**Letter to the Editor.**

# Streptokinase induced Purpura like lesions.

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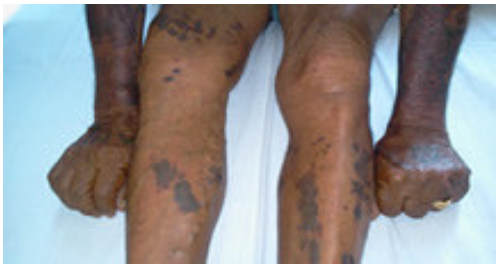
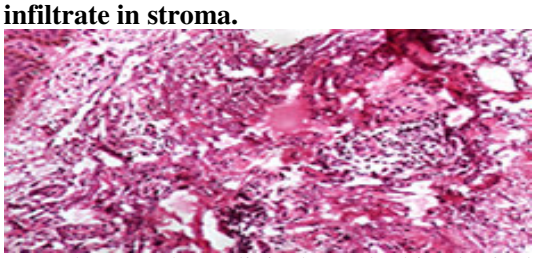
We present a case of Streptokinase induced vasculitis presenting as purpuric lesions immediately after streptokinase therapy. Streptokinase is a frequently used thrombolytic agent in clinical practice. The common unwanted effects include hemorrhage, fever, and an immediate allergic reaction . Serum sickness [1,2] and crescentic glomerulo-nephritis have also been described after streptokinase treatment. In each case the patient had a purpuric rash . A further two papers describe non-specific rashes caused by streptokinase . We report the development of small vessel vasculitis in a patient treated with streptokinase for acute myocardial infarction. Eighty year old male was admitted with myocardial infarction. He was known hypertensive and was on treatment with beta blockers, asprin and statins. His pre thrombolysis bleeding parameters were within normal limits and was thrombolysed with streptokinase 150000 IU. Just after thrombolysis he developed multiple purpuric lesions involving the entire body, predominantly distributed on the extensor aspects of the extremities associated with itching (Fig 1).

**Fig1: Multiple purpuric lesions on extremeties.**



There was no associated arthralgias, fever and joint pains. His blood counts, urine and stool analysis, liver and renal function tests were within normal limits. He had not been started on any oral or injectable anticoagulants or other medications and had no history of any infections in the recent past. Differential diagnosis of drug induced purpura or hypersensitivity vasculitis were considered. Routine investigations were within normal limits. A skin biopsy from a lesion on the left leg revealed hyperkeratosis of stratum corneum, below which there was dense mononuclear infiltrate in both intravascular and perivascular region. Some areas showed extravasation of red blood cells (RBC), edema, fibrinoid degeneration and keryorrhexis (Fig 2).

**Fig 2. (H&E 200 x) Photomicrograph showing numerous blod vessels infiltrated with fragmented neutrophils, marked derma edema and dense neutrophilic infiltrate in stroma.**



Based on the clinical and histological findings, a diagnosis of streptokoinase induced vasculitis was made. The lesions did not aggravate further and started resolving with conservative treatment.

Streptokinase is a non-enzymatic protein (47 kD) produced by group C streptococci . The reported frequency of allergic reactions to the drug varies between 1-7% and 18% [3] of which the majority is mild and self-limiting, taking the form of acute immediate-type hypersensitivity reactions including urticaria, angioedema, bronchospasm and anaphylaxis, and delayed cutaneous lymphocytic reactions have been reported [4]. Although leucocytoclastic vasculitis and serum sickness have been reported after streptokinase treatment, Streptokinase induced vasculitis is a rare entity. The pathogenesis is uncertain but may be a direct toxic reaction to the initial high dose of the drug damaging the capillaries of the dermo-vascular loop with local deposition of immune complexes [5]. There are close antigenic similarities between group A streptococci, which include the common human pathogen S.pyogenes, and group C streptococci from which streptokinase isproduced. Pre-sensitized by a previous streptococcal infection may result in the formation of antibodies cross-reactive with streptokinase resulting in rapid and vigorous secondary immune response when that antigen was administered exogenously resulting in immune complex deposition . Skin lesions in this rare reaction to anticoagulant drugs is known to become dramatically evident between the third and tenth day of therapy. In our case the lesions developed immediately on the day of streptokinase therapy. Such early onset of lesions has not been described so far. Awareness of this complication induced by streptokinase must be known to cardiologists as well to dermatologists. The distinction must be drawn between the long term benefit of myocardial salvage and the small risk of a transient allergic reaction that seems on present evidence to be benign.

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