An expanding forearm phlegmon and subsequent compartment syndrome

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ABSTRACT

We present the case of a young, fit woman who developed a forearm phlegmon with subsequent compartment syndrome. Further investigation found her to be suffering from endocarditis and she had had recent dental work. Presentation and management is discussed, with learning points highlighted.

KEYWORDS

Compartment syndrome – Phlegmon

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Case history

A 28-year-old cleaning manager was admitted in late May 2015 under the medical team with a 3-day history of fever as well as left elbow and forearm pain and swelling. This was occurring on a background of five months of anorexia and weight loss. There was no history of trauma, animal bite or any obvious triggering events but she had been treated for cellulitis with oral antibiotics a month prior to presentation. She was otherwise fit and well with no co-morbidities (sickle cell trait only). She was a non-smoker, had no past surgical history, no family or drug history of note and no allergies.

Initial clinical examination showed a painful, swollen left elbow and forearm but with no erythema or cellulitis and soft compartments. The patient was haemodynamically stable. An x-ray showed no evidence of osteomyelitis, fracture or foreign body. She had an elevated white blood cell count of $17.8 \times 10^9/l$, C-reactive protein of 302mg/l, an erythrocyte sedimentation rate of 112mm/hour and a creatine kinase level within the normal reference range. Renal function was normal.

The initial differential diagnoses included upper limb deep vein thrombosis (DVT) and gout. Initial management included elevation, anti-inflammatories and analgesia. Subsequent venous Doppler ultrasonography found no evidence of DVT, only subcutaneous oedema at the elbow and forearm. The patient had a urate level and rheumatoid factor within the normal reference range and her autoantibody screen was negative.

Over a 48-hour period, owing to the crescendo in pain and swelling and a diagnostic dilemma, magnetic resonance imaging (MRI) of the left forearm was requested to investigate further. This showed appearances suggestive of

an inflammatory phlegmon of the proximal deep flexor musculature of the forearm, with diffuse flexor compartment myositis, and features suggestive of infective myositis, including loss of muscle definition. The median nerve appeared compressed by the proximal collection and oedema in the musculature, and the patient was believed to be at risk of compartment syndrome.

A prompt referral was made to the plastic surgery team. Repeat examination by our team showed clinical features in keeping with compartment syndrome with pain out of proportion to the physical findings, tight tender compartments, pain on passive extension and paraesthesia in the hand in the distribution of the median nerve. Movements in any direction were limited by pain. Empirical intravenous co-amoxiclav therapy was commenced. Given the evolving clinical picture and MRI findings, the diagnosis was not in significant doubt and compartment pressures were not formally measured.

The patient was taken to theatre and a fasciotomy performed as far as possible through a standard access incision. The muscles of the deep flexor compartment were friable and the dissection proceeded to the area of the phlegmon as indicated by the MRI, just distal to the insertion of the biceps tendon (Fig 1). The phlegmon was washed out and samples were taken for microbiology. Light dressings were applied. Following surgery, the forearm was elevated in a Bradford Sling® (Capatex, Nottingham, UK). Subsequent pain scores and analgesic requirements showed a downward trend, as did inflammatory markers. The patient was taken back to theatre 48 hours later for a second look, further washout and direct closure of the wound.

Tissue samples had a negative Gram stain and subsequent culture grew no organisms but blood cultures grew

Figure 1 Left forearm dissection into deep flexor compartment. Friable muscle noted in the area of the phlegmon. Fascia was released beyond the visible skin incision.

Streptococcus gordonii (viridans) sensitive to penicillin. Further clinical examination picked up a pansystolic murmur. Echocardiography was organised, which showed findings consistent with native mitral streptococcal endocarditis.

Further history from the patient found that she had received a temporary dental filling two months prior to presentation. Following consultations with cardiology and microbiology, a peripherally inserted central catheter was put in place and the patient was started on a six-week course of intravenous antibiotics. She was discharged after a three-week hospital stay, with her forearm wound healed, and made an uneventful recovery, having follow-up review with the cardiology team and her dentist.

Discussion

This case highlights the need for wide ranging and repeat assessments of a patient presenting with progressive symptoms without obvious cause. It shows the importance of maintaining an open mind diagnostically when the clinical picture is evolving rapidly or not clear. Furthermore, it emphasises the need for prompt decompression in the context of an evolving compartment syndrome.

Recent dental treatment should be elicited in the history and a complete examination of the patient with auscultation of heart sounds should be performed to look for evidence of haematogenous spread from a heart valve. The

benefits of MRI to delineate the nature and extent of the pathology, guide surgical exploration, and facilitate prompt surgical intervention in the management of compartment syndrome to rescue threatened muscles and nerves is also demonstrated by this case.

There are of course other causes of atraumatic progressive deep swelling and pain in a limb without skin changes. DVT and gout were among the initial differential diagnoses. Fatemi *et al* have described muscle infarction in a patient with diabetes mellitus; again, MRI was a useful diagnostic tool in this case.¹ Early radiography to look for osteomyelitis, evidence of gas forming organisms and surgical emphysema as well as foreign bodies and fracture is still useful given its ease of access and low cost. Necrotising myositis should also be included in the differential diagnoses as this does not have the typical skin features of necrotising fasciitis although it is more common in penetrating injuries, which go subfascial. In both diseases, however, the patient is systemically unwell, which our patient was not although we appreciate that young, fit people can compensate much better.

Kawalec and Włodarczyk reported a lumbosacral epidural abscess as a distant iatrogenic sequela of a phlegmon of the forearm following injection.² A phlegmon is an expanding inflammatory mass (which may or may not be related to infection) that can often complicate existing clinical scenarios. It can synergise with other pathological processes like ischaemia and can compound clinical situations, causing diagnostic dilemmas.^{3,4} They are best treated by following first principles: release of the relevant compartment, taking samples for microbiological surveillance and subsequent directed therapy, excision of the phlegmon and any devitalised tissue, thorough washout, elevation and antibiotics, and arranging a second look. Finally, our case highlights the importance of shared care, good communication and a multidisciplinary approach to such complex patients.

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