Clinical guidelines

The assessment of acute calf pain

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The assessment of the acutely painful calf can pose diagnostic problems in the Accident and Emergency department. The primary diagnostic concern is to establish or exclude the diagnosis of deep vein thrombosis, which can lead to mortality as well as morbidity if recognised.

Clinical signs suggestive of deep vein thrombosis are known to be nonspecific and unreliable in isolation. Hence, in dealing with acute calf pain the clinician needs to be aware of several diagnostic and therapeutic pitfalls. Anticoagulation in the absence of radiological proof of deep vein thrombosis is contraindicated. Anticoagulation is not without risks of its own, and the inappropriate use of anticoagulants can aggravate the symptoms and complications of many of the nonthrombotic causes of acute calf pain.

This review discusses the causes of acute calf pain. The basis of clinical assessment is to select patients who require further investigation from those who do not. As venous thrombosis can be symptomatic of other diseases or of thrombotic disorders, once the diagnosis is likely or proven, clinical assessment must be targeted accordingly.

Clinical assessment

Assessment of acute calf pain starts with accurate history taking. A history of acute onset calf pain while walking, running or engaging in sporting activity should suggest a calf muscle tear involving the gastrocnemius–soleus complex. It is doubtful whether the so-called plantaris tear exists as a discrete entity. Typically, there is pain, tenderness and swelling localised to the medial head of the gastrocnemius muscle. Symptoms are aggravated by passive stretching (dorsiflexion of the ankle) or active restricted contraction of the muscle. With delayed presentation, surface bruising is often present.

Rupture of a popliteal cyst is another common cause of acute calf pain. This entity was first described by Morrant Baker who noted the association with knee joint disease. The cyst is essentially a distended gastrocnemius–semimembranosus bursa, which communicates with the knee joint in about 60% of cases via a possible one-way valve mechanism. A history of pre-existing arthropathy of the knee joint, which may be osteoarthriosis, rheumatoid arthritis, pyogenic arthritis, neuropathic arthropathy, or even colitic arthropathy is often present. A knee effusion may be demonstrable. The term pseudothrombophlebitis syndrome has been given to acute rupture of a popliteal cyst leading to acute calf pain. Occasionally, popliteal cyst rupture may occur in the absence of obvious arthropathy of the knee. To add to diagnostic problems, popliteal cyst rupture may coexist with deep vein thrombosis. Popliteal cysts can indeed produce extrinsic vein compression predisposing to thrombosis. Venography or combined arthropathy and venography may be required if the two conditions are thought to coexist.

Blind anticoagulation in the presence of ruptured popliteal cysts produces haemarthrosis, which may progress to acute posterior calf compartment syndrome. Usually, prompt reversal of anticoagulation is required.

Bruising of the calf from direct blunt trauma or post-operative trauma, producing intramuscular haemorrhage, can mimic deep vein thrombosis. This may occasionally lead to gravitation of extravasated blood producing the sign of crescentic bruising around the malleoli. This sign might predict an increased likelihood of local complications if anticoagulation is used.

Calf cellulitis is commonly quoted as a cause of acute calf pain. Deeper infections, such as necrotising fasciitis, can cause local signs of acute inflammation, fever, blistering and bullae after one to two days. Ultrasonography may show fluid deep to the soleus. Localised myositis has also been described as a source of pain in this situation.

Acquired immune deficiency syndrome has been associated with a syndrome of calf pain, swelling and tenderness, along with cutaneous hyperaesthesia to light touch. In a series of five patients, all had venography which excluded deep
vein thrombosis. In these cases, the pain is believed to be due to an intense local inflammatory response, termed hyperalgesic thrombophlebitis.22

Clinical signs, as described for deep vein thrombosis, are unreliable. The signs usually described include calf muscle or direct venous tenderness, local pitting, oedema, muscle induration, distended superficial veins and skin discolouration. It is recognised that the more proximal and occlusive the thrombus the more marked are the symptoms and physical signs. Homans described a sign of calf muscle pain on ankle dorsiflexion, which is now believed to be unreliable and possibly dangerous.23

Pre-existing venous abnormalities, such as varicose veins and congenital malformations can both predispose to deep vein thrombosis and occasionally produce calf pain in the absence of thrombosis. Venous haemangioma may present with calf pain, mimicking deep vein thrombosis and can be diagnosed on venography. Contrast-enhanced computed tomography is confirmatory.24

While previous deep vein thrombosis is a risk factor for subsequent thromboembolic phenomena, a ‘thromboneurosis’ may occur. The characteristics include atypical clinical features, pain and tenderness out of proportion to the physical findings, a history of multiple hospital admissions for treatment of recurrent venous thrombosis and long-term anticoagulant therapy. These patients have no evidence of coagulopathy predisposing to thrombosis, and no evidence of deep vein thrombosis on venograms.25

A thrombotic tendency associated with neoplasm has been recognised in the association of migratory superficial thrombophlebitis with cancer. A follow-up of patients with deep vein thrombosis may reveal an increased likelihood of subsequent malignancy.26 Plasmin, compared to controls. Lower extremity and pelvic neoplasia are particularly associated with deep vein thrombosis in the leg. For practical purposes, however, most of these neoplasia should be diagnosable on clinical examination at initial presentation with thrombosis. The use of duplex ultrasound scanning reveals not only the thrombosis but also the coexisting neoplastic or aneurysmal lesion. It is a cost-effective technique suitable for the assessment of the ambulatory patient.27

Popliteal space lesions other than cysts, such as exostoses and aneurysms, may predispose to deep vein thrombosis secondary to local venous compressions. Particularly in the young patient one should actively look for palpable lesions in the popliteal area.28

Certain subgroups of patients with deep vein thrombosis may require further investigation for thrombotic disorders, especially if they are under 45 years old, have a history of previous thrombotic episodes or have a family history of thromboembolic disorders.29

I wish to thank Ms K Melia for typing the manuscript.

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**Figure** Algorithm for calf pain assessment in Accident and Emergency; DVT=deep vein thrombosis

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**Learning points**

- Calf pain is the usual mode of presentation of deep vein thrombosis to an Accident and Emergency department
- The history can help determine the need for further investigations
- The clinical findings are nonspecific
- Radiological confirmation should be obtained before commencing treatment for deep vein thrombosis

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**Common causes of acute calf pain**

- Calf muscle tear
- Ruptured popliteal cyst
- Deep vein thrombosis
- Cellulitis
- Intramuscular haematoma
- Superficial thrombophlebitis

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**History and physical examination**

- **Not DVT** (eg immediate preceding trauma)
  - No risk factors
    - No action
  - Risk factors
    - Consider contrast venography
- **Possible DVT**
  - Duplex Doppler ultrasound scan
  - No risk factors
    - Treat
  - Risk factors
    - Treat
    - Consider further investigations

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Medical Anniversary
JOSEPH THOMAS CLOVER, 28 FEBRUARY 1825

Joseph Thomas Clover (1825–1882) was born in Aylesham, Norfolk, qualified at University College Hospital and became anaesthetist at University College, Westminster, and the London Dental Hospitals. He was a pioneer anaesthetist inventing apparatus for the use of nitrous oxide, chloroform and for ether (which was first used in 1846). His patients included Princess Alexandra, Robert Peel and Florence Nightingale. He died on 27 September 1882 and is buried in the Brompton Cemetery, West London. — DG James