Case report

Henoch-Schonlein Purpura: An unusual presentation of Disseminated Tuberculosis

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Abstract:

Henoch-Schönlein purpura (HSP) is common in children and adolescents between 3 and 15 years of age and is more frequent in boys than in girls . It is recognized as a putative IgA-mediated immune vasculitis. Diagnosis is based on the clinical tetrad of arthritis, abdominal pain, hematuria, and nonthrombocytopenic purpura. Herewith we presented a case of 60 year old man fruit vendor by occupation presented to the medical emergency with complaints of fever, anorexia and weight loss for one month which was associated with simultaneous multiple symmetric and painful joint swelling in bilateral proximal interphalengeal joints, wrist, elbow, knee and ankle, along with blood stained stools and cramping abdominal pain for three days. The association of tuberculosis with HSP is very rarely reported. The underlying pathogenic mechanisms are not yet clear. It may be triggered-off by tuberculosis or by antituberculosis treatment. In our patient symptoms were cured with anti tuberculous treatment without the addition of steroids, thus we conclude that HSP was triggered by Tuberculosis.

Keywords: Henoch-Schönlein purpura, vasculities

Introduction

Henoch-Schönlein purpura (HSP) is common in children and adolescents between 3 and 15 years of age and is more frequent in boys than in girls (1.5:1). It is recognized as a putative IgA-mediated immune vasculitis. Diagnosis is based on the clinical tetrad of arthritis, abdominal pain, hematuria, and nonthrombocytopenic purpura. The classic purpuric skin rash (palpable purpura) is generally regarded as essential for diagnosis. HSP is relatively uncommon in adults and often more severe[1].

Tuberculosis being an endemic disease in India and one of the common causes of morbidity makes the disease of great clinical concern[2]. Here we describe a case of disseminated pulmonary tuberculosis which was seen to be associated with Henoch-schonlein purpura in an adult patient. Though the causative factors of HSP are not known but an abnormal immune response to an infection has been seen as a cause in many cases.

Clinical record

A 60 year old man fruit vendor by occupation presented to the medical emergency with complaints of fever, anorexia and weight loss for one month which was associated with simultaneous multiple symmetric and painful joint swelling in bilateral proximal interphalengeal joints, wrist, elbow, knee and ankle, along with blood stained stools and cramping abdominal pain for three days. Patient developed painless, non pruritic bluish-red rashes on the both the ankles and legs during the sixth day of hospital stay. There was a history of Pulmonary Koch's fifteen years back for which the patient had taken anti tuberculous medication for six months and

was labelled as cured. On examination patient was febrile (101.4° F), the pulse rate was 110/ min and the blood pressure was 108/72 mm Hg. Fine crepitations were present in the left upper zone of the chest. Affected joints were tender, swollen, had limited movements but no deformity or instability. During the hospital stay patient developed multiple discrete, well defined, non blanchable, palpable purpura on lower limbs (Figure 1). Rest of the examination was normal. Blood investigations revealed moderate anaemia (Hb 9.4 gm %), leucocytosis (TLC 23,400 cells/ mm^3) and raised ESR (57 mm/hr). Peripheral smear showed neutrophilic leucocytosis with toxic granules, normocytic, normochromic RBC's and mild anisocytosis. Blood urea (22 mg/dl) and serum creatinine (0.7 mg/dl) were within range. Liver function tests were normal. The coagulation profile and platelet count were within range. Urine examination revealed proteinuria (2+) with active sediment (13-15 RBC/HPF, 75% dysmorphic RBCs). 24 hr urinary protein had 270 mg of protein per day. Stool routine and microscopy showed 8-10 RBC/HPF with no evidence of pus cells, bacteria and ova/cyst. Stool culture was sterile. Induced sputum was positive for acid fast bacilli. Left upper zone consolidation was seen on a chest X-ray. ANA, CRP, rheumatoid factor, c-ANCA, p-ANCA, HIV, Hbs Ag and Anti- HCV IgM antibody were all negative. CECT abdomen showed long segment circumferential enhancing thickening of jejunal loops with maintained mural stratification with few discrete mildly enhancing mesenteric lymph nodes in the central abdomen with mild free fluid in peritoneal cavity. Colonoscopy was within normal limits. The kidney biopsy revealed mild increase in mesangial matrix and cellularity (Figure 1A) with focal endocapillary proliferation in two glomeruli associated with a partial cellular crescent in one glomerulus (Figure 1B). Immunofluorescence revealed a diffuse granular deposition of IgA (Figure 2 A and B) pattern in the peripheral capillary walls and mesangium.

Patient was started on Category II DOTS (Isoniazid, streptomycin, rifampicin, ethambutol, pyrazinamide) according to RNTCP. After 15 days of inpatient care patient was symptomatically better, afebrile, arthritis and rash had disappeared and thus the patient was discharged. The patient was followed up at 3months and 9 months, was found to be asymptomatic and sputum was negative on both the occasions and was declared as cured. Routine urine examination as well as stool microscopy at 3 months and 9 months showed no abnormality.

Table 1: Case reports of TB with HSP.

S. No	Author/ year	Age (years)/ Sex	Place	Form of TB	Treatment
1.	Pacheco A et al (1987)[4]	33/ Male	Spain	Pulmonary TB	ATT, Systemic steroids
2.	Washio M et al (1988)[5]	21/ male	Japan	Tuberculous pleuritis	ATT
3.	Saatci U et al (1989)[6]	7/ Female	Turkey	Urinary tract TB	ATT
4.	Pacheco A et al (1991)[7]	33/ Male	Spain	TB lymphadenitis	ATT, Systemic steroids
5.	Mishima Y et al (1994)[8]	34/ Male	Japan	Pulmonary TB	ATT, Systemic steroids
6.	Han BG et al (1995)[9]	41/ Male	Korea	Disseminated TB	ATT
7.	Islek I et al (2002)[10]	8/ Female	Turkey	Pulmonary TB	ATT
8.	Chemli J et al (2004)[11]	12/ female	Tunisia	Renal TB	ATT
9.	Kuboi S et al (2006)[12]	46/ Male	Japan	Tuberculous pleuritis	ATT, Topical steroids
10.	Kitamura H et al (2007)[13]	38/ Male	Japan	Pulmonary TB	ATT, Systemic steroids
11.	Isobe Z et al (2008)[14]	54/ Male	Japan	Pulmonary TB	ATT, systemic steroids
12.	Alvarez C et al (2012)[15]	6/ Male	Spain	TB lymphadenitis	ATT

TB: Tuberculosis, HSP: Henoch-Schönlein purpura, ATT: Anti tuberculous treatment

Figure 1: Multiple discrete, well defined, non blanchable, palpable purpura on lower limbs.

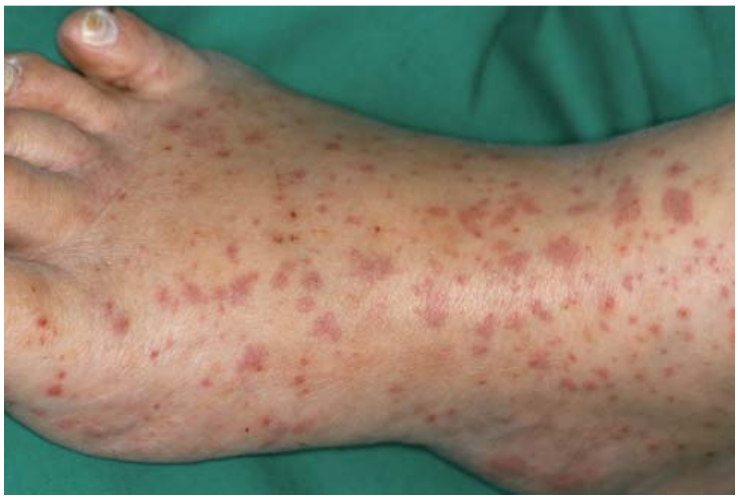

Figure 2: Renal Biopsy

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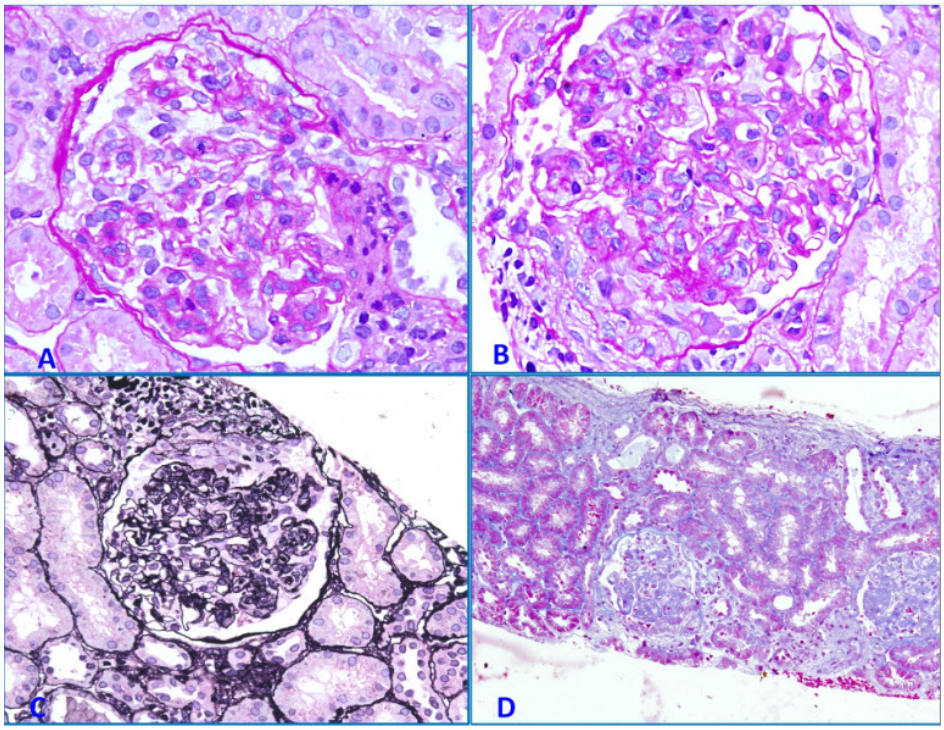
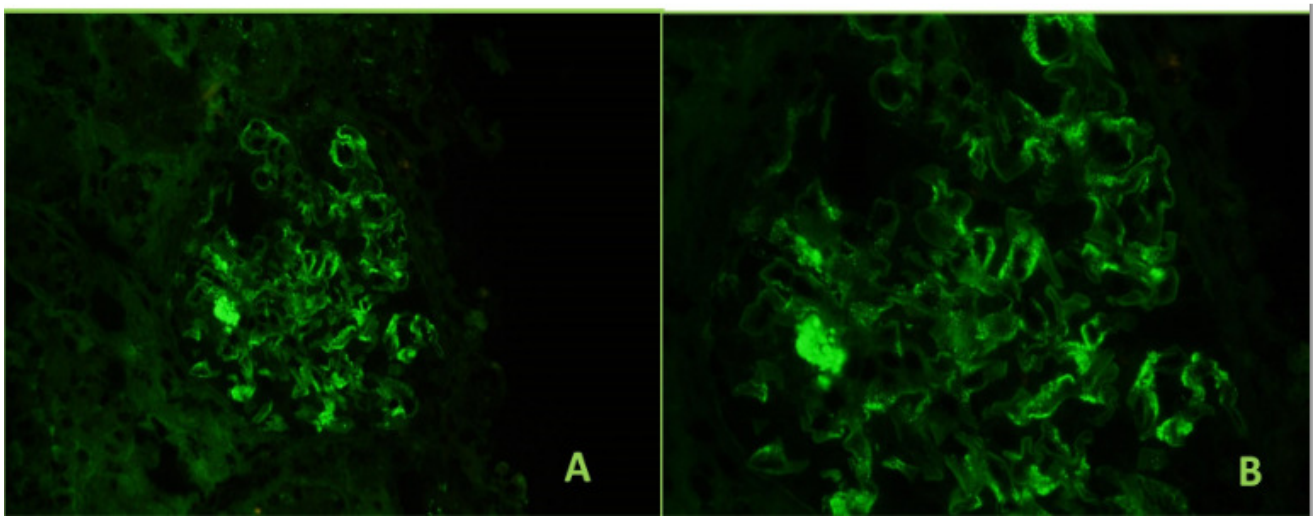


A: Periodic acid Schiff (PAS) stained photomicrograph at high power showing glomerulus displaying increase in mesangial matrix and cellularity (PAS 400x); **B:** Glomerulus displaying a partial cellular crescent and increased cellularity (PAS 400x); **C:** Jones methenamine silver stained section showing unremarkable basement membranes in the glomerulus (JSM 200x); **D:** Masson's trichrome stained section at low power displaying well maintained tubulo-interstitial compartment (MT 100x)

Figure 3: Immunoflouroscence



A & B: Photomicrograph of IgA stained frozen section at low power and high labelled with Fluorescein isothiocyanate (FITC) demonstrating deposition of IgA in the mesangium and peripheral capillary walls (IgA 20x and 40x respectively)

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Discussion

HSP is a leukocytoclastic vasculitis that usually affects children. Several precipitating factors have been implicated, including infections, environmental chemicals, toxins, insect bites, physical trauma, Complement C_2 deficiency and malignancy[3]. A Medline search on pub med for "Henoch Schonlein purpura" and "tuberculosis" showed 12 cases which have been summarized in Table 1.

The association of tuberculosis with HSP is very rarely reported. The underlying pathogenic mechanisms are not yet clear. It may be triggered-off by tuberculosis or by antituberculosis treatment. In our patient symptoms were cured with anti tuberculous treatment without the addition of steroids, thus we conclude that HSP was triggered by Tuberculosis.

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