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LOSARTAN INDUCED HENOCH SCHONLEIN PURPURA: A CASE STUDY.

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ABSTRACT

Henoch-Schonlein purpura is an acute small-vessel vasculitis, characterized clinically by a nonthrombocytopenic purpuric rash, nondeforming arthritis, gastrointestinal involvement, and nephritis. The incidence of HSP is about 10 cases per 100,000 per year, but 90% of cases are found in those less than 10 years of age. Kidney involvement occurs in 30-50% patients. A male patient of age 55yrs have hypertension and on losartan produce Henoch Schonlein Purpura. Proper mechanism behind this is unknown, we think it may be due some immune mediated mechanism by this given drug.

KEYWORDS: Henoch schonlein purpura, thrombocytopenic purpuric rash, nephritis.

INTRODUCTION

Henoch-Schonlein purpura (HSP) is a leukocytoclastic vasculitis involving small vessels with the deposition of immunoglobin A which can affect the skin, joints, bowel and kidneys.[1] It is also known as IgA vasculitis (IgAV). IgA is a form of antibody that we all make, to protect the lining of the airway, throat, and gut.[4]

The incidence of HSP is about 10 cases per 100,000 per year. It affects all ages, but 90% of cases are found in those less than 10 years of age, with the median age at presentation being 6 years.[3]

CASE REPORT

A male patient of age 55yrs, admitted in general medicine department, had the past history of hypertension and was on T. Losartan since 2015. On the last week, he was admitted to hospital and diagnosed as typhoid fever. And he was treated with T. Azithromycin 500mg/day and T. Cefoperazone & Sulbactum 2g/day for 7 days after testing the antibiotic sensitivity by disc diffusion method. And after a week of diagnosis and treatment, he admitted to hospital with the complaints of fever, abdominal pain with distension and nausea. While referring the record we found that he had no family history of this illness and have no habitual history of smoking, drinking alcohol etc. The past medication history reveals that he only taking T. Losartan and the antibiotic drugs, prescribed for typhoid fever.

Physical examination of the patient shows that he had an elevated blood pressure of 180/116mmHg and had body temperature of 100^0F. And the laboratory data shows that he had a high Complete Blood Count, 12,450cell/mm^3, Erythrocyte Sedimentation Rate of 54mm/hr, high serum creatinine value, 2mg%, proteinuria (50mg/dl), urinary lymphocytes, haematuria, high phosphate level in urine (3g/day), high protein creatinine ratio (1.4mg/mg). After the laboratory investigation, patient was treated with medications such as Inj. Ceftriaxone 2gm IV BD, Inj. Pantoprazole 40mg OD, Inj.Ondanserton 4mg SOS, Inj. Acetaminophen 1g IV. Because of this elevated renal parameters, the patient is referred for nephrology consultation. He diagnosed it as Chronic Tubulo-Intestitial Disease. After two days of admission bright red coloured rashes appears on the left leg of the patient and it progress as day passes and produce oedema on the same leg. So he is referred for dermatology consultation. Then the patient is admitted for skin biopsy and add on the prescription with T. Vitamin C 500mg OD, T. Hydroxyzine 10mg BD, T. Multivitamin OD, Fusidin H cream for local application. Skin biopsy shows he had leucocytoclastic vasculitis, Henoch Schonlein Purpura.

After the final diagnosis of the disease by skin biopsy, we stopped the antihypertensive medication and replaced it with Amlodipine (given only if BP >180/100mmHg) show a greater reduction in the progression and spreading of rash. To asses this we conduct further laboratory investigation showing a reduction in Complete Blood Count, Erythrocyte Sedimentation Rate, i.e.32mm/hr, Blood Urea Nitrogen, reduced serum creatinine level, 1.6mg%, reduction in proteinuria and urinary lymphocytes level.

DISSCUSSION

Henoch-Schonlein purpura is an acute immunoglobin A mediated disorder characterised by genaralised

vasculitis. Skin findings show erythrematous macular or urticarial lesions and the renal findings shows Acute Glomerular lesions, endocapillary proliferation, necrosis, blood in urine and leukocyte infiltration.[6] Although there is a general agreement that HSP nephrotic proteinuria is more in children than in adults.[5]

Ig A complexes with complement deposited in target organs, resulting in elaboration of inflammatory mediators including prostacyclin play a central role in the pathogenesis of HSP. An antigen stimulate the production of IgA which in turn cause vasculitis. [6] Drugs such as losartan, quinidine, monteleukast, adalimumab, cytarabine and the antibiotics such as erythromycin, [6] azithromycin, [8] ampicillin, pencillin [6] and cephalosporins, cefoperazone- subactum.[9] were also act as antigens. Here the patient is on losartan since 2015 and a week before admission he was treated with antibiotics such as azithromycin and cefoperazone sulbactum. And after a week he was diagnosed as HSP. And he had no family history of this disease. So in this case the HSP is drug induced by losartan, that he had taken it for a long period and is aggravated by combined consumption of azithromycin and Cefoperazone-sulbatum.

Alternation in the production of interleukins (ILs) and growth factors may also play a pathogenic role.[6] Cytokines has also been implicated in the pathogenesis of HSP and endothelins (ET), which are vasoconstrictor hormone produced by endothelial cells, may also have a role. Level of ET-1 are substantially higher during the acute phase of the disease.[6]

No specific medication to shorten the duration of HSP to any degree. Management include immediate discontinuation to any exposure to antigenic stimulants includes drugs and adequate hydration. Used NSAID such as acetaminophen, naproxen, Ibuprofen for joint pain. In some cases, corticosteroid can also be used. Nephropathy is treated supportively by maintaining fluid and the electrolyte balance, restrict salt intake and antihypertensive should be prescribed when needed. Because of this, Amlodipine is prescribed only for BP >180/100mmHg. Various drugs (eg: corticosteroids, azathioprine, and cyclophosphamide) and plasmapheresis have been used to prevent renal disease from progressing. [7] Rate of complete remission of 72.5% at an average of 20 months follow up. However one third of patients have one or more recurrences.[6]

Henoch Schonlein purpura is very rare in adults. The case of 55yr old male patient is Drug induced HSP caused by Immunoglobin A activation produced by the combined effect of Losartan, Azithromycin and Cefoperazone- Sulbactum. This Ig A complexes with complement deposited in target organs, resulting in elaboration of inflammatory mediators including vascular prostaglandins such as prostacyclin, there by producing vasculitis. Proper mechanism behind this is unknown. The proper treatment for curing the disease so far. But there is only some relevant supportive measures such as discontinue the given drugs that stimulate antigenic exposure, give antihypertensive such as Amlodipine, Clonidine, Atenolol, etc. only if necessary and maintain fluid and electrolyte balance. Patients with this disorder suffered a lot due to the lack of adequate medication, so the necessity of finding proper treatment is so high.

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