later life, should be surgically treated in childhood. Options include open flexor tenotomy as described by Pollard and Morrison (1975); Ross and Menelaus (1984) thought that this was preferable to a flexor-to-extensor transfer.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

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PYODERMA GANGRENOSUM AFFECTING THE HAND

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Pyoderma gangrenosum is an ulcerative necrotic dermatosis of unknown aetiology (Gasparini et al 1993). The disease may mimic other disorders and is usually diagnosed by exclusion (Perry 1982). It may occur at various anatomical sites (Perry and Brunsting 1957) and in association with other conditions (Holt et al 1980), but has rarely been reported in the hand. Disastrous results can occur if the condition is not treated properly. We report a case that mimicked a local infection of the hand.

Case report. A 63-year-old, right-hand-dominant woman presented with a lesion on her left index finger. She had sustained a paper cut three weeks earlier and had noticed erythema and mild swelling for one week, becoming ulcerated in the last 48 hours. She had no history of fever or gastrointestinal complaints. She was generally well except for intermittent and unexplained tinnitus, and a history of fluid retention, for which she took Aldactazide. She was married, with four children, smoked one pack of cigarettes per day, and denied alcohol or drug use. She had taken a holiday in the Caribbean three months earlier.

Her index finger showed an ulcer 1 cm × 1 cm on its dorsoradial aspect adjacent to the proximal interphalan-geal joint. The ulcer was surrounded by a 1 cm blue-black rim, which was further encircled by a small zone of erythema. There was mild oedema and tenderness and serous fluid could be expressed. All finger movements were normal, and there was no erythematous streaking of the arm or axillary or epitrochlear adenopathy. Neu- rological examination of the hand was normal as was capillary refill in the fingers. A radiograph was normal.

On a diagnosis of bacterial infection, treatment was by antibiotics, immobilisation, and elevation, but after 24 hours, swelling and erythema had increased and the necrotic border of the ulcer had widened (Fig. 1). In view of this, an operation was performed to excise the ulcerated area to apparently healthy tissue. The proximal interphalan-geal joint was opened on the ulnar side of the finger to reveal normal joint surfaces and normal synovial fluid, which was also normal to microscopy.

One day later, necrotic ulceration had reappeared at the border of the lesion, extending into previously
uninvolved tissue. Histological examination of the excised tissue showed evidence of pyoderma gangrenosum, with no signs of bacterial, fungal or mycobacterial involvement.

High-dose steroid therapy with 50 mg of prednisone twice daily was started. During the next 24 hours, the necrotic lesion began to heal and the patient was discharged four days later on oral steroids. Within two weeks the ulcerated area had re-epithelialised, and by four weeks it was completely healed (Fig. 2).

Fig. 2

Discussion. Pyoderma gangrenosum is idiopathic in about 40% of cases, but is more usually associated with other conditions, such as non-specific polyarthritis, rheumatoid arthritis and Crohn’s disease (Hickman and Lazarus 1980). In these cases the skin lesions often improve after treatment of the associated condition (Newell and Malkinson 1982). A history of antecedent injury is found in about 40% of patients (Powell et al 1985).

Pyoderma gangrenosum usually starts with sterile pustules that become ulcerated, nodular plaques (Hurwitz and Haseman 1993). Histopathologically, it is a folliculitis with occasional secondary leucocytoclastic vasculitis. It progresses from a folliculitis to a granulomatous dermatitis and panniculitis (Hurwitz and Haseman 1993).

Our case presented as a single nodular ulcer, probably because of the delay after the first appearance of the lesion. At that stage, the weeping ulcer had the appearance of a local infection.

The diagnosis of pyoderma gangrenosum was made by the absence of bacteria, fungus or mycobacteria, the exuberant reappearance of the necrotic lesion within 24 hours of surgical debridement, and the presence of neutrophils and lymphocytes with evidence of vasculitis (Powell et al 1985). The deterioration after debridement is particularly interesting; this so-called pathergic response (Shands et al 1987) is one of the hallmarks of the disease.

The only other reported case of pyoderma gangrenosum of the hand which we could find was in a patient with diffuse bullae (Papilion and Bergfield 1990). The eventual diagnosis was of an atypical form of pyoderma gangrenosum; the lesions responded to high-dose steroids. The differential diagnosis of pyoderma gangrenosum includes brown recluse spider bite, erythema multiforme, and atypical bacterial, fungal or mycobacterial infections (Wustrack and Zarem 1978). Systemic steroids are the best treatment (Papilion and Bergfield 1990), but other primary and adjunctive agents have been proposed including potassium iodide (Richardson and Callen 1993) and low-dose cyclosporin (Schmitt et al 1993).

The lesion in our case improved dramatically with systemic steroids and local wound care. The outcome of serial debridement and aggressive wound care would probably have been disastrous. The diagnosis of pyoderma gangrenosum must be considered for ulcerative lesions of the hand which follow an unusual clinical course with conventional treatment.

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