



MEDICAL JOURNAL OF WESTERN INDIA

THE OFFICIAL PUBLICATION OF RESEARCH SOCIETY OF BJMC AND SGH, PUNE

WEBSITE: www.mjwi.org

ISSN NO.: 0972-9798

EISSN No.: 0972-9798

CLINICAL

Adult IgA vasculitis (Henoch schonlein purpura) : A case report and review of literature

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ARTICLE INFO

Article history:

Date of Web Publication 31 Mar 2022

Date of Receipt: 31 Mar 2022

Date of Acceptance: 18 Apr 2022

Date of Publication: 01 Jan 1970

Article No: 182

ABSTRACT

Here we report a case of adult male with IgA vasculitis (HSP, Henoch Schonlein purpura), presenting with cutaneous and musculoskeletal manifestations. Skin biopsy revealed a leukocytoclastic picture. Showed complete resolution of symptoms to steroid therapy. HSP is the most common cause of childhood vasculitis, characterized by tetrad of arthritis or arthralgia, palpable purpura, gastrointestinal involvement and renal involvement mainly in the form of glomerulonephritis. It is multisystem disease where after exposure to inciting agents like infections, immunisation, various drugs antigen-antibody (IgA) complexes are formed, which in turn activate the alternate complement pathway, resulting in small vessel vasculitis. Systemic Steroids are advised for management of moderate to severe HSP, whereas mild diseases resolve spontaneously, symptomatic treatment is sufficient. Main prognostic factor is involvement of renal system. Hence early diagnosis especially in patients of age group outside for typical age group of 4-7 years, as in our adult patient and early intervention can prevent organ damage hence providing better outcome.

KEY WORDS

IgA vasculitis, Henoch schonlein purpura

Adult IgA Vasculitis (Henoch Schonlein Purpura) : A Case Report And Review Of Literature

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Vasculitis (Henoch schonlein
purpura): A case report and review of
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1 -1 DR Anita Basavaraj 31/03/2022

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Type of article: Case Report

Title of the article:

Running title : Adult IgA Vasculitis
(Henoch schonlein purpura): A case

report and review of literature

Contributors

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Corresponding Author:

Total number of pages: 9

Total number of photographs: 3

Word counts

for abstract: 159

for the text: 2218

Source(s) of support: NONE

Presentation at a meeting: NONE

Organisation NA

Place NA

Date NA

Conflicting Interest (If present, give more details): NONE

Acknowledgement:

Department of general medicine, government medical college, Miraj

Department of pathology, government medical college, Miraj

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KEY WORDS – IgA vasculitis, Henoch schonlein Purpura

CASE REPORT

A 24 year old male from Gomewadi, Maharashtra presented with rashes over both lower limbs since last 7 days and lower limb joint pain since last 5 days. He developed erythematous, non pruritic, non painful rashes over both his lower limbs especially over tibial region whose number gradually increased over the course. On 3rd day of his disease course he developed pain in multiple joints of lower limbs especially bigger joints like knee and ankle joints, aggravated by ambulation. Later both lower limbs till knee became swollen. He didn't give any positive history related to other systems. He didn't have any significant comorbidity. His personal and family history also were not significant. There was no history of any addiction or drug abuse. On physical examination, there was palpable, non tender, non blanching, non pruritic, purpuric rash over both lower limbs especially over tibial areas associated with local non pitting edema. Laboratory tests showed Hb 12.8 g/dL, Hct 42%, TLC 15900/microL, platelet count 2.8 lakh/micorL, serum creat 0.8 mg/dL, BUL 28 mg/dL, RBSL 88 mg/dL, bilirubin total 0.6 mg/dL, direct 0.2 mg/dL, indirect 0.4 mg/dL, ALT 27 IU/L, AST 32 IU/L, ALP 110 IU/L, proteins Total 6.7 g/dL, Albumin 4 g/dL, Globulin 2.7 g/dL, prothrombin time 12 sec (control 12), INR 1, aPTT 24 sec, ESR 48mm/hr, CRP 4.4 g/dL, ASO titre 80 IU/mL, RA factor 10 IU/mL, ANA negative, p and c ANCA negative, HIV negative, HbsAg negative, anti Hbc IgG negative, Dengue profile negative, PS for MP and MP card test negative, Weil felix test negative, urine routine normal, USG abdomen pelvis showed grade 1 fatty liver. Skin punch biopsy showed Leukocytoclastic vasculitis with pustules formation. The patient was diagnosed with IgA nephropathy (HSP) as per American college of Rheumatology and European League Against

Rheumatism (EuLAR) and Pediatric Rheumatology Society (PReS) criteria. He was treated with intravenous fluids, painkillers initially. After biopsy report he was started on Oral prednisone 50mg/day divided in 2 doses (dose 1mg/kg/day, patient weight 51kg) given for 2 weeks, which was gradually tapered over period of 2 more weeks with resolution of symptoms and corresponding decrease in ESR and CRP.

Figure 1

Figure 2

REVIEW OF LITERATURE

INTRODUCTION

HSP is a multisystemic, non granulomatous, autoimmune complex mediated small vessel vasculitis. Etiology is unclear but is associated with infections, immunisation, various drugs, tumors (blood malignancy, esophageal cancers, non small cell cancer of lung), insect bite. HSP is the most common vasculitis in children accounting for 90% cases [1-3].

EPIDEMIOLOGY

The annual incidence varies geographically from 6.2 to 70.3 per 1,00,000 in children of age less than 17 years with slight male predominance (M : F = 1.2 : 1). Peak age of incidence is 4-7 years and 90% cases occur before the age of 10 years. Worldwide Asians have the highest incidence and least incidence found in Africa. Incidence in adults varies between 3.4 - 14.3 per million people. As the disease is self limited and many of the mild cases undergo unreported, it's true incidence may be underreported [4,5]

PATHOPHYSIOLOGY

Antigen and antibody complexes, mainly IgA, form as a result of various inciting agents like infections, vaccines, drugs and Autoimmune mechanisms. These Ag Ab complexes deposit in small vessel walls and activate alternate complement pathway which leads to neutrophil accumulation resulting in inflammation and vasculitis without a granulomatous reaction. This can involve multiple systems including skin, GIT, renal system, musculoskeletal system.

Figure 3

CLINICAL FEATURES

HSP is characterized by classic tetrad of non thrombocytopenic palpable purpura, arthritis, gastrointestinal and renal involvement. Other systems like lungs, CNS, genitourinary involvement is less common [4,6,7]. In Pediatric population GIT involvement is most common, where as in adults skin and joint

involvement predominates.

Skin manifestations include non thrombocytopenic rash which evolves from erythematous rash to non blanching palpable purpura with petechiae and ecchymosis. This is the most common presenting feature in adults. These lesions occur in groups and may persist up to 7-10 days. Classically it is symmetrical in distribution involving dependent areas such as lower extremities and buttocks but can also be seen in upper extremities. Face and trunk involvement can be seen. Initially the lesions are single but later may coalesce to form ecchymosis. Rarely Hemorrhagic bullae, ulcers can be seen. In three fourth of the patients skin manifestations precede gastrointestinal manifestations. On histopathology leukocytoclastic vasculitis, characterized by neutrophilic infiltration and prominent nuclear fragmentation, involving the upper and middle layers of dermis with IgA deposition in immunofluorescence is seen.

Abdominal pain, colicky type with worsening on food intake is the most common gastrointestinal presentation. Other symptoms include nausea, vomiting, hematemesis, melena, hematochezia. These symptoms are secondary to mesenteric vasculitis. Rarely intussusception, ischemic necrosis of bowel wall, intestinal perforation, pancreatitis, Biliary cirrhosis may occur. Endoscopic findings include erythema, edema, petechiae, ulcers, nodular changes, ecchymotic lesions, strictures. Ulcers are usually small, less than 1cm size, superficial, multiple, irregular. The second portion of duodenum is most commonly involved.

Joint involvement is seen in up to two third cases. This is more common in adults compared to Pediatric HUS. In one fourth cases it may be the initial presenting complaint. Typically symmetrical, non migratory, non destructive polyarthralgia occurs. Knees and ankles are the most commonly involved joints.

Glomerulonephritis is most common renal manifestation. Hematuria is most common finding. Proteinuria is also seen usually along with hematuria. Most HSP nephritis cases resolve spontaneously, only 5% progress to ESRD at 5 years. Persistent hematuria and proteinuria predict development of ESRD. It is the most important prognostic factor, it is the most common cause of mortality in HUS patients.

DIAGNOSIS

HSP is a clinical diagnosis but when the presentation is atypical, tissue biopsy may be helpful. Two diagnostic criterias are proposed. American college of Rheumatology criteria, 1990 and newer one is EuLAR/PReS criteria (European League Against Rheumatism and Pediatric Rheumatology Society), 2006 [8,9].

A. American college of Rheumatology criteria, 1990

Three of the following four criteria are needed

1. Age of 20 years or less at disease onset
2. Palpable purpura

3. Acute abdominal pain with GI bleeding
4. Biopsy showing granulocytes in the walls of small arterioles or venule in superficial layers of skin.

B. EuLAR/PreS criteria, 2006

Mandatory criteria- Palpable purpura

PLUS

At least one of the following four criteria

5. Diffuse abdominal pain
6. IgA deposition in any biopsy
7. Arthritis/arthralgia
8. Renal involvement (hematuria and /or proteinuria)

DIFFERENTIAL DIAGNOSIS

9. HUS/TTP
10. Hypersensitivity vasculitis
11. Drug induced vasculitis
12. IgA nephropathy
13. Crohns disease
14. Wegener's granulomatosis
15. Infective endocarditis

TREATMENT

HSP has self limiting course. Symptomatic treatment is sufficient for symptoms such as rash and arthritis. Acetaminophen and NSAIDs can be used. Aspirin should be avoided in children because of risk of Reye syndrome.

Oral Steroids are indicated in patients with moderate to severe HUS presenting with severe rash, edema, severe colicky abdominal pain, renal involvement. Prednisone is The drug of choice, started at dose of 1mg/kg/day for 1-2 weeks then gradual tapering over next two weeks. Steroids shown to prevent major complications like GI bleeding [10,13,14,15]. High dose IV pulse Steroids of 500mg to 1 gram, are indicated in patients with nephrotic range proteinuria and mesenteric vasculitis. Nephrologist opinion and renal biopsy is recommended in patients with renal involvement.

Immunosuppressive drugs like Cyclophosphamide, azathioprine, cyclosporine A, mycophenolate mofetil in combination with high dose IV pulse Steroids are recommended if there is no benefit from Steroids alone especially in patients with rapidly progressive glomerulonephritis, hemorrhagic involvement of lungs or brain [10,11,12].

Plasmapheresis or high dose IVIg therapy may be recommended if patients are refractory to steroids or

immunosuppressive drugs [16,17,18]. Some studies also show beneficial effects of colchicine especially in chronic HSP.

PROGNOSTIC FACTOR [19,20,21,22]

Most cases are self limited, with very good prognosis and 5 years survival rate of 95%. One third patients have relapses but usually involving the same organs. Prognosis depends on the age of onset, extent of renal involvement and its course, extent of skin involvement especially in upper extremities, neurological involvement. Following are worse prognostic factors- age more than 8 years, repeated relapses, higher creatinine level, proteinuria of more than 1gram/day, purpura above waist line, persistent purpura, raised ESR, raised IgA level at time of diagnosis, low factor XIII levels.

CONCLUSION

HSP presents in adults often without a classic preceding upper respiratory tract infection. Underlying cancers, drug exposures, vaccination may be present. In some no inciting event is found.

Although HSP is uncommon in adult age group, non thrombocytopenic palpable purpura with systemic involvement of gastrointestinal, renal and musculoskeletal system should make one consider HSP as one of the differential diagnosis.

Diagnosis is clinical but can be confirmed by biopsy showing leukocytoclastic vasculitis and IgA deposits.

Treatment is supportive and corticosteroids are useful in moderate to severe HSP, though their role in preventing progression of end organ damage is uncertain.

Early diagnosis and treatment can prevent the complications and limit the organ damage providing better prognosis.

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Acknowledgement

No

Conflict of Interest

Financial Support and Sponsorship

No

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How to cite the Article

<http://mjwi.org/article-detail.php?artid=182>

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