The ischaemic leg

Introduction

Peripheral vascular disease often affects the lower limb and the resulting ischaemia can be a considerable source of morbidity and mortality. Presentation may either be acute, chronic or acute on chronic, and accurate history and examination is key to successful management of the patient.

Acute lower limb ischaemia

Acute lower limb ischaemia is associated with considerable mortality and morbidity and warrants immediate referral to the vascular team. Left undiagnosed and untreated it will, within 6 hours, progress to irreversible tissue damage requiring amputation. Early diagnosis and restoration of arterial blood flow is critical for limb salvage.

Aetiology

Acute limb ischaemia can occur either acutely or as a result of deterioration of a more chronic process – this can usually be determined from the history. The aetiology in most cases is thrombosis in a vessel with pre-existing atherosclerosis (60%) and the superficial femoral and popliteal arteries are most commonly affected. Embolus accounts for about 30% of all cases and the majority (80%) of emboli originate from the left side of the heart in patients with ischaemic heart disease and/or atrial fibrillation (*Table 1*). An embolus typically will lodge at the bifurcation of a vessel and the femoral, popliteal and aortic (a so-called saddle embolus) bifurcations are frequently involved. Other causes of acute limb ischaemia include trauma, iatrogenic damage, acute aortic dissection and aneurysmal disease.

Presentation

The management of thrombotic and embolic disease differs, so it is important to distinguish between them. The extent and reversibility of the condition must also be assessed. Patients classically present with the 6Ps: Pain, Pallor, Perishing coldness, Pulselessness, Paraesthesia and Paralysis. However, the severity of disease does not correlate with the severity of symptoms and signs.

Miss Kaji Sritharan *is Clinical Research Fellow, and* **Mr Alun H Davies** *is Reader in Surgery and Consultant Vascular Surgeon, Department of Vascular Surgery, Charing Cross Hospital, London W6 8RF*

Correspondence to: Mr AH Davies

History

Pain usually precedes paraesthesia and is often sudden in onset, continuous and severe in nature as a result of vascular spasm. Some relief may be gained by hanging the leg from the bed, indicating dependency of the blood flow within the limb.

In acute or chronic disease there is often a history of intermittent claudication. The patient is likely to have one or more risk factors for peripheral vascular disease, e.g. diabetes, hypertension, hypercholesterolaemia, smoking, cerebro- and renovascular disease and a positive family history. In contrast, in purely embolic disease (*Figures 1* and *2*) a history and pre-existing vascular disease is absent and there may be evidence of an embolic source, e.g. a history of chest pain or a murmur on examination.

Table 1. Aetiology of acute lower limb ischaemia

Thrombosis (60%)	Occlusive atherosclerotic disease
	Graft stenosis usually caused by intimal hyperplasia
	Aneurysmal atherosclerotic disease
	Coagulopathy, thrombophilia
Embolus (30%)	Atrial fibrillation
	Mural cardiac thrombus, e.g. post MI
	Congestive cardiac myopathy
	Infective endocarditis
	Rheumatic heart disease
	Prosthetic heart valves
	Thrombus from peripheral aneurysm, e.g. AAA, popliteal aneurysm
	Atheroma, cholesterol emboli
	Tumour, e.g. atrial myxoma
Trauma	Fracture
	Joint dislocation
Iatrogenic	Arterial cannulation
	Intra-arterial drug administration
Aortic dissection	

AAA = abdominal aortic aneurysm; MI = myocardial infarction

Examination

In the early stages of acute ischaemia, arterial spasm gives rise to a marble white leg and venous guttering. Over subsequent hours vasodilation occurs and the presence of stagnant deoxygenated blood turns the limb a mottled purple colour.

Coldness of the limb, pallor, a sluggish capillary refill time (> 2 seconds) and absent distal pulses are commonly seen signs. Sensory and motor dysfunction, calf tenderness and a bulging anterior compartment (which indicates muscle necrosis) and fixed (i.e. non-blanching) mottling are late signs which suggest that the limb is no longer viable. Examination of the contralateral limb can further aid diagnosis and a weak or absent pulse points towards thrombosis as the most likely cause.

Investigations

Duplex scanning is an accurate, non-invasive and useful first-line test for assessing the site and extent of occlusive and aneurysmal disease. Angiography, however, still remains the gold standard investigation but is not always required. If an abdominal aortic aneurysm or aortic dissection is suspected a computed tomography scan with contrast should be performed.

Additional investigations are aimed both at establishing the cause and identifying the complications associated with limb ischaemia (*Table 2*).

Figure 1. Acute onset digital gangrene.

Figure 2. Digital gangrene: the distribution suggests the aetiology is embolic.

Management

Acute limb ischaemia has a mortality rate of about 20%. Resuscitation (airways, breathing, circulation) of the patient is paramount with subsequent management being supportive (rehydration, catheterization, analgesia) as well as geared towards limb salvage and treatment of the underlying cause. The former will be influenced by both the viability of the limb and aetiology.

If the limb is clinically viable and an arterial embolus is suspected, balloon catheter embolectomy is the treatment of choice and a completion angiogram may be performed as necessary after the procedure.

In patients where the limb is viable and thrombosis is suspected, angiography, intra-arterial thrombolysis with streptokinase or tissue plasminogen activator (which typically takes between 4 and 24 hours to effectively dissolve a thrombus), angioplasty, balloon catheter thrombectomy, reconstructive surgery and fasciotomies (only should compartment syndrome develop) all form part of the repertoire for management. Intravenous heparin is usually instigated in this group, unless contraindicated (aiming for an activated partial thromboplastin time ratio of between 2.0 and 2.5), to prevent further propagation of the thrombus. Following revascularization, the patient should be closely monitored for reperfusion injury and compartment syndrome.

Where ischaemia is irreversible and the limb non-salvageable, amputation may be required urgently to stem the systemic sequelae of tissue necrosis, including sepsis, metabolic acidosis, rhabdomyolysis, hyperkalaemia and acute renal failure. *Table 3* gives a preoperative management checklist.

Table 2. Investigations

Blood tests	Full blood count
	Urea and electroytes (looking for acute renal failure)
	Creatinine kinase (a marker of rhabdomyolysis; exclude myocardial infarction)
	Clotting and thrombophilia screen (Protein C, S factor V Leiden and antithrombin III deficiencies and lupus anticoagulant) needs to be performed before starting anticoagulation and is especially useful in young patients with no risk factors for vascular disease
Arterial blood gas	Metabolic acidosis
	Lactate (marker of ischaemia)
Urinalysis	Myoglobinuria (a result of rhabdomyolysis)
Electrocardiogram	Ischaemia, myocardial infarction, atrial fibrillation
Echocardiogram	To identify a cardiac source of thromboembolism
24-hour Holter monitor	To identify arrhythmia

Chronic lower limb ischaemia

The term chronic limb ischaemia embraces a spectrum of disease ranging from intermittent claudication (a cramp-like pain bought on by exercise, which is relieved with rest) to rest pain (pain in the forefoot which is typically worse at night, often waking the patient from their sleep and relieved by hanging the foot over the edge of the bed), ulceration and gangrene (irreversible tissue necrosis, which when superinfected is termed wet gangrene) (*Figure 3*).

Aetiology

Occlusive atherosclerotic disease is implicated in the majority of disease (*Table 4*) and the extent and location of the lesion often correlates with symptoms. Patients with aorto-iliac disease classically present with buttock claudication and impotence, those with iliofemoral disease with thigh claudication and those with femoropopliteal involvement, calf claudication.

Intermittent claudication is prevalent in 5% of men and between 2 and 4% of women over 40 years of age. Approximately 20% of patients with symptoms of intermittent claudication will progress to develop critical limb ischaemia where the viability of the limb then becomes threatened.

Critical limb ischaemia

The European Working Group defines critical limb ischaemia as the presence of ischaemic rest pain requiring analgesia for more than 2 weeks, or ulceration, or gangrene of the lower extremity with an absolute ankle systolic blood pressure of <50 mmHg and/or toe systolic pressure of <30 mmHg.

History and examination

On examination the patient usually shows stigmata of peripheral vascular disease (*Table 5*) and palpation and auscultation of the upper and lower limb pulses, as well as examination of the abdomen for an abdominal aortic aneurysm, is mandatory. Pulses should be recorded as: 0 – absent; 1 – reduced volume; 2 – normal volume; 3 – ectatic; 4 – aneurysmal or B – bruit.

Buerger's test with the assessment of Buerger's angle should also be performed. The latter is the angle to which the straight-

Table 3. Preoperative checklist

Ensure adequate resuscitation: oxygen, airways, breathing, circulation
Intravenous access and intravenous fluids
Analgesia – opiate
Catheterize
Keep nil by mouth
Mark side
Consent
Crossmatch blood
Anticoagulation therapy (if appropriate)
Antibiotic prophylaxis
Ensure radiological films are at hand in theatre
Notify radiographer, organize image intensifier in theatre if required
Book high dependency unit/intensive therapy unit bed if necessary

Figure 3. Wet gangrene of the foot with associated cellulitis.

ened leg must be raised before it turns white. In a normal person the leg can be raised to 90° and will still remain pink. A vascular angle of <20° suggests severe ischaemia. Following elevation of the leg, to complete the test, the foot is hung to the ground. In a healthy limb the foot will remain pink, however, if ischaemic the foot will turn purple-red as a result of reactive hyperaemia (blood filling dilated capillaries).

Ankle brachial pressure indices (ABPIs) using a hand-held Doppler probe and sphygmomanometer can usefully be established at the bedside. An APBI >1.0 is considered normal. However, readings can be falsely elevated as a result of incompressible calcified vessels, as seen in patients with diabetes. An ABPI of between 0.8 and 0.6 is associated with claudication and an ABPI <0.5 indicates multi-level occlusive disease and is seen in patients with critical limb ischaemia. If the ABPIs at rest are unequivocal, they should be repeated following a fixed period of exercising on a treadmill. A fall in ABPI with a slow recovery following rest indicates a positive exercise test and significant occlusive atherosclerotic disease.

In addition to ABPIs, the audible Doppler waveform yields useful information. A triphasic waveform is normal. A biphasic waveform suggests loss of arterial wall compliance and a monophasic waveform poor vessel compliance – as seen in atherosclerotic disease.

Table 4. Causes of chronic limb ischaemia

Causes	Occlusive atherosclerotic disease	
	Vasculitides, e.g. Buerger's disease, giant cell arteritis, Takayasu's (pulseless) disease, systemic lupus erythematosus	
	Popliteal entrapment syndrome	
	Cystic adventitial disease	
	Haematological disorders	
	Collagen disorders: Ehlers–Danlos syndrome and Marfan's syndrome predispose to popliteal aneurysms	
Differential diagnosis	Neurological	Spinal canal stenosis (neurogenic claudication)
	Musculoskeletal	Osteoarthritis
	Vascular	Venous claudication in a post-phlebitic limb

Investigations

Duplex is widely used in the first instance, although the gold standard still remains an angiogram. However, invasive tests should only be used in patients where vascular intervention is being actively considered.

Management

Chronic limb ischaemia

Management is geared towards optimizing risk factors: cessation of smoking; control of diabetes, hypertension and cholesterol levels; weight loss; and initiation of antiplatelet therapy (aspirin 75 mg daily). Supervised exercise programmes are also of benefit in claudicants and are thought to encourage the development of collaterals. At 5 years a third of claudication patients with conservative treatment will have improved symptoms, a third will remain stable and the remainder will progress and require surgical intervention.

Although there are no hard and fast rules, intermittent claudication of <80 m should be managed conservatively and shorter distance, more debilitating claudication, with angioplasty or surgery. However, the mortality in this group at 3 years is 25% – largely as a result of myocardial infarction or stroke, and fitness for surgery will greatly influence management.

Critical limb ischaemia

Critical limb ischaemia requires revascularization to salvage the limb. As well as optimizing risk factors, angioplasty, surgical endarterectomy and bypass procedures or a combination of these should be considered. Availability of a suitable conduit, adequate inflow, run-off and fitness of the patient will all influence management and where revascularization is not possible amputation, lumbar sympathectomy and prostanoids may have a role to play. BJHM

Table 5. Stigmata of peripheral vascular disease

Nicotine staining	
Xanthoma	
Xanthalassma	
Lower limb	Cold peripheries
	Marbled skin
	Trophic changes
	Venous guttering as a result of poor blood supply
	Arterial ulcers – overlying lateral malleolus
	Gangrene (wet/dry)
	Prolonged capillary refill time
	Buerger's test positive
	Weak/absent distal pulses

Conflict of interest: none.

Further reading

Baker D, Mahadevan V, Tsui J (2001) Lower limb ischaemia. In: Hamilton G, ed. *Vascular Surgery.* Royal College of Surgeons of England MRCS STEP Course. Royal College of Surgeons of England, London: 85–149

Beard JD, Gaines P (1999) Acute leg ischaemia. In: Davies AH, Beard JD, Wyatt MG, eds. *Essential Vascular Surgery.* WB Saunders, London: 198–214

Browse NL (2001) The arteries, veins and lymphatics. In: Browse NL, ed. *An Introduction to the Symptoms and Signs of Surgical Disease.* Arnold, London: 162–95

Cheshire NJW, Wolfe JHN (2000) Critical leg ischaemia: amputation or reconstruction? In: Wolfe JHN, ed. *ABC of Vascular Surgery.* BMJ Publishing Group, London: 19–21

Fligelstone L, Davies AH (2001) Vascular surgery. In: Fligelstone L, Davies AH, eds. *Essential Postgraduate Surgery.* Churchill Livingstone, London: 91–109

Hallett JW, Brewster DC, Darling RC (1995) Lower extremity claudication and threatened limb loss. In: Hallett JW, Brewster DC, Darling RC, eds. *Handbook of Patient Care in Vascular Surgery.* Little, Brown, Boston: 157–98

MacPherson GAD, Wolfe JHN (2000) Acute ischaemia of the leg. In: Wolfe JHN, ed. *ABC of Vascular Surgery.* BMJ Publishing Group, London: 15–18

Shaw JE, Beard JD (1999) Chronic leg ischaemia. In: Davies AH, Beard JD, Wyatt MG, eds. *Essential Vascular Surgery.* WB Saunders, London: 215–42

KEY POINTS

- Acute limb ischaemia is a vascular emergency associated with significant morbidity and mortality.
- To maximize the chances of salvaging the limb blood flow must be restored within 6 hours of the insult, so early diagnosis and referral to the vascular team is vital.
- Surgical management is determined largely by the viability of the limb at presentation and the aetiology of the ischaemia.
- Chronic limb ischaemia presents clinically as intermittent claudication, rest pain, ulcers and gangrene.
- Claudication is common, but can progress to limb-threatening ischaemia – critical limb ischaemia.
- Management of chronic limb ischaemia includes risk factor modification, structured exercise programmes and interventional or surgical procedures.