Acute compartment syndrome complicating deep venous thrombosis

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A 40-year-old, heavily-built man initially presented to his general practitioner two days before admission with recent onset of pain and swelling in his left calf. A duplex ultrasound scan demonstrated an occlusive deep vein thrombosis (DVT) extending from the calf through the popliteal and into the femoral vein 15cm above the knee. He was started appropriately on enoxaparin 130mg bd. Despite the treatment, the pain got worse, particularly when standing, and he was admitted for symptom control. Five weeks earlier he had undergone arthroscopic repair of the anterior cruciate ligament and partial meniscectomy of the left knee. The operation went well (tourniquet time 57 minutes). The patient was mobile and discharged after 24 hours. He had low DVT risk and therefore was not given any form of prophylaxis. Ten days after surgery at routine follow-up the wound was healing and examination of the knee was satisfactory.

On examination he was mildly distressed with a low-normal blood pressure (110/70mmHg) and a persistent low-grade fever (on the observation chart his temperature was documented between 37.5 and 37.8°C on four occasions over the first 48 hours). The left leg was slightly warm, moderately diffusely swollen and pink in colour (Figure 2). The pain was localised to the posterior compartment which was also tender and tense to palpation. It was exacerbated by dorsiflexion of the foot (Homan’s sign). There were no signs of joint effusion, thrombophlebitis, lymphadenopathy or cellulitis. Pulses were preserved and there was no neurological deficit. Initial results included a normal full blood count and routine biochemistry but a very high C-reactive protein of 233mg/L (<5) and D-Dimer of 14,988µg/L (<500). Coagulation tests were mildly deranged: international normalised ratio 1.3 (0.8–1.2), activated partial prothrombin time 37s (24–38), thrombin

Figure 1: MRI of the left lower leg; fat suppressed axial T2 and sagittal T2 weighted sequences demonstrating extensive soft tissue oedema involving the soleus muscle and deep posterior muscle compartment [blue arrows]. Note absence of flow void in the deep calf veins [white arrows] consistent with deep venous thrombus.
clotting time 16s (15–21) and fibrinogen 7g/L (1.5–4.0). Two sets of blood cultures were negative and the creatinine kinase level was 40µU/L (30–180). His enoxaparin was continued, and he remained unable to weight-bear despite regular morphine (10mg q4h). Flucloxacillin 2g was started IV q6h as a precaution, in view of the very high CRP. On day 2, magnetic resonance imaging demonstrated extensive enhancing soft tissue oedema involving the soleus muscle and muscles of the deep posterior compartment of the lower leg (Figure 2). There was some fascial fluid but no soft tissue gas or abscess. There was loss of the normal flow voids within the popliteal and deep calf veins consistent with deep venous thrombosis. Normal flow voids were seen in the popliteal artery. On day 3, orthopaedic assessment was obtained, and on the basis of the clinical findings and the MRI, the diagnosis of posterior acute compartment syndrome (ACS) was made. Urgent fasciotomies were performed to release the the pressure in the superficial and deep posterior compartments via a 20cm incision down the posterior-medial border of the tibia. Muscle pressure measurements were not undertaken because the surgeon felt that fasciotomies were mandatory, regardless of the result. At surgery, tense fascial compartments were released with significant muscle herniation after fascial incision. The muscle fibres were oedematous, but retained appropriate colour and contractility, and remained viable. One of the deep flexors (tibialis posterior) was biopsied and the wound was left unsutured. Thereafter his symptoms improved. His wound was re-opened on day 5 and all muscles appeared viable, therefore the skin was sutured. Microscopy and cultures from the deep posterior compartment aspirate were negative. Histology from tibialis posterior demonstrated endomysial oedema, small segmental necrosis and infiltration with CD68 histiocytes. There was no evidence for myositis, suppuration or vasculitis. He was started on oral anticoagulants and discharged wearing a compression stocking. He was seen after three months and the leg appeared to be normal. Ultrasound to review long-term outcome of the DVT and risk of postphlebitic changes is planned.

He was discharged on oral anticoagulants and has subsequently regained normal leg function without any signs or symptoms of post-thrombotic leg syndrome. Venous recanalisation studies have not been undertaken.

Acute compartment syndrome (ACS) of the limb is a serious condition and most commonly a complication of fracture, crush or reperfusion injury.1 Myocyte damage is the initial insult, leading to swelling of muscle tissue within non-elastic fascial compartments. As pressure increases, pain and paraesthesia (secondary to nerve compression) are the initial symptoms, followed by vascular embarrassment, muscle ischaemia, further swelling and a vicious cycle resulting in myonecrosis. Occasionally, ACS occurs after relatively mild trauma in the setting of therapeutic anticoagulation.2,3 The terms ‘spontaneous’ or ‘atraumatic’ ACS have been used to describe these cases and the mechanism usually involves rapid swelling of the muscle secondary to minor injury or bleeding. Occasionally, atraumatic ACS can also complicate a DVT. In some patients venous drainage from the compartment...
is insufficient due to the obstructing DVT. This leads to increased pressure in the compartment, decreased arterial inflow and finally, venous ischaemia.

Compartmental pressure may be further increased by an acute arterial bleed into the muscle. This may be spontaneous or secondary to needle injury and is more likely when the patient is anticoagulated.5–7

The diagnosis in our patient was difficult because there was no history of even minor trauma, the signs of posterior ACS were subtle and our blood tests did not demonstrate over-anticoagulation. Furthermore, there was no evidence of a spontaneous bleed into muscle (on MRI or at operation). DVT is often associated with a modest increase in CRP but in this patient, the very high level was secondary to complications of the ACS (early muscle necrosis and interstitial oedema), not infection.8 Finally, as is often the case in the early phase of this condition, the CK level was normal.4 This case demonstrates that occasionally a moderate DVT is sufficient to trigger an atraumatic ACS despite two days of therapeutic anticoagulation. Unfortunately, sensory changes and muscle weakness are late complications of ACS and occur when myonecrosis is already underway. The aim of urgent fasciotomy is to release compartmental pressure before the myocytes undergo irreparable damage. Therefore the main clinical feature of pain, out of proportion to that expected for a DVT was the reason for expedited surgery.

Doctors should be aware of this unusual (but treatable) complication of DVT and have a low threshold for investigating pain “disproportionate” to the appearances of the limb. Unless there are clear signs of cellulitis, they should probably consider ACS before infection in an appropriately anticoagulated patient. Untreated ACS is potentially limb threatening. When DVT is the cause of the ACS its appropriate management is important with regard to the duration of anticoagulation and follow up to exclude the development of a postphlebitic syndrome. Finally, the absolute risk of symptomatic DVT following knee arthroscopic surgery is approximately 2% and can be halved by standard prophylaxis.10

Competing interests:
Nil.

Acknowledgements:
The authors would like to thank Dr Alan Pithie for his help with the diagnosis.

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