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Martorell's Ulcer: Diagnostic and Therapeutic Challenge

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Key Words
Martorell's ulcer · Hypertensive ulcer · Hypertension · Ischemia

Abstract
Martorell's ulcer is an uncommon ischemic and extremely painful lesion located in the distal portion of the lower limb, resulting from severe systemic and poorly controlled hypertension. It is common in women between 50 and 70 years of age. The diagnosis is clinical and mostly belated, following exclusion of other causes. The response to treatment takes time and is unsatisfactory. A combination of several drugs associated with surgery may be required for wound healing. The authors present a case of Martorell's hypertensive ulcer, with emphasis on the diagnostic and therapeutic difficulties.

Introduction

The most common causes of ulcers in the lower limbs are arterial insufficiency and venous stasis [1]. Martorell's hypertensive ulcer is an ischemic lesion of the tissue caused by obstruction of the small arterioles of the medial artery.

Haxthausen [2], in 1940, for the first time reported the association of ischemic ulcers as a consequence of arterial hypertension complications, and Martorell, five years later, published four cases correlating systemic arterial hypertension, arteriolar lesion and ulcer for-

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mation [3, 4]. These findings were supported by Hines and Farber from the Mayo Clinic [5]. The clinical criteria described by Martorell for the diagnosis of hypertensive ulcer were: location on the inner side of the distal third of the lower limbs; diastolic arterial hypertension in the lower limbs; 'hyperpulsatility' of the arteries of the lower limbs, absence of arterial calcifications; absence of chronic venous insufficiency; symmetrical lesions or residual hyperpigmentation of previous ulceration in the inner side of the inferior limb; increased pain in the horizontal position, with greater prevalence in women [3, 4, 6].

It is believed that the pathogenesis of hypertensive ulcer is related to local factors of the microvasculature and not to involvement of large vessels, as observed in the most common peripheral vascular diseases. Trauma can be a triggering factor, although half of the ulcers arise spontaneously [7]. From the pathophysiological point of view, the dermal arterioles with uncontrolled systemic arterial hypertension present arteriosclerotic changes, such as hyperplasia of the core elements of the middle layer cells in the vessels with thickening of the elastic lamina. Such a process, called hyalinosis [7–9], leads to an increase in the dermal arterioles lumen diameter [6, 10]. Luminal stenosis produces increased local vascular resistance and reduced skin perfusion pressure [11].

Hypertensive ulcers are more common in women between 50 and 70 years of age, often symmetrical and located in the distal inner side of the lower limbs. Pain in greater intensity than in ulcers of other etiologies, referred to as being disproportionate to the size of the lesion, is the most relevant symptom. Inefficiency of ordinary analgesics is common [1, 12]. Classically, the ulcer has variable depth, necrotic base and violaceous edges. The presence of satellite lesions and irregular edges are also features that suggest additional cutaneous ischemia. The diagnosis is usually belated and requires the exclusion of other causes. The healing process is slow and the response to treatment is poor [13].

Proposed treatments are antihypertensive therapy, especially with drugs that reduce vasoconstriction, such as calcium channel blockers and inhibitors of angiotensin-converting enzyme. Pacifico et al. [14] in 2011 suggested the use of prostaglandin E (PGE_1).

Case Report

A black woman, a 40-year-old housewife born in Rio de Janeiro, was referred for ulcers on the toes for 2 months. She reported intense pain in the lesions with a progressive increase in size. At the primary care clinic, she was treated with benzathine, penicillin and ciprofloxacin in addition to local care, however without presenting significant improvement. She had systemic arterial hypertension upon irregular monitoring, without adequate blood pressure control. She was taking digoxin 0.25 mg/day, carvedilol 3.125 mg/day, captopril 100 mg/day, furosemide 80 mg/day, spironolactone 50 mg/day and simvastatin 20 mg/day. Both parents had suffered from hypertension and had died from encephalic strokes.

At the initial physical examination, the patient did not present fever, but was hypertensive (blood pressure 240/120 mm Hg), with mild edema of the lower limbs. There were two irregular edge ulcers and necrotic bases in the lower limbs located in the 3rd and 4th right and left toes (fig. 1, fig. 2, fig. 3). She did not show any signs of inflammation or wound secretion in the region. Peripheral arterial pulses of the posterior tibial and posterior tibial arteries were palpable bilaterally. Radiography of the feet excluded osteomyelitis. Eye fundus examination showed Keith-Wagener-Baker hypertensive retinopathy grade III (arteriolar narrowing, with silver wire arteries, increased vascular tortuosity with A/V pathological groups). Transthoracic echocardiography demonstrated parietal concentric hypertrophy of the left ventricle with diastolic dysfunction grade I and left atrial enlargement. Doppler study

of the carotid and vertebral arteries only revealed mild bilateral intimal thickening without atheromatous plaques. In echo color Doppler of the lower limbs, arterial thickening and irregularities were detected without parietal diffuse atherosclerosis and with normal bilateral flow.

Histopathological examination of the biopsy of the ulcer in the left leg showed a nonspecific chronic inflammation process with ulceration, consistent with the diagnosis of Martorell's ulcer. The clinical picture and laboratory tests showed that the ulcers had a hypertensive etiology. The patient was admitted for a proper treatment for systemic arterial hypertension and surgical debridement of the ulcer (fig. 4). The local dressings were made with petrolatum and the ulcer progressed with healing.

Discussion

Martorell's hypertensive ulcer is an unusual injury and, although cases have been seen in men, it is more common in women. It is frequently symmetrical and located in the distal third and anterolateral surfaces of the lower limbs [15].

The most common histopathological findings are thickening of the elastic lamina, proliferation of the intimal layer and hyperplasia of the middle layer with hyalinization resulting in luminal narrowing [16]. Studies have shown that the presence of intimal hyperplasia is the primary event for ulcer formation. The shear stress, caused by persistently high blood pressure levels, stimulates endothelial remodeling with thickening of the wall vessel, luminal narrowing and consequent obstruction of the blood flow. It was also admitted that there is a loss of the offsetting vasodilation physiological reflex of the distal arterioles in the obstructed region, further compromising the tissue perfusion. When the perfusion reaches critical levels, cutaneous ischemia occurs, with epidermal necrosis and ulcer formation [11].

Total ulcer healing requires the combination of various therapies. Reduction of blood pressure levels is critical to the success of the treatment and not only prevents increased vascular damage, but improves blood supply through local vasodilation. Nonselective beta-blockers are contraindicated for reducing cardiac output, worsening the skin perfusion pressure. The drugs of choice are calcium channel blockers and angiotensin-converting enzyme inhibitors, and all patients must be anticoagulated with heparin or coumarin [17].

Surgical treatments involve the debridement of devitalized tissue with second intention closure or skin grafting and lumbar sympathectomy. Surgical closure of lesions >4 cm^2 is recommended [18]. Lumbar sympathectomy aims at promoting vasodilation, improving perfusion of the affected limb and controlling pain. The result is variable, from complete recovery to little impact on healing [19].

Mild to moderate pain can be managed by the use of ordinary painkillers such as nonsteroidal anti-inflammatories, and in severe cases opioids are the drugs of choice. The use of spinal cord stimulation therapy, with good results in reduction of pain and healing of ulcers, has been mentioned and consists of the use of small electrodes that stimulate certain points of the spinal cord, influencing the transmission of the pain signals to the brain [20].

The type of therapy called hyperbaric oxygen therapy consists in the supply of pure oxygen in a pressurized environment above atmospheric pressure. Its action mechanism is not entirely understood, but it can be used as an adjunct in the treatment of chronic wounds, although the level of evidence in patients with Martorell's ulcer is low [21].

Behavioral measures, such as avoiding trauma, stopping to smoke and wearing compressive stockings (25 and 30 mm Hg) must be encouraged [22].

Conclusion

In the case presented, the patient featured four out of the seven criteria set up by Martorell. Complementary tests allowed the exclusion of other etiologies. The lower limbs Doppler excluded chronic venous insufficiency and coagulopathy, while the histopathological examination did not show any signs of cutaneous vasculitis. The observation of lesions secondary to hypertension in other target organs also contributed to the diagnostic conclusion. The patient underwent daily dressing while hospitalized. After the optimization of oral antihypertensive medication doses, obtaining acceptable pressure levels allowed the lesion to heal.

Hypertensive ulcer should always be considered in the differential diagnosis of painful lesions of the lower limbs.

Statement of Ethics

Our patient gave her written authorization for the publication of her case and the authors followed all ethical guidelines.

Disclosure Statement

The authors declare no conflict of interest.

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Fig. 1. Ulcer with irregular borders and dry necrosis at the base of the 4th left toe.

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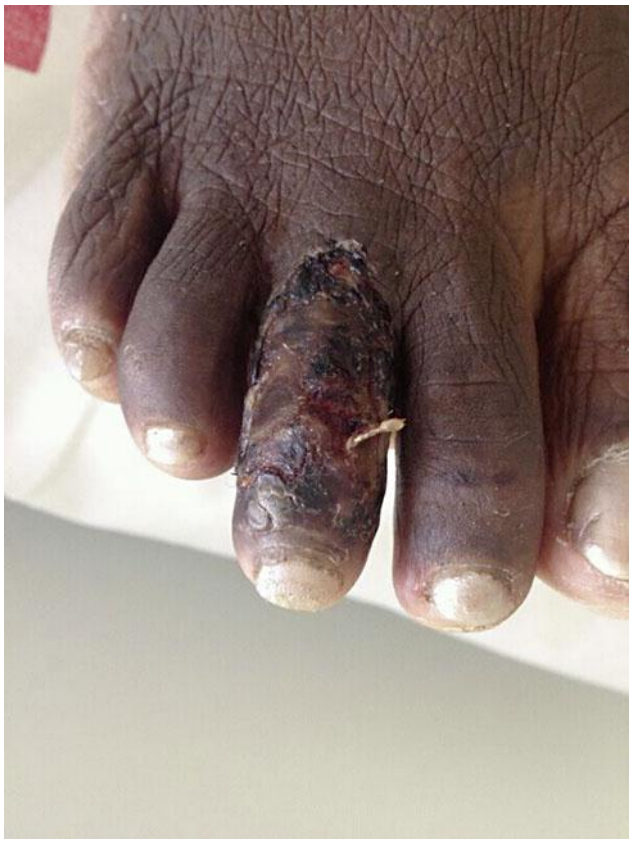

Fig. 2. Ulcer involving all of the 3rd right toe with dry necrosis.

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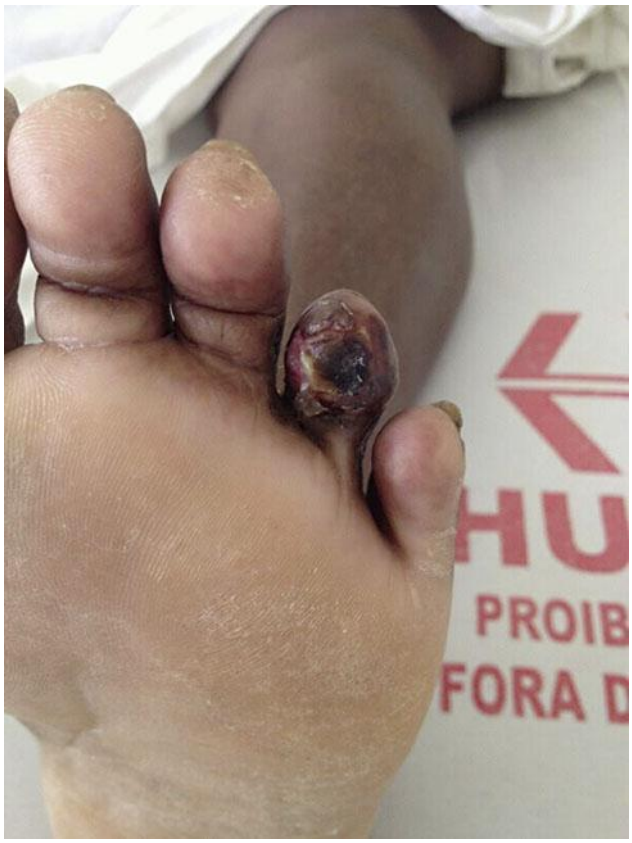

Fig. 3. Detail of the extension of the lesion.

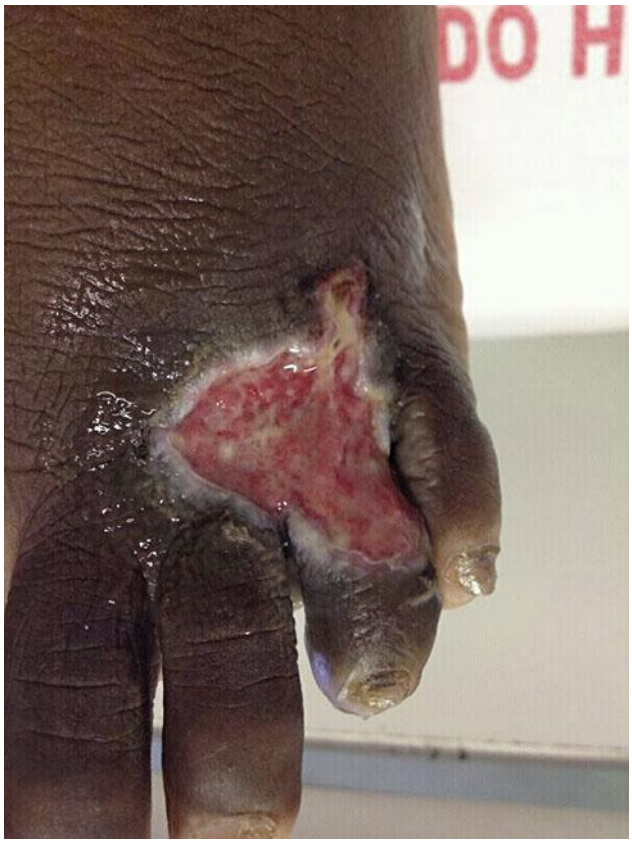

Fig. 4. After surgical debridement.