has been no report of pyrazinamide associated with vasculitis but other cutaneous adverse reaction such as erythema multiforme, urticaria and morbilliform rash was found out to be a bit higher.⁷

The possibility of anti-tuberculosis medication-associated vasculitis in this patient is unlikely, even though the lesions developed after starting on anti-tubercular drugs. The patient successfully completed the drug challenge test, tolerated the drugs without any cutaneous adverse reaction and completed the treatment.

Tuberculosis induced vasculitis seems to fit in this patient. Patient was diagnosed as disseminated tuberculosis as he was microbiologically confirmed pulmonary tuberculosis and USG whole abdomen suggestive of abdominal tuberculosis. He developed cutaneous vasculitis which disappeared with continuation of anti-tubercular drugs without the addition of steroids. Thus we conclude that this is a case of Tuberculosis induced vasculitis confirmed by histology, immunofluorescence and drug challenge test. Although rare, we should be careful in considering rifampicin and pyrazinamide as a possible cause of cutaneous vasculitis.

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