cystitis. The spleen was enlarged and congested with one obvious micro-abscess.

Comment

This patient appears to have had longstanding urinary tract infection in addition to chronic glomerulonephritis. Probably her recent illness developed with an *E coli* bacteraemia originating from the urinary tract, as the central nervous system was seeded with the same strain of *E coli* 09 found in the urine. No enteropathogens were isolated. The septicaemia and uraemia may have accounted for the bloody diarrhoea.[2]

In view of the frequency with which *E coli* septicaemia occurs in adults it is surprising that *E coli* meningitis is rare. The reason for this is not known,[3] though possibly a patient develops *E coli* meningitis only when the immune mechanisms are severely compromised.[3] The uraemia in this patient is one possible cause of immunological anergy[4] and in at least one other report *E coli* meningitis was associated with uraemia.[5] The mortality of *E coli* meningitis is high,[3] partly owing to the difficulty in establishing an early diagnosis.[1] As *E coli* septicaemia and uraemia are not infrequently associated, an awareness of the possibility of meningitis may lead to earlier recognition of this complication.

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Subcorneal pustulosis and IgA myelomatosis

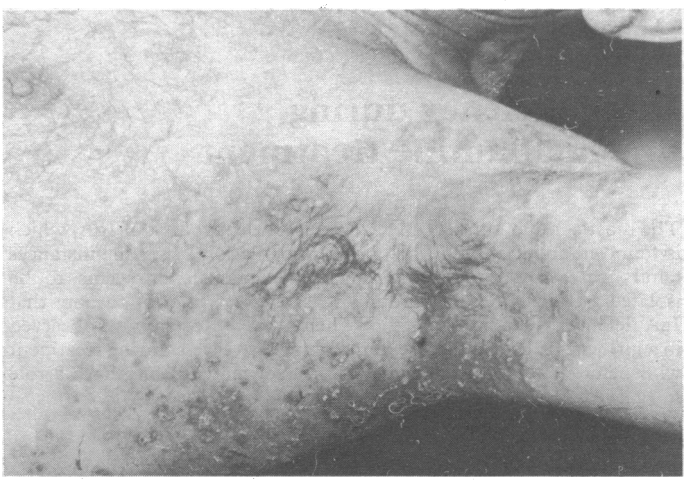
Subcorneal pustulosis is a relapsing condition affecting the groins, axillae, submammary areas, and flexor aspects of the limbs. The cause and pathogenesis remain unknown. It has not hitherto been shown to be associated with any other disorder.[1] We describe a patient with subcorneal pustulosis and multiple myelomatosis.

Case report

A man aged 61 presented in 1975 with a blistering eruption over the axillae and upper thighs. In 1970 a moderately well differentiated adenocarcinoma of the sigmoid colon had been resected. Examination showed erythematous areas with crops of small superficial pustules (see figure) over the axillae, thighs, groins, buttocks, and flanks.

Investigations—Skin biopsy: subcorneal pustule, no amyloid, IgA fluorescence throughout section; erythrocyte sedimentation rate 130 mm in first hour (Westergren); haemoglobin 11·1 g/dl; serum proteins: total 71 g/l, paraprotein (IgA-λ; 37 g/l) serum immunoglobulins: IgG 3·4 g/l, IgM 0·5 g/l; urine: free λ-chains 0·6 g/l; bone marrow: myeloma cells 20% of nucleated cell count; serum complement: CH_{50} 1225 MU/l (control 1000 MU/l), C3 60% (low), total alternative pathway 140% (normal); no serum autoantibodies present against epidermal intercellular substance or basement membrane zone; antinuclear factor and thyroid, gastric, mitochondrial, and smooth-muscle antibodies not detected; blood urea, electrolytes, urate, Ca^{++}, aspartate and alanine transaminases, and cholesterol normal; chest radiograph and skeletal survey normal; no occult blood detected; Wassermann reaction negative; platelet and white cell, and differential counts normal; cryoglobulin absent.

Before myelomatosis was diagnosed he was started on dapsone 100 mg/day with only transient improvement. The dose was increased to 150 mg/day but the haemoglobin fell sharply and the drug was stopped. By this time the diagnosis of myeloma had been confirmed and he was started on intravenous cyclophosphamide 600 mg/m² every two weeks. He also began a course of sulphapyridine tablets, initially 1 g twice daily. The skin failed to improve until the sulphapyridine had been increased to 4 g/day, at which dose the rash was almost completely suppressed, with only occasional pustulation.



Appearance of eruption over axilla.

Comment

The aetiology and pathogenesis of subcorneal pustulosis are unknown. It resembles pustular psoriasis of the von Zumbusch type and dermatitis herpetiformis. Abnormalities of the jejunal mucosa, however, which are common in dermatitis herpetiformis, do not occur in subcorneal pustulosis. Antibodies to stratum corneum have been found in pustular psoriasis and pemphigus foliaceus. Using the same technique Krogh and Tönder[2] studied two patients with subcorneal pustulosis and found antibody to stratum corneum in the roofs of the pustules. No antibody was detected in the blister fluid itself possibly because of depletion, and in one patient the titre of antibody in the serum fell during exacerbations.

The association with multiple myelomatosis in our patient may have been fortuitous, but an IgG cryoglobulin has been found in a patient with subcorneal pustulosis.[3] Our patient's myeloma was producing an IgA paraprotein, and abnormalities of serum IgA have been noted in several cutaneous disorders. In psoriasis, one feature of which is pustulation, raised serum concentrations have been reported.[4] Fraser *et al*[4] also noted raised serum IgA concentrations in dermatitis herpetiformis, which is characterised by deposits of IgA along the basement membrane zone. Few reports of subcorneal pustulosis give the immunoglobulin values. In most instances they were normal, though Peterson *et al*[5] noted increased serum IgA concentrations on immunoelectrophoresis in two patients. Thus there is scant evidence to suggest a relationship between IgA and subcorneal pustulosis. Nevertheless, the emergence of an IgA antibody implicated in the pathogenesis of subcorneal pustulosis need not result in a rise in the total serum IgA.

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