Leucocytoclastic vasculitis: A complication after streptokinase therapy for acute myocardial infarction

Sir,

Leucocytoclastic vasculitis (LCV) is a reaction pattern of small dermal vessels, almost exclusively postcapillary venules, characterised by a combination of vascular damage and infiltrate composed largely of neutrophils.[1] Vasculitis has been reported as a rare complication of intravenous streptokinase but histopathological examination (HPE) has been rewarding. Less than 10 cases have been reported in the literature so far on Pubmed search.

A 62-year-old male patient presented to the emergency with severe chest pain. After all the investigations, the diagnosis of acute myocardial infarction was made for which he was given 30 units of intravenous streptokinase, without heparin. After 6 h of treatment, he developed palpable purpuric rashes on the extensor aspects of arms and legs, with few on the abdomen. All routine investigations including complete blood counts, blood urea, serum creatinine, liver function tests and clotting studies were normal. Urine microscopy was also negative. Special tests for antinuclear antibodies, rheumatoid factor and cryoglobulins were also negative. Biopsy from the lesion was obtained and sent for HPE. Haematoxylin and eosin (H and E) stained sections revealed perivascular inflammatory cell infiltrate, mainly neutrophils and their fragmentation (leucocytoclasia). Dermal vessels show swelling of endothelial cells and deposits of strongly eosinophilic strands of fibrin within and around the vessel wall. Extravasation of erythrocytes was also present along with dermal oedema [Figures 1 and 2]. Staining by direct immunofluorescence showed C3 and IgM deposits around the blood vessels. These findings were consistent with LCV.

The term LCV means karyorrhexis or fragmentation of nuclei. A large number of different disease processes can be accompanied by neutrophilic small vessel vasculitis mainly infections, immune-complex-mediated disorders like Henoch-Schonlein purpura, cryoglobulinaemia, serum sickness, autoimmune diseases, drug-induced paraneoplastic process, Behcets disease, antineutrophil antibody-associated like Wegner's granulomatosis, microscopic polyangitis, Churg-Strauss syndrome and polyarteritis nodosa. A hypersensitivity reaction to a drug can cause LCV. Drugs responsible are penicillin, thiazides, sulphonamides and rarely after, intravenous

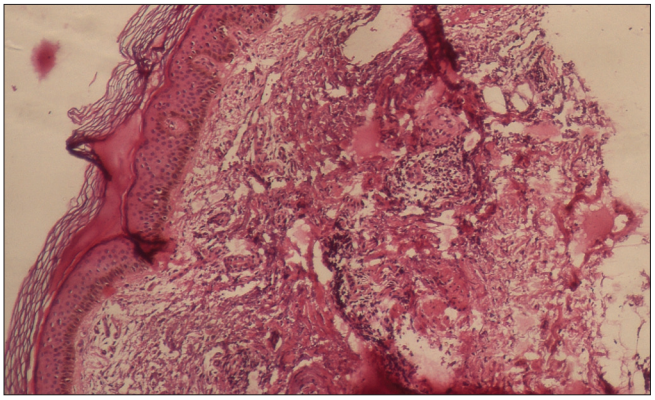

Figure 1: Photomicrograph of skin biopsy specimen showing leucocytoclastic vasculitis showing inflammatory infiltrate and fibrinoid necrosis surrounding the blood vessels, along with dermal oedema and extravasation of RBCs (H and E, ×100)

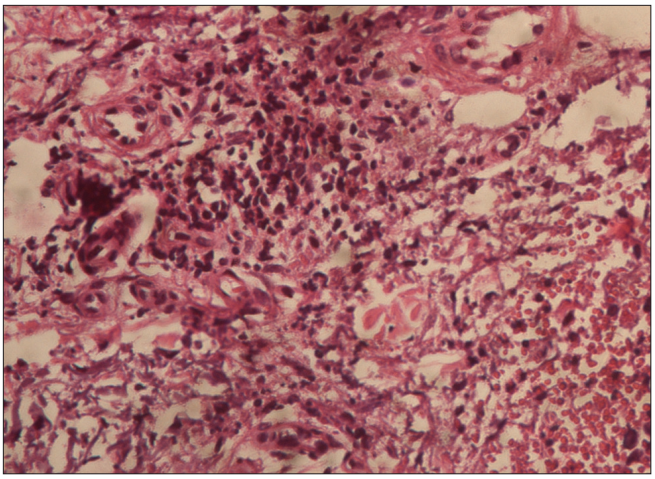

Figure 2: Photomicrograph showing dense perivascular neutrophilic infiltrate and extravasation of erythrocytes (H and E, ×400)

streptokinase. Clinical and histological manifestations are non-specific for particular category of vasculitis. It is, therefore, important to interpret the histological findings in context of clinical information along with other laboratory data to reach an appropriate diagnosis. Exposure to any potential allergen, such as a drug that might have elicited a hypersensitivity reaction should be sought. Antibody mediated inflammation plays a prominent role in pathogenesis of small vessel vasculitis. Its final pathway typically involves neutrophilic and monocyte activation with adherence to endothelial cells, infiltration of vessel wall and subsequently the release of lytic enzymes and toxic radicals.[1,2]

LCV is a seldom reported side-effect of streptokinase and may be caused by Type-3 hypersensitivity syndrome. Smithson *et al*., in their study on skin lesions associated vasculitis with intravenous streptokinase in three patients.[3] A review of recent trials do not mention

purpuric rashes, though this complication was found in 0.8% of patients receiving it.[4] Patients usually recover spontaneously.

We, hereby, report early development of purpuric rash as a sole complication, after treatment with intravenous streptokinase.

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