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Case Report
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Difficulty in the Diagnosis of the Acute Charcot Foot

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Abstract

Charcot's foot or Charcot's neuroarthropathy consists of a progressive deterioration of the bones and joints, mainly the ankle and foot, in patients with severe neuropathy. Diabetes is currently the most frequent cause of neuropathic arthropathy, although it can occur in other neurological processes. The greatest difficulty is that it can be confused with other processes, such as cellulitis, osteomyelitis and Deep Vein Thrombosis (DVT), among others, delaying the diagnosis and appropriate treatment, which determines the development of deformities and important complications. We present a clinical case of acute Charcot foot.

Introduction

Charcot's foot or Charcot's neuroarthropathy consists of a progressive deterioration of the bones and joints, mainly the ankle and foot, in patients with severe neuropathy [1]. Diabetes is currently the most frequent cause of neuropathic arthropathy, although it can occur in other neurological processes such as syringomyelia, leprosy, heavy metal deposition or peripheral nerve trauma. The prevalence in the diabetic population is from 0.8% to 7.5%, and the most frequently affected joint is that of Lisfranc [2,3,4]. The greatest difficulty is that it can be confused with other processes, such as cellulitis, osteomyelitis and Deep Vein Thrombosis (DVT), among others, delaying the diagnosis and appropriate treatment, which determines the development of deformities and important complications.

Case Report

A 65-year-old male with a history of hypertension and diabetes mellitus of 20 years of evolution in insulin treatment, attended by pain-free inflammation of the right foot for two months. Seen in the emergency room a month ago, he was diagnosed with cellulitis and was prescribed oral antibiotic (amoxicillin clavulanic 500 mg/vo/8hours/7 days). Physical examination revealed edematous, hyperemic, hot, deformed and unstable foot, together with a plantar ulcer and signs of severe distal neuropathy type alteration of the sensitivity detected with Semmes-Weinstein monofilament (Figure 1 and 2). The analytical practice showed a normocytic anemia, leukocytes 9,000/mm^3, neutrophils 75.2% and a blood glucose level of 254 mg/dl. The patient was a febrile. X-ray of the foot reported bone resorption and dislocation of tarsal bones. The venous echo-Doppler performed ruled out the DVT of the right lower limb. Hospitalization of the patient was decided and a treatment of absolute rest with the high extremity with Brown splint, debridement of slough of the plantar ulcer, sampling for culture and intravenous empirical antibiotic therapy with ertapenem 1 gr/iv/24hours. The result of the culture was positive for Staphylococcus aureus. The MRI of bone and soft parts of the right foot reported flattening of the plantar vault, erosions and inflammatory changes in the midfoot and ankle, plus degenerative changes in the talar dome (Figure 3). At two weeks of hospital stay, when the edema of the foot decreased, the patient was placed an orthosis of total discharge in the lower right limb to be able to walk without supporting the foot. Given the favorable evolution, the patient was discharged after six weeks of hospitalization, being currently controlled in outpatient clinics and referred to Orthopedic Surgery to assess the treatment of instability and residual foot deformity.

Discussion

From the etiopathogenic point of view, there are two hypotheses, that of sensory neuropathy and the theory of autonomic neuropathy, which differ from the initial cause, but both agree that peripheral neuropathy together with repeated microtraumastisms produced during walking or minor or major trauma sometimes unnoticed, trigger mechanical and vascular alterations that lead to changes in the bone structure (resorption, hypertrophy and finally deformity) [1-4]. This determines an anomalous distribution of the load that favors the appearance of ulcers, fractures and calluses.

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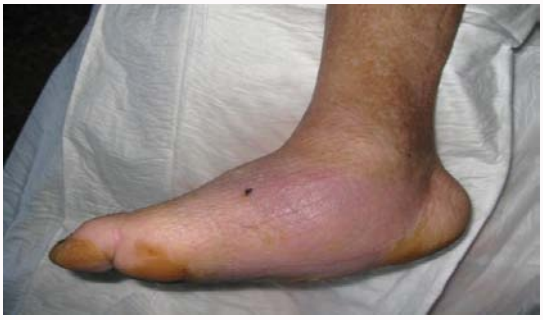

Figure 1: Acute Charcot foot, with total loss of the plantar arch.

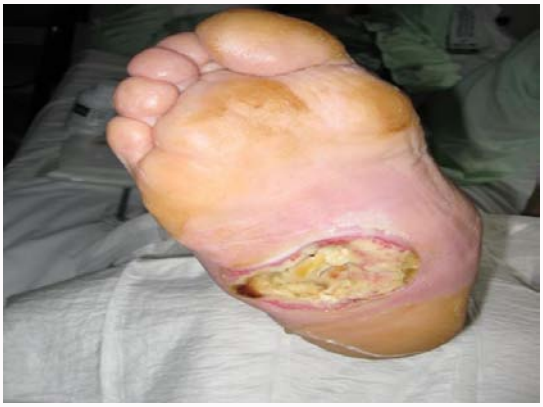

Figure 2: Plantar ulcer with bone exposure.

The diagnosis of Charcot's foot is fundamentally clinical. Suspicion should be established in the presence of a long-standing diabetic patient with edema, heat and unilateral flushing of the foot, with absence or mild pain, and deformity and instability of the joint. The course of the disease was described by Eichenholtz, establishing itself in four phases: Stage 0 or Clinical where edema, erythema and increased temperature in the foot can be seen [2-5]. Stage 1 or Acute or Fragmentation, where periarticular fractures, dislocations, instability and deformed foot occur. Stage 2 or Repair, where inflammatory signs decrease, and finally Stage 3 or Consolidation, where the foot is stabilized, although residual deformity persists and ulcerations may appear. In a retrospective study of Pakarinen T6 feet, 36 feet of Charcot were detected from 1994 to 2000, showing that the diagnosis was delayed by an average of 29 weeks. The differential diagnosis of acute Charcot foot should be made primarily with cellulitis, osteomyelitis and DVT, in addition to other processes such as arthritis, gout, sympathetic reflex dystrophy [1-3,6]. Characteristically, when the affected limb is elevated for about ten minutes, the redness disappears, whereas in cellulitis this does not occur (Brodsky's test) [1,3,4]. The plain radiograph may be normal in the early stages. Scintigraphy, CT and MRI help to differentiate neuroarthropathy from other diseases such as osteomyelitis [5].

The fundamental treatment of acute Charcot foot consists of rest with elevation of the limb to reduce edema. The most commonly used procedure is the placement of a total contact cast, although it has its limitations and a specially trained staff is also needed to apply it [3,4]. In our hospital there is no experience in the placement of the same, so we opted for the splint Brown. When a reduction of the edema

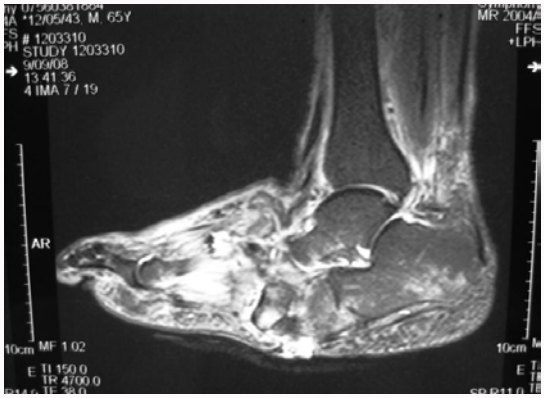

Figure 3: Magnetic Resonance right foot. There is a total loss of the plantar arch called "foot in rocking chair" (rocker bottom foot).

is achieved, the plaster can be replaced by a total discharge orthosis (ankle-foot type, CROW or PPWB) until bone consolidation is achieved [1,3,4]. Finally, the patient will need custom-made footwear. Recent studies show positive results with the administration of bisphosphonates to stop the inflammatory reaction, although more studies are needed to establish their role in the treatment of neuroarthropathy [1,2,3]. Surgical treatment will be indicated when there is an affectation of soft parts, the foot is unstable, or its adaptation to the footwear is not possible [7,8].

When all treatment options have failed or there are complications, you should opt for an infracondylar amputation. We can conclude that, in the presence of inflammation and foot edema in a patient with diabetes and severe neuropathy, once cellulitis, osteomyelitis and DVT have been ruled out, one should think of a Charcot neuroarthropathy.

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