Vascular and Neuropathic Foot

14

Adolfo V. Zavala

Diabetic foot is one of the most frequent complications of diabetes, with a high morbimortality, with important health cost, and causing alterations in the quality of life of the patients.

The presence of an ulcer in the feet indicates a high risk of amputation and death. Fifty percent of patients with it die within the next 5 years.

Patients with a history of foot ulcers have a high risk of ulcer recurrence or the involvement of the collateral limb. Therefore, the patient must be considered at a great risk of developing ulcers, and protective and corrective measures must be indicated so that he does not ulcerate.

Diabetic foot is the distal ankle involvement, caused by various causes, mainly due to the interaction of peripheral vasculopathy, neuropathy and alterations in foot biodynamics.

By decreasing the vitality of the foot, and lack of recognition of trauma due to loss of protective sensitivity, pre ulcerative lesions are caused, which, if not detected and treated in time, ulcerate, become infected, causing necrosis leading to amputation, and even the loss of the patient's life.

It is noteworthy that injuries do not occur suddenly, that there is a timeline for the diabetic foot, which must be known to avoid its progression.

We highlight what Bernard Swan said: "I marvel at how much people pay for cutting a leg, and how little they pay to avoid it."

A.V. Zavala
Diabetic Foot and Vasculopathies Clinic at Diabetes Division, Internal Medicine Department, Hospital de Clínicas José de San Martín, University of Buenos Aires, Buenos Aires, Argentina
e-mail: info@fuedin.org; fpuchulu@gmail.com

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Neuropathic Foot

Neuropathy is one of the most frequent complications, being able to be present in more than 50% of diabetics, after 10 years of the disease. The prevalence depends on the diagnostic methods used, increasing when using electromyogram and biopsy.

It can be asymptomatic and is the cause of 80% of foot ulcers (60% pure neuropathic and 20% neuroischemic).

It is noteworthy that it can be present and be asymptomatic, and this is one of the causes of the lesions when the patient does not recognize the presence of pathology and consequently do not take preventive measures, which is why the annual examination of lower limbs is emphasized. By itself, it increases morbidity and mortality, being associated with other complications of diabetes.

The calcification of the middle tunic (Monkerberg's disease) is more frequent in diabetics than in the general population, its pathogenesis is not known with certainty. Edmons et al. argue that it is produced by neuropathy, which we corroborated in a study in diabetics patients with or without calcification. The denervation of arterial walls alters tissue oxygenation, with lack of blood flow regulation, and thus facilitates arterial calcification, with an auto-sympathectomy.

Neuropathy is caused by the individual or associated action of the following processes: sorbitol accumulation, reduction of nerve myo-inositol, reduction in synthesis, quantity and transport of intra-axonal proteins, reduction of ATPase Na/K in the nerve, reduction of the incorporation of glycolipids and amino acids to the myelin, excessive accumulation of glycogen, increased glycosylation of nerve proteins, and nerve hypoxia.

In addition to hyperglycemia, there has been an increased incidence of neuropathy in patients with increased elevated total cholesterol and LDL, increased triglycerides, increased BMI, high levels of von Willebrand factor, microalbuminuria, hypertension and smoking. This highlights the need of a good metabolic control and the reduction of cardiovascular risks factors.

The pathophysiology of neuropathy is not well understood, and is due to metabolic alterations triggered by hyperglycemia. We observe in the figure these alterations: (Fig. 14.1).

To highlight is the elevation of four metabolic pathways:

1. Polyols pathways, with the increase of the sorbitol and decrease of the myo-inositol. This causes increase of the nerve edema and a decrease of the sodium potassium pump.
2. Glycosylation of the nerve proteins (axon and myelin)
3. Oxidative stress, by increasing reactive oxygen species (ROS).
4. Alterations of the δ-6-desaturase, causing prostacyclins decrease and thromboxane increase.

There would be other factors that contribute to nerve damage, such as deficiency of essential fatty acids, immunological and haemorreological alterations.

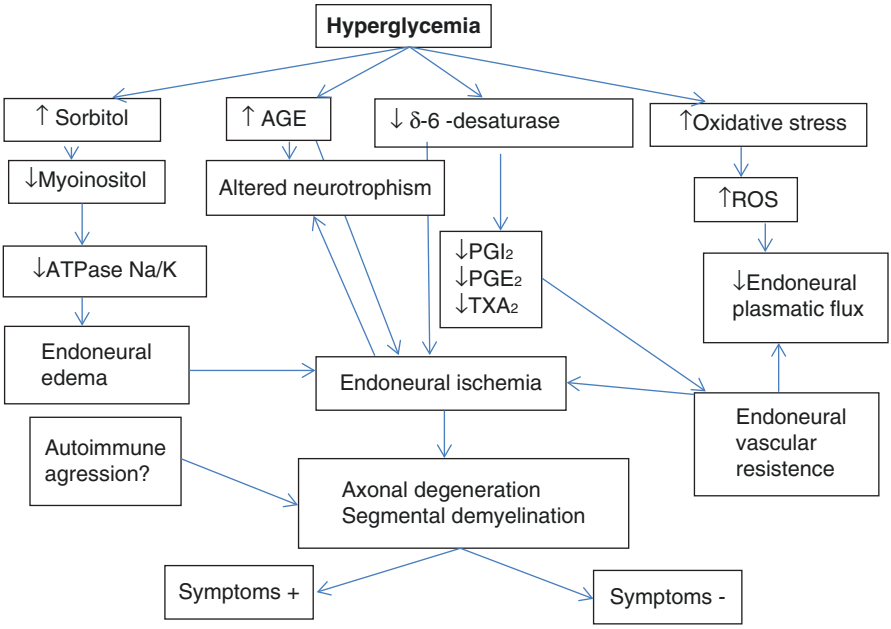

Fig. 14.1 Possible mechanisms by which hyperglycemia determines neuropathy

Table 14.1 Differences between acute and chronic sensitive neuropathy

	Acute sensitive neuropathy	Chronic sensitive motor neuropathy
Start mode	Fast	Insidious, slow
Symptoms	Severe pain and terebrante	Paresthesia, numbness
Severity symptoms	+++	0 to ++

Types of Neuropathy

Neuropathy can affect the foot of the diabetic by individual action or in conjunction with the following categories:

Sensitive Neuropathy

Premature cramps occur specially at night, sensation of walking on cottons, hypoaesthesia and hyperesthesia.

Sensory fibers are affected, losing protection to external and internal aggressions (loss of protective sensitivity).

Sensitivity is altered: tactile, valued with the Semmes-Weinstein filament, vibratory sensitivity (tuning fork or biothesiometer), thermal sensitivity, sensitivity to pain and osteotendinous reflexes.

Acute sensitive neuropathy should be differentiated from chronic, as seen in the Table 14.1.

Motor Neuropathy

The foot muscles (interosseous and lumbrical) are especially affected. This does not permit the toes separation, favoring the action of the extensor tendon, which produces the hammer or claw toes. Foot support points are altered, and chronic hypersupport may result in mallet toes.

Motor neuropathy is potentiated with sarcopenia, which is more frequent in old people, sedentary, with poor diets and with associated chronic pathologies, especially nephropathy.

Autonomic Neuropathy

The skin is dry, with hyperkeratosis, hot with venous dilatation. The temperature is increased and the calcification of the interosseous is favored.

The temperature is elevated and in patches, caused by the increase of flow and the presence of arteriovenous shunt. This causes auto-sypathectomy with calcification of the middle tunic of the vessels.

Osteoarthropathy

With autonomic neuropathy, osteopenia is produced, which facilitates osteoarticular alterations.

The most common involvement is the ankle joint, tarsometatarsal and metatarsophalangeal. It is especially seen in poorly controlled diabetics with peripheral and autonomic neuropathy.

There is hyperkeratosis and edema of the foot, usually without pain. It should be suspected in every patient with long-term Diabetes, with a hot and swollen foot, with peripheral and autonomic neuropathy, with other diabetic complications. Dislocations and fractures occurs which, if not diagnosed early, can produce permanent deformations with metatarsal overloaded and recurrent ulcers.

Neurological Evaluation

For the diagnosis of peripheral neuropathy, a complete clinical and instrumental examination should be performed. It should be done once a year to all diabetic patients despite been asymptomatic, and should be performed more frequently in the presence of symptoms.

Neuropathy should be suspected in long-standing, poorly controlled diabetic patients and with other diabetic complications. In many cases it can be asymptomatic, so it is always necessary to evaluate with instrumental maneuvers.

- Symptoms: pain, especially nocturnal leg cramps, hypoesthesia and hyperesthesia, pallesthesia, tingling, coldness sensation, walking on cotton sensation, etc.
- Evaluation of motor neuropathy: atrophy of the interosseous and lumbrical muscles. Evaluate the magnitude of the muscle strength, the walk and the spreading of the toes. Photo: normal case before separating the toes, and then opening them. In cases of pathological spreading sign, it must be evaluated if there is atrophy of the interosseous spaces (Fig. 14.2).

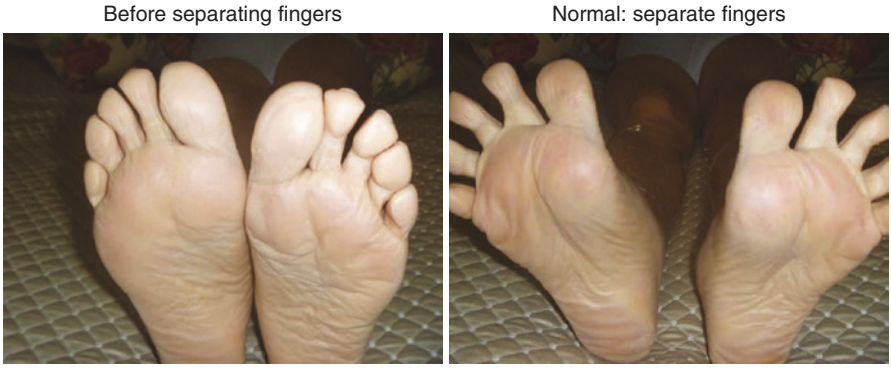

Fig. 14.2 Evaluation of motor neuropathy (**a**) before separating fingers; (**b**) normal: separate fingers

- In the autonomic neuropathy the skin is hot with venous dilatation. At first is hyperhidrosis, but subsequently is dry, it develops an auto-sympathectomy. It is manifested by the decrease or absence of secretion of the sweat and sebaceous glands, causing a decrease in the cutaneous trophism and favoring trophic lesions and/or infections. It is very useful in these cases to measure the temperature by hand and if possible with infrared thermometers, which are more accurate.
- There are other instruments to measure the temperature, including a new podimetric system, which is a mat that marks the temperature, predicting 97% of the recurrence of ulcers.

Score of Sensory Signs

Several scores can be made, we use the University of Texas guidelines, assessing sensitivity to pain, discrimination against cold and heat, vibratory sensitivity, tactile sensitivity with Semmens Weinstein 5.07 monofilament and aquilian reflex.

At least the tactile sensitivity should be measured with the Semmens Weinstein filament (high predictability), vibration sensitivity with the 128 Hz tuning fork or a quantitative meter (Biothesiometer) and the temperature with the infrared thermometer. The latter is highly predictive of the risk of developing ulcers in cases of preulcerative lesions, especially with bone deformations and hyperkeratosis.

Can be seen in the photo, an infrared thermometer, in some countries, patients are indicated to buy it to control their foot temperature. If bone deformities exist, with hyperkeratosis and one or more degree centigrade difference with another zone, it indicates a high risk of ulceration. The patients should rest and consult to the health team. Consensuses mention that it is the most accurate and predictive method of the risk of ulceration.

It is of great value also in the Charcot's foot. In this case, the difference of 2°Centigrades from one zone to another indicates oxidative stress, specific to the acute stage of the disease and when the remission stage begins (Fig. 14.3).

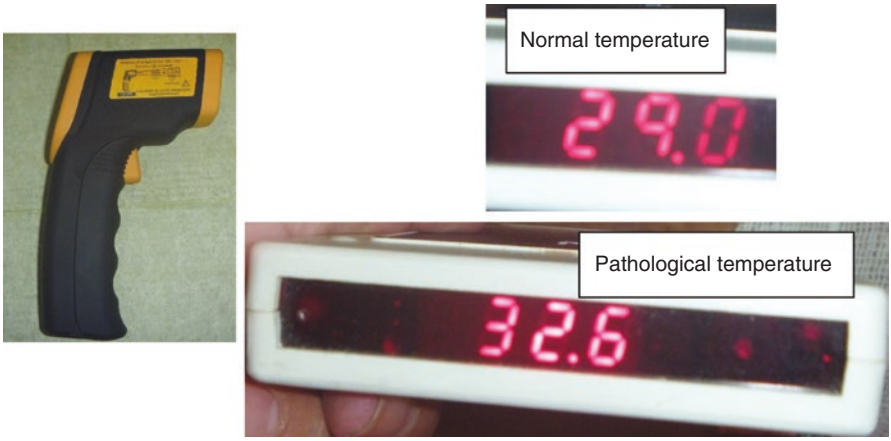

Fig. 14.3 Accessible thermometer. One or more degree centigrade difference with another zone, indicates a high risk of ulceration

Foot Temperature Control

Sensitive Score

There are several schemes; we recommend the evaluation of ten places of the foot (first, third and fifth finger, first, third and fifth metatarsal head, inner edge of foot, external edge foot, heel and back foot. Score and add both feet: 0–3 normal, 3–6 slight alteration, 6–9 moderate alteration and more than 9, lack of protective sensitivity.

This facilitates the production of internal or external traumas, which are warned belatedly by the patient. The diabetic patients can have a bone fracture and continue walking on the affected leg.

- Cold heat sensitivity: Normal 0, pathological 1.
- Pain sensitivity: Normal 0, pathological 1.
- Vibrational sensitivity: Normal 1, pathological 1.
- Tactile sensitivity: Normal 0, altered in some places 1, further altered in most places 2.
- Aquilian reflex: normal 0, decreased 1, absent 2 points.

The Latin American Association of Diabetes states that sensitivity tests can be done in three places (first, third and fifth finger). If there are diagnostic doubts, it is better to do the score in the ten places mentioned.

The autonomics tests and sensory and motor nerve conduction speed with electromyogram are not routine studies in the evaluation of patients, and should be done in case of diagnostic doubts.

Instrumental Studies

- Pallesthesia quantitative measuring devices: the biothesiometer is an objective method of assessment, a threshold lower than 15 mV is normal, and it is pathological when it exceeds 25 mV
- Infrared thermometry. The normal temperature of the skin is between 28 and 31°C. Less than 26° indicates ischemia and there is inflammation when it exceeds 31.5°C. In case of neuropathy, the temperature is not uniform and there may be an elevation of 1–2°C in an area with bone deformation and hyperkeratosis. As mentioned above, this indicates oxidative stress, and a high risk of ulceration.

In Charcot's foot, the temperature is not uniform and there are 2°C more in the affected area. It is an early sign of activity; and the coalescence stage when the temperature decreases.

Electromyogram

It should be done with motor and sensory nerve conduction velocity. Must evaluate the proximal and distal latency, the evoked potentials and the spontaneous activity.

The electromyogram shows fibrillation, loss of motor units, potential for reinnervation and slower conduction velocity. The major compromise is in the distal muscles, with predominance of the lower limbs.

Changes in conduction velocity may reflect underlying pathological structural changes of axons, such as demyelination and atrophy.

These findings suggest a diffuse abnormality of the peripheral nerves function, being able to show subclinical damage in asymptomatic patients.

Neuropathy Classification

According to the findings the patients can be classified:

- **Without clinic neuropathy**: without symptoms, without motor or autonomics signs and a sensitive score lower than 3, being doubtful between 3 and 6. If there are no pathologies, foot must be controlled annually.
- **With clinical neuropathy**: symptoms, other signs and a sensitive score greater than 6. Optimal control of Diabetes, etiological treatment of neuropathy (gamma linoleic or alpha lipoic acid, vitamin E, magnesium), orthosis if indicated and protector shoes should be done. Extreme care of the foot and control of risk factors. Patients should be monitored every 3 months.
- **With severe neuropathy**: With rest pain, ulcers, deformations, Charcot's foot. Treatment must include, local treatment, insoles if necessary, correction of eventual deformations, etc. It is important to consult a team specialized in diabetic foot. Control monthly and after the improvement every 3 months.

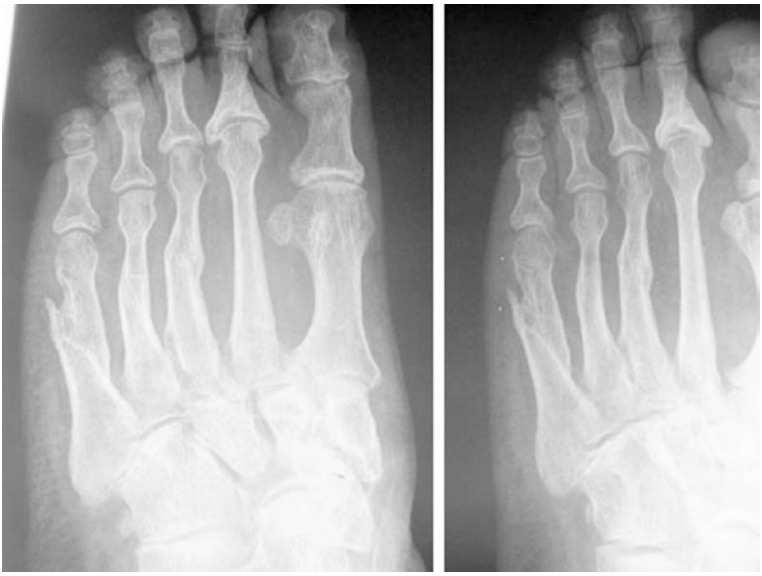

Fig. 14.4 Radiography of a patient with consolidated fractures, in the stage of remission

Charcot's Foot

Is the chronic affection of bones, joints and soft tissues, produced by peripheral and autonomic neuropathy, characterized by an inflammatory process in the initial stages, with the development of bone loss, joint dislocation and fixed deformations.

It is often not diagnosed, with a prevalence of 0.4–13%, increasing to 29%, using radiography, and higher prevalence with nuclear resonance (Fig. 14.4).

Physiopathology

Several factors contribute to its determinism, considering two theories: neurotraumatic and neuro-vascular due to sympathectomy.

It is currently considered that in susceptible individuals with peripheral neuropathy, an inflammatory process is developed, by activation of the polypeptide receptor activator of nuclear factor kappa B ligand (RANKL), which causes maturation of the osteoclasts. At the same time the nuclear factor kb (NF-kB) stimulates osteoprotegerin production of osteoblasts. The repetitive trauma with lack of pain induces the production of inflammatory cytokines and osteoclasts, causing local osteolysis.

Another possible cause is the decreased secretion of the calcitonin gene, in addition to protein glycosylation, the oxidative stress and the oxidized lipids.

Diagnosis

In the presence of neuropathy, especially autonomic, the clinical presentation with edema, erythema and an increase of 2 °C in the area makes the diagnosis of Charcot's foot.

The radiograph has little sensitivity and its alteration is delayed, being more specific the nuclear magnetic resonance.

Both radiography and nuclear magnetic resonance have different sensitivity according to the stage of development, coalescence or reconstruction.

Treatment

The treatment is conservative, being emphasized the immobilization, the gold standard of the treatment is the total contact cast. Initially it must be changed within 3–7 days, and then every 15 days. Once in the stage of reconstruction, according to the present deformations it must be indicated supportive insoles to correct structural misalignment and orthopedic shoes. In many cases, the patellar dislocation discharge boot is necessary.

Another not so effective possibility is the CROW (Walker).

Bisphosphonates and calcitonin may be indicated to improve osteopenia, and in cases of irreducible deformations, with recurrent ulcers, corrective surgeries must be considered.

Treatment of Neuropathy

The first and fundamental treatment is the improvement of diabetes control, nutrition status and cardiovascular risk factors.

In addition to these measures, normalization of altered metabolic pathways (sorbitol pathway, oxidative stress, proteins glycation, etc.) should be achieved and avoid its consequences. There is evidence of the beneficial effect of lipoic acid.

Haemorheological changes must be improved, avoiding anoxia.

Indication of a healthy diet, with antioxidants, and must be considered the supplementation of taurine, essential fatty acids and myoinositol.

In case of painful neuropathy, the nocturnal pain must be treated to improve quality of life. Can be used antidepressants, mexitilen, pregabalin, opioids, alone or in combination. In specific cases, electrostimulation or nerve blockade are indicated.

Protective and corrective measures are fundamental in order to avoid ulcers.

Early detection is crucial for early immobilization (total contact cast) to avoid deformation.

Must be taken into account that patients who have had a standing ulcer have 50–70% chance of a new ulcer, dying the 50% of the patients in 5 years.

The use of arginine, and nerve growth factors has been postulated.

In case of ulcers, the area must be protected, correct the structural misalignment and the excessive contact, debride all necrotic, hyperkeratotic and devitalized tissues. If there is infection, a tissue sample cultivation must be done with antibiogram, taking the sample with scraping, curettage or biopsy. Gram staining and a colony count should be done, and according to the result, a suitable antibiotic plan, which should be assessed according to the clinical response.

If there is associated osteomyelitis, it is essential to discharge, remove all necrotic bone and establish and adequate antibiotics treatment. According on the depth of the ulcer and the progression, larger debridement, or partial amputations, may arise.

Vascular Foot

Vascular alteration of the lower limbs is less frequent than neuropathy, but is more serious because of the risk of gangrene, with more immediate and long-term mortality, with healing delay, and a higher percentage of amputations.

Approximately 30% of diabetic patients develop peripheral arteriopathy.

In the NHANES study of 1999–2004, was found a prevalence of 5.9% of peripheral vasculopathy (valued with ankle/arm indexes less than 0.9).

Non-diabetics have more affected the aortoiliac or femoral territory. In diabetes is more prevalent the distal affectation or a multiple compromise.

Vasculopathy appears at earlier ages, with several territories affected, and in each area more diseased arteries, with pathology in collaterals, and coronary disease; not being difference between sexes.

20% of people aged over 65 have evidence of peripheral vasculopathy, and only a quarter have symptoms. In the presence of peripheral vascular disease, it is necessary to search the affectation of other territories, especially the heart.

As already mentioned, the international consensus on diabetic foot advises that all diabetics undertake an annually comprehensive foot examination, including vascular evaluation at rest and after exercise. Perform the clinical examination, and the ankle- brachial pressure index. Interrogate on intermittent claudication and resting pain.

Peripheral vasculopathy is more frequent in poorly controlled diabetics with higher cardiovascular risk factors, with greater morbimortality, even if it is asymptomatic. It also has higher mortality than several types of cancer, which highlights its early search and appropriate treatment.

In the BARI 2D study, it was found that every 1% increase in glycosylated hemoglobin it was associated with the 21% increase of peripheral vasculopathy risk at a 5-year follow-up. Diabetic patients have a 50% higher risk of having amputations when they have peripheral vasculopathy.

The prevention of peripheral vasculopathy would be oriented to the intensive treatment of cardiovascular risk factors, and the vascular assessment of the patient should be performed in an integral manner.

Similarly in diabetics as in non-diabetics, the value of vascular risk factors for the development and recurrence of peripheral vasculopathy has been shown; classical risk factors: smoking, dyslipemias, hypertension, obesity, sedentarism, hyperuricemia, microalbuminuria and duration of diabetes; chronic inflammation and oxidative stress, has now been highlighted.

Other factors are associated with hyperglycemia such as: (1) typical diabetic metabolic modifications; (2) variation of insulin levels, (3) microangiopathy and microcirculation disorders; (4) hormonal modifications; and (5) metabolic alterations of the arterial wall, with greater platelet adhesiveness and aggregation, with thrombogenesis increase.

There is a greater susceptibility to complications in certain individuals, relating to family history and various metabolic pathways, especially the decline of antioxidant enzymes.

The natural evolution of 10-year vasculopathy is that of 20 patients, 12 die of vascular events in the heart, brain or aorta, four remain stable, one will need amputation and three vascular surgery.

The early detection of peripheral vasculopathy is essential, to control cardiovascular risk factors and the diabetes, by maximizing the protective and corrective measures of ulcers.

Disease progression, correlates with poor diabetes control, LDL cholesterol elevation, oxidative stress and smoking. This is more atherogenic in diabetics than in the general population, in addition to damage of the endothelium, increases insulin resistance and delays the healing of ulcers.

Classification of Peripheral Vasculopathy

WHO indicates that the peripheral vascular disease presents these stages:

(0) Normal. (1) Asymptomatic foot, but lesion is demonstrated with special studies. (2) Intermittent claudication. (3) Resting pain of ischemic origin. (4) Necrosis or gangrene.

It is common the description of the intermittent claudication pain. It is a pain that appears with the walk and disappears when you suspend it, calming the pain immediately when stopping walking, not needing to lie down or raise the leg.

The most common location is at calf level, but may appear in other areas (buttock, thigh, feet) indicating the area of stenosis. Aortoiliac lesions affect the buttocks or thigh. The femoris in the calf, and those of the tibial peroneal trunk are expressed in the ankle and foot.

Resting pain indicates greater severity of ischemia and a greater need for surgical help. It appears when the patient adopts the horizontal position, relieving himself in the seated position. This is transitory, as it can cause edema, which aggravates ischemia.

Resting pain worsens with cold or walking; being important to differentiate with the resting pain of neuritic origin. For the differential diagnosis, clinical facts must

be taken into account, complementing with the doppler flow study. An ankle/arm index greater than 0.50 or an ankle absolute pressure of 90 mmHg is indicative that such pain cannot be due to ischemia.

In any foot lesion, should be determined, if there is critical ischemia with clinical and ankle/arm or finger/arm indices, the patient must be evaluated by a vascular surgery team.

Other Classification

Some authors consider that the WHO classification offers doubts in the clinical management of the patients, so we classify the peripheral vasculopathy in: (a) Normal. (b) Asymptomatic vasculopathy. (c) Clinical vasculopathy (symptoms or signs). (d) Severe ischemia.

It is considered that there is severe ischemia when there is resting pain, ulcers of more than 10 weeks of evolution without improvement with an adequate treatment, necrosis that progresses, ankle/arm index lower than 0.5 and finger/arm index less than 0.3, and transcutaneous oxygen tension less than 30 mmHg.

Valuation of Ischemia

- Symptoms: presence of cold feet and legs, heaviness, intermittent claudication, resting pain mainly at night.
- Skin examination: The skin is cold, shiny, and atrophic, with diminished nail growth, lack of hairs, especially on the fingers (the presence of hairs indicates adequate blood flow). There is also atrophy and loss of subcutaneous fat in the dorsum of the feet.
- The temperature is decreased, less than 26°C with the infrared thermometer, in a room with an adequate temperature.
- Pulse, murmurs, venous filling and capillary refill should be examined at rest and if there are doubts (especially in type 2 diabetic patients, elderly and long-term diabetic patients) the examination must be performed after an exercise or an hyperemia test.
- Take the ankle/arm index, and finger/arm index, with a bidirectional doppler. Pressure controls can also be performed on other areas of the leg and thigh (see below).
- In suspected cases of severe ischemia, pulse waves can be seen, and in more complex equipment, the plethysmographic waves and the evaluation of the transcutaneous oxygen tension.

Ischemia Degrees

According to the findings we classified the patients in:

- **No significant ischemia**: Without signs or symptoms, the ankle /arm index is higher than 0.9, the finger/arm index higher than 0.5 and the transcutaneous

oxygen tension over 60 mmHg. Improve diabetes control, foot control (insoles, hyperkeratosis, nails care, fungal infections, etc.) and the correction of cardiovascular risk factors is indicated; patients should be monitored annually.

- **Clinical ischemia**: There is intermittent claudication or some pathological finding (pulses, murmurs, etc.). The ankle/arm index is between 0.9 and 0.5, the finger/arm index between 0.5 and 0.3, and the oxygen tension between 30 and 60 mmHg. In addition to the measures to be taken in the previous category, it is necessary to indicate the vasculopathy treatment (hygienic dietary and vasoactive measures), extreme foot care and control it every 3 months.
- **With severe ischemia**: have resting pain, especially nocturnal, necrosis and gangrene. The ankle/arm and finger/arm indices, and the oxygen tension are lower than the previous category. They must be studied to evaluate whether angioplasty and/or bypass can be performed. If after the procedure there is remaining ischemia, the intravenous prostaglandins treatment is a possibility, and if there is no improvement, consider amputation. Diabetes care, cardiovascular risk factors and foot care must be extreme to avoid recurrence and the involvement of another member; as well as death due to a vascular episode (cardiac, cerebral or abdominal).

Special Studies

Noninvasive

Doppler Indices

Although there are no symptoms or signs for all diabetics over 35 years of age or with more than 10 years of evolution of the disease, annual determination of ankle and arm pressure must be done with the bidirectional doppler. It is done at rest and if there are doubts, after exercise or the test of hyperemia.

There is autonomic neuropathy in many diabetics, which leads to false positive results due to the opening of the arteriovenous shunt and the calcification of the interosseous arteries. In that case it is more valuable to do the finger/arm index.

Ankle/Arm and Finger/Arm Indices

The pedis and tibial pressure is taken, and the highest value is divided on the arm highest value. A suitable gel should be used, and place the sensor with an adequate pressure and at a 45° angle. The doppler first signal appearance indicates the pressure which must be registered and calculate the ratio with the arm values.

The handle of the gauge should be two centimeters above the malleolus in the leg and sensors in the pedis, or the tibial arteries. At the arm 2 cm above the arm bend and the sensor is placed in the radial artery.

The **index values** are:

- Ankle/arm (brachial) index:
  - Normal: 0.91–1.30
  - Clinical ischemia: 0.5–0.9

  - Severe ischemia: <0.5
  - False positive (autonomic neuropathy, interosseous calcification) over 1.30.
- Finger/arm index:
  - Normal: >0.5
  - Clinical ischemia: 0.3–0.5.
  - Severe ischemia: <0.3.

Sometimes **absolute pressures** must be considered:

- In order to cure an injury to the foot there must be at least an ankle pressure over 50 mmHg.
- In order to cure an injury to the finger, there must be a finger pressure over 30 mmHg.
- An ankle pressure of 90 mmHg discards an ischemic resting pain and suggests a good vascularization for an infrapatellar amputation.
- In case of uncontrolled arterial hypertension, with high arm values, is convenient to consider the absolute pressure at the ankle to evaluate the perfusion.
- Pressure can be taken on the upper and lower thigh and on the calf, determining the segmental pressures, thus suggesting the occlusion site a drop of more than 30 mm of Hg between each sector.
- A thigh index of 1.20 or more indicates the absence of a significant aorto-iliac obstruction. An index of 0.80–1.20 suggests an important but not complete aorto-iliac stenosis and an index of less than 0.80 indicates a probable complete occlusion.
- At the calf level the normal index is from 1 to 1.30; a value from 0.65 to 1 suggests incomplete occlusion of the distal femoral. A complete obstruction at this level is found with an index <0.65.
- At the ankle the normal index is from 1 to 1.30. Incomplete obstruction: 0.5–1, and a complete obstruction of the tibial peroneal trunk or its branches: <0.5.
- As mentioned in pulses and murmurs, the flow study by doppler can show normal values at rest, so it must be done after an exercise or the hyperemia test. The normal result of these test is the increase of the pulse and lower limb pressures. In the case of peripheral arteriopathy, four types of results can be found:
  - Type I: there is a very slight pressure elevation that produces the disappearance of diastolic sound.
  - Type II: the pressure falls, almost 50% of the initial value, but with recovery in 5 min.
  - Type III: there is a more than 50% decrease in pressure values recovering the initial values after 10 min.
  - Type IV: systolic blood pressure drops to almost zero, with a very slow recovery to baseline values (more than 15 min).

Pulse Wave. Plethysmography

The determination of the pulse wave can recognize the arterial stiffness. The normal wave is three phasic, being altered according rigidity and obstruction in biphasic, monophasic and disappearing.

The pneumoplethysmography evaluates the volume changes of the limb according to the systole and diastole; and photoplethysmography studies the change in volume of blood passing through the capillaries.

In each of them are four types of waves: normal, without the dicrotic sign, flattened and lack of wave.

Transcutaneous Oxygen Tension (TCOM)

Is a non-invasive method of measuring the oxygen level of the tissue below the skin. Since oxygen is carried by the blood, TCOM can be used as an indirect measure of blood flow to the tissue. Since blood flow is important for wound healing, TCOM is often used to gauge the ability of tissue to effectively heal.

The transcutaneous oxygen tension may decrease because of the less saturation due to respiratory or cardiac problems, by peripheral vasculopathy, by tissue edema, by microangiopathy, peripheral neuropathy with autonomic alteration with opening of arteriovenous shunts, infections with increased oxygen demand with oxidative stress and local compression (osseous enhancements).

The normal transcutaneous oxygen tension is >60 mmHg, indicating ischemia when it is <30 mmHg. To be healed an amputation there must be a minimum tension in the cutting area of 20 mmHg.

Keep in mind that when an amputation is done, it has to be an adequate flow in the distal area, being useful this study.

In the area to be amputated infection must not exist and we should think about the functionality, the orthosis and avoiding new areas of repeated friction or pressure. The younger the patient's the more the function should be valued.

To amputate it must be extreme the control of Diabetes and the nutritional status, the early rehabilitation of the patient and the prevention of the lesion recurrence and the involvement of the contralateral foot are the clue of a successful treatment.

Contrast Studies

When there is a severe ischemia, in danger of losing the leg and/or life, it must be considered the vascular permeability and the possibility to perform arterial surgery. This requires a contrast arteriography, digital angiography or magnetic resonance angiography.

In order to make an arteriography the contrast substance can be introduced into the artery through the direct injection due to vessel puncture, but it is more convenient introducing a catheter.

The study of the vessels can be done perfectly with this procedure, having very little morbimortality, probably related to the team's experience than the procedure itself.

One advantage of catheterization methods are that allows the study of other vascular territories simultaneously, especially cardiac, carotid arteries, brain and abdomen.

It is essential the visualization of the distal vessels. When there is a chronic arteriopathy it should always be seen contrast substance in the main trunks or collaterals, but the absolute absence of contrast in the leg and/or foot only indicates that insufficient quantity was injected or that the exposure of the radiography was

untimely. It should always try to include the foot in the studio, but when it isn't seen permeable vessels useful for the reconstruction in the leg it should be required the presence of contrast in it, either in the collaterals or demonstrating a pedis, posterior tibial or plantar that allows a bypass.

The arterioscopy allows observing the permeability, the vessel diameter and also a stent application.

The magnetic resonance image (MRI), with or without contrast offers a better visualization of the arteries, with less contrast and rays exposure.

Treatment of Vasculopathy

It is also important the adequate control of Diabetes and the cardiovascular risk factors.

Careful must be taken with the use of inhibitors of sodium glucose transport (SGLT2), which may increase the risk of amputations.

It was observed the preventive and curative effect of a diet rich in vegetables and fruits, being detrimental exaggerated red and processed meat consumption.

Very useful is the realization of physical activity. Although patients have intermittent claudication, it must be emphasized walking. It has been observed that tolerance to walk becomes better with simple stretching exercises of the calf. This improves vascular endothelial function, which improves the ability to walk.

Walk works best when exercise lasts a minimum of 30 min, it may be staggered due to pain.

The reduction of the oxygen tension of the muscle or the metabolic alterations on it due to vasculopathy is the angiogenic stimulus.

Training increases flow redistribution from inactive to active muscles; with improvement or reversal of haemorheological disorders.

It is better to include the patients on a controlled exercise program. There is no efficacy data showing the message "go home and walk," which is the most common prescription.

After 4 months of an exercise plan, intermittent claudication (IC) patients require less oxygen at a same physical exigency, improving walking distance with a better quality of life.

To control the walk, pedometers can be used, with a more adherence to treatment. A pedometer that counts steps and measure the whole activity can be used by patients (Fig. 14.5).

Besides health and diet treatment, which should never be missed, it can be indicated an antioxidant and cilostazol treatment in 200 mg per day doses.

In cases of severe ischemia, it should be sent to vascular surgery, to see the possibility of surgery (bypass and/or angioplasty); and if remains residual ischemia or vascular surgery cannot be performed, consider prior to amputation the possibility of treatment with series of intravenous or intra-arterial injections of prostaglandins.

It must be highlighted that the peripheral vascular disease is a marker of systemic atherosclerosis. Therefore, the prevention is oriented to the intensive treatment of the cardiovascular risk factors.

Fig. 14.5 Pedometer

The vascular patient assessment must be comprehensive, ruling out the involvement of coronary and cerebral arteries.

Bibliography

1. Aragon-Sanchez J, et al. Conservative surgery of diabetic for foot osteomyelitis: how can I operate on this patient without amputations. Int J Low Extrem Wounds. 2015;14(2):108–31.
2. Boulton AJM. The pathway to foot ulceration in diabetes. Med Clin N Am. 2013;97:775.
3. Forsythe RO, et al. Peripheral arterial disease and revascularization of the diabetic foot. Diabetes Obes Metab. 2015;17(5):435–44.
4. Javed S, et al. Treatment of painful diabetic neuropathy. Ther Adv Chronic Dis. 2015;6:15.
5. Gmae FE, et al. Effectiveness of interventions to enhance healing of chronic ulcers of the foot in diabetes: a systematic review. Diabetes Metab Res Rev. 2016;32(supl 1):154.
6. Petrova N, et al. Acute charcot neuro/osteoarthropathy. Diabetes Metab Res Rev. 2016;32(suppl 1):281.
7. Ponirakis G, et al. Nerve check for the detection of sensory loss and neuropathic pain in diabetes. Diabetes Technol Ther. 2016;18(12):800–5.
8. Mascarenhas J, et al. Pathogenesis and medical management of diabetic charcot neuroarthropathy. Med Clin N Am. 2013;97:857.
9. Lipsky B. Treating diabetic foot osteomyelitis primarily with surgery or antibiotics: have we answered the questions. Diabetes Care. 2014;37:593.
10. Nicole Lou for PAD, Walking tolerance boosted with simple calf stretching. American Heart Association; May 5, 2017. www.medpagetoday.com/meetingcoverage/additionalmeetings/65055
11. FDA adds boxed warning to canagliflozin for amputation risk. www.fda.gov/mewatch/report.
12. Armstrong D, Lavery LA. Clinical care of the diabetic foot. 3rd ed. Los Angeles: American Diabetes Associations; 2005.
13. Ogilvie R, et al. Dietary intake and peripheral arterial disease incidence in middle aged adults: the Atherosclerosis Risk in Communities (ARIC) Study. Am J Clin Nutr. 2017;105:651–9.
14. Heffron S, et al. Greater frequency of fruit and vegetable consumption is associated with lower prevalence of peripheral artery disease. Arterioscler Thromb Vasc Biol. 2017;37(6):1234–40. https://doi.org/10.1161/ATVBAHA.116.308474.
15. Jeffcotate WJ. Charcot foot syndrome. Diabet Med. 2015;32:760.