Special Feature: "Clinical image"

Drug-induced cutaneous hypersensitivity vasculitis

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A 20-year-old male patient known to have rheumatic heart disease, severe mitral stenosis and moderately severe mitral regurgitation, mild pulmonary arterial hypertension underwent mitral valve replacement surgery and had been discharged from the hospital. Five days after discharge he presented to medicine outpatient service with cutaneous lesions on both upper and lower limbs of 2 days duration. There was no history of fever, arthralgias, and abdominal pain. Physical examination revealed palpable purpuric lesions predominantly on both lower and upper limbs suggestive of cutaneous small vessel vasculitis (Figure 1). There were no peripheral signs of infective endocarditis. Cardiovascular system examination revealed prosthetic valvular click in mitral area. Rest of the physical examination was unremarkable. The patient was evaluated for the aetiology of small vessel vasculitis and other organ involvement. The following laboratory investigations were done: complete haemogram including platelet count, serum biochemistry including liver and renal functional tests, prothrombin time, activated partial thromboplastin time, urinalysis and chest radiograph (postero-anterior view) were all found to be normal. Serological testing for human immunodeficiency virus, hepatitis B surface antigen, anti-hepatitis C immunoglobulin M antibodies, anti-nuclear antibody profile, cytoplasmic antineutrophil cytoplasmic antibodies(C-ANCA) and perinuclear antineutrophil cytoplasmic antibodies (P-ANCA) was negative. Review of drug history revealed that he was receiving oral ciprofloxacin for the preceding 5 days. A diagnosis of ciprofloxacin-induced cutaneous small vessel vasculitis was considered based on the American College of Rheumatology 1990 criteria for the classification of hypersensitivity vasculitis (Table 1).[1] The patient was advised to stop treatment with ciprofloxacin and a skin biopsy was being considered as the next step in diagnostic

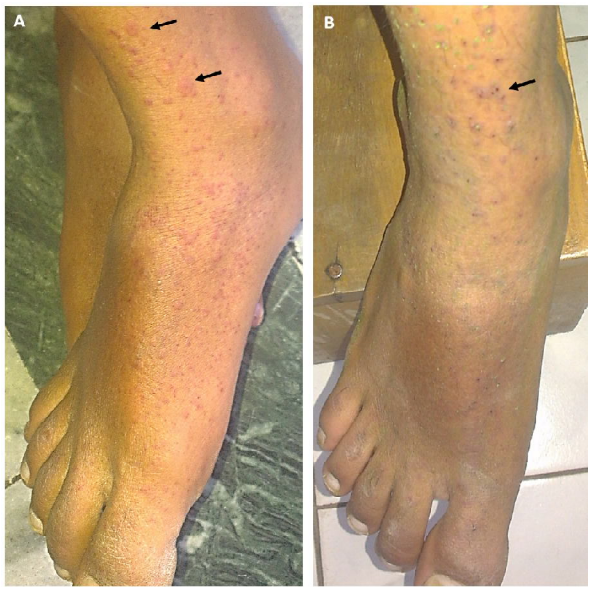

Figure 1: Clinical photograph at the time of initial presentation (**A**) showing palpable purpuric lesions (arrow headss) and petechiae on right foot. Clinical photograph of the same patient obtained after one week (**B**) showing healed lesions with hyper pigmentation (arrow)

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Table 1: The American College of Rheumatology 1990 criteria for hypersensitivity vasculitis*

Age >16 years at disease onset
History of taking a medication at onset that may have been a precipitating factor
The presence of palpable purpura,
The presence of maculopapular rash
A biopsy demonstrating granulocytes around an arteriole or venule

* Presence of 3 or more of these 5 criteria was associated with a sensitivity of 71.0% and a specificity of 83.9% for the diagnosis of hypersensitivity vasculitis

Source: reference 1

evaluation. Over the next one week, the purpuric lesions healed with hyperpigmentation (Figure 1). As there was significant clinical improvement, skin biopsy was not done.

Some of the important causes of small vessel vasculitis include granulomatosis with polyangiitis (Wegener's granulomatosis), eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome), microscopic polyangiitis, Henoch-Schönlein purpura [immunoglobulin A (IgA) vasculitis], cryoglobulinemic vasculitis, hypersensitivity vasculitis, and vasculitis secondary to connective tissue disorders**.** Common drugs implicated in drug induced hypersensitivity vasculitis are penicillins, cephalosporins, sulphonamides, phenytoin, and allopurinol among others including fluoroquinolones (ciprofloxacin, ofloxacin and levofloxacin).[2,3]

Early recognition of drug-induced hypersensitivity vasculitis is important because withdrawal of the offending drug can prevent the progression of the disease, which may be life threatening and avoid subjecting the patient to unnecessary investigations.

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