EMERGENCY CASEBOOK

Necrotising myositis: a surgical emergency that may have minimal changes in the skin

G H Smith, J S Huntley, G F Keenan

Emerg Med J 2007;**24**:e8 (http://www.emjonline.com/cgi/content/full/24/2/e8). doi: 10.1136/emj.2006.041723

Necrotising myositis is a surgical emergency. It is underappreciated that it may present without changes in the skin. Diagnosis is therefore often delayed. We describe a case of necrotising myositis necessitating glenohumeral disarticulation. Remarkable features were the absence of skin signs and the rapidity with which the patient became extremely septic. A review of the literature has shown the importance of early diagnosis and quick decision making to minimise mortality.

A previously fit and healthy 47-year-old man presented to the receiving unit with a painful left forearm. The history was 9 days of a flu-like illness (generalised myalgia, fever and night sweats) followed by 2 days of increasing pain in the left forearm. There was no history of trauma apart from a small insect bite on his left shin.

On examination, he had fever (37.9℃), tachycardia (115 beats/min) and hypotension (blood pressure 100/50 mm Hg). He had swelling in the forearm but no cutaneous changes, and was tender over the flexor aspect. His hand was warm and well perfused, but he could not make a fist or fully extend his fingers. There were no sensory changes.

Investigations showed a white cell count of 18 000/mm^3, C reactive protein concentration of 200 mg/l and creatine kinase concentration of 100 U/l. His renal function was normal. Doppler ultrasound scan showed no deep vein thrombosis or collection. The medical team started ceftriaxone for a possible cellulitis. Later that day, the pain and swelling increased. He also developed dysaesthesia in the median nerve territory. He was referred to the orthopaedic team for assessment. A compartment syndrome due to infection was diagnosed. An urgent repeat ultrasound confirmed no collection but abnormal stranding of the muscle architecture. Repeat testing showed an increase in the creatine kinase concentration to 1100 U/l.

Examination in the theatre was deemed mandatory. In the minutes between ultrasound and examination in the theatre, he became precipitously septic, requiring inotropic support. The theatre findings were interstitial ''dishwater-like'' fluid and discoloured non-contractile muscle in the flexor compartment of the forearm. Pockets of necrosis and pus were present. Samples were sent for urgent microscopy, which showed likely streptococcus (that was later confirmed). Extensive debridement of the flexor musculature followed. The tissues were irrigated with 10 l of saline. He started receiving benzylpenicillin, clindamycin and meropenem.

The patient remained intubated, and became progressively septic and acidotic. His requirement for inotropic support increased, and his renal function declined. Twelve hours post operation, the deterioration was such that he returned to theatre for further debridement. Although the extensor arm musculature appeared healthy, the biceps, the brachialis and the rest of the flexor compartment were non-contractile and discoloured. The decision was made to proceed with a glenohumeral disarticulation. The same day, histological analysis confirmed the margins as being clear of infection.

Over the next 24 h, he became anuric and required continuous veno-veno haemofiltration. A 96-h course of activated protein C was initiated. Antibiotics were tailored to sensitivities (benzylpenicillin and clindamycin). He returned to the theatre for two more washouts but no further debridement. An improvement was seen over the ensuing 72 h. By day 7 post disarticulation, he was extubated, off continuous veno-veno haemofiltration and the wound was closed.

DISCUSSION

Necrotising fasciitis or myositis is potentially a fatal condition with mortality from 6%–80%.[1] The infection travels along fascial planes and later involves deeper muscles with resultant myositis and myonecrosis. There are two main groups of causative organisms. Type I infections display synergistic polymicrobial qualities (usually non-group A streptococci, aerobic and anaerobic organisms), and tend to occur in patients with multiple comorbidities. Type II infection is caused by *Streptococcus pyogenes* or staphylococci. Patients with type II infections tend not to be immunocompromised.[2]

Chin-Ho *et al*[3] reported a series of 89 patients with necrotising fasciitis. Only 14.6% were diagnosed correctly on admission. They suggested that the difficulty of making the correct diagnosis is due to the paucity of early cutaneous findings. Most patients present with a triad of exquisite pain, swelling and fever. In 44% of patients, there was an intermediate stage, with the formation of small bullae filled with serous fluid. The major predictor of death was a delay of >24 h to surgery. A total of 22.5% of patients underwent an amputation to control the infection.

The ''finger test'' can be used when there is doubt or when the theatre is unavailable. This involves a 2-cm incision under local anaesthetic over the area of concern, allowing visual and digital inspection and biopsy for microscopy.[4] Positive findings include ''dishwater'' coloured fluid, absence of normal blood flow and tissue dissection with minimal resistance. Other diagnostic aids include gas on plain radiographs (17–57%) and crepitus (37%).[3] Magnetic resonance imaging and ultrasound are useful in ruling out a collection or deep vein thrombosis, but should not postpone exploration in the theatre.

The management of necrotising myositis is immediate fluid resuscitation, debridement, appropriate antibiotic treatment and supportive measures. Quick decision making is paramount in reducing death and morbidity.

In conclusion, we have described a case of necrotising myositis necessitating glenohumeral disarticulation. Remarkable features were the absence of skin signs and the rapidity with which the patient became extremely septic.

Authors' affiliations
G H Smith, J S Huntley, G F Keenan, Department of Orthopaedics, Royal Infirmary Edinburgh, UK

Competing interests: None.

Patient consent has been gained for publication.

Correspondence to: G Smith, Department of Orthopaedics, Royal Infirmary Edinburgh, UK; georgesmith@doctors.org.uk

Accepted 4 September 2006

REFERENCES

1 **McHenry CR**, Piotrowski JJ, Teprinic D, *et al*. Determinants of mortality for necrotising soft tissue infections. *Ann Surg* 1995;**221**:558–65.
2 **Elliot D**, Kufera JA, Myers RA. The microbiology of soft tissue infections. *Am J Surg* 2000;**179**:361–6.
3 **Chin-Ho W**, Haw-Chong C, Pasupathy S, *et al*. Necrotising fasciitis: clinical presentation, microbiology, and determinants of mortality. *JBJS* 2003;**85**:1454–60.
4 **Childers BJ**, Potyondy LD, Nachreiner R, *et al*. Necrotising fasciitis: a fourteen-year retrospective study of 163 consecutive patients. *Am Surg* 2002;**68**:109–16.