CLINICAL MANAGEMENT extra

Arterial Disease Ulcers, Part 1: Clinical Diagnosis and Investigation

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This continuing educational activity will expire for physicians on September 30, 2015.

Editor's note: Part 2 of this article series will appear in the October issue of *Advances in Skin & Wound Care*.

PURPOSE:

To provide information regarding the clinical diagnosis, assessment, and investigations related to arterial disease ulcers of the lower extremity.

TARGET AUDIENCE:

This continuing education activity is intended for physicians and nurses with an interest in skin and wound care.

OBJECTIVES:

After participating in this educational activity, the participant should be better able to:

1. Describe the etiology and pathophysiology of peripheral arterial disease (PAD) and arterial ulcers.
2. Identify the clinical features of PAD and arterial ulcers as a result of arterial insufficiency of the lower limb.
3. Compare assessment modalities to determine the extent of arterial insufficiency and appropriate interventions.

ABSTRACT

Arterial disease (peripheral vascular disease) is the result of narrowing of the blood vessel lumen. The classic clinical signs need to be recognized early before progression to arterial predominant disease and limb ischemia. Arterial ulcers or tissue breakdown can result from trauma, infection, or other etiologies with diabetes, smoking, increasing age, and hypertension the most important risk factors. Diagnostic testing starts with a palpable pulse with special investigation including handheld Doppler for ankle brachial pressure index ratios, segmental duplex leg Doppler waveforms, and more specialized procedures, including transcutaneous oxygen saturation.

KEYWORDS: arterial disease, Doppler, ankle-brachial pressure index, ischemia, hypoxia

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INTRODUCTION

Adequate delivery of blood and oxygen is essential to sustain life. This truth was recognized in 1727 BC in the Code of Hammurabi of Mesopotamia that contains a phrase: ''to pour out his life-blood like water.'' In the Babylonian creation myth of Enuma Elish, blood was considered the essential ingredient in the creation of mankind. Ugaritic and Egyptian sources also recognized the importance of blood as a life source.[1] Indeed, not only life, but also wound healing, depends on it.

Ischemia is defined as a restriction in blood supply, generally due to factors related to blood vessels, such as stenosis and/or occlusions, leading to damage or dysfunction of tissue.[2] This is a clinical manifestation of advanced peripheral arterial disease (PAD), also known as critical limb ischemia.

Hypoxia is defined as a condition in which tissues are deprived of oxygen, regardless of the cause. This distinguishes it from hypoxemia, which is a reduction in blood oxygen content specifically.[2] Whether due to ischemia or hypoxemia, hypoxia is a final common pathway in many causes of wound healing failure (eg, venous stasis ulcers, pressure ulcers, arterial ulcers).[3,4]

The Inter-Society Consensus for the Management of Peripheral Arterial Disease (TASC II) recommends the term ''critical limb ischemia'' be used for all patients with (*a*) chronic ischemic rest pain and/or (*b*) ulcers or gangrene, attributable to objectively proven arterial occlusive disease. The term implies severity and chronicity and is to be distinguished from acute limb ischemia.[5]

This continuing education article will help the clinician to describe the etiology and pathophysiology of PAD and arterial ulcers and to recognize the clinical features of PAD and arterial ulcers as a result of hypoxia and/or ischemia of the lower limb.

ETIOLOGY AND PATHOPHYSIOLOGY

Under ideal conditions, wound repair is a perfectly orchestrated symphony of highly integrated biologic and molecular events of cell migration and proliferation, and of extracellular matrix deposition and remodeling. Certain pathophysiologic and metabolic conditions can significantly alter this normal course of events so that healing is impaired or delayed, resulting in chronic, nonhealing wounds. Wound hypoxia impairs essentially all the components of healing.[6] In the absence of adequate oxygen delivery, the process of wound healing becomes a cacophony of deafening alarm bells.

Chronic wounds of the lower extremities may develop because of venous disease, arterial disease, infections, infestations, neuropathy, trauma, diabetes, malignancy, burns, coagulopathy, vasculitis, immobility (eg, pressure ulcers), or psychosis (eg, factitious ulcers). The most common ulcers on the leg are venous, arterial, neuropathic, or a combination of these.[3] Approximately 8% to 10% of patients with leg and foot ulcers have pure arterial insufficiency.[7]

Transport and delivery of oxygen to tissue are the primary function of blood flow. Under normal physiologic conditions, blood flow and blood oxygen content are matched to ensure normal function of body tissues. In the event of an injury, increased oxygen demands may be met by an increase in the extraction of oxygen from hemoglobin, as well as by an increase in perfusion. These mechanisms offer limited physiologic reserve. Importantly, both mechanisms depend on a patent conduit whereby oxygen can be transported from the alveoli to the affected tissue.

Peripheral arterial disease results from the narrowing of the vessel lumen by the accumulation of cholesterol plaque and other tissue debris. Critical limb ischemia is an advanced state of PAD. It is characterized by severe impairment of blood flow to the limb, whereby the metabolic requirements of the tissue at rest are not met. Multiple occlusive lesions of the limb arteries, coupled with functional and structural changes in the microcirculation, are responsible for inadequate tissue perfusion, leading to the formation of skin ulcers and necrosis. Inflammatory mediators and endogenous procoagulants contribute to development and progression of critical limb ischemia. Blood components, such as red blood cells, white blood cells, and platelets, aggregate and perturb blood flow in the microcirculation.[8]

The processes that contribute to wound healing, such as fibroblast replication, collagen placement, angiogenesis, intracellular leukocyte bacterial destruction, and infection resistance, are all reliant upon oxygen.[9,10] Local tissue hypoxia and tissue ischemia impair healing profoundly.[4] It is widely accepted that lower-extremity ulcers with a partial oxygen pressure (transcutaneous oxygen pressure [$TcPo_2$]) of less than 30 mm Hg will not heal. Anemia might further exacerbate tissue hypoxia.

Arterial ulcers may develop because of atherosclerosis, arterial obstruction, arterial trauma, cholesterol embolism, diabetes, thromboangiitis

obliterans (Buerger disease), HIV-related vascular disease,[11] popliteal artery entrapment,[12] cystic adventitial disease, fibromuscular dysplasia, vasculitis, radiation damage, arteriovenous malformation, increased blood viscosity, and platelet adhesiveness.

Precipitating events for arterial ulcers vary. Limbs with arterial compromise may have minimal but adequate blood flow to maintain tissue viability. Ischemic lower-extremity ulcers are often precipitated by trauma or infection. The location of traumatic ulcers varies depending on the cause, but these wounds are commonly found on the foot or on the anterior tibial area of the lower leg. Traumatic ulcers may be caused by an acute physical injury, such as a blunt trauma–like bumping into a piece of furniture or dropping a heavy object on the foot. They can also be caused by acute or chronic pressure, such as the continual pressure from ill-fitting footwear. Several other conditions may be responsible for tissue breakdown, including thermal extremes, chemicals, or a localized clot or embolus, which can also lead to decreased cellular nutrition from impaired arterial flow. Regardless of the cause, wound healing is inhibited when ischemia is present.

Infection results when the host defense equilibrium is upset in favor of the bacteria. Infection plays various roles in the etiology, healing, operative repair, and complications of arterial and mixed ulcers. Restoration of flow is crucial to infection control in arterial ulcers and must be addressed first, especially in patients with diabetes or who are immunocompromised.[13]

Although some wounds heal in the presence of ischemia, arterial inflow must often be improved for healing to occur. Repair of the injured tissue requires more oxygen, as well as an increase in other nutrients. Diminished arterial flow and tissue hypoxia in PAD can eventually lead to gangrene or tissue necrosis. In the presence of an arterial ulceration, the natural history is one of disease progression. This can reach a sudden tipping point with extreme physical discomfort and pain, increased risk of infection, and even limb loss. This tipping point can be averted by revascularization.

EPIDEMIOLOGY

Peripheral arterial disease is very common but severely underdiagnosed.[14] It affects approximately 14% to 20% of the adult population, with a ratio between symptomatic and asymptomatic individuals of 1:3 to 1:4.[15] Patients with PAD may also have a history of cerebrovascular disease or coronary artery disease.[3,16] Diabetes and cigarette smoking are strongly associated with PAD: 90% of patients with PAD possess at least 1 of these 2 risk factors.[17] The risk of mortality due to PAD increases with age and severity of arterial insufficiency. The 10-year mortality is approximately 60%.[18,19]

Only 3% of all patients with PAD have critical limb ischemia. The incidence of critical limb ischemia is estimated between 300 and 1000 persons per million per year. Of the patients with critical limb ischemia, 30% will undergo an amputation within the first year after diagnosis, and 25% of these patients will have died because of cardiovascular or cerebrovascular events.[5,20] The 5-year mortality for patients with critical limb ischemia is 50% to 70%, with 35% of these being cardiovascular deaths.[15]

A unique study has demonstrated that the prevalence of leg ulcers can be decreased within a defined geographical population, probably as the result of more liberal use of arterial interventions, involvement of podiatrists, and improved wound care. Medical interventions aimed at decreasing cardiovascular morbidity, with statins and platelet inhibitors and smoking cessation, were considered important factors.[21,22] Despite the fact that clinicians are dealing with a population at risk, their endeavors are not futile.

DIAGNOSIS

The importance of making the diagnosis of PAD derives from the prognostic information implicit with its diagnosis.[23] A concerted effort to detect arterial disease in patients with wounds is paramount, along with selecting an appropriate therapy when arterial insufficiency is identified as a significant or primary etiology.[14] All patients with lower-extremity ulcers should be assessed for arterial disease.[5,13] If arterial disease is detected or even suspected, a vascular surgeon should be consulted.

The first component of the wound bed preparation (WBP) paradigm is to identify and treat the cause(s) of the ulcer.[24] Sibbald et al[24] suggest that for any ulcer the clinician should determine if there is adequate blood supply for the ulcer to heal. The second component of the WBP paradigm is to identify the cause(s) as specifically as possible and to make appropriate referrals. In a recent community-based comprehensive interprofessional assessment of leg and foot ulcer patients, more than 60% of diagnoses were changed or made more specific. This led to the implementation of best practices, facilitating the optimization of the WBP paradigm and improving chronic wound healing rates.[25] The third component of the WBP paradigm is to review cofactors/comorbidities (systemic disease, nutrition, medications) that may delay or inhibit healing.

DOCUMENTATION

Quod on in actis, non est mundo. [''Anything not written down is nonexistent''].

The authors briefly remind clinicians that an integral step in all diagnostic procedures is to document all interactions with a patient. This is a very important legal and professional requirement.

HISTORY

The patient often provides the observant clinician with the diagnosis while a comprehensive history is taken. The medical history is the foundation of the physician-patient interaction.[23]

The patient often presents with symptoms related to PAD. Progressive intermittent claudication (ie, ischemic muscular pain on

exertion) is a very common complaint and indicates mild to moderate PAD. Cessation of activity relieves the exercising muscle's demand-supply mismatch and enables restoration of oxidative metabolism. Approximately 25% of patients with claudication deteriorate in terms of clinical stage, most often in the first year after diagnosis.

Rest pain might precede or accompany the development of an arterial ulcer or gangrene, which indicates advanced PAD. Patients with rest pain may describe waking at night with pain across the distal metatarsal area of the foot. Pain due to an arterial ulcer is very intense and increases on elevation of the leg.[3] Elevating the limb would cause an increase in pain due to a decrease in perfusion. The pain is temporarily relieved by placing the leg in a dependent position. This increases blood flow through collateral vessels because of increased hydrostatic pressure facilitated by gravity. Patients often prefer to sleep with the leg dangling from the bed or while sitting in a lounge chair. This is an important diagnostic indicator not to be overlooked in history taking. The subsequent edema, due to the dependent position of the limb, could further impair wound healing and could mimic chronic venous insufficiency.

Some patients with ischemic ulcers may not experience pain, because of extensive sensory neuropathy (eg, diabetic neuropathy). On the contrary, these patients may experience such intense hyperesthesia associated with the neuropathy that they cannot even bear the light touch of bandages. Ulcers in patients with neuropathy are typically found on the plantar side of the foot and are surrounded by calluses from long-term local pressure. These patients may describe the sensations of burning, stinging, shooting, and stabbing pain (neuropathic pain), rather than the more characteristic gnawing, aching, throbbing, and tender pain (nociceptive pain) associated with PAD.

Ulceration, gangrene, and rest pain all indicate critical limb ischemia, which occurs when limb blood flow is inadequate to meet the metabolic demands of the tissues at rest (Rutherford categories 4 to 6; Rutherford grades II to IV; Fontaine classes III to IV).

Patients with a history of atherosclerosis risk factors (eg, smoking, diabetes, hypertension, hypercholesterolemia, advanced age, obesity, hypothyroidism) should be assessed for PAD.[13,16] Historical evidence of coronary artery disease or cerebrovascular disease should be sought and documented. Other systemic causes of impaired healing, including autoimmune diseases and medication (systemic steroids, immunosuppressive drugs, and chemotherapy), should be identified and corrected.

The risk factors for arterial disease are outlined with the mnemonic **ABCDE'S**[26]**:**

• **A**$_{1c}$: Hemoglobin A_{1c} refers to the personal or family history of diabetes or arterial disease.

• **B**lood pressure: Determine whether it is elevated and if the patient is using medications.

• **C**holesterol: Elevated cholesterol is a risk factor. Use of statins (cholesterol-lowering agents) may reduce this risk.

• **D**iet and obesity: Increased weight, especially a body mass index greater than 25 kg/m^2, indicates an increased risk for coronary disease, peripheral vascular disease, and type 2 diabetes.

• **E**xercise: Individuals who exercise regularly have a lower risk of peripheral vascular disease and can build up a greater tolerance to overcome compromised circulation. In general, individuals with leg pain at rest or when in bed have severe ischemia. Those who have pain or claudication (aching and throbbing calf muscles) when walking up a few stairs or less than 50 m have moderate disease. Individuals with symptoms after walking 1 or 2 blocks have mild disease.

• **S**moking: One cigarette decreases circulation by 30% for 1 hour. The longer a patient smokes, the greater the risk of disease. Ask patients about their tobacco usage and how many years they have been smoking. Patients with symptomatic vascular disease may aggravate their symptoms by using tobacco, nicotine gum, or nicotine patches for smoking cessation.

All allergies should be documented. Iodine sensitivity is very important in the context of contrast imaging studies (eg, arteriography, computed tomographic angiography). Previous operations should also be documented, with emphasis on previous bypass procedures and/or endovascular interventions.

CLINICAL EXAMINATION

Vital signs should be assessed and recorded. Blood pressure should be measured in both arms. A thorough, systemic clinical examination should be done. The vascular examination includes inspection, palpation, and auscultation of vascular structures throughout the body (Figure 1). Using the mnemonic of the 6 P's can help remind the clinician of the clinical signs of arterial ulcers: pulselessness, pain, pallor, polar, punched-out defect, and pressure sites.[27]

On inspection, the skin could demonstrate trophic changes, including distal hair loss and a dry, shiny appearance. The toenails might appear thickened and yellow (onychogryphosis). The muscles of the affected limb might appear wasted (ischemic atrophy). Discoloration of digits could also indicate advanced disease. Ischemic tissue initially appears pale, then blue-gray, followed by purple and, finally, black. Gangrenous tissue eventually becomes black, hard, and mummified. The hardened tissue is not painful, but significant pain may be present at the line of demarcation between the gangrene and the viable but ischemic tissue. Gangrene may involve a small skin area or extend to an entire limb depending on the location of the arterial lesion. If a small patch of skin is affected, the skin will dry and fall off, exposing a skin lesion.

Arterial ulcers appear on the area where the arterial supply is the poorest and at pressure points and bony prominences: on the tip of the toes, dorsum of the foot, the heel, and the shin. The ulcers are sharply defined, punched out, and deep. The base of the wound is pale, nongranulating, and often necrotic.[28] The ulcer may penetrate

Figure 1.
PALPATING THE POSTERIOR TIBIAL ARTERY

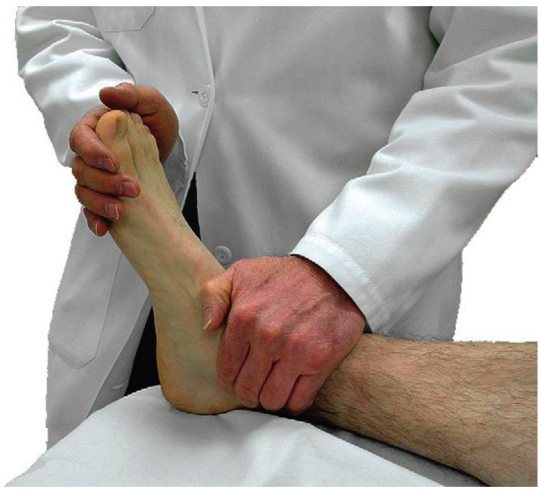

Photo courtesy/Dr. Gregory R. Weir

the deep fascia, even exposing the tendons at the base of the wound. The presence of varicose veins does not exclude the diagnosis.[3]

The ulcer can be photographed and described using the MEASURE mnemonic: **m**easure size, **e**xudate amount and characteristics, **a**ppearance, **s**uffering (pain), **u**ndermining, **r**eevaluate, and **e**dge.[29]

In ulcers that appear ischemic, the clinician should also consider and look for contributing factors other than atherosclerosis that involve the arterial system (macrovascular vs microvascular), such as thromboangiitis, vasculitis, Raynaud, pyoderma gangrenosum, thalassemia, or sickle cell disease.[13]

The pulse examination of the arms and legs is a critical part of the vascular examination. Asymmetry, decreased intensity, or absence of pulses provide clinical evidence of PAD and indicate the location of stenotic lesions.[23] According to TASC II,[5] pulses are graded as 0 (absent), 1 (diminished), or 2 (normal). The American Heart Association's guideline adds a grade of 3 for bounding pulses, which may be evidence of aortic valve insufficiency.[20] To ensure consistency, adhere to local policy. A palpable pulse indicates an arterial pressure of 80 mm Hg or higher and does not exclude PAD. The pedal pulses are usually absent if PAD is the primary etiology of a leg ulcer. Despite a palpable pulse, the nonhealing wound might be situated in a different angiosome that has to be 1 in order to induce healing.

The posterior tibial pulse should be present. Its absence is diagnostic for PAD. In contrast, the dorsalis pedis pulse may be absent in 8% of healthy persons.[30] The sensitivity of an undetectable pulse for the diagnosis of PAD can be 17% to 32%, whereas the specificity is 97% to 99%.[31] The foot is often colder than the contralateral limb or the proximal part of the same limb, and topical infrared surface thermometry may be a valuable diagnostic tool to provide objective numerical data, if available.

There may be a delay in capillary refill response. Normal capillary refill time is less than 3 seconds from pallor to normal skin color. A delay of 10 to 15 seconds in the return of color when raising the leg to 45 degrees for 1 minute (Buerger test), with rubor when the limb is placed in a dependent position (reactive hyperemia), also indicates advanced PAD. On occasion, the increase in flow velocity and poststenotic turbulence caused by a stenosis may create a thrill in the artery on palpation. This is often accompanied by a bruit that can be heard with auscultation with a stethoscope.

An objective neurologic assessment must be completed to exclude neuropathy. The mnemonic SAM reminds the clinician to test for **s**ensory, **a**utonomic, and **m**otor function. The 5.07 monofilament has been accepted as the medical standard for screening of the minimum level of protective sensation in the foot. The reproducible buckling stress force required to bend the 5.07 monofilament is 10 g of force.[32] Autonomic (sympathetic) denervation is demonstrated by impaired hair growth or impaired sweating. Sensorimotor neuropathy is demonstrated by lack of vibratory sense. The presence of a neuroischemic ulcer is considered a sign of multiorgan disease.[33]

Ankle-brachial pressure measurement is absolutely essential in the examination of any patient with a wound of the lower extremity.[5,34] It should be performed as the first-line noninvasive test for the diagnosis of PAD.[35] All clinicians involved in the management of patients with lower-limb ulcers should have direct access to 8-MHz handheld Doppler devices, and this should not be considered a special investigation limited to a vascular laboratory. A handheld Doppler ultrasound transmitting probe sends a signal, which is reflected from an object to the receiving probe. If the signal strikes a moving object such as blood cells, a frequency shift is detected and reflected as sound (Doppler principle). The audible signals of arterial flow patterns can then be determined. The handheld Doppler is used to detect an audible signal on the dorsum of the foot or ankle (dorsalis pedis artery and posterior tibial artery). A blood pressure cuff is then placed around the lower calf and inflated until the audible signal disappears. The cuff is then slowly deflated, and when the signal returns, the systolic pressure is determined from the reading on the cuff gauge.

An ankle-brachial pressure index (ABPI) is determined by dividing the highest Doppler pressure of a limb at the level of the ankle, by the highest Doppler pressure of either upper limb at the level of the fossa cubiti. An ABPI of less than 0.9 or higher than 1.4 is considered abnormal. Lower indices (<0.9) are indicative of PAD. An ABPI of less than 0.5 should be followed by an urgent referral to a vascular surgeon, especially in the presence of an ulcer. This is highly suggestive of critical limb ischemia, and conservative management is unlikely to succeed. Higher indices (>1.4)

might indicate severe, circumferential atherosclerosis of the tibial arteries with poor compressibility. Diabetes, chronic renal disease, or rheumatoid arthritis should then be excluded. Toe pressures are more sensitive in this group of patients, because of the fact that the digital arteries are not often circumferentially calcified and should be done to exclude or confirm PAD.[35] The presence of an abnormal ABPI is also associated with an increased risk of cardiovascular morbidity and mortality.[16,35,36] A scientific statement from the American Heart Association recommended that the measurement and interpretation of the ABPI should be part of the standard curriculum for medical and nursing students.[35]

In patients with severe atherosclerosis (whether due to diabetes, chronic renal failure, or advanced age), the tibial vessels become circumferentially calcified. This renders them incompressible. Toe pressure tests measure the flow through the large toe where the vessel is small enough that calcium deposits do not involve the entire vessel and compressibility is usually present. A toe pressure of 50 mm Hg or higher, even in a person with diabetes, is usually adequate for healing. Toe pressures of 20 to 30 mm Hg usually indicate vascular compromise with impaired wound healing. Pressures less than 30 mm Hg may be adequate if the skin is intact, but as soon as injury occurs and disrupts the cutaneous barrier, the vascular supply is often inadequate for the repair process. An arterial ulcer with a toe pressure of less than 30 mm Hg is an indication for intervention.[37]

SPECIAL INVESTIGATIONS

The relevance and value of any special investigation should be based on whether it will influence the management of the patient. Thomas Hunt was among the first to investigate the role of oxygen in wound healing. His major contributions to the field include the observations that all wounds show some degree of hypoxia.[38]

Transcutaneous Oxygen Pressure

Transcutaneous oxygen pressure is a useful diagnostic tool in PAD.[39] It is finding increasing application as a diagnostic tool to assess the periwound oxygen tension of wounds, ulcers, and skin flaps. It must be remembered that $TcPo_2$ measures the oxygen partial pressure in adjacent areas of a wound and does not represent the actual partial pressure of oxygen within the wound. Tissue hypoxia is defined as a $TcPo_2$ of less than 40 mm Hg. In patients breathing air, a $TcPo_2$ of less than 30 mm Hg indicates critical limb ischemia. The clinician should be aware that a low $TcPo_2$ value obtained while breathing normobaric air may be caused by a diffusion barrier, such as edema.

A $TcPo_2$ obtained while breathing normobaric air can assist in identifying which patients will not heal spontaneously.[40] In fact, $TcPo_2$ has been proposed both as a screening tool to select patients for revascularization and to evaluate the efficacy of endovascular procedures.[41] An increase in $TcPo_2$ to greater than 40 mm Hg during normobaric air breathing after revascularization (surgical or endovascular) is usually associated with subsequent healing, although the increase in $TcPo_2$ may be delayed because of the initial ischemia-reperfusion phenomenon and subsequent edema. As a result of a successful intervention, $TcPo_2$ progressively increases to reach a peak after 4 weeks.[42] Transcutaneous oxygen pressure has been validated as a predictor of lower-limb amputation healing complications.[40,43] And, $TcPo_2$ measured in a hyperbaric chamber provides the best single discriminator between success and failure of hyperbaric oxygen therapy using a cutoff of 200 mm Hg.[44]

For any objective technique to be used in practice, validity, reliability, and reproducibility need to be established. Transcutaneous oxygen pressure is considered a reliable method to measure tissue viability. Clinical interest in other techniques (laser Doppler flowmetry, $TcPCo_2$, near-infrared spectroscopy) has not yet been fully validated, and their uses are mostly confined to the research laboratory at present.[45]

Laser Doppler Flowmetry

Laser Doppler provides semiquantitative measurements of perfusion.[39] This has also been used as a noninvasive test in patients with ischemic ulcers. There is a significant correlation between laser Doppler and $TcPo_2$.[46] Some studies used multiple tests to assess peripheral circulation and oxygen delivery. Laser Doppler appears to be useful in cases where false-low $TcPo_2$ values were obtained because of acute edema or inflammation.

Doppler Arterial Waveforms

Doppler arterial waveform disparities and dampened pulse volume recordings are also associated with PAD. These noninvasive tests are usually performed in a vascular laboratory. Recording the Doppler shift demonstrates the normal triphasic signal representing the 3 phases of the pulsation in a normal peripheral artery. The first wave represents forward flow of blood and arterial distention. The second phase represents the arterial relaxation and subsequent retrograde flow of blood. The third phase of the triphasic Doppler signal is believed to represent the bulging of the aortic valve, which occurs during diastole. Another theory suggested by some authors is that the third phase represents the rebound of the compliant, elastic arterial wall. The third phase of the triphasic arterial signal is first lost as an artery becomes less compliant and is followed by loss of the second phase of the triphasic Doppler signal. With worsening occlusive disease proximal to the area of auscultation, the normally sharp first wave becomes flattened and broader. In severely diseased arteries, the Doppler signal can be a monophasic, low-amplitude wave.

Segmental Doppler Pressures

Segmental pressures can be used to determine the location of arterial vascular lesions. Pressures obtained at the level of the

thigh, above the knee, calf, and ankles are compared with each other and with pressures in the other leg. An arterial lesion can be isolated with a 20–mm Hg gradient between cuff pressures. If no pressure gradient exists on a limb with claudication, the patient is asked to exercise, and subsequent pressures are obtained.

Imaging Studies

Imaging studies help to determine the location and the extent of the arterial disease. These studies could be used to confirm a clinical diagnosis of PAD but are more appropriate when used by the vascular surgeon as a roadmap in deciding and planning on the type of and extent of intervention required to revascularize a limb. Be sure to discuss the choice of imaging study with the vascular surgeon on the wound care team. Not all patients with arterial ulcers will require intervention,[33,47,48] which means that not all patients will require these expensive investigations.

A vascular surgeon should be consulted in cases where the patient shows evidence or suspicion of PAD. The presence of rest pain, gangrene, discoloration, ulceration, ABPI of less than 0.5, or $TcPo_2$ of less than 40 mm Hg warrant urgent referral.[40]

CONCLUSION

The assessment and diagnosis of the patient with insufficient arterial supply to the lower limb are of vital importance for timely clinical intervention and provision of much needed pain relief in cases of critical limb ischemia. This article highlights the clinical picture of the patient presenting with arterial disease and identifies assessment modalities available to guide informed decisions regarding the clinical pathway most beneficial to patients within their individual set of circumstances. After reading this article, the clinician will have a better understanding regarding the urgency of approach and appropriate use of available modalities to guide referral for appropriate intervention.

PRACTICE PEARLS

- Timely identification of the patient at risk for arterial disease can make the difference between salvage possibilities and the inevitable loss of a limb.
- The **6 P's** help remind the clinician of the clinical signs of arterial ulcers: pulselessness, pain, pallor, polar, punched-out defect, and pressure sites.[27]
- The risk factors for arterial disease are outlined with the mnemonic: **ABCDE'S**[26]:
  - **A**$_{1c}$: hemoglobin A_{1c}
  - **B**lood pressure
  - **C**holesterol
  - **D**iet and obesity
  - **E**xercise
  - **S**moking

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