We describe a case of pyoderma gangrenosum which presented with severe wound breakdown after elective hip replacement. The patient was treated successfully with minimal wound debridement and steroids. This diagnosis should always be considered when confronted with an enlarging painful skin lesion which does not grow organisms when cultured and fails to respond to antibiotic therapy, especially if there are similar lesions in other sites. In patients who have a past history of pyoderma gangrenosum, prophylactic steroids may be indicated at the time of surgery or may be required early in the postoperative period.

Pyoderma gangrenosum is a condition causing progressive necrotising ulceration of the skin. The exact aetiology is unknown. It may be associated with several chronic diseases including ulcerative colitis, Crohn’s disease, diverticulitis, hepatitis, rheumatoid arthritis, polycythaemia rubra vera and malignancy. In about 20% to 30% of patients there is no underlying cause. It is a rare condition, but previous reviews have found many reports in the literature of its occurrence after surgery.

Case report

A 69-year-old man was transferred to the Oxford Bone Infection Unit 13 days after total hip replacement. He had an open wound with a large surrounding area of ulceration and necrotic tissue (Fig. 1a). The fascia lata was visible in the base of the wound. He also had an ulcerating lesion on the back of his hand at the site of an intravenous cannulation. He had been fit before operation. One year before he had sustained dog bites to his legs which had ulcerated. They had been extensively debrided at another hospital before a diagnosis of pyoderma gangrenosum had been made. He had required substantial skin grafting and systemic steroids for that problem, but the wounds were healed and required no treatment at the time of the hip replacement.

The wound of the hip arthroplasty broke down two days after surgery and he became systemically unwell. No organisms were grown from superficial swabs or deep tissue samples. Despite intravenous administration of teicoplanin, cefotaxime and metronidazole, and two debridement procedures the wound deteriorated. His C-reactive protein level was >300 mg/l, his ESR 121 mm/hour and his WBC 3.4*10^9/l. A presumptive diagnosis of necrotising fasciitis was made. Histological specimens showed a non-specific acute inflammatory infiltrate with patchy necrosis. No organisms were seen with Gram staining. The histological findings were consistent with a diagnosis of necrotising fasciitis or pyoderma gangrenosum.

He was therefore given prednisolone (40 mg daily) and improved dramatically over the next two days. Both wounds stopped spreading; each was cleaned and dressed daily with flamazine dressings. Two weeks later he had a minor debridement of the hip wound, the deep part of which was closed over gentamicin-impregnated methylmethacrylate beads. Antibiotics were continued (piperacillin 4.5 g t.d.s. for three weeks and metromidarole 400 mg t.d.s for six weeks) contamination of the hip prosthesis. Skin grafting was considered but there was concern about healing at the donor site; it proved to be unnecessary and the wound slowly healed spontaneously (Fig. 1b). The patient was mobile 11 weeks after the initial operation with healed wounds. He was discharged on a reducing course of oral steroids (prednisolone).

Discussion

Clinically, pyoderma gangrenosum presents with pain followed by the formation of pustules. A haematoma-like lesion forms which becomes a sterile abscess spreading...
concentrically, undermining the skin edges to become a large necrotic ulcer with raised blue/purple borders. Histological examination shows no pathognomonic features, only a non-specific acute inflammatory reaction. The wounds are sterile and the organisms cultured are considered to be contaminants due to secondary bacterial colonisation. Pathergy is a feature of pyoderma gangrenosum with new lesions arising at sites of minor trauma, such as intravenous cannulation, as occurred in our patient.

When making the diagnosis it is important to consider a past history of the disease or of any of the known associated systemic disorders. The diagnosis should always be considered if there is an enlarging, painful skin lesion, which fails to respond to antibiotic therapy, especially if there are similar appearances at other sites.  

Orally administered steroids, local skin care and a search for underlying systemic disease are the mainstays of treatment. Only conservative local debridement should be undertaken since aggressive surgery can incite the disease process. Treatment with steroids, if started early, will usually rapidly improve the condition preventing much morbidity. The prophylactic use of steroids has not been reported but they may be indicated for patients with a clear history of a previous episode of pyoderma gangrenosum if elective surgery is contemplated.

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References