A treatment algorithm for Charcot foot arthropathy

Algoritmo do tratamento na artropatia do pé de Charcot

Michael S. Pinzur[1]

Abstract

Charcot arthropathy of the midfoot imparts a severe negative impact on health related to quality of life. Treatment has been historically accommodative. Surgery was reserved for those who could not be accommodated with an orthosis, or developed ulcers or deep bony infection. The remaining ulcer and infection free as well as the possibility of maintaining the walking capacity with commercially available therapeutic footwear have been previously defined as favorable outcome. Using this definition of a favorable outcome, favorable results can be achieved in 60% of patients without surgery. When surgery is advised, a treatment algorithm is presented.

Keywords: Foot/pathology; Arthropathy, neurogenic/therapy; Orthopedic procedures/ métodos; Quality of life

Resumo

A artropatia de Charcot do mediopé implica numa grave repercussão sobre a qualidade de vida de seus portadores. O tratamento clássico tem sido conservador. A cirurgia tem sido reservada para os pacientes que não obtiveram conforto com uso de órteses, ou que desenvolveram úlceras ou ainda infecções ósseas profundas. A ausência de úlcera e infecção e a convivência com calçados comercialmente adequados para os pés insensíveis foram definidas como os objetivos de um resultado favorável. Usando esta definição de resultado satisfatório, foi obtido 60% de sucesso sem cirurgia. Quando a cirurgia é indicada, um algoritmo dos procedimentos é apresentado.

Descritores: Pé/patologia; Artropatia neurogênica/terapia; Procedimentos ortopédicos/ métodos; Qualidade de vida

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INTRODUCTION

In 1868, Jean-Martin Charcot provided the first in-depth description of a destructive hypertrophic osteoarthropathy that affected joints of patients with tertiary syphilis[1-3]. Penicillin has virtually eradicated tertiary syphilis, while insulin has allowed diabetics to survive and develop the longstanding peripheral neuropathy that appears to be the precursor for the development of a neuropathic (Charcot) osteoarthropathy. More recently, the Research Committee of the American Orthopaedic Foot & Ankle Society has twice rated Charcot Foot "neuro-arthropathy" as one of the most important problems confronting the Orthopaedic foot and ankle specialist[4-5]. The goal of this review is to provide a simple decision tree to use in evaluating and treating this very complex patient population (Figure 1).

PATHOPHYSIOLOGY

The development of peripheral neuropathy in individuals with diabetes is attributed to a complex interaction of glycosylated hemoglobin with arterioles of both central and peripheral nerves. The resultant progressive loss of function is first appreciated in the smallest nerve fibers, leading to conduction defects in sensory, motor and autonomic nerves. This complex pathophysiologic process at the arteriolar level is likely responsible for many of the co-morbidities associated with diabetes[6]. While there are many methods for detecting the presence of clinical peripheral neuropathy, the accepted clinical tool is Semmes-Weinstein 5.07 monofilament. The threshold for assigning the diagnosis of peripheral neuropathy in diabetic patients is the lack of perception of ten grams of applied pressure, *id est*, the pressure exerted by the Semmes-Weinstein 5.07 monofilament[7]. While not universally true, most patients who develop Charcot foot arthropathy have this threshold level of peripheral neuropathy[8-9].

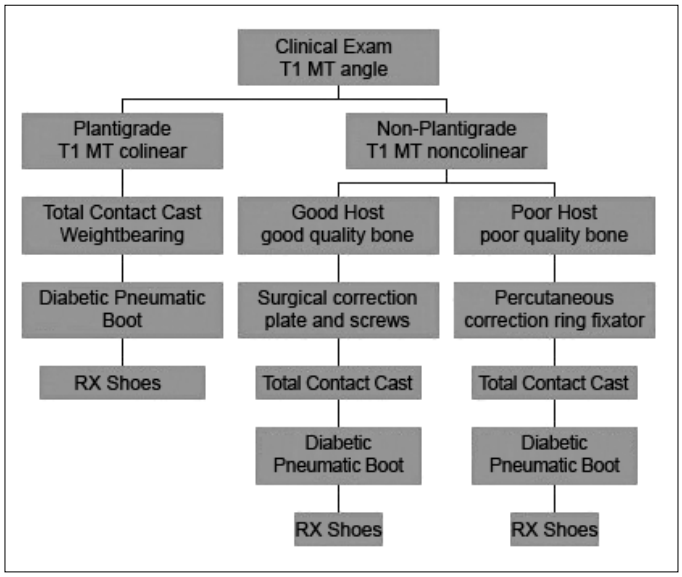

Figure 1 - Treatment algorithm for Charcot foot arthropathy.

Our current understanding of the effect of glycosylated hemoglobin on arterioles within central and peripheral nerves, leading to neuropathy creates an excellent foundation for explaining the pathologic process. Some inciting event, likely trauma, initiates the production of specific cytokines which upregulate osteoclast activity and start the destructive process[10-11]. The motor neuropathy, which initially affects smaller nerves and muscles, leads to a motor imbalance which the larger and stronger foot and ankle plantar-flexors overpower the smaller dorsiflexors. Static contracture and stiffness within the gastrocnemius-soleus muscle-tendon unit creates both a static and dynamic ankle equinus deformity. During terminal stance phase of gait, a bending moment creates mechanical overloading at the midfoot level, which may well be responsible for the mechanical breakdown and development of the neuropathic arthropathy[12-15]. The ending result is a complex set of deformities within the foot and ankle that imparts a severe negative impact on health related to the quality of life for the affected individuals[16].

We now understand that some inciting event triggers cytokines to activate osteoclasts to initiate the destructive process. Patients are generally in their sixth or seventh decades, have been diabetic for a long period of time and are generally insensate to the Semmes-Weinstein 5.07 monofilament. Most are morbidly obese. This stage is characterized by redness and swelling of the involved foot with no systemic signs of infection. Contrary to classic teaching, many of the patients will have pain associated with weight-bearing. Expert opinion has long advised treatment with a non-weight bearing total contact cast until the active phase of the disease resolves. Many recent publications advise early arthrodesis to prevent the development of late deformity. Based on retrospective reviews, we now appreciate that approximately 60% of such patients can be successfully treated without surgery[17-18].

DECISION # 1

The first decision point in the algorithm (Figure 1) is based on clinical examination and weight-bearing radiographs. Patients who are clinically plantigrade and have a colinear lateral talar-first metatarsal axis, as measured from weight-bearing AP radiographs, are predictably treated with a weight-bearing total contact cast. The cast is changed every two weeks (average 6.8) until

the swelling and redness subside and clinical stability returns (Figure 2). Patients then progress to a removable diabetic walking boot, until the size of the limb stabilizes. They are then fit with commercially-available therapeutic footwear, *id est*, commercially available non-custom fabricated depth-inlay shoes and custom accommodative foot orthoses.

DECISION # 2

The next decision must be made on patients who are clinically non-plantigrade, have a non-linear lateral latar-first metatarsal axis, or develop deformity during treatment with a weight-bearing total contact cast. These patients are advised to undergo correction of their deformity and surgical stabilization[17-20].

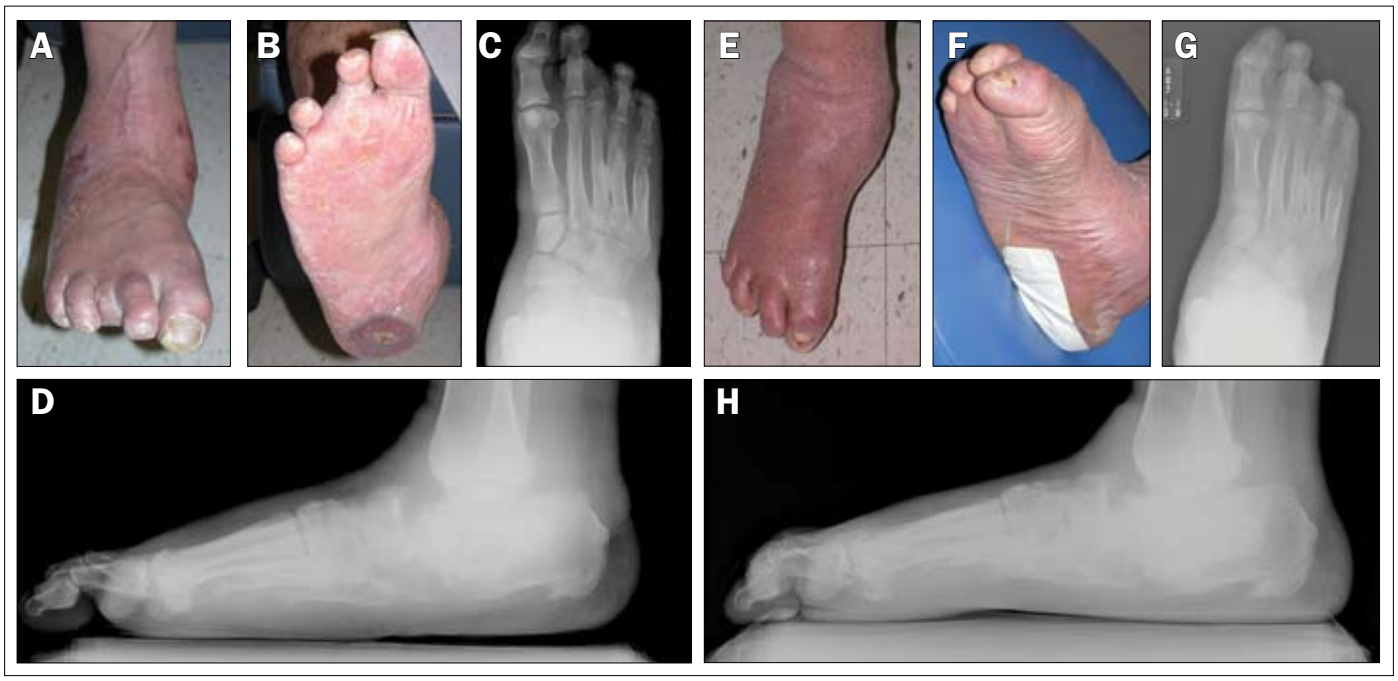

Figure 2 - A, B, C and D: this 54 year old physician presented with this deformity. He has already had a heel ulcer treated with skin grafting. Not that, in spite of the deformity, the foot is plantigrade and the lateral talar-first metatarsal axis is colinear. E, F, G and H: one year after closed treatment with a weight-bearing total contact cast and progression to therapeutic footwear.

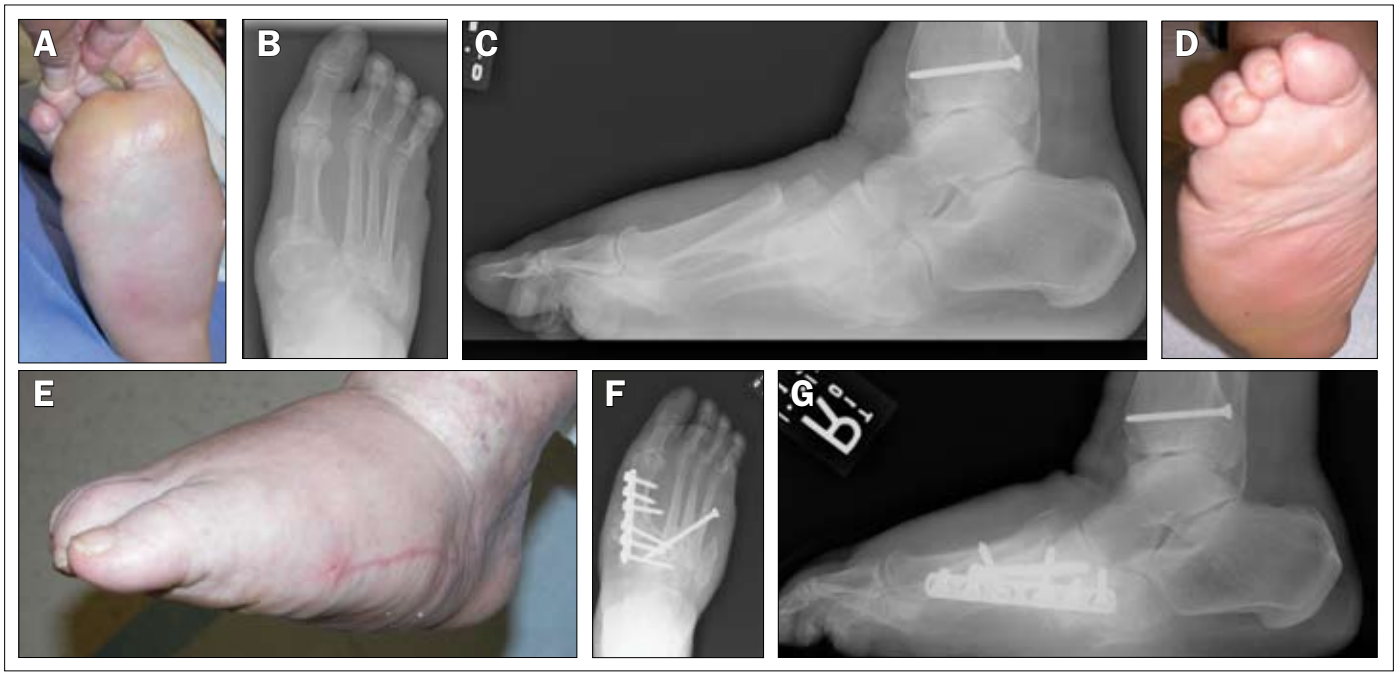

Figure 3 - A, B and C: this woman presented with a non-plantigrade deformity. D, E, F and G: two years following surgery.

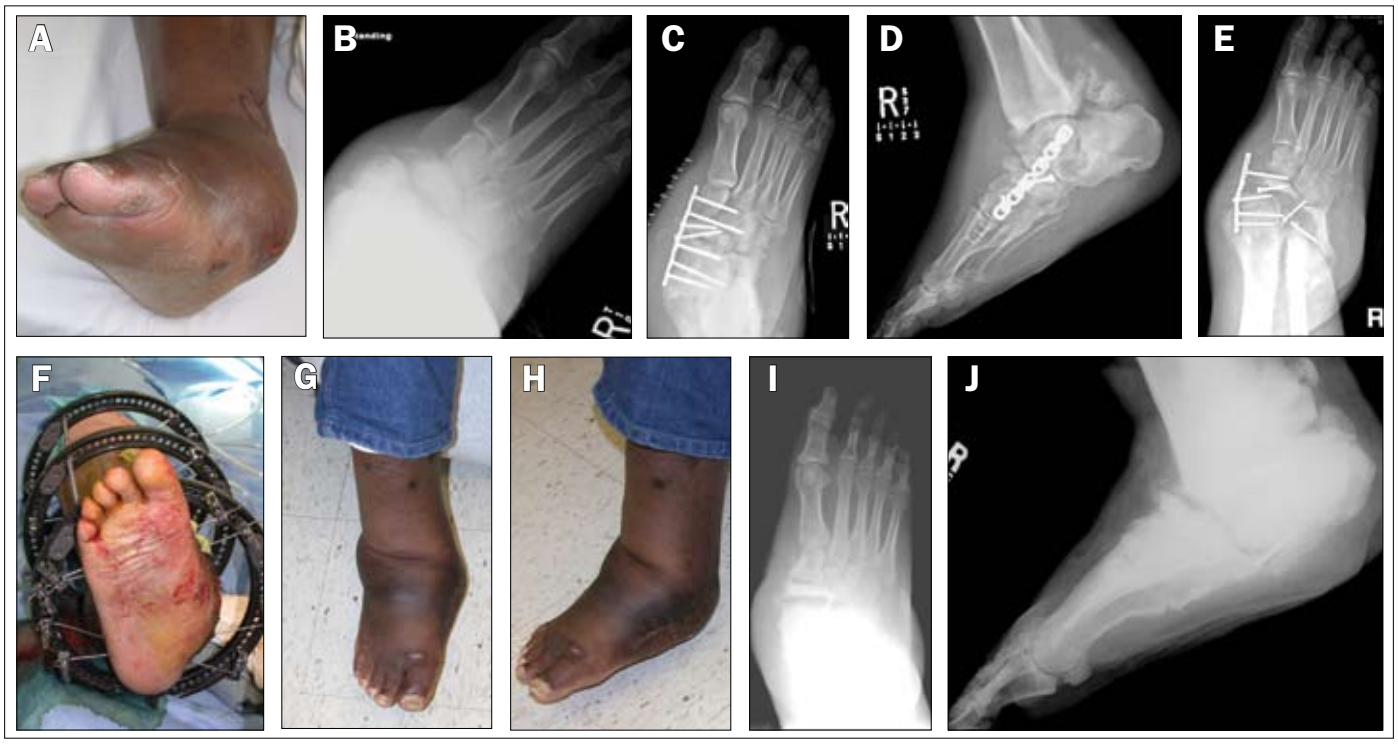

Figure 4 - A and B: This 400 pound mentally challenged diabetic presented with this deformity. C and D: he underwent surgical correction of his deformity with internal fixation. He attempted to remain non weight-bearing during the perioperative period. E: he was not successful. F: he underwent revision of his correction, this time maintaining correction with a three level neutral ring external fixator. G, H, I and J: at two years, he is able to ambulate with commercially available therapeutic footwear.

DECISION # 3

The next decision is made based on surgical risk. Patients are divided into relatively "good" surgical hosts and relatively "bad" hosts. "Good" hosts are reasonably normal sized individuals with no open wounds or osteomyelitis and good quality bone density. Surgery in these individuals involves Tendon Achilles lengthening, correction of the deformity and stabilization with standard methods of internal fixation (Figure 3). Following successful surgery, these patients also progress to a fracture boot and eventually into commercially available therapeutic footwear.

DECISION # 4

These patients are poor hosts for standard surgery. They are generally morbidly obese, have large open wounds with underlying osteomyelitis, and have poor quality bone stock. With standard methods of internal fixation, these individuals are prone to develop deep infection/osteomyelitis, or failure of internal fixation. They appear to be best treated with a more extensive surgical procedure. Tendon Achilles lengthening is performed to achieve muscle balance. A wedge of bone is resected at the apex of the deformity to achieve a plantigrade foot. Patients are treated with a prolonged course of parenteral culture-specific antibiotic therapy. Maintenance of the correction is achieved with a neutrally applied three level ring external fixator[20] (Figure 4). The fixator is generally maintained for eight weeks approximately, followed by a period of ambulation with a weight-bearing total contact cast. Upon healing, patients progress to appropriate therapeutic footwear.

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