Peripheral arterial disease

A practical guide for GPs

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The incidence of peripheral arterial disease (PAD) is increasing globally, largely attributed to the growing burden of atherosclerotic risk factors, as well as ageing populations and smoking rates. Patients with PAD may be asymptomatic or have symptoms ranging from atypical leg discomfort to intermittent claudication and chronic limb-threatening ischaemia. With symptomatic and asymptomatic forms of the disease potentially leading to limb and life-threatening complications, a robust understanding and practical framework for addressing PAD are indispensable.

Lower limb peripheral arterial disease (PAD) can be defined as obstructive atherosclerotic disease affecting the arteries from the distal aorta to the foot, leading to clinical symptoms, signs or abnormalities detected through noninvasive or invasive vascular testing or medical imaging. This stenosis or obstruction results in disturbed or impaired circulation to one or both lower extremities.[1]

Globally, an estimated 237 million people were affected by PAD in 2015, and this number is expected to increase by 30 to 50% by 2045.[2] The rise in PAD cases can be attributed to the growing burden of atherosclerotic risk factors such as diabetes, obesity, metabolic syndrome and hypertension as well as ageing populations and smoking rates.

PAD encompasses a wide range of symptomatology and disease severity. Surprisingly, up to 50% of patients with PAD do not experience any clinical symptoms.[3] However, when symptoms are present, they can vary from atypical leg discomfort to intermittent claudication (IC) and chronic limb-threatening ischaemia (CLTI).[3] Importantly, PAD is strongly associated with mortality, irrespective of symptomatology, as it serves as a significant predictor of future myocardial infarction and stroke.[4]

GPs have a vital role in the early detection, risk factor modification and initial management of PAD. This article, based on recent guidelines, provides an accessible and systematic approach to managing this common yet complex disease in primary practice.

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Clinical features and symptomatology

The symptomatic burden of PAD can range from IC – a reproducible exertion-induced leg discomfort relieved on rest – to more advanced stages where patients experience persistent ischaemic rest pain, nonhealing ulcers or gangrene, known as CLTI. The traditional term critical limb ischaemia (CLI) has been replaced by CLTI to better encapsulate the broader spectrum of limb-threatening ischaemia seen in contemporary clinical settings.[5]

Intermittent claudication

IC is a hallmark of PAD, manifesting as predictable and reproducible pain in the legs

Key points

- **Peripheral arterial disease (PAD) encompasses a wide range of symptomatology and disease severity. Although up to 50% of patients with PAD are asymptomatic, others can experience symptoms ranging from atypical leg discomfort to intermittent claudication (IC) and chronic limb-threatening ischemia (CLTI).**
- **Although IC is generally a relatively stable vascular condition, it is a powerful indicator of systemic atherosclerotic disease.**
- **CLTI is a serious manifestation of advanced peripheral circulatory compromise, warranting prompt assessment and consideration of intervention.**
- **The Wound, Ischemia and foot Infection (WIfI) classification system, which grades the three key risk factors leading to amputation, is invaluable, especially in the context of diabetes-associated PAD, where it has a pivotal role in guiding therapeutic decisions.**
- **Objective confirmation of lower extremity PAD is made via the Doppler ankle-brachial index. However, in patients with medial arterial calcification, especially prevalent among individuals with diabetes or chronic kidney disease, the absolute toe pressure or toe-brachial index should be measured.**
- **The primary objective of IC therapy is to improve cardiovascular health and enhance the patient's overall quality of life rather than prevent amputation.**
- **Effective revascularisation procedures are crucial for limb salvage in CLTI; however, managing this complex condition requires consideration of multiple factors.**

that is elicited by exercise and rapidly alleviated by rest. Although claudication often affects the calf muscles, the varying levels of arterial involvement mean that patients may also experience discomfort in the buttocks, hips, thighs or feet. Patients with PAD may sometimes present with less typical symptoms, such as a sense of fatigue during exercise that surprisingly does not compel the sufferer to stop – a sign that can delay recognition and treatment. The underlying pathophysiology of IC stems from a metabolic mismatch between the oxygen demanded by working muscles and the supply constrained by atherosclerotic stenosis.[1,6]

Evaluating the impact of IC is focused on identifying the 'claudication distance' or the maximal walking distance before the onset of pain and understanding its repercussions on daily activities and overall quality of life. The intensity of IC can range widely, from mild inconvenience only noticeable during extensive ambulation or walking uphill to severe interference that significantly affects employment and the performance of everyday tasks.

Despite the discomfort it causes, IC is generally a relatively stable vascular condition and the potential for progression to CLTI remains low, with less than a 1% annual risk of limb loss.[5,6] However, it is critical to recognise PAD and, by extension, IC as powerful indicators of systemic atherosclerotic disease. The cardiovascular implications are

Grade	Wound	Ischaemia	Foot infection
0	No ulcer and no gangrene	ABI ≥0.80 or TP≥60 mmHg	No signs or symptoms of infection
1	Small ulcer and no gangrene	ABI 0.7-0.79 or TP 40-59 mmHg	Mild infection: ≤2 cm of cellulitis
2	Deep ulcer or gangrene limited to toes	ABI 0.4-0.59 or TP 30-39 mmHg	Moderate infection: >2 cm of cellulitis or involving deep structures (e.g. abscess, osteomyelitis, septic arthritis)
3	Extensive ulcer or extensive gangrene	ABI ≤0.39 or TP <30 mmHg	Severe infection: presence of systemic response/SIRS

Estimated amputation risk at one year for each combination (green = very low risk; yellow = low risk; orange = medium risk; red = high risk).

	Ischaemia 0				Ischaemia 1				Ischaemia 2				Ischaemia 3			
Wound 0																
Wound 1																
Wound 2																
Wound 3																
foot Infection	fI 0	fI 1	fI 2	fI 3	fI 0	fI 1	fI 2	fI 3	fI 0	fI 1	fI 2	fI 3	fI 0	fI 1	fI 2	fI 3

Abbreviations: ABI = ankle-brachial index; fI = foot infection; SIRS = systemic inflammatory response syndrome; TP = toe pressure.
Adapted from: Mills JL Sr, Conte MS, Armstrong DG, et al. The Society for Vascular Surgery Lower Extremity Threatened Limb Classification System: risk stratification based on wound, ischemia, and foot infection (WIfI). J Vasc Surg 2014; 59: 220-234.e1-2.[8]

Figure 1. Summary of the Wound, Ischaemia and foot Infection (WIfI) classification system.

profound: within a five-year timeline, nearly 20% of patients with IC may suffer a major cardiovascular event, and mortality rates fluctuate between 10% and 15%.[7] These statistics underline the need for a comprehensive approach to PAD – addressing both limb and life – in routine primary care.

Chronic limb-threatening ischaemia

Patients with CLTI comprise a heterogeneous group with varying degrees of ischaemia that may delay wound healing and increase amputation risk.[7] The diagnosis of CLTI is based on evidence of PAD in association with ischaemic rest pain or the presence of tissue loss, – such as ulceration or gangrene.[7] Although only a subset of patients with PAD – estimated to be between 1% and 10% – experience CLTI, it is a serious manifestation of advanced peripheral circulatory compromise, warranting prompt assessment and consideration of intervention. It is often accompanied by several key conditions, in particular diabetes and chronic kidney disease.[5]

Classically, ischaemic rest pain is localised to the forefoot and is exacerbated in positions such as lying flat, whereas limb dependency typically affords some relief. Such rest pain should be present for more than two weeks, and be associated with noninvasive confirmation of severe ischaemia, including an ankle-brachial index (ABI) of less than 0.4 or toe pressures below 30 mmHg (Wound, Ischemia and foot Infection [WIfI] criteria ischaemia grade 3; see Figure 1).[8] Tissue loss in CLTI (gangrene or prolonged ulceration) is associated with significant anatomical and haemodynamic evidence of PAD, as suggested by a WIfI ischaemia grade of at least 1 (ABI ≤0.79 or toe pressure ≤59 mm/Hg).[5]

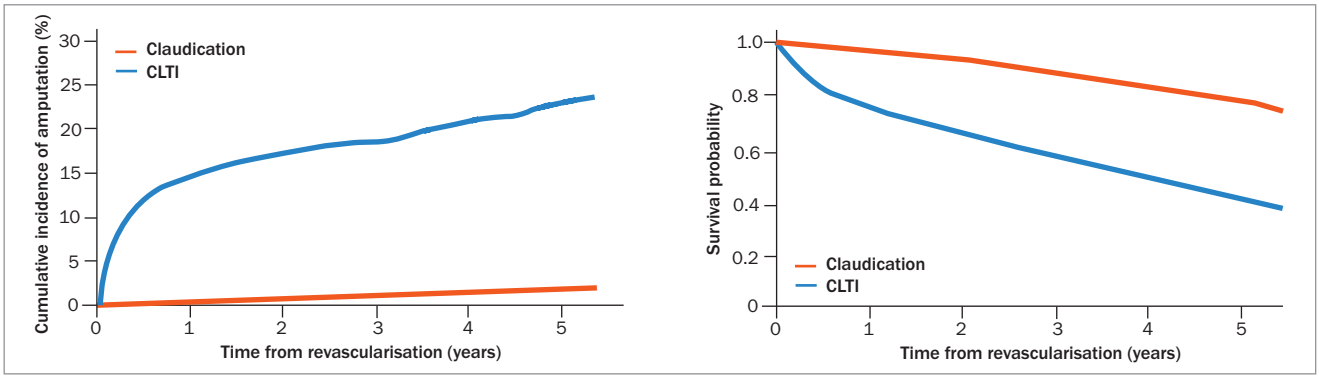
The implications of CLTI go beyond the limb threat. It is also associated with very high cardiovascular morbidity and mortality. It is estimated that one in five patients with CLTI die within a year of diagnosis. Without appropriate therapeutic intervention, the risk of limb loss can be as high as 25% within the first year of diagnosis, demonstrating the dire picture of the CLTI trajectory if left unmanaged.[9] These data highlight the necessity for an aggressive, multidisciplinary treatment approach aimed at mitigating the risks associated with CLTI.

Figure 2 illustrates the difference in outcomes for patients with IC and CLTI.[10]

Classification systems

The Fontaine and Rutherford classification systems are widely used to describe the severity of PAD. The Fontaine classification is based solely on clinical symptoms, whereas the Rutherford classification considers clinical findings, Doppler assessment and measurements of the ABI (Table).[11]

Complementary to the Fontaine and Rutherford systems, the WIfI classification system represents a more recent advance, grading the three key factors that precipitate amputation risk: wound severity, presence and degree of ischaemia and presence and severity of foot infection. Each component is evaluated on a graded scale from 0 to 3, with 0 depicting the absence of the condition and 3 signifying severe involvement (Figure 1). The WIfI classification system is invaluable, especially in the context of diabetes-associated PAD, where it has a pivotal role in guiding therapeutic decisions, reflecting the benefit of revascularisation and risk of limb loss.[8]



Figures 2a and b. Visual comparison of the cumulative incidence of amputation (a, left) and survival probability (b, right) for patients with revascularised claudication and chronic limb-threatening ischaemia (CLTI).

Adapted from: Fridh EB, Andersson M, Thuresson M, et al. Amputation rates, mortality, and pre-operative comorbidities in patients revascularised for intermittent claudication or critical limb ischaemia: a population based study. Eur J Vasc Endovasc Surgery 2017; 54: 480-486.[10]

Diagnostic assessment

The diagnosis of PAD commences with the clinical history and physical examination (see the Flowchart). The clinician must distinguish IC – a symptom strongly indicative of PAD – from lower limb discomfort due to nonvascular aetiologies such as nerve impingement syndromes or various forms of arthritis.

Classic symptoms of PAD-induced leg pain are precipitated by physical exertion and present in specific locations such as the calves, thighs or buttocks. Patients often experience a compelling need to decelerate their pace or cease walking altogether, with symptoms dissipating typically within 10 minutes of resting. Calf claudication often arises from pathology in the common and superficial femoral arteries and popliteal artery, whereas claudication of the hips, thighs and buttocks is often linked to occlusive disease of the aorta and iliac arteries.

A comprehensive physical examination is essential, including a systematic palpation of peripheral pulses. Importantly, foot ulcers often manifest as signs of severe PAD.

Objective confirmation of lower extremity PAD is made via the Doppler ABI – a diagnostic cornerstone (Figure 3). An ABI ≤0.90 is conventionally interpreted as indicative of PAD. The method for calculating the ABI may vary depending on whether it is used as a cardiovascular risk marker or for gauging the severity of PAD in symptomatic individuals, particularly in follow-up consultations after revascularisation.[1]

When used as a cardiovascular risk stratifier, a more sensitive approach for detecting PAD is recommended, using the lowest recorded systolic blood pressure at the ankle level divided by the highest brachial systolic pressure to calculate the ABI. Conversely, when the ABI is used to assess the severity of PAD in patients manifesting symptoms – or to gauge the efficacy of revascularisation interventions – the ABI should be derived using the highest systolic pressure measured at the ankle divided by the highest systolic pressure measured at the brachial artery. This approach provides a robust measure of the perfusion dynamics under the most favourable circulatory conditions and is particularly advantageous during

Table. Classification systems for severity of lower limb ischaemia[11]

Fontaine		
Stage	**Clinical description**	
1	No symptoms	
2a	Intermittent claudication after more than 200 m of walking	
2b	Intermittent claudication after less than 200 m of walking	
3	Rest pain	
4	Ischaemic ulcers or gangrene	
Rutherford		
Category	**Clinical description**	**Objective criteria**
0	No symptoms	Normal treadmill test
1	Mild claudication	Completes treadmill exercise;* AP after exercise >50 mmHg but at least 20 mmHg lower than resting value
2	Moderate claudication	Between categories 1 and 3
3	Severe claudication	Cannot complete standard treadmill exercise* and AP after exercise <50 mmHg
4	Rest pain	Resting AP <40 mmHg, flat or barely pulsatile ankle or metatarsal PVR; TP <30 mmHg
5	Minor tissue loss – nonhealing ulcer, focal gangrene	Resting AP <60 mmHg, ankle or metatarsal PVR flat or barely pulsatile; TP <40 mmHg
6	Major tissue loss – extending above TM level	Same as category 5

Abbreviations: AP = ankle pressure; PVR = pulse volume recording; TP = toe pressure; TM = transmetatarsal.

* Five minutes at 2 mph on a 12% incline.

Adapted from: Rutherford RB, Baker JD, Ernst C, et al. Recommended standards for reports dealing with lower extremity ischemia: revised version. J Vasc Surg 1997; 26: 517-538.[11]

Peripheral arterial disease clinical decision algorithm for GPs

Patient presents with leg pain, cramping, fatigue or nonhealing wounds

Assess risk factors: age, smoking, diabetes, hypertension, hyperlipidaemia, family history of cardiovascular disease

Take a history and perform a physical examination:
- Characterise leg pain: onset, duration, location, relation to exertion/rest
- Check for pulse deficits, bruits, skin changes, ulceration and temperature changes in the lower extremities
- Consider differential diagnoses (spinal stenosis, neuropathy, musculoskeletal issues)

Organise diagnostic testing: measure the ABI and, if relevant, TBI, toe pressures, exercise ABI

If ABI >1.40, suspect calcified, noncompressible vessels; consider toe pressures and/or TBI measurement*

- If TBI >0.70, diagnosis of PAD is unlikely. Look for other causes for the symptoms
- If TBI ≤0.70, diagnosis of PAD is confirmed. Toe pressure <30 mmHg signifies severe ischaemia

If ABI 0.91 to 1.40 (normal), consider alternative diagnoses. If still clinical suspicion of PAD, consider exercise ABI

- If no reduction in ABI after exercise, PAD is unlikely. Look for other causes for the symptoms
- If after exercise, ABI reduction exceeds 20% or >30 mmHg reduction in absolute ankle pressure, PAD is confirmed

If ABI ≤ 0.90, diagnosis of PAD is likely

Classify symptoms:
- Determine if symptoms are consistent with IC or CLTI
- Note presence of nonhealing wounds or rest pain

Manage risk factors:
- Initiate smoking cessation counselling
- Optimise management of diabetes, hypertension and hyperlipidaemia
- Prescribe antiplatelet therapy (aspirin or clopidogrel) for symptomatic patients unless contraindicated
- Start or adjust high-intensity statin therapy to achieve LDL targets

Management for IC:
- Recommend supervised exercise therapy or home-based exercise
- Schedule follow up to reassess symptoms and ABI
- If IC is significantly affecting function or quality of life, refer patient to a vascular specialist

Management for CLTI:
- Refer urgently to a vascular specialist for consideration of need for revascularisation
- Initiate wound care and antibiotic therapy if infection is present

Management for asymptomatic PAD (incidentally found ABI ≤0.90):
- Provide patient education about PAD and lifestyle modifications
- Implement risk-factor reduction and start statin therapy

Special considerations:
- In patients with diabetes, ensure regular foot examinations by podiatrist
- Address patient's lifestyle, including diet and exercise
- Consider referral to multidisciplinary teams as necessary for comprehensive care

*GPs can organise toe pressure or TBI measurements, but these tests are more available to specialists. Refer patient to a vascular specialist if needed.
Abbreviations: ABI = ankle-brachial index; IC = intermittent claudication; CLTI = chronic limb-threatening ischaemia; PAD = peripheral arterial disease; TBI = toe-brachial index.

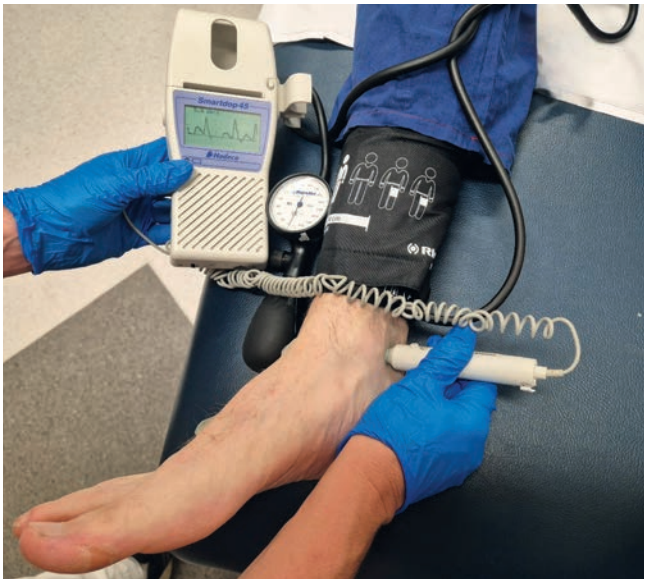


Figure 3. Measurement of the ankle-brachial index (ABI). The measurement is made in the supine position and by using a manual blood pressure cuff and a handheld Doppler. The cuff is placed just above the ankle. Flow signal is identified with a Doppler probe held over the posterior tibial and dorsalis pedis arteries, respectively. The ABI is calculated by dividing the recorded ankle pressure by the highest recorded arm pressure. Normal waveforms are noted on the screen in this case.

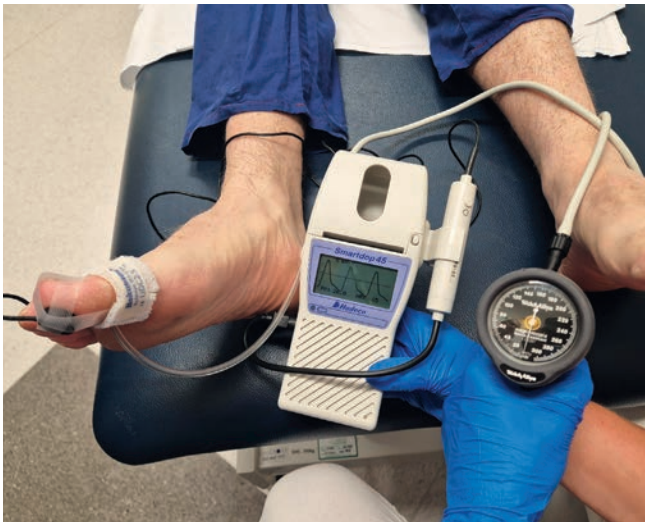


Figure 4. Toe pressure measurement. The toe cuff is placed securely around the base of the hallux. The photoplethysmography (PPG) probe is placed on the distal pulp of the hallux and affixed with tape. The cuff is inflated until 20 mmHg above the point at which the PPG waveform becomes flat. The cuff is then slowly deflated. The first regular cyclical waveform on the screen is the point at which the pressure in the artery is greater than the pressure in the cuff and is considered the toe systolic pressure. The toe-brachial index (TBI) is calculated by dividing the recorded toe pressure by the highest arm pressure recorded with doppler.

longitudinal patient assessments where incremental changes in perfusion may guide clinical decision-making and therapeutic adjustments.

Nonetheless, ABI readings may not always be reliable, particularly in the context of medial arterial calcification (MAC), which stiffens the crural arteries, diminishing their compressibility. This phenomenon is especially prevalent among individuals with diabetes or chronic kidney disease. In these cases, clinicians are advised to measure the toe-brachial index (TBI) – a ratio that is less likely to be affected by MAC and provides a more accurate reflection of perfusion status. A TBI of 0.70 or below establishes a diagnosis of PAD and absolute toe pressures below 30 mmHg signify severe ischaemia (Figure 4).[1]

When clinical suspicion for IC persists despite normal resting ABI values, exercise testing with pre- and postexercise ABI measurements is a valuable alternative. A decrease in ABI after exercise (reduction exceeding 20% or an absolute ankle pressure drop of more than 30 mmHg) confirms PAD.[1]

Imaging modalities for PAD

Assessment of the anatomy of the arterial system from the aorta to foot is recommended when revascularisation is considered.

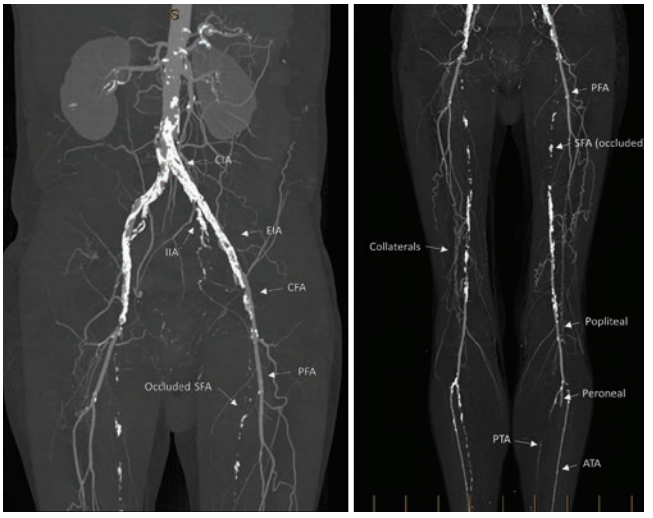
Duplex ultrasound is usually the first imaging modality for assessment of PAD due to its noninvasive nature and the absence of nephrotoxic contrast agent and radiation requirements. This modality, however, is operator dependent and its accuracy can be affected by patient factors such as body habitus, presence of oedema and arterial calcification.[1,5]

Computed tomography angiography (CTA) generates high-resolution images and can create three-dimensional reconstructions, serving as a powerful tool for pre-interventional planning (Figure 5). However, it has limited utility in assessing calcified tibial vessels. Moreover, the necessary use of a significant contrast load can limit its application, especially in individuals with severe renal compromise, in whom the risk of contrast-induced nephropathy must be carefully weighed against the diagnostic benefits.[1,5]

The use of magnetic resonance angiography (MRA) for lower limb vascular imaging is increasing because it avoids the use of ionic contrast material and can image the full arterial network. However, MRA might misidentify severe stenoses as occlusions and cannot be used in patients with some metal implants or in those with significant kidney problems due to the risk of a rare condition called nephrogenic systemic fibrosis.

New noncontrast MRA techniques (quiescent-interval single-shot [QISS] and three-dimensional fast spin echo [3D-FSE] MRA) are emerging as valuable alternatives for patients who cannot be safely exposed to gadolinium contrast.[12]

Digital subtraction angiography (DSA) remains an integral component of the imaging arsenal available to visualise the vasculature of the lower extremities. As an invasive modality offering dynamic and detailed vascular mapping, DSA is predominantly used when intervention is planned (Figure 6).



Figures 5a and b. CT angiogram maximum intensity projection (MIP) reconstruction. (a, left) MIP reconstruction of aortoiliac segment. Presence of severe calcification can be seen. Occlusions of iliac arteries are present. (b, right) MIP reconstruction of lower limb arterial system showing presence of bilateral superficial femoral artery (SFA) occlusions and severe tibial artery disease.

Abbreviations: ATA = anterior tibial artery; CFA = common femoral artery; CIA = common iliac artery; EIA = external Iliac artery; IIA - internal iliac artery; PFA = profunda femoris artery; PTA = posterior tibial artery; SFA = superficial femoral artery.

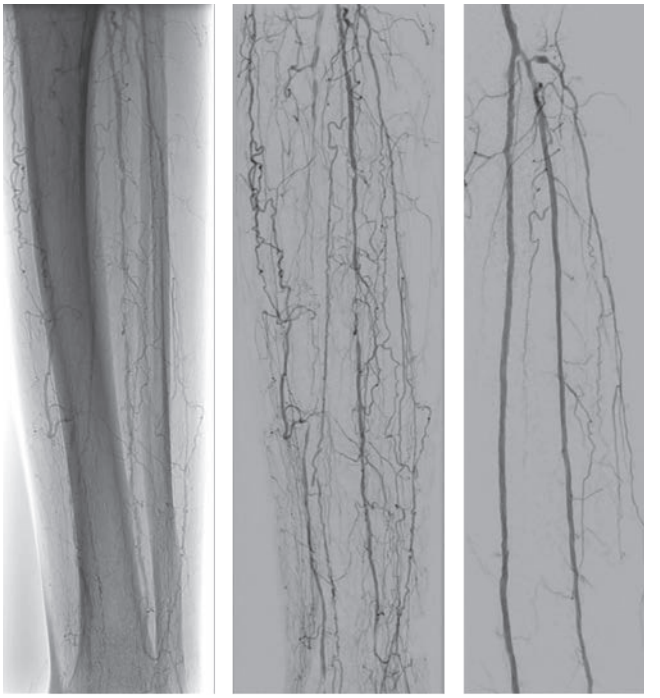


Figure 6. Patient with diabetes admitted with left hallux gangrene who underwent left leg angiography that demonstrated severe below-knee disease. (a, left) Unsubtracted angiogram. (b, middle) Digital subtraction angiogram shows severe occlusive disease of all tibial arteries. (c, right) Angioplasty of posterior tibial artery and peroneal artery was performed and these two arteries are now in flow. The angioplasties were followed by hallux amputation.

Management

Management guidelines

Comprehensive recent guidelines on the management of patients with PAD have been published by international associations and are available online. These include the following:

- *European Society for Vascular Surgery (ESVS) 2024 Clinical Practice Guidelines on the Management of Asymptomatic Lower Limb Peripheral Arterial Disease and Intermittent Claudication*[1]
- *The Intersocietal IWGDF, ESVS, SVS Guidelines on Peripheral Artery Disease in People with Diabetes Mellitus and a Foot Ulcer*[13]
- *Global Vascular Guidelines on the Management of Chronic Limb-Threatening Ischemia*.[5]

Managing atherosclerotic risk factors

Effective management of PAD includes both lifestyle modifications and pharmacological interventions, with the goals of decreasing the progression of lower limb disease and mitigating the broader cardiovascular risks associated with atherosclerosis.

A crucial part of PAD management is the cessation of tobacco use, a risk factor implicated not only in the development and progression of PAD, but also associated with increased incidences of revascularisation failure, progression to CLTI and eventual amputation. Consequently, robust smoking cessation programs and support systems are vital in the therapeutic regimen for patients with PAD.

Other modifiable risk factors – obesity, dyslipidaemia, hypertension and, notably, diabetes mellitus – have an integral role in the comprehensive management of PAD. Targeted strategies to reduce cholesterol is critical, with specific emphasis on lowering LDL-cholesterol levels to below 1.4 mmol/L. For patients not achieving these targets with statin therapy alone, the adjunctive use of ezetimibe may be advisable.[1,5] High-intensity statins are recommended for patients with PAD regardless of symptomatic presentation, aiming for optimal lipid profile control. Statin treatment is advocated not only to reduce cholesterol levels but also due to its pleiotropic effects and ability to reduce mortality.[1,14] Proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors can be used selectively as a third-line option for patients with PAD at very high risk of ischaemic events who did not reach their stipulated LDL target despite high-dose statin and ezetimibe treatment.

Diabetes dramatically amplifies the risk profile for PAD, often yielding a more aggressive, multisegmental, and bilateral manifestation of the disease.[15] It is also implicated in the development of peripheral neuropathy, which predisposes to foot deformity and ulceration. Moreover, individuals with concomitant diabetes, PAD and foot ulcers requiring amputation face prognostic outcomes comparatively as severe as those associated with several common malignant conditions, with survival rates plummeting below 50%

over five years.[13,16] In the subset of patients with PAD and diabetes, rigorous glycaemic control is warranted, with an HbA_{1c} threshold of ideally 7% or lower, although this may not always be feasible.

In the hypertensive cohort of patients with PAD, a cascading approach is adopted to carefully titrate blood pressure. Suggested thresholds are ≤120 to 129/80 mmHg for those under 70 years of age and ≤130 to 139/80 mmHg for those aged 70 years and above to taper the incidence of major adverse cardiovascular episodes effectively.[1]

Antiplatelet therapy is only recommended for the treatment of symptomatic individuals. Refer to the above management guidelines for further details on antiplatelet therapy.

Specific management

Intermittent claudication

In addition to lifestyle modifications and pharmacological interventions for PAD risk factors, effective treatment options are available that focus on relieving IC limb symptoms and improving quality of life. Examples of such interventions include exercise therapy, endovascular procedures and surgical revascularisation.

Unlike patients with IC, patients with CLTI have a very poor limb prognosis with conservative therapy. Therefore, effective revascularisation procedures are crucial for limb salvage in CLTI

It is important to note that in patients with IC, the prognosis for limb function is generally favourable, meaning that the condition does not typically result in severe limb complications.[17] The primary objective of therapy for IC is to improve cardiovascular health and enhance the patient's overall quality of life rather than prevent amputation.[1] In most cases, the risk-to-benefit ratio favours initial medical therapy over revascularisation.

One highly recommended noninvasive and evidence-based intervention for IC is walking exercise therapy. Walking exercise therapy has been shown to improve walking distance comparable to the effects of revascularisation.[18,19] Ideally, patients with IC should undergo a supervised walking exercise program, which involves intermittent walking on a treadmill to a moderate level of claudication pain, followed by a brief period of rest. These sessions should be conducted for a total duration of 30 to 60 minutes, three times a week, for three to six months. In cases where supervised exercise is not feasible, a structured home-based exercise program should be considered.[1]

For patients who continue to experience debilitating symptoms despite undergoing medical therapy, revascularisation procedures may be considered as a viable option. Revascularisation techniques encompass a range of procedures that include open surgery, endovascular procedures or hybrid (combined open and endovascular) approaches. The selection of the most appropriate approach should be determined based on various factors, including patient comorbidities, the anatomy of the vascular disease and the presence of suitable venous conduits. Endovascular therapy is particularly attractive for claudicants requiring intervention due to its relatively low morbidity and mortality rates.

Chronic limb-threatening ischaemia

Unlike patients with IC, patients with CLTI have a very poor limb prognosis with conservative therapy.[5] Therefore, effective revascularisation procedures are crucial for limb salvage in CLTI. However, managing this complex condition requires consideration of multiple factors. Although most patients with CLTI are candidates for limb salvage, shared decision-making may lead to appropriate treatment options such as primary amputation or palliation for some individuals. It is essential to promptly refer patients with CLTI to a vascular specialist. The Global Vascular Guidelines on CLTI management recommend an individualised approach called the PLAN approach, comprising the following three integrated steps.[5]

- Patient risk estimation, which involves assessing the patient's suitability for limb salvage, procedural risk and life expectancy.
- Limb staging, which entails evaluating the disease severity based on tissue loss, ischaemia and infection severity using the WIfI staging system.
- The ANatomy of vascular disease, which is assessed to determine the optimal strategy for vascular intervention.

Other factors to consider include the availability of a suitable vein to be used as the bypass conduit, patient preferences and surgeon experience in determining the most appropriate revascularisation strategy (open, endovascular or hybrid).[5]

A successful limb salvage intervention should be associated with low postprocedural morbidity and mortality, preservation or restoration of independent ambulation, improved quality of life and reduced healthcare costs.

Conclusion

GPs have a crucial role in the early detection, risk factor modification and initial management of PAD. With symptomatic and asymptomatic forms of the disease potentially leading to limb and life-threatening complications, a robust understanding and practical framework for addressing PAD are indispensable. **CT**

References

A list of references is included in the online version of this article (www.cardiologytoday.com.au).

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