History and Physical Exam of Chronic Critical Limb Ischemia

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Rajmony Pannu and Steven M. Dean

Introduction

"Journey of thousand miles begins with a single step" Lao-tzu: 604 BC - 531 BC

History taking and physical examination are the first steps in patient evaluation. This chapter describes in detail the components of the physical exam that are essential in decision making when confronting patients with critical limb ischemia. The methods described can be applied as a standalone modality or in conjunction with vascular tests to diagnose and follow up vascular disease. This review will allow an astute clinician to recognize common complaints associated with critical limb ischemia and correctly associate them with the characteristic physical manifestations. Consequently, this highly morbid disease should not go unrecognized.

History

The medical history initiates the physician-patient relationship, guides the uncovering of relevant physical findings, facilitates appropriate vascular testing, and then assists treatment choices. Per one report, a skilled history can lead to the correct diagnosis 75 % of the time [1]. The most common presenting symptom for limb ischemia is pain, and knowing its severity, location, frequency, exacerbating, and/or relieving factors along with the duration assists in distinguishing vascular vs nonvascular leg discomfort.

R. Pannu, MD, RPVI
Vascular & Endovascular Medicine, Columbus Vascular Vein & Wound Center, 895 S State street, Columbus, OH 43081, USA

S.M. Dean, DO, RPVI (✉)
Division of Cardiovascular Medicine, Department of Internal Medicine, The Ohio State University Wexner Medical Center, Columbus, OH 43017, USA

Critical Limb Ischemia

A distinction must be made between chronic critical limb ischemia (cCLI) and acute critical limb ischemia (aCLI). aCLI is a medical emergency related to abrupt arterial occlusion which requires immediate treatment. The pathophysiology of cCLI is related to slowly progressive, inadequate arterial limb perfusion that is below the threshold needed to meet the metabolic demands of the limb, resulting in resting ischemia with pain, skin breakdown, and eventual tissue necrosis [2].

Acute Critical Limb Ischemia

aCLI most commonly occurs due to embolism or in situ thrombosis. Arterial dissection or trauma can also cause aCLI but at a much lower rate. Unfortunately, the presenting symptoms do not predict the cause of aCLI. Patients can present with an asymptomatic loss of pulse, acute deterioration in previously stable claudication or as sudden onset of severe rest pain in the affected limb. These symptoms may develop over several days or over few hours. Acute arterial occlusion may result in any one or all of the notorious five "Ps":

1. Pain
2. Paresthesias
3. Pallor
4. Paralysis
5. Poikilothermia

These manifestations typically occur due to lack of collateral blood flow and reestablishment of primary arterial flow in timely fashion is the key.

The pain in aCLI may be evanescent and pallor may quickly give way to cyanosis. This discomfort is very different than that of cCLI. For instance, it is not localized to the

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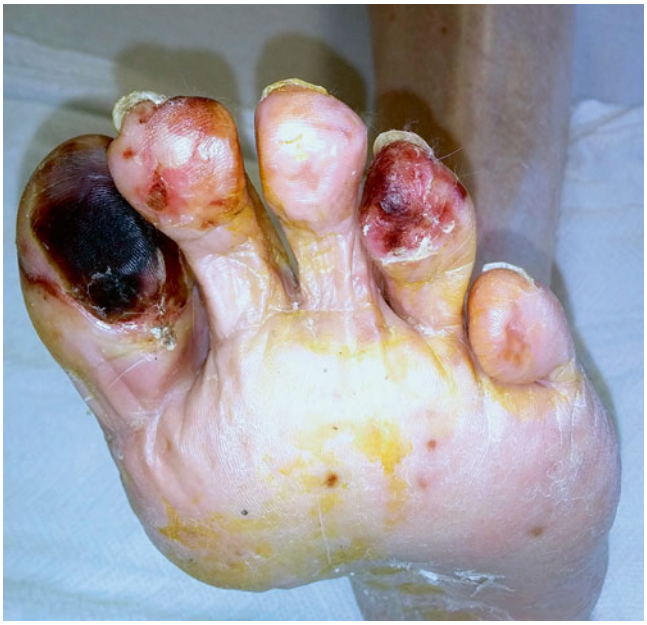

Fig. 6.1 Arterial emboli after endovascular procedure

acral portion of the foot and is not affected by gravity. The pain is usually diffuse and can extend above the ankle in severe cases. It is usually of sudden onset and may quickly increase in intensity when caused by arterial emboli (Fig. 6.1). Patients can describe the feeling as being struck in the limb after which they feel weak. In case of arterial thrombosis, the pain develops less rapidly, but patient is aware of some change in their baseline status. The rapid peak in pain intensity is also absent in arterial thrombosis. However, in the case of *massive* arterial thrombosis where the limb is threatened, the pain quickly and unexpectedly changes in intensity. The pain may subside in intensity after the initial vasospasm subsides and collateral flow is recruited. Finally, the discomfort may completely resolve if the collateral supply is able to meet the functional demands of the foot or it may convert into the pain typically seen in cCLI patients.

Pulses are usually difficult to palpate since the majority of this patient population have preexisting peripheral arterial occlusive disease.

The sensory deficit may be minimal and can easily be missed early in the course of presentation. Patients may lose sensation of light touch, the ability to differentiate two points, and experience altered vibratory perception and proprioception before deep pain ensues. Loss of motor function may present very late in the process as the majority of foot movements are produced by muscles originating below the knee. It can be challenging to test for the motor function of the foot as these muscle groups may not be developed at baseline in many PAD patients. The tests that are generally advocated in evaluating the intrinsic foot muscles are the paper grip test and intrinsic positive test:

- Paper grip test: The patient attempts to grip a standard paper sheet between two toes while the physician tries to pull it away. Patient may curl their toes to grip the paper and this action is hypothesized to activate the long extrinsic toe flexors. Therefore, the paper grip test is repeatable but has questionable validity as a measure of intrinsic weakness because is likely to be assessing both intrinsic and extrinsic muscle strength [3].
- Intrinsic positive test: The test involves the participant extending the great toe while simultaneously attempting to flex the lesser toes at the MTP joint and extend the interphalangeal joints. The strength of the intrinsic muscles is determined by the type of lesser toe flexion demonstrated which includes either (1) intrinsic positive pattern, which involves flexion at the MTP joint and extension at the interphalangeal joints, or (2) intrinsic negative pattern, where the participant is unable to actively flex the MTP joint and extend the interphalangeal joints. This test has not been extensively validated and level of strength required to perform the test is unknown [4].

More objective testing can be performed in centers that have access to a handheld dynamometer. This instrument can measure toe flexor strength [5].

The persistence of pain, particularly if followed by numbness and/or weakness suggests the threat of limb loss.

Chronic Critical Limb Ischemia

Inability of the blood flow to meet the functional demands of tissue produces pain that has two very distinct characteristics: intermittent claudication and ischemic rest pain.

Intermittent claudication is discomfort associated with exercise that is relieved by rest. Depending on the anatomical location and extent of arterial occlusive disease, the patient may present with buttock, thigh, and/or calf claudication. Calf claudication is the most common presenting symptom and is reported as cramps in the calf that are brought on by walking. This should not be confused with nocturnal cramps that some elderly people manifest. These cramps have no known vascular origin and are thought to be the result of an exaggerated neuromuscular response to stretching. Chronic exertional compartment syndrome can also produce calf tightness provoked by exercise, but the distinguishing feature is that the patient is usually a younger athlete without atherosclerotic risk factors and large calf muscles. Increased muscle pressure due to impaired venous outflow is usually the cause and this pain is not relieved quickly by rest.

Thigh and buttock claudication is different than calf claudication as the exertional pain is much less pronounced. Instead, patients typically complain of an exertional ache or discomfort that is associated with weakness. Patients might

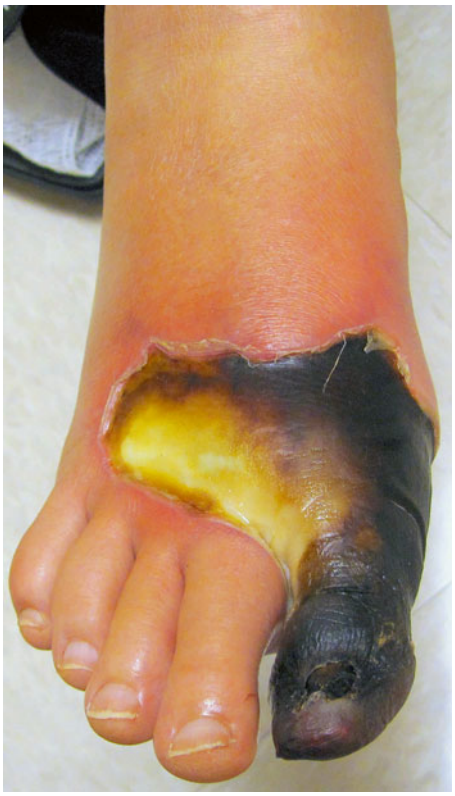

Fig. 6.2 Gangrene of the great toe and forefoot

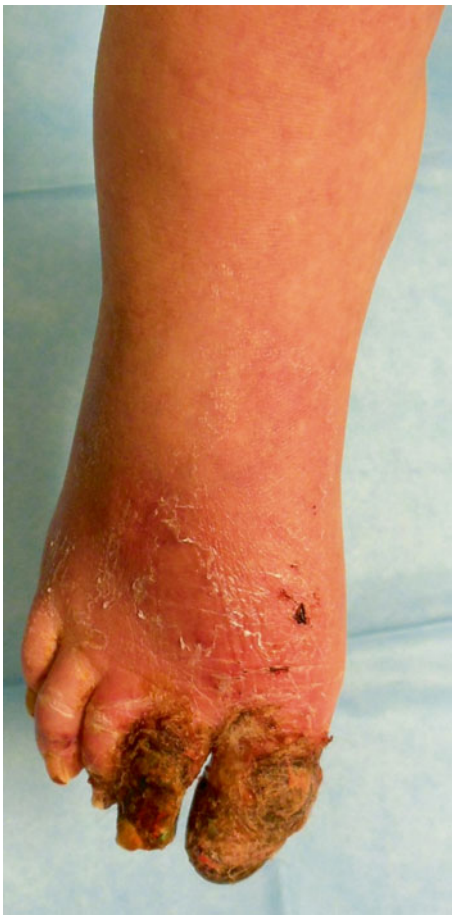

Fig. 6.3 Nonhealing ischemic ulceration

say that their hip or thigh "gives out" or "tires" after they have walked a stereotypical distance. Thigh and buttock claudication may be somewhat similar to the pain of hip osteoarthritis, but the amount of exercise that provokes discomfort in osteoarthritis is variable; the pain does not subside promptly upon cessation of activity, and there is considerable day-to-day pain variability. Neurospinal compression may also cause exertional buttock and thigh pain, but the main differentiating feature from claudication is associated limb numbness and that the symptom complex can also be produced by prolonged standing. Additionally, the pain and numbness of neurospinal compression can also involve the perineum. Affected patients often relate their symptoms are improved with truncal flexion such as when they shop in the supermarket and lean on the shopping cart. However, straightening of the lumbar spine is likely to exacerbate their symptoms. Since patients presenting with lumbar neurofo-raminal compression are often elderly, coexistent PAD with claudication can confound the diagnosis and it is paramount that the predominant complaints be matched to the disease. Treating mild arterial occlusive disease in this patient will not relieve their symptoms. Thigh claudication can also be part of the postthrombotic syndrome that usually occurs when the patient has history of ilio-femoral deep vein thrombosis (DVT) and the collateral venous outflow fails to match the increased arterial inflow during exercise. The pain is often described as "bursting" or a severe tightness or heavy sensation that is relieved by cessation of exercise, but the improvement is not as rapid as in arterial claudication.

Claudication involving the foot is uncommon and can occur independently or in association with calf claudication. Foot claudication is more common in the setting of thromboangiitis obliterans because of the distal distribution of occlusive arterial lesions. Foot claudication secondary to atherosclerotic occlusive disease is frequently associated with ischemic rest pain. There might be reference of acral numbness and/or cold sensation and patients may refer to feeling a "wooden foot." Inflammatory processes of the foot can cause similar focal pain but the variability of the discomfort with exercise that is not quickly relieved by exercise cessation helps differentiate the two (ischemic rest pain is not quickly relieved with exercise). Additionally, non vascular pain may not relate to activity level, may be affected by weight bearing.

cCLI occurs from severely reduced arterial blood flow resulting in ischemic rest pain, nonhealing ischemic ulceration(s), and/or gangrene (Figs. 6.2 and 6.3). The pain is usually located in the acral portion of the leg, toes, or heels, is severe, and is persistent. The feet may be insensitive to cold, the joints could be stiff, and/or patients may suffer from hyperesthesias. Patients note pain relief when feet are

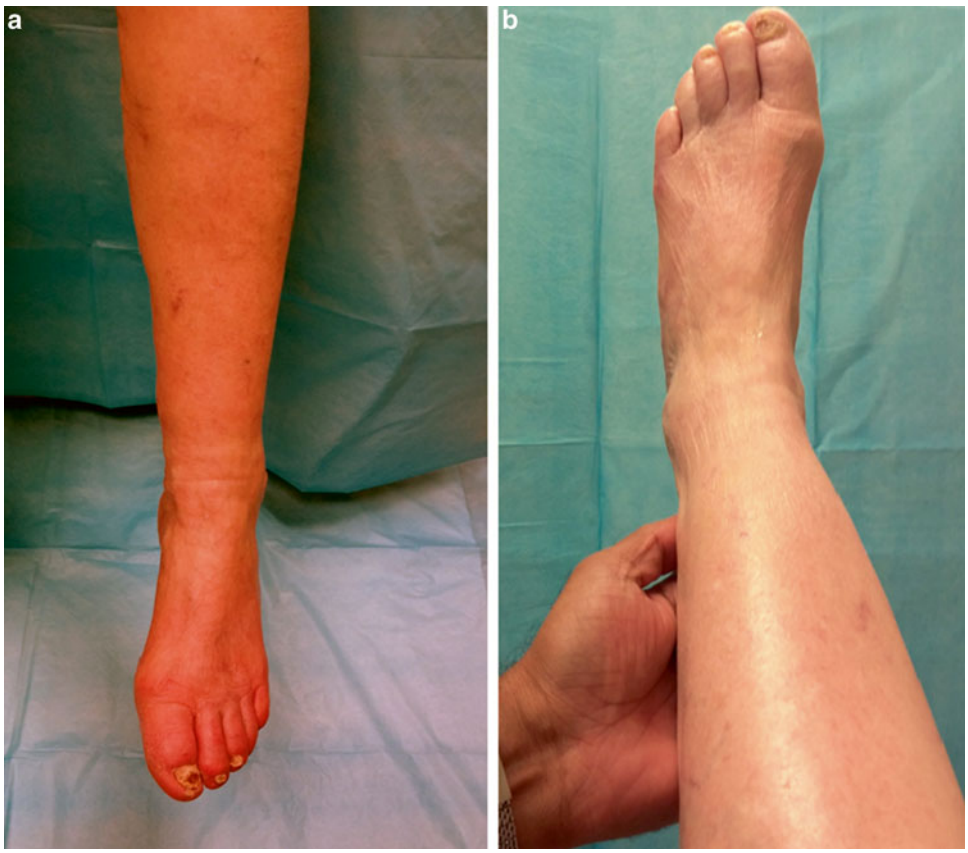

Fig. 6.4 Buerger's sign with dependent rubor and elevation pallor. (**a**) Dependent rubor in critical limb ischemia due to impaired vasoconstriction. (**b**) Elevation pallor in critical limb ischemia

in dependent position and conversely an increase in pain if the feet are at or above the level of heart. Patients will often narrate how they have pain in the feet at night that improves when they hang their feet over the bedside. Thus, they eventually learn to sleep with their feet hanging down or in a chair with their feet dependent. A clinician may be deceived by pedal rubor, but it is important to stress that this "dependent rubor" (Fig. 6.4) is matched by "elevation pallor" (Buerger's sign) which is consistent with severe limb ischemia. Pain may be sharply localized to an ischemic ulcer or gangrenous toe. However, the initial presentation of cCLI may be acute gangrene or a minimally painful wound with associated advanced diabetes mellitus and peripheral neuropathy. It is very important to recognize that this particular patient subset is insensate and may not manifest rest pain. If cCLI is untreated, gangrene may ensue with the eventual loss of the limb (from amputation or mummification) and perhaps life (from sepsis).

Physical Examination

A comprehensive physical exam must include vital signs, including the blood pressure, heart rate, and respiratory rate. The blood pressure should be measured in both arms, with an appropriately sized blood pressure cuff. The patient's overall appearance should be noted. The vascular examination includes inspection, auscultation of vascular structures, and palpation of axial pulses, and if the distal pedal pulses are not palpable, documentation of same should be completed with a handheld Doppler if available. A systemic approach is advocated and all the body systems should be examined. Examination of the abdomen should include more than palpation for an aortic aneurysm. Lower abdominal bruits may provide the sole clue to arterial occlusive disease in the patient with thigh and buttock claudication as there may be no sign of chronic ischemia and the femoral pulse exam maybe normal at rest. It is outside the scope of this chapter to detail the heart, lung, musculoskeletal, neurological, etc., examinations.

Visual Inspection

The limbs should be inspected carefully, assessing their appearance, symmetry, and color, and for evidence of edema or muscle wasting. Special attention should be given to any areas of discoloration, and if present, the patient should be interrogated about the timeline of discoloration appearance as this may clarify the acuity of presenting symptoms. Subtle

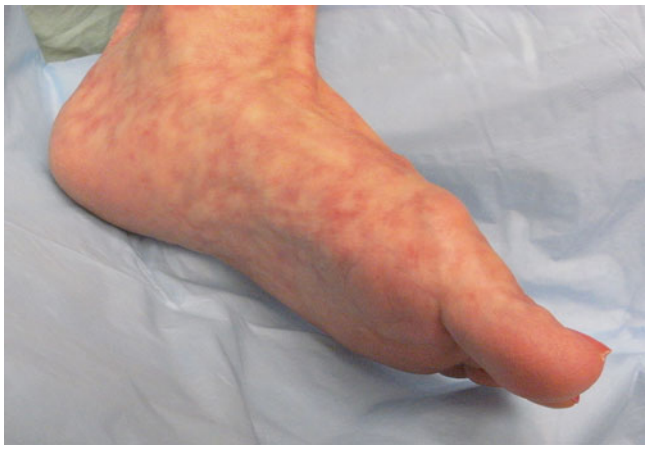

Fig. 6.5 Livedo reticularis is rash resembling fishnet pattern

findings of distal hair loss, trophic skin changes, and hypertrophic nails should be sought and may suggest severe underlying PAD. Patients presenting with lower extremity ulcerations suspected of having critical limb ischemia should be examined for the fishnet-like rash of livedo reticularis (Fig. 6.5). Also, the skin around the ulcerations should be inspected for changes in texture, color, elasticity, etc. It is important to visually inspect the base and margins of any presenting wound. In patients with neuropathy, the plantar foot surface should be inspected for the presence of unknown foreign bodies in the skin.

Arterial ulcers typically occur on the toes, heels, and bony prominences of the foot. They appear "punched out" with well-demarcated edges and a pale, nongranulating, and often necrotic base. The surrounding skin may be cool to touch and can exhibit dusky erythema. Examination of the arterial system demonstrates decreased or absent distal pedal pulses.

In contrast to the arterial ulceration, the majority of the venous leg ulcers develop in the gaiter area of the limb, usually around the malleoli (Fig. 6.6). The ulceration may be circumferential or discrete, and the ulcer bed is often covered with a fibrinous layer mixed with granulation tissue surrounded by irregular margins. Pitting-dependent edema is often present and may predate the ulcer. Extravasation of erythrocytes into the skin occurs, resulting in the deposition of hemosiderin within macrophages, which stimulates melanin production, pigmenting the skin brown.

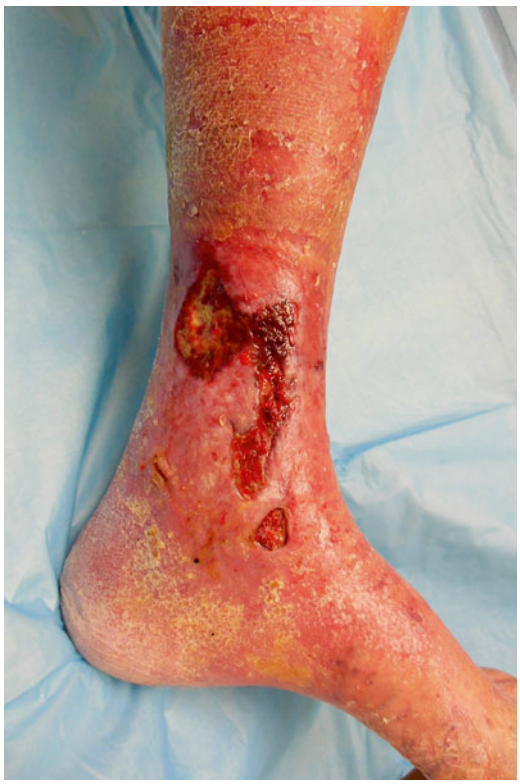

Fig. 6.6 Gaiter area venous ulcer

Pulse Exam

The pulse exam of upper and lower extremities is a critical part of the vascular exam. The exam should be conducted in warm and well-lit room. Asymmetry, decreased intensity, or the absence of pulses suggests arterial occlusive disease and provides clues to the location of disease. There are two widely used numeric scales used to grade pulses ranging from 0 (absent) to 4 (aneurysmal). We recommend using a simpler 0 (absent), 1 (diminished), and 2 (normal) scale as there may be a substantial inter-user variability in grading pulses on 0–4 scale [6]. A bounding pulse may suggest aortic valvular insufficiency and an expansive pulse may suggest the presence of an arterial aneurysm.

Pulse exam of the lower extremity includes palpation of the femoral, popliteal, posterior tibial, and dorsalis pedis arteries and the examination should performed with the patient in supine position.

The femoral pulse is located below the inguinal ligament, approximately midway between the iliac spine and symphysis pubis. It may be challenging to palpate this pulse in muscular or obese patients. In order to facilitate femoral pulse detection, the hips should be externally rotated so the artery can be palpated over the pubic ramus of the ilium approximately 1–2 finger breadths lateral to the pubic tubercle where there is less fat.

Palpation of popliteal artery is often difficult and has led to premise that if the popliteal pulse is very easily palpable, it should raise suspicion for an aneurysm. The leg should be straight, slightly flexed at the knee joint. The leg should be relaxed enough that it "falls" on to the examiner's hands. The popliteal pulse is usually palpated with three fingers from each hand and the thumb applying a moderate opposing

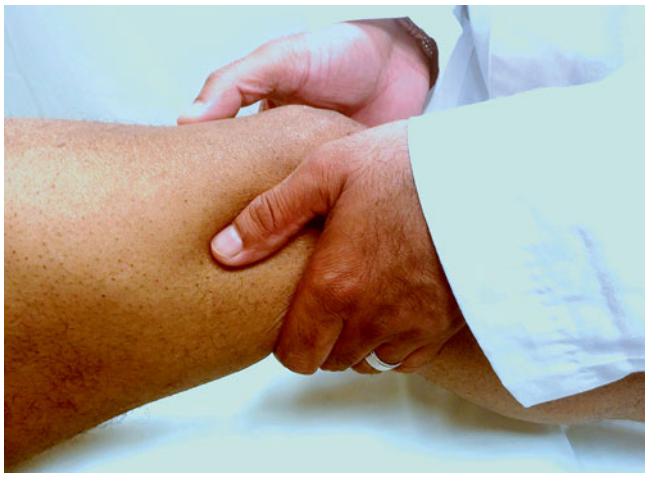

Fig. 6.7 Palpation of popliteal artery

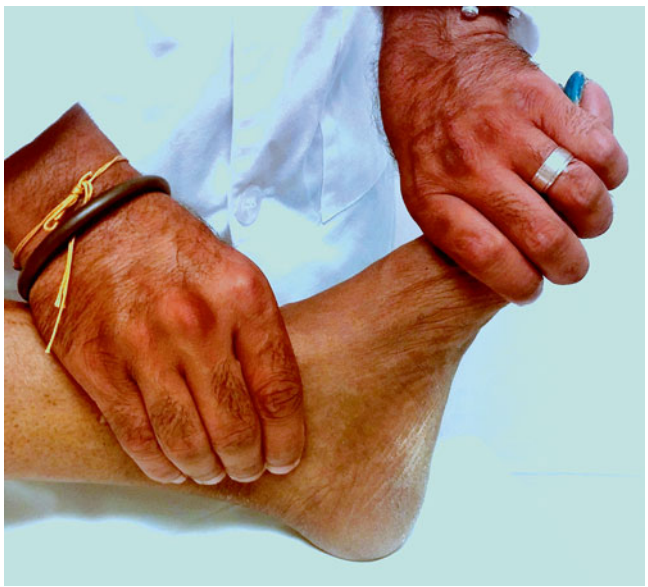

Fig. 6.8 Palpation of posterior tibial artery

force to the top of the knee (Fig. 6.7). The pulse can be felt at the junction of medial and lateral third of the popliteal fossa.

The posterior tibial pulse is located slightly behind the medial malleolus. The examiner should approach the depression behind the malleolus from the lateral side and apply their digits to the lower curvature. Passive dorsiflexion of the foot may aid the palpation of the posterior tibial pulse (Fig. 6.8). The absence of posterior tibial pulse is diagnostic for peripheral arterial disease.

The dorsalis pedis pulse is palpated along the dorsum of the foot between the first and second metatarsal bones. Ideally, arteries should be palpated with the sensitive palmer surface of fingers (Fig. 6.9). The dorsalis pedis artery may be absent in 5–10 % of the population. In such cases, the lateral tibial artery, the terminal branch of peroneal artery should be

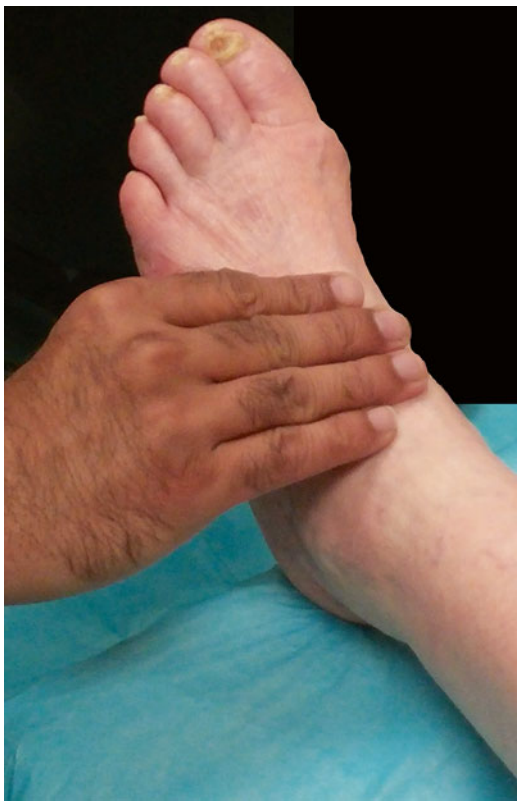

Fig. 6.9 Palpation of dorsalis pedis artery

sought. This artery can be palpated higher up in the foot, below the lateral ankle, and medial to fibular prominence.

With wide availability of handheld Doppler devices, it is imperative to document the presence of Doppler signals in the pedal arteries that are not palpable. This assumes increased significance in endovascular interventions below the knee and could help determine if pedal vessels could potentially be used as an access for complex interventions. To perform the Doppler exam, a copious amount of gel is applied to the medial malleolus area where the posterior tibial pulse is expected to be found and the Doppler probe is held between the index finger and thumb. It is important to apply minimal pressure with the probe as even slight compression can obliterate flow through a diseased artery (Fig. 6.10). The Doppler flow can be further documented as triphasic, biphasic, or monophasic. Similar maneuvers should be performed on dorsalis pedis artery (Fig. 6.11). Examiners can use the handheld Doppler along the course of dorsalis pedis artery toward the hollow between the first and second phalangeal bones to ascertain the patency of pedal plantar arch. If the dorsalis pedis artery is not audible, place the Doppler probe on the dorsum of the foot between the first and second phalangeal bones to evaluate for the presence of flow within deep plantar artery (Fig. 6.12); subsequently, try to follow its course back to the expected location of dorsalis pedis artery between the first and second metatarsal bones.

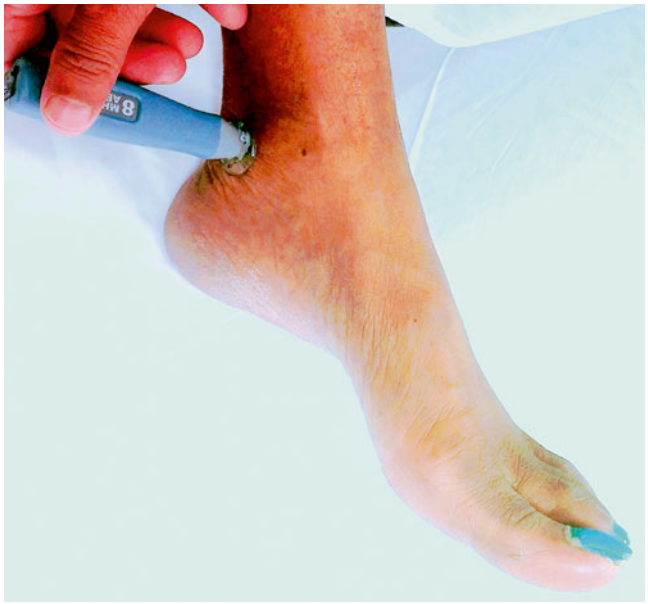

Fig. 6.10 Doppler of posterior tibial artery with liberal gel and slight pressure on the artery

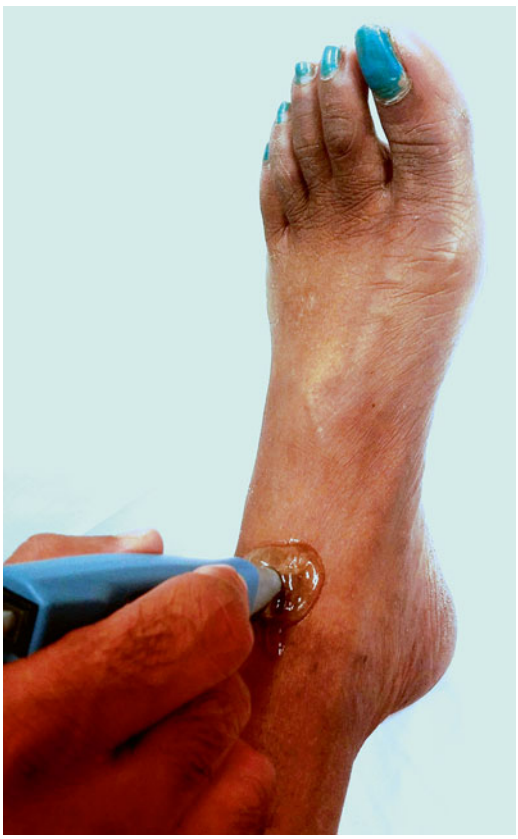

Fig. 6.11 Doppler of dorsalis pedis artery

The examiner should document the farthest point that the flow is detected. This aids in planning future interventions and may even help ascertain if the disease is even amenable to intervention. Wounds secondary to thromboangiitis obliterans are not amenable to endovascular interventions.

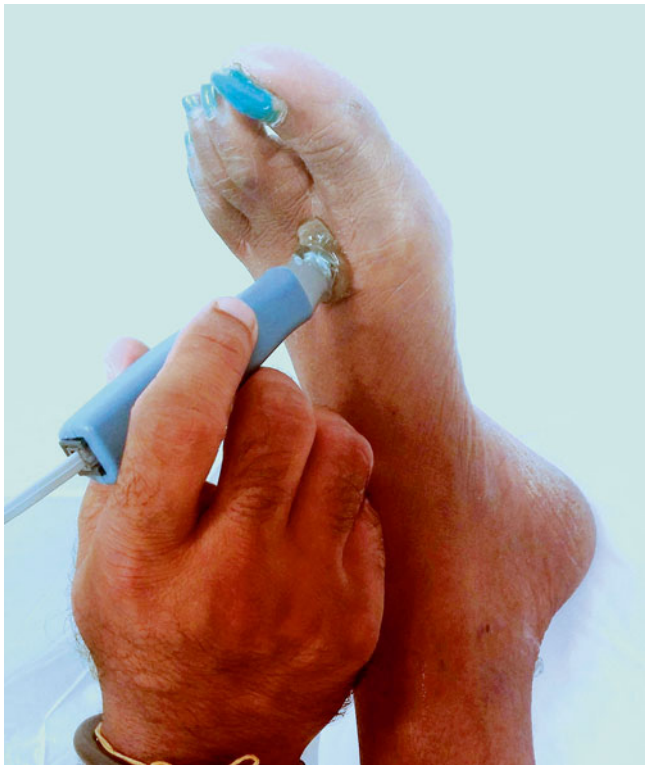

Fig. 6.12 Doppler of deep plantar artery

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