



OUTPATIENT DEPARTMENT

This chapter outlines the main arterial and venous diseases that are likely to be seen within a vascular outpatient setting. It also highlights the role of the vascular nurse specialist wherever appropriate.

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1.2 Carotid artery disease

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PERIPHERAL ARTERIAL DISEASE

1.1 PERIPHERAL ARTERIAL DISEASE

S. Dorgan and H. Al-Khaffaf

Peripheral arterial disease (PAD) is generally used to describe diseases of the arteries outside the heart and the brain. However, specifically it refers to atherosclerosis of the arteries that carry blood to the legs (and to a lesser extent to the arms).

ATHEROSCLEROSIS

Atherosclerosis is a complex and insidious condition, and one of the primary causes of death in the UK.

Generally, arteries have smooth linings, allowing unimpaired blood flow. Atherosclerosis is a degenerative arterial disease and refers to “hardening of the arteries”, whereby muscle and elastic tissues are replaced with fibrous tissue, and calcification might occur.

It is characterised by atheromatous plaques, which are deposits of fatty material in the lining of medium- and large-sized arteries. These arteries then become narrowed and rough as more fat is deposited. Blood clots form more easily due to their roughness, further narrowing the artery, and thus potentially limiting blood flow.

A reduction of blood supply to the organs and tissues means that they are unable to perform as well, and the plaques are very liable to break down and form ulcers. Thromboses may then develop as a result of the roughening and ulceration of the inner coat of the arteries (Figure 1.1.1).

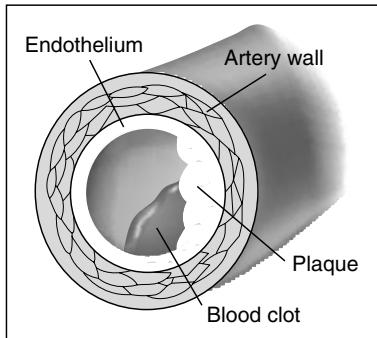


Fig. 1.1.1: A cross section of a diseased artery.

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Table 1.1.1: Areas of the body that are affected by atherosclerosis

Affected area	Disease type	Clinical effects
Brain	Cerebral vascular disease	TIA/amaurosis fugax/CVA
Heart	Coronary artery disease	Myocardial infarction Angina
Abdomen	Aortic artery disease	Aortic aneurysm
Renal	Renal artery disease	Renal failure Hypertension
Lower limbs	Peripheral arterial disease	Intermittent claudication Chronic limb ischaemia Acute limb ischaemia

Signs and symptoms

Atherosclerosis causes:

- The narrowing of small arteries which reduces the blood supply to various organs and tissues.
- Occlusion as a result of thrombosis which occurs in the diseased arteries.

In many arteries, atherosclerosis might have little effect, however, in certain organs it does produce well-recognised diseases.

- In the leg arteries results in intermittent claudication and peripheral thrombosis with gangrene of the limb.
- In the cerebral arteries it leads to cerebral thrombosis (one form of “stroke”).
- In the coronary arteries it leads to angina pectoris and coronary thrombosis.
- In the kidneys it causes renal artery stenosis, which may lead to hypertension and renal failure (Table 1.1.1).

CARDIOVASCULAR RISK FACTORS

PAD risk factors are those factors that are statistically associated with the incidence of the disease. The probability of developing PAD, coronary heart disease (CHD) or cerebrovascular disease is mainly dependent on a person's risk profile. PAD usually co-exists with extensive atherosclerotic disease elsewhere and, therefore, the risk of all types of vascular events for these patients is extremely high (Mikhailidis, 2000). Therefore, patients presenting with intermittent claudication require a critical assessment of all the vascular risk factors (Table 1.1.2), with the aim of reducing coronary and cerebral events risk (Belch, 1999).

Table 1.1.2: Cardiovascular risk factors for PAD

Modifiable risk factors	Non-modifiable risk factors
<ul style="list-style-type: none"> • Cigarette smoking • Hypercholesterolaemia • Hypertension • Physical inactivity • Obesity • Diabetes mellitus • Homocysteine (raised levels) • Thrombogenic factors 	<ul style="list-style-type: none"> • Age • Gender • Family history

Ref.: Pasternak *et al.*, 1996.

Evidence of the benefit of correction of associated risk factors is strong (Hiatt, 2001). Conservative treatment centres on modifying the following risk factors:

- smoking cigarettes (Levy, 1989);
- hypertension (Strano *et al.*, 1993);
- diabetes mellitus (Beks *et al.*, 1995);
- hyperlipidaemia (Fowkes *et al.*, 1995);
- thrombotic abnormalities, such as platelet aggregation (Belch *et al.*, 1984), increased plasma fibrinogen (Lowe *et al.*, 1991) and decreased fibrinolysis (Smith *et al.*, 1993).

Cigarette smoking

Some 90% of PAD patients are smokers or recent ex-smokers (Mikhailidis, 2000).

Smoking is a major risk factor in lower limb atherosclerosis progression and moderate cigarette smoking (15 cigarettes a day) almost doubles the risk of developing PAD, as compared to non-smokers, and the risk may increase in relation to the number of cigarettes smoked (Hughson *et al.*, 1978; Kannel & McGee, 1985).

Studies show a four- to nine-fold increased risk of PAD in patients smoking in excess of 20 cigarettes per day, as compared to non-smokers (Hughson *et al.*, 1978).

As smoking tobacco increases the risk of intermittent claudication and contributes to its progression (Krupski, 1991), stopping smoking could prevent patients from progressing towards critical limb ischaemia and is probably the most important action a claudicant can take.

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Hypertension

Hypertension increases the incidence of PAD by 4 times in women and 2.5 times in men (Kannell & McGee, 1985).

Guidelines issued by the British Hypertension Society (1999) recommend that therapy should be commenced in all patients with sustained systolic blood pressure >160 mmHg or sustained diastolic blood pressures >100 mmHg. Where there is evidence of cardiovascular disease (to include intermittent claudication), drug therapy should be initiated when patients have sustained systolic blood pressures between 140 and 159 mmHg or diastolic blood pressures between 90 and 99 mmHg.

In addition to patients commencing on any required antihypertensive medications, lifestyle modification should also be emphasised which includes:

- reduce salt intake,
- decrease alcohol consumption,
- weight reduction,
- increase exercise.

Diabetes

PAD is present in some 40% of diabetic patients aged over 40 years, and its prevalence is 7 times greater in non-insulin-dependent diabetic patients than in non-diabetics: 22% versus 3% (Beach *et al.*, 1988).

The exact nature of how diabetes increases risk of atherosclerosis is not known, however, Colwell (1987) suggests it may be due to the endothelial damage caused by free fatty acids, hyperlipidaemia, hyperglycaemia and glucose metabolism.

Levels of low- and very-low-density lipoprotein (LDL) cholesterol are often raised in people with diabetes, both of which enhance plaque formation. Conversely, high-density lipoprotein cholesterol, considered to protect against atherosclerosis, is reduced by diabetes.

Approximately 50% of all patients with diabetes will have evidence of PAD 10–15 years after the onset of diabetes. It is important for all patients with diabetes to have good diabetic control, but perhaps this is even more important in those with any form of established vascular disease.

Hyperlipidaemia

Raised serum total cholesterol, low serum high-density lipoprotein cholesterol and raised serum triglycerides are recognised risk factors that lead to PAD.

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Raised lipid levels can lead to early vascular damage (fatty streaks), cholesterol rich plaques can occlude and narrow vessels, and finally endothelial function is disturbed which affects flow in the microcirculation (Belch *et al.*, 1984).

Therefore, lowering plasma lipid levels with drugs such as statins benefits patients at risk of cardiovascular events.

The current recommendation of treating only total cholesterol over 5 mmol/l has been challenged by evidence that statins may have a direct beneficial effect on atherosclerosis (Shearman, 2002). Results from studies currently underway are needed to evaluate this in greater detail, in particular the cost-effectiveness of more widespread use of these drugs (Leng *et al.*, 2001).

Antiplatelet medication

Patients with established atheromatous vascular disease are advised to take 75 mg aspirin daily, which significantly reduces cardiovascular events – 27% relative risk reduction (Antiplatelet Trialists' Collaboration, 1988).

A randomised clinical trial comparing clopidogrel (an antiplatelet agent) with aspirin (CAPRIE Trial) illustrated a significant reduction in anticipated cardiovascular events, with clopidogrel fairing slightly better (relative risk reduction of 9%). The subgroup with PAD appeared to benefit most and there appears little doubt that all claudicants should be on an appropriate antiplatelet agent (CAPRIE Steering Committee, 1996).

Gender and age

Previous studies also indicate that 2–3 times more middle-aged men than women suffer from PAD, a ratio which appears to even out over the age of 70 years (Kannel & McGee, 1985).

Approximately 20% of men over the age of 50 years are affected (Schroll & Munck, 1981). Prevalence increases with age and the condition is likely to become increasingly common as the proportion of elderly people in the population increases (Kannel, 1996).

Role of nurses in the management of cardiovascular risk factors

- *Smoking:* Advise on how smoking affects the circulation. Refer to local specialist smoking cessation service. Give constant support and encouragement to help patient quit.

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- *Cholesterol:* Ensure patient has had fasting lipid profile undertaken in the last 12 months and that total serum cholesterol (TC) levels are <5 mmol/l and LDL <3 mmol/l. If above these levels request for patient to be commenced on statin (cholesterol lowering medication – for life) and advise regarding low-fat diet.
- *Hypertension:* Target levels – no diabetes <140/85 mmHg; diabetes <140/80 mmHg.
- *Antiplatelet medication:* Aspirin 75 mg once daily if no contraindications. Clopidogrel (Plavix) 75 mg once daily if unable to tolerate aspirin.
- *Exercise:* Refer to local community sports centre or hospital-based exercise programme if available. Home exercise programme should be discussed if preferred or if no other suitable alternative.
- *Diabetes:* Test patients for undiagnosed diabetes by undertaking fasting glucose test and urinalysis. Abnormal results should be passed onto the Diabetes Care Team for further assessment and management.

CHRONIC LOWER LIMB ISCHAEMIA

Chronic lower limb ischaemia (LLI) is one of the most common referrals to a vascular outpatient clinic. It becomes increasingly common with age and approximately 5% of people over 50 years have LLI. It is more common in men with a ratio of males to females of 2:1.

Symptoms

- Many patients with LLI have no symptoms.
- Only less than half of these patients typically experience cramp-like pain of the leg muscles, which is known as *intermittent claudication* (derived from the Latin word *claudicatio*, to limp).
- Typically the pain is brought on by exercise and relieved by rest.
- The pain only affects the muscle group being exercised: claudication involving the calf muscles only indicates disease of the superficial femoral artery while claudication of the thigh and calf together suggests an occlusion of iliac arteries. Buttock claudication occurs when there is occlusion of the internal iliac artery.
- Claudicants may use different terms to describe the discomfort associated with ambulation and activity like; “cramping”, “aching”, “weakness”, “tightness” and “giving out” (Wright, 1996). This symptom is a result of reduced blood flow and inability of the collateral circulation to meet the oxygen demands of the exercising muscle.
- Approximately 10% of patients will progress to critical ischaemia (see Section 1.1).

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- Chronic LLI can be classified into the following stages according to Fontaine's classification:
 - Stage I: Asymptomatic
 - Stage II: Intermittent claudication
 - Stage III: Ischaemic rest pain
 - Stage IV: Ulceration or gangrene, or both.

Signs

- In many patients there may be no significant skin changes. However, in some patients there may be evidence of hair loss or the skin may look dry and scaly.
- The affected limb may feel slightly cooler than the normal limb.
- Pulses in the leg and foot may be reduced or absent. It's important to remember that patients with isolated stenotic lesions may have normal pulses at rest and it is only after exercise that the pulses become reduced or absent (Table 1.1.3).

Assessment

Assessment of patients with PAD should include the following:

- History of the presenting illness.
- Social history.
- History of risk factors: cardiac, diabetes, hypertension and hypercholesterolaemia.
- Physical examination: as part from routine general examination a thorough examination of the legs should be conducted. This should include inspection to detect any trophic changes as well as palpation of all pulses.

Table 1.1.3: Clinical features of LLI

Symptoms

- Pain in calf, thigh, buttock, brought on by exercise and relieved by rest
- Walking distance is limited
- Numbness, pins and needles in skin of the foot
- Impotence in patients with aorto-iliac disease
- Symptoms of coronary or cerebrovascular atherosclerosis, i.e. angina, TIAs

Signs in affected leg

- Affected leg may feel cooler
- Diminished peripheral pulses, or absent distal to level of involved segment

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- If the pain is atypical, a full examination of the lumbar spine, hip and knee joints should also be done to exclude spinal stenosis and osteoarthritis as a cause of the patient's symptoms.
 - No vascular examination is complete without recording the ankle brachial pressure index (ABPI).

The ankle brachial pressure index

- The ABPI is an inexpensive, non-invasive diagnostic test that is both highly sensitive and specific for PAD, and this test can quantitatively clarify the severity of PAD in nearly all affected individuals, whether symptomatic or not.
 - The ABPI is also an accurate predictor of poor prognosis.
 - Patients with an ABPI of 0.90 or less are diagnosed to have PVD. It is important to note that an ABPI can be difficult to measure in patients with long-standing diabetes or other older patients with calcified calf arteries, not compressible by the blood pressure cuff.

How to record the ankle brachial pressure index

- Rest the patient in a supine position.
 - Measure the systolic blood pressure in both arms.
 - Measure the ankle systolic blood pressure from the left and right dorsalis pedis (DP) and posterior tibial (PT) arteries.
 - The value of one ankle is taken from the higher of the DP and PT readings.
 - The ABPI is calculated by dividing the highest ankle pressure by the highest brachial pressure (McKenna *et al.*, 1991) (Figure 1.1.2).



Fig. 1.1.2: Recording the ABPI.