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BEYOND CHILDHOOD: ADULT-ONSET IgA VASCULITIS PRESENTING AS LEUKOCYTOCLASTIC VASCULITIS — A CLINICOPATHOLOGICAL CASE REPORT

General Medicine

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ABSTRACT

Henoch-Schönlein Purpura (HSP), commonly known as IgA vasculitis, is an acute IgA-mediated self-limiting small-vessel vasculitis predominantly affecting children under 10 years of age. Here we discuss a case of an adult female who presented with purpuric rash over bilateral lower limbs. Histopathological examination from the lesion demonstrated leukocytoclastic vasculitis, and Direct Immunofluorescence demonstrated IgA vasculitis, further supporting the diagnosis of HSP. Although self-limiting, it can lead to life-threatening conditions such as end-stage renal failure. Hence, early diagnosis and appropriate intervention can limit organ damage.

KEYWORDS

Henoch-schönlein Purpura, Iga Vasculitis, Leukocytoclastic Vasculitis, Direct Immunofluorescence, Renal Failure

INTRODUCTION

IgA vasculitis is a small-vessel vasculitis characterised by palpable purpura, arthralgias, gastrointestinal involvement and glomerulonephritis. It is commonly seen in children aged 4–7 years; the male to female ratio is 1.5:1.[1] It tends to be more prevalent in winter and autumn, reaching its peak between January and March.[2]

Henoch-Schönlein Purpura is named after the German physician Dr. Johann Schönlein and his student Edward Henoch. The association between joint pain and purpura was identified by Schönlein, and Henoch identified gastrointestinal and renal involvement. However, the English physician William Heberden was the first to describe the disorder in the early 1800s.[3]

CASE REPORT

A 45-year-old female presented with multiple palpable purpuric lesions involving both lower limbs for the past one month. She gave a history of itching of the lesions which had led to ulceration at the time of presentation. She was a known case of type 2 diabetes mellitus and hypertension with poor glycaemic control. There was no history of fever, abdominal pain or haematuria, but she gave a history of arthritis. Laboratory investigations revealed raised ESR, CRP, HbA1c, FBS, and PPBS, suggestive of an acute inflammatory process with uncontrolled diabetes. There was no renal involvement, as urine protein and RBCs in urine examination were absent with normal serum creatinine (Tables 1 and 2).

TABLE 1: ROUTINE BLOOD WORKUP

Test	Value	Reference Range
Haemoglobin	10	11.5–16.5 g/dL
ESR	77	<20 mm/hour
CRP	58.3	<5 mg/L
HbA1c	13.3	4.8–5.6%
FBS	287	70–99 mg/dL
PPBS	429	<140 mg/dL
Serum uric acid	7.9	2.4–5.7 mg/dL
Serum creatinine	0.91	0.5–1.1 mg/dL
Total protein	8.6	6.4–8.3 g/dL
Globulin	4.5	2.3–3.5 g/dL
Albumin	4.1	3.5–5.2 g/dL
Albumin/Globulin ratio	0.9	1.1–1.8

TABLE 2: URINE ROUTINE

Test	Results
Urine glucose	2+
Urine ketones	Absent
Bilirubin	Not detected
Nitrite	Not detected
Red blood cells	Nil
Dysmorphic RBCs	Absent
Pus cells	4–6/hpf

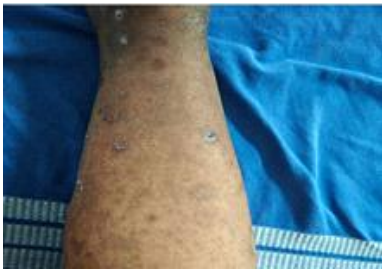

Figure 1: Purpuric lesions with ulceration over lower limb

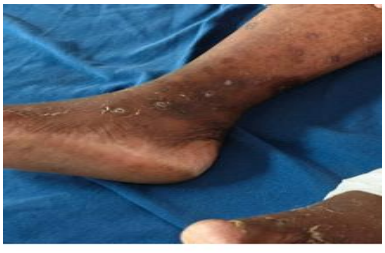

Figure 2: Purpuric lesions with ulceration over lower limb and ankle

A clinical diagnosis of HSP was considered and a punch biopsy from the lesion was sent for histopathological examination and immunofluorescence.

Histopathology showed leukocytoclastic vasculitis consistent with Henoch-Schönlein Purpura, further confirmed by immunofluorescence, which showed granular staining of blood vessel walls with IgM, IgG, IgA, C3 and fibrinogen — suggestive of immune complex vasculitis.

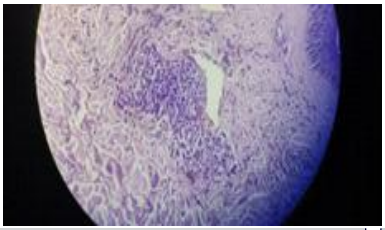

Figure 3: Perivascular inflammatory infiltrate with predominant neutrophils, nuclear dusting and extravasated RBCs (leukocytoclastic vasculitis)

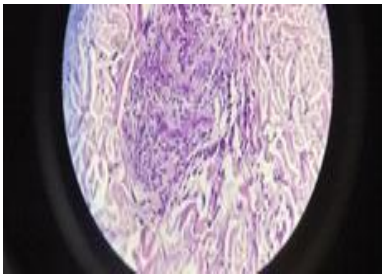

Figure 4: Moderate to marked perivascular mixed inflammatory infiltrate with nuclear dusting

DISCUSSION

The pathogenic mechanism of HSP is immune-complex deposition. Various inciting antigens have been suggested, including upper respiratory tract infections, drugs, foods, immunisation, and insect bites.[1]

The criteria established by EULAR/PRINTO/PRES for the diagnosis of HSP include palpable purpura or petechiae located primarily on the lower limbs and gluteal region, in conjunction with at least one of the following: (1) diffuse abdominal pain; (2) arthritis; (3) renal involvement; (4) biopsy showing leukocytoclastic vasculitis with predominant IgA deposition.[4]

In the present case, the patient presented with palpable purpura and biopsy from the lesion showed moderate to dense superficial and deep perivascular mixed inflammatory infiltrate with predominant neutrophils, nuclear dusting and extravasated erythrocytes — features of leukocytoclastic vasculitis. Direct immunofluorescence demonstrated granular deposition of IgA, IgM and fibrinogen along dermal vessel walls, confirming immune complex-mediated vasculitis consistent with IgA vasculitis.

The presenting symptoms in adults are most frequently related to skin and joints, while initial complaints related to the gut are less common,[1] consistent with our case. Renal involvement in HSP is one of the main indicators of morbidity and increases with the age of onset. Around 30–50% of patients present with haematuria and/or proteinuria within six weeks of disease onset.[5] Severe renal involvement with progression to nephrotic syndrome and end-stage renal failure is more common in adults.[6] Despite this increased risk, our patient showed no renal involvement at presentation.

The prognosis of HSP is excellent.[1] Most patients recover completely and some do not require treatment, especially for cutaneous involvement.[7] Prednisolone 1 mg/kg per day in tapering doses has been shown to be useful in GI and joint involvement.[1]

CONCLUSION

This case highlights three important clinical and pathological lessons:

1. The importance of clinical suspicion of IgA vasculitis in an adult presenting with purpura.
2. The central role of histopathological examination and direct immunofluorescence in diagnosing immune complex-mediated vasculitis.
3. The importance of follow-up of adult HSP patients with urine examination and serum creatinine for subsequent renal involvement.

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