

# Acute Compartment Syndrome Complicating Deep Venous Thrombosis

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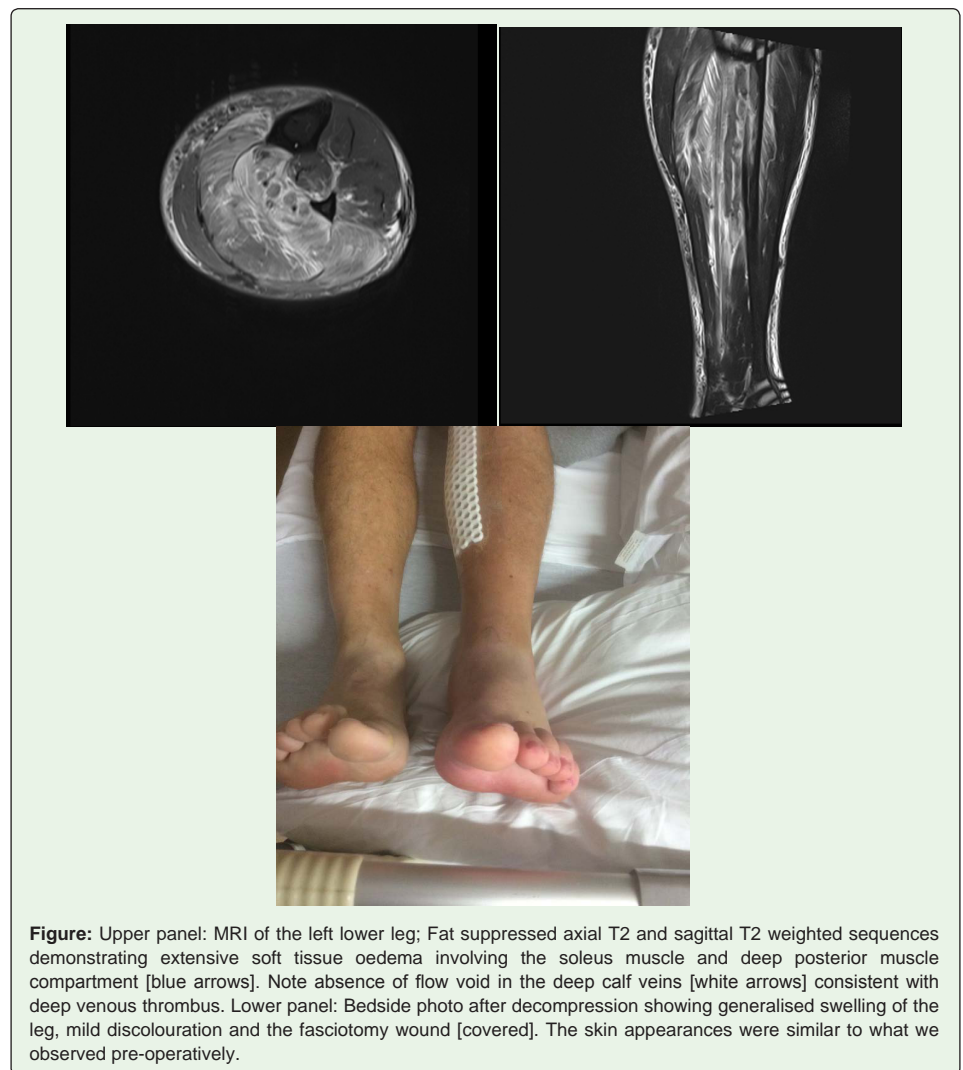
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A 40-yr old, heavily-built man initially presented to his general practitioner 2 days before admission with recent onset of pain and swelling in his left calf. A duplex ultrasound scan demonstrated a popliteal and lower leg deep vein thrombosis [DVT], extending 15 cm above the knee into the femoral vein. He was started appropriately on enoxaparin 130 mg bd. Despite the treatment, the pain got worse, particularly when standing, and he was admitted for symptom control. Five weeks earlier he had undergone anterior cruciate ligament repair of the left knee, [without heparin prophylaxis] and made a satisfactory recovery (Figure 1).

On examination he was mildly distressed with a low-normal blood pressure [110/70 mmHg] and a persistent low-grade fever [temperature ranged from 37.0-37.8 °C]. The left leg was moderately diffusely swollen, slightly warm and darker in colour [figure]. The pain was localised to the posterior compartments and was exacerbated by dorsi-flexing the ankle and squeezing the gastrocnemius. 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



**Figure:** Upper panel: 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. Lower panel: Bedside photo after decompression showing generalised swelling of the leg, mild discoloration and the fasciotomy wound [covered]. The skin appearances were similar to what we observed pre-operatively.

and routine biochemistry [serum creatinine 111  $\mu\text{mol/L}$ ] but a very high C-reactive protein [233 mg/L] and D-Dimer [14,988  $\mu\text{g/L}$ ]. Coagulation tests were mildly deranged: international normalised ratio 1.3 [0.8-1.2], activated partial prothrombin time 37s [24-38], thrombin clotting time 16s [15-21] and fibrinogen 7 g/L [1.5-4.0]. Two sets of blood cultures were negative and the creatinine kinase level was 40  $\mu\text{U/L}$  [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]. 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 acute compartment syndrome [ACS] was made. Urgent fasciotomies were performed. 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. The muscles were drained via a 20cm incision down the posterior-medial border of the tibia. 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 endomyosial oedema, small segmental necrosis and infiltration with CD68 histiocytes. There was no evidence for myositis, suppuration or vasculitis. He was discharged on oral anticoagulants and has subsequently regained normal leg function.

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. ACS

can also complicate a DVT. In this situation, the venous obstruction becomes sufficient to impair muscle perfusion despite a normal blood pressure and intact peripheral pulses [4]. Consequently the bigger the DVT, the more likely a traumatic ACS is to occur, particularly when the patient is over-anticoagulated [5]. In these patients, the mechanism for atraumatic ACS is usually an arterial bleed into muscle, which may be either spontaneous or secondary to needle injury [6,7]. The diagnosis of ACS in our patient was difficult because there was no history of even minor trauma, the DVT was only moderate, and our blood tests did not demonstrate over-anticoagulation. There was no evidence of a spontaneous bleed into muscle on MRI or at operation. We therefore suggest that the moderate DVT was sufficient in itself to predispose him to atraumatic ACS after only 2 days of therapeutic anticoagulation. 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 [8].

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### Contributions

Dhayalan and Jardine investigated the patient, Dr Goh reported the imaging and Mr Lash undertook the surgery. All authors were involved in the care of the patient and contributed to the manuscript.

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