Acute Charcot' joints in diabetics: surgical approach

Artropatia de Charcot aguda: abordagem cirúrgica

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Keywords:
Diabetes mellitus; Diabetes neuropathies; Diabetic foot; Arthropathy, neurogenic Surgical procedures, operative

ABSTRACT

Objective: The Charcot osteoarthropathy is a progressive, disfiguring, and debilitating condition characterized by joint subluxation and dislocation, fracture, and extensive osseous destruction of the foot architecture due to severe peripheral neuropathy. This progressive disease process is precipitated with repetitive trauma that goes unrecognized because of a loss of sensation and reactive hyperemia. **Methods:** We have been operated 20 Acute Charcot arthropathy at the last 3 years, with Achiles lengtheting, exostectomy, joint realignment, debridement, static external fixation (one stage approach) and internal fixation with cannulated screws (staged approach) immediately to external fixation removal, to prevent future instability-collapse and ulcer recurrence, in active patient with no evidence of bone infection. **Results:** Follow-up was performed through a multidisciplinary team to maintain metabolic and glycemic control, provide education strategies to prevent recurrence of lesions, and also physical therapy for adequate function and protection. During the study, none of the patients presented with deep infection or non plantigrade or unstable foot. Two patients presented superficial infection and underwent managed appropriately with antibiotics, skin care and local protection. **Conclusion**: Acute correction of Charcot joint using static external fixation alone minimizes the need for extensive surgical exposure and may provide a means of reducing deformities while maintaining the reduction during consolidation.

Descritores:
Diabetes mellitus; Neuropatias diabéticas; Pé diabetic; Atropatia neurogência; Procedimentos cirúrgicos operatórios

RESUMO

Objetivo: A osteo-artropatia neuropática de Charcot é considerada um condição clínica progressiva e debilitante caracterizada por desarranjos articulares, fraturas, extensa destruição óssea com perda da arquitetura do pé, devido a grave processo neuropático periférico. O progresso de progressão da doença é precipitado por trauma repetitivo decorrente da perda da sensibilidade e da expressão inflamatória predisponente. **Métodos:** Foram manejados 20 casos de Charcot Agudo nos últimos 3 anos, por meio alongamento percutâneo do tendão calcaneano, reposicionamento articular, desbridamento (se associado à ferida complexa) e fixação externa neutra estática (primeiro estágio) e fixação interna com parafusos canulados (tratamento estagiado) imediatamente à retirada do fixador externo, prevenindo futuros colapso ou instabilidade e recorrência de feridas, em pacientes ativos e sem evidência de infecção. **Resultados**: O seguimento foi dado por equipe multidisciplinar capacitada objetivando a manutenção do controle glicêmico, a educação assistida evitando recorrência de lesões, trabalho fisioterápico para reestabelecimento da função adequada e proteção. Durante o estudo, nenhum dos pacientes apresentou infecção profunda ou pés não plantígrados ou instáveis. Dois pacientes apresentaram infecção superficial e foram submetidos a antibioticoterapia sistêmica, tratamento local e proteção. **Conclusão:** A correção aguda da Artropatia de Charcot por meio de fixação externa estática sozinha (montagem apresentada) já minimiza a necessidade de extensa exposição cirúrgica além de proporcionar a manutenção anatômica até a consolidação.

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INTRODUCTION

Charcot neuroarthropathy is a noninfective, deforming and destructive process that can lead to increased patient morbidity. This pathology can be activated by a neurotraumatic stimulus associated with insensate peripheral neuropathy that manifests as dislocation or periarticular fracture[1].

Peripheral neuropathy secondary to diabetes mellitus is the most common etiology of Charcot neuroarthropathy, however, peripheral neuropathy from leprosy, alcoholism, syphilis, syringomyelia, rheumatoid arthritis, multiple sclerosis, and traumatic injury also may be associated. Among the complications related to diabetes, Charcot neuroarthropathy continues to be an enigmatic pathologic entity.[2,3]

Although this disease has been associated with other bodily sites in the diabetic patient[4,5] almost exclusively of the foot and ankle joints.

The prevalence of Charcot arthropathyis unknown,[6] and this may be due to the lack of standardized clinical criteria or wrong diagnosis as infection, gout, arthritis, fracture, venous insufficiency, or tumors.

An incidence of Charcot arthropathy is approximately 3%. Most of the patients have a period of unexplained swelling that resolves without deformity, being explained as tenosynovitis, gout or cellulitis. The only ones that we see are the ones that develop the clinical picture that we understand. The cytokines that stimulate the process are likely triggered by trauma. We think that trauma turns on osteoclasts in patients with vitamin D deficiency and osteoporosis. They keep weight bearing because of their loss of protective sensation and develop a pattern that looks like a hypertrophic nonunion, the so-called Charcot arthropathy described by Eichenholtz.[7-9]

The Charcot osteoarthropathy is a progressive, disfiguring, and debilitating condition characterized by joint subluxation and dislocation, "fractures", and extensive osseous destruction of the foot architecture due to severe peripheral neuropathy.[1] This progressive disease process is precipitated with repetitive trauma 1that goes unrecognized because of a loss of sensation and reactive hyperemia.[10]

The pattern of risk factors for the development of Charcot foot is similar to that for the development of diabetic foot ulcers, even for the fact that individuals who develop a Charcot foot deformity, generally have no evidence of peripheral vascular disease (PVD).[11] These people have PVD as evidence by calcification within the vessels, but they not have ischemic disease.

Surgery of the foot for other diagnoses has been reported to induce, or to be followed by the development of Charcot's arthropathy,[12,13] however, obesity is probably most important. There is more deforming force in someone morbidity obese than in someone who is small. Whether the mechanism in these cases was local trauma, a change in the mechanics of the foot, or incitement of a vascular hyperemia is not clear.[11]

Diabetic patientes with Charcot arthropathy and ulceration have statistically greater mortality rates than diabetics without Charcot arthropathy.[14] A recent large retrospective study revealed that obese diabetic patients are statistically more likely to develop Charcot arthropathy.[15]

The first theory of the Charcot development is neurotraumatic *destruction* and hypothesizes that the joint destruction, fractures, and collapse of the foot occur as the result of cumulative mechanical trauma, which could be minor trauma, major trauma, or unrecognized microtrauma in a joint that has been rendered insensitive to proprioception and pain.[16] The second theory is an autonomic neuropathy of vasoregulation leading to a hyperemic state and anincreased blood flow and bony resorbtion in the affected footthe joint dissolution occurs from bone resorption and ligamentous weakening as a result of a neurally stimulated vascular reflex interpreted as an autosympathectomy. It has been suggested that the natural course of the Charcot process, which consists of disintegration followed by new bone formation and bony consolidation, is related primarily to the self-limiting nature of the autonomically mediated hyperemia.[17]

We believe in a modern theory, which is a combination of the old neurovascular and neurotraumatic.

In this paper we will focus the discussion on our surgical approach of acute Charcot arthropathy.

METHODS

Clinical evaluation

When clinically evaluated patientes with Charcot arthropathy appears to combinevascular and a traumatic cause. They typically presents as a warm, swollen, and erythematous foot and ankle. The appearance of the extremity may be indistinguishable from infection, and almost all afflicted patients have severe peripheral neuropathy. It is this lack of protective sensation that delays identification of bony stress injuries that may overload the insensate limb, leading to an active Charcot process.[16]

Classification

Eichenholtz[9] published a landmark article on Charcot arthropathy based on radiographic appearance and its

physiologic course. Dividing the condition throughout its process, he described three separate but linear stages: developmental or acute, coalescent, and reconstructive or chronic stages. (Table 1). This classification is currently being applied by the majority of foot and ankle physicians to Charcot arthropathy patients in the staging of the disease. Others authors have also anatomically classified the characteristics of Charcot arthropathy by observing the patterns of destruction to the foot and ankle.[16,18-20]

Table 1. Modified Eichenholtz stages[9]

Stage	Phase	Description
0	Inflammatory	Localized warmth, swelling and redness; minimal to no radiographic abnormalities; MRI may show nondisplaced pathologic fracture(s) and increased marrow edema to the foot and/or ankle
1	Development	Localized warmth, marked swelling, and redness; radiographic presence of bony debris, fragmentation of subchondral bone, periarticular fracture, subluxation, and/or dislocation
2	Coalescence	Continued but decreased warmth, swelling, and redness; radiographic presence of absorption of fine debris, new bone formation, coalescence of fragments, fusion of joints (ankylosis), and/or sclerosis of bone ends
3	Remodeling	Marked decrease or absence of warmth, swelling, and redness; physically enlarged fixed ("healing") deformity; radiographic appearance of remodeled and new bone formation, decreased sclerosis, and/or possible gross residual deformity

Modified from Eichenholtz (1966).[9]

Preparation

A multidisciplinary team approach must be necessary, quickly and previously the surgical management. Usually, 2 or 3 intensive days has been enough to prepare the patient to the operation.

Operation

We have been operated 20 acute Charcot Arthropathy at the last 3 years, with Achiles lengthening, exosctectomy, joint realignment by handle distraction without osteotomy, debridement and static external fixation (one stage approach). Vaccum wound care therapy was used as adjuntive treatment in cases associated with large neuropathic ulcers.[21,22] (Figures 1-4).

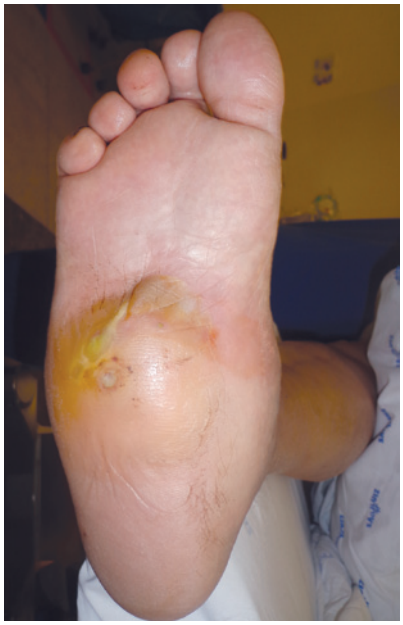

Figure 1. Acute Charcot foot

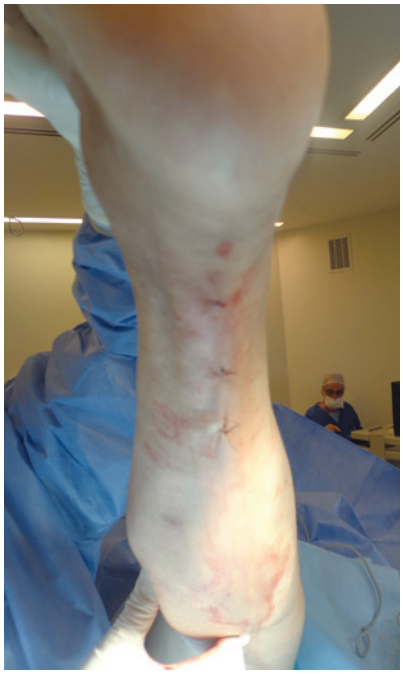

Figure 2. Percutaneous achiles lengthenhing

Internal fixation with cannulated screws (staged approach) to prevent future instability-collapse and ulcer recurrence, in active patient with no evidence of bone infection, demonstrated by histological analyses, is performed immediately external fixation removal (chronic phase).[23] (Figure 5).

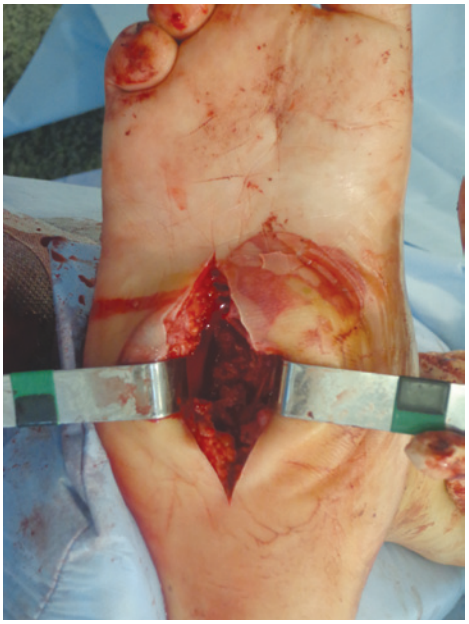

Figure 3. Direct exostectomy

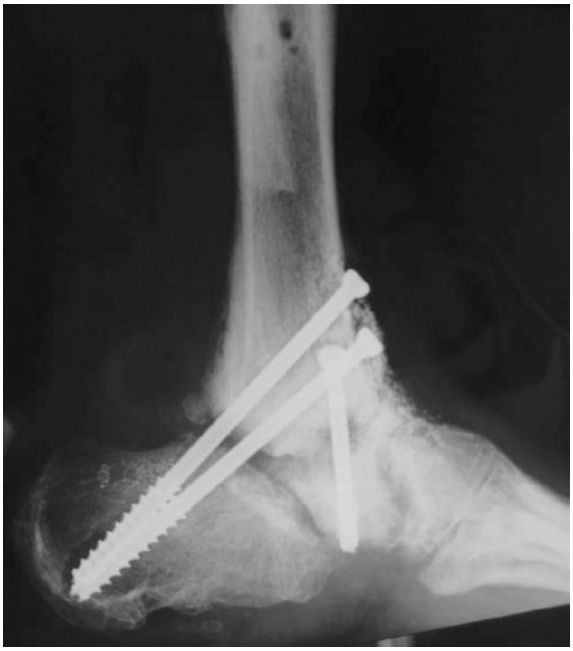

Figure 5. Staged approach (Charcot hindfoot internal fixation immediately external fixation removal)

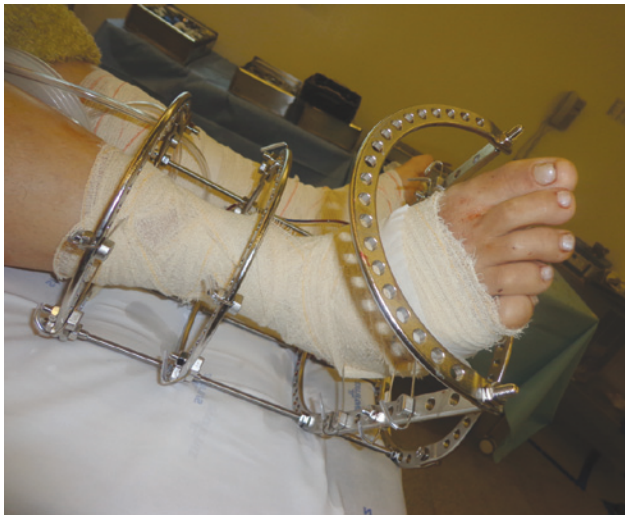

Figure 4. Static external fixation

RESULTS

Follow-up was performed through a multidisciplinary team to maintain metabolic and glycemic control, provide education strategies to prevent recurrence of lesions, and also physical therapy for adequate function and protection.

After external fixation removal (3 months on average), patients underwent to one stage approach was encouraged to use a no weight bearing total contact cast for 2 weeks and custom walking boots for at least 2 months; patients underwent to staged approach was encouraged to use a no weight bearing total contact cast for 6-8 weeks and custom walking boots for at least 2 months.

Thereafter, custom deep shoes and insoles were prescribed.

During the study, none of the patients presented with deep infection or non plantigrade or unstable foot.

Two patients presented superficial infection and underwent managed appropriately with antibiotics, skin care and local protection.

DISCUSSION

The goal in any treatment regimen involving the diabetic Charcot foot deformity is to create a plantigrade and stable foot free from significant risk for further breakdown or infection.[2,15,24,25].

The actual decision between conservative and surgical intervention on acute Charcot arthropathy depends on an assessment of the risks and benefits of each in terms of the deformity present, the expected recovery course, the ability of the patient to comply, comorbidities, nutritional status, psychosocial issues and family support capabilities.[11]

Here we are going to focus our discussion on acute Charcot Arthropathy surgical treatment.

Operative treatment

The objective in any treatment regimen involving the diabetic Charcot foot deformity is to create a plantigrade and stable foot without significant risk for wound breakdown or infection.[2,15]

Two basic surgical techniques are specific to the chronic Charcot foot. One is excision of the bony prominences that cause ulceration and lead to infection; the other is arthrodesis to realign the deformed area and to reconstruct the architecture of the foot to produce a plantigrade foot and to relieve pressure on the soft tissue. There are many options to achieve the main objective, this include open and minimally invasive techniques.[10,26]

Acute correction of Charcot joint using static external fixation alone minimizes the need for extensive surgical exposure and may provide a means of reducing deformities while maintaining the reduction during consolidation. With external fixation surgeons can perform a single multiple staged reconstruction and include ulcer resection, biopsy, wedge osteotomy, exostectomy, Achilles lengthening, wound care and primary arthrodesis.

The use of internal fixation including screws, plates, staples, or intramedullary nails is usually not advisable in the diabetic acute Charcot.[13] In addition, internal fixation may be a source of a seeded infection and loosening.

The use of an external fixation device alone becomes suitable in these surgical reconstruction procedures because it provides a source of rigid fixation and stabilization. Only when there is absence of a concomitant ulcer or infection, a combination of internal and external fixation techniques may be entertained.[19]

CONCLUSION

Acute correction of Charcot joint using static external fixation alone minimizes the need for extensive surgical exposure and may provide a means of reducing deformities while maintaining the reduction during consolidation.

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