When infection isn't infection

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ABSTRACT

Following open reduction and internal fixation for a proximal humerus fracture a 71-year-old man re-presented with wound changes consistent with infection and elevated inflammatory markers. No significant improvement was seen with concomitant intravenous antibiotics and multiple debridement procedures. This case was further complicated by soft tissue breakdown at the site of a left thigh haematoma also requiring debridement. Surgical site infections represent the most common cause of morbidity postoperatively. This case highlights the importance of considering a number of differential diagnoses. A diagnosis of pyoderma gangrenosum prompted systemic corticosteroid therapy giving rapid clinical improvement.

KEYWORDS

Humeral fracture – ORIF – Open reduction and internal fixation – Pyoderma gangrenosum

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Background

Surgical site infections represent the most common cause of morbidity postoperatively. This case highlights the importance of considering a number of differential diagnoses. A diagnosis of pyoderma gangrenosum prompted systemic corticosteroid therapy giving rapid clinical improvement.

Case history

A 71-year-old white European man presented with a left proximal humeral fracture and left thigh haematoma following a fall. His significant past medical history included previous total hip replacements and his postoperative recovery following his right hip arthroplasty was complicated by infection, diagnosed as necrotising fasciitis and treated with multiple debridements. The left subsequent hip arthroplasty proceeded without complication. At the time of his presentation, he was on aspirin, smoked three cigarettes per day and drank approximately 30 units of alcohol per week.

Following clinical and radiological assessment, it was considered that his proximal humerus fracture should be managed surgically and he was treated with an open reduction and internal fixation using a five-hole locking plate. Intravenous teicoplanin and cefuroxime were administered on induction of anaesthesia and two further doses of intravenous cefuroxime were administered postoperatively. An extended deltopectoral approach was used. No intraoperative problems were encountered. There were no concerns during the patient's hospital admission and discharge and initial rehabilitation proceeded routinely. Follow-up was arranged for two weeks' time.

Seven days postoperatively, the patient made contact, explaining that his surgical wound had become red. Consequently, the patient was readmitted, wound swabs were taken and intravenous antibiotics commenced. Blood tests revealed a white cell count of 8.3 and a C-reactive protein of 170. The patient underwent surgical debridement (postoperative day 8) and the operative findings surmised deep infection was likely. Postoperatively, he was admitted to the critical care unit to continue intravenous antibiotic therapy. Blood cultures were negative, as were biopsy cultures on samples taken intraoperatively.

Serial debridements (postoperative days 12 and 15) were performed and on each occasion the wound edges were found to be necrotic. A vacuum pump was fitted.

During the treatment for his shoulder wound, the skin overlying the left thigh haematoma became necrotic (Fig 1). This was initially aspirated (postoperative day 15) alongside a third debridement of the left shoulder wound. No growth was seen upon culture of the aspirate.

A further five debridements of the left thigh were performed (postoperative days 20, 21, 25, 29 and 33), each combined with debridement and washout of the shoulder wound, such that the number of procedures post ORIF totalled eight.

Despite aggressive medical treatment and surgical intervention, the patient deteriorated. Inflammatory markers peaked 20 days postoperatively with a white cell count of 41.4 and C-reactive protein of 356. In addition, the patient developed renal failure. Serum creatinine increased to 315mmol/l from a baseline of approximately 95mmol/l and medical review surmised antibiotic mediated nephrotoxicity the most likely cause.

Tissue samples taken during each debridement were sent for culture but no positive microbiology was obtained. Cytology demonstrated inflammatory cells only, with no crystals identified. Furthermore, mycobacterium culture was negative at six weeks. A vasculitic process was considered but no consistent features were identified on histopathology, including fascia and muscle from the left thigh and temporal artery biopsy. Subsequent tissue biopsy, analysed by a skin pathologist, identified prominent infiltrate of neutrophils and, following clinical review by a dermatologist, a diagnosis of pyoderma gangrenosum was confirmed.

The patient was commenced on prednisolone 60mg daily, which resulted in rapid clinical improvement. He was discharged home and has since made a good recovery. The metalwork remains in place, the patient is pain free and is living independently.

Discussion

Surgical site infections are recognised to contribute significantly to mortality and morbidity. In particular, infection following orthopaedic surgery can be difficult to manage, given the extensive use of exogenous materials and the subsequent need for revision surgery.¹ Public Health England data indicate that 1% of all surgical procedures involving the reduction of long-bone fractures are complicated by surgical site infections.² The most common causative organism is *Staphylococcus aureus*.¹ Given the impact that surgical site infections have on patient outcomes, it is important that surgeons take actions to mitigate the risk of infection. Concomitant use of antibiotic therapy and surgical debridement forms the mainstay of treatment in such cases.⁵ While infection represents the most common cause of morbidity postoperatively,¹ further

differentials must remain under consideration when reviewing clinical progress.

In this case, the disproportionate way in which the skin deteriorated while other tissues appeared to improve, combined with the spontaneous deterioration of wounds away from the primary site of injury, generated clinical suspicion of a systemic process.

Pyoderma gangrenosum is a rare, painful condition in which ulcerative lesions develop on the skin. The initial stages are characterised by erythema surrounding sterile pustules. These ulcerate leaving well-demarcated central areas of necrotic tissue, purple or red in colour.⁴

Pyoderma gangrenosum displays a female preponderance and has an incidence of approximately 0.63 per 100,000 person-years. It is associated with inflammatory bowel disease, rheumatoid arthritis and, to a lesser degree, haematological disorders.⁵ A background of minor trauma is reported roughly 30% of the time, as is demonstrated in this case.⁴

Of note, the literature reports pyoderma gangrenosum being commonly misdiagnosed as infection. Characteristically, the lesions associated with pyoderma gangrenosum are sterile, so culture results are consistently negative, as was the case here.⁴ Diagnosis therefore relies on classical clinical presentation combined with biopsies excluding alternative pathology. In addition, histopathological examination typically reveals lymphocytic infiltration followed by neutrophilic infiltration and haemorrhage.^{4,5}

Treatment of pyoderma gangrenosum involves both local wound care and systemic medical therapy, most commonly high-dose corticosteroids. Alternatively, immunosuppressants such as intravenous immunoglobulin or biological agents such as infliximab are used. There is no marker specific to pyoderma gangrenosum, so regular clinical review is essential. Given the numerous disorders associated with the condition, further management should be overseen by a wider multidisciplinary team.⁴

Conclusion

It is prudent for clinicians to regularly reassess the clinical situation and consider a number of differentials throughout the care of a patient. In this case, examination, clinical experience and blood results pointed to an infectious process, but this was not the case.

References

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Figure 1 Left thigh lesion.