Development of Henoch-Schoenlein Vasculitis Following Ranitidine Administration in a Geriatric Patient.

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Abstract:

Henoch-Schoenlein Vasculitis following drug administration is not so common. Here, a case of Henoch-Schoenlein Vasculitis caused by Ranitidine and Naproxen is described which presented with fever, vomiting bloody diarrhea, palpable purpura and azotemia. Histology of the skin showed leukocytoclastic vasculitis of superficial vessels and direct immunofluorescence revealed immunoglobulin A (IgA) in superficial dermal vessels and so confirmed the diagnosis. The patient was treated with Ranitidine discontinuation and Prednisone administration. A short review of literature is also made.

Key Words: Henoch-Schoenlein Vasculitis, Ranitidine, Naproxen.
Introduction:

Henoch-Schönlein purpura (HSP) is a self-limited systemic vasculitis. It is the most common systemic vasculitis of childhood and is characterized by a tetrad of clinical manifestations that vary in their presence and order of presentation. Adult patients with HSP present with the same clinical manifestations as seen in children\(^1\).

In 1990, the American College of Rheumatology Classification established criteria to classify vasculitides including HSP\(^2\). Based upon these criteria, the diagnosis of HSP required the following: Palpable purpura without thrombocytopenia and coagulopathy, Age at onset 20 years, Gastrointestinal bleeding, Biopsy which showed granulocytes around arterioles and venules. Two or more of the criteria had a sensitivity and specificity of approximately 90 percent in separating adult patients with HSP from those with other causes of vasculitis\(^3\). There are case reports about development of HSP in patients with ranitidine administration.\(^4, 5\)

We describe here a case of henoch-schoeinlien purpura after ranitidine consumption.

Case Presentation:

A 74 year old man was admitted with vomiting and bloody diarrhea. He was well until 2 weeks before admission when due to musculoskeletal pain received naprooxen and ranitidine and after 8 days developed abdominal pain, bloody diarrhea, joint pain, general malaise and purpuric rashes on lower extremities and buttock. He discontinued his medication including ranitidine and naproxen and symptoms were removed after one week. His dyspepsia continued so he started ranitidine again. While He was taking ranitidine alone for about 5 days he developed nausea vomiting, bloody diarrhea, joint pain and purpuric rash. On admission the patient was febrile (38.1 c), blood pressure, heart rate and respiratory rate were within normal range. Conjunctiva was pale, lungs were auscultated symmetrically clear bilaterally. Heart sound was normal. Abdomen was soft without any organomegaly but with mild diffuse tenderness. Multiple round, confluent, purpuric lesions with some area of residual pigmentation was seen on both upper and lower extremities and buttock.

Laboratory examination revealed severe azotemia (BUN:116 mg/dl, Cr: 6.5mg/dl). Urinalysis showed proteinuria of 0.9 g/24 h and microscopic hematuria. Urine culture was negative. C reactive protein was 96mg/dl. Hemoglobin was 8.6 mg/dl, leukocyte count was 12000/mL and platelet was 450000/mL. Prothrombin time and partial thromboplastin time were normal. Antistreptolysin O, antinuclear antibodies, anticardiolipin antibody, antinutrophil cytoplasmic antibody, cryoglobulins, and hepatitis B, C and HIV serologies were all negative. Kidney sonography showed increase of cortical echogenecity of both kidneys in favor of parenchymal damage. Histology of the skin showed leukocytoclastic vasculitis of superficial vessels and direct immunofluorescence revealed immunoglobulin A (IgA) in superficial dermal vessels.
Ranitidine was discontinued and prednisone (1 mg/kg/day) was started and the arthralgia, hematochezia, and eruption resolved over the next 7 days. Renal function gradually improved and patient did not need hemodialysis. He was discharged while was on prednisone and omeprazole.

Discussion:

The underlying cause of HSP is unknown. It has been postulated that HSP is an immune-mediated vasculitic disorder that is triggered by an immunoglobulin A (IgA) dominated response to an infection or chemical antigen. Drug induced hypersensitivity vasculitis and HSP are diseases that were reported with respiratory tract and intestinal infection, food allergy, insect bites, immunization and various drugs such as: Ranitidine, Acetaminophen codeine, Etanercept, Clarithromycin, Propyliouracil, Metoclopramide, Carbidopa/levodopa, Angiotensin converting enzyme inhibitors, Co-dydramol, Ampicillin and amoxicillin, NSAIDS, Diclofenac, Piroxicam and Naproxen, Streptokinase, Calcium channel blockers, Quinidine. Histologic findings in skin biopsy of this patient were in favor of hypersensitivity vasculitis and also a clinical diagnosis of HSP could be made based on features characteristics of the syndrome. This patient's problem occurred while was taking both naproxen and ranitidine and after discontinuation of these medication his purpuric rash and other symptoms disappeared. Hyper sensitivity vasculitis was reported with both naproxen and ranitidine but in this patient after reconsupmption of ranitidine alone his purpuric rash, arthritis and bloody diarrhea reappeared thus the relapse of clinical symptoms on rechallenge with ranitidine, strongly suggest that ranitidine was causally related to the acute Henoch-Schonlein Purpura. Upon rechallenge renal involvement is more likely to occur and to be severe in this adult patient. Adults are at increased risk for developing significant renal involvement requiring more aggressive therapy.

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