Scabietic Leucocytoclastic Vasculitis with Focal Glomerulonephritis

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Two cases of leucocytoclastic vasculitis in patients with scabies are reported. One of them developed a focal glomerulonephritis. (Received March 19, 1984.)

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The clinical manifestations of scabies depend on the host response to the mite. Immunologic type I and type IV reactions (Commb's classification) have been documented (1, 2, 3). A generalized cutaneous vasculitis has been reported in two patients with scabies, one of them with the scabies crustosa type (4, 5). Two additional cases are presented. One of these was further complicated by a focal glomerulonephritis.

CASE I

The patient was a 62-year-old male suffering from Parkinson's disease. During 6 weeks had had an intensely itching, widespread eczematous eruption. Treatment with prednisone 5 to 20 mg a day had been given for one week and additional local application of steroid ointments. Twenty-four hours before admission haemorrhagic skin lesions appeared. Examination showed excoriated papules all over the body except of the face. Several scabies burrows surrounded by palpable purpura were seen on his hands. From one of the burrows a live scabies mite was isolated. On the lower legs and in the genital area purpuric palpable lesions 2 mm to 20 mm in diameter were seen. Some of the hemorrhagic elements were bullous, others were necrotic and ulcerated. On the lower legs and on the feet a moderate oedema was present.

Examinations

Bacterial swabs from excoriated lesions showed growth of *Staphylococcus aureus*, phage type 3A/3A/71. S-albumin was significantly lowered (243 mmol/1; normal range 540-800); C-3 pro-activator: 2.8 (0.7-1.6); C-4: 0.9 (0.6-1.4); C1q: 1.3 (0.6-1.3). Urine analysis revealed microscopic haematuria without any erythrocyte or hyaline casts. There was a significant albuminuria that reached a maximum of 3.5 g a day. S-creatinine increased up to 193 (50-130 mmol/1). A punch biopsy from a purpuric lesion on the leg showed leucocytoclastic vasculitis involving capillaries and arterioles of the dermis and subcutis. A kidney biopsy showed a focal glomerulonephritis.

Clinical course. The patient was treated with gamma benzene hexachloride (Hexicid® lotion 1%) which was repeated after one week. Initially the skin improved rapidly, but disperse new lesions continued to develop for several weeks. The haematuria, proteinuria and S-creatinine too normalized gradually over the next 5 months.

CASE II

The patient was a 51-year-old women who in 1975 had undergone operation for parathyroid adenoma and nephrolithiasis. Since 1977 she had had insulin dependent diabetes mellitus. She was admitted to hospital because of an abscess on her right buttock. After surgical intervention, intravenous ampicillin 4 g daily were given for 6 days. Five weeks before admission a generalized itchy eruption had started. The first day after operation extensive haemorrhagic elements developed on the legs.

Examination showed excoriated eczematous lesions on her arms and trunk. On her legs 1 to 3 cm large haemorrhagic palpable elements were present, some of them with bullae and ulcerations. On hands and feet several scabies burrows with a palpable purpura were seen. Live mites wree isolated from the burrows. A slight oedema was present on her feet and lower legs.

Examinations

Bacterial culture from the abscess showed *Staphylococcus aureus*, phage type 3C/55 u. C-3 proactivator: 3.8 (0.7–1.6 u); C-4: 0.9 (0.6–1.4 u). Total IgE: 1 000 u/l (1–120 u/l). Urin analyses revealed an intermittent microscopic haematuria. S-creatinine was repeatedly normal. A punch biopsy from the lesion on the leg showed a leukocytoclastic vasculitis involving the dermal and subcutaneous capillaries and venules. Biopsies from scabies burrows on the hands and feet showed similar changes and contained in addition subcorneal scabietic burrows with eggs and mites.

Clinical course. The patient was treated with Hexicid® lotion 1% which was repeated after one week. After the first Hexicid® treatment, no additional skin lesions appeared and the haemorrhagic vasculitis subsided. After additional five months she was readmitted because of a new Staphylococcus aureus abscess. No signs of vasculitis lesions developed.

DISCUSSION

In the two cases the clinical diagnosis of vasculitis was confirmed by microscopy. No signs of an actual or recent infection with B-haemolytic streptococci was established. In both patients staphylococcus aureus was isolated, which is an unusual cause of allergic vasculitis. In case 2 ampicillin treatment was started two days prior to the onset of vasculitis. The drug is not a likely cause, because after the treatment for scabies the vasculitis regressed in spite of a continuous supply of ampicillin.

In the two presented cases a prolonged scabies infestation is the most likely trigger of the reaction. Apart from allergic vasculitis on the lower legs, both patients had a clinically and histologically verified vasculitis surrounding the scabies burrows. We consider the vascular reaction related to the burrows as being comparable to a positive skin test. IgE antibodies to house-dust mites may be demonstrated in patients with scabies (1), but none of our patients had IgE antibodies or precipitating antibodies to house-dust mites.

Acute glomerulonephritis may follow a scabies epidemic (6) due to secondary infection with haemolytic streptococci. In the cases reported here, the scabies mite itself may be the primary cause of the glomerulonephritis, but a substantial proof for this explanation has not been established.

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