Surgical management of the Charcot foot

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ARTICLE POINTS

1 Charcot foot is a relatively rare complication of diabetes.

2 Charcot may be misdiagnosed as infection.

3 The mainstay of treatment is non operative.

4 Surgery needs to be carefully planned.

KEY WORDS
- Charcot foot
- Deformity
- Exostectomy
- Arthrodesis

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Introduction

Charcot foot is a relatively rare complication of diabetes but one which can lead to chronic ulceration, marked deformity and amputation. This article describes the development of the Charcot foot. Although the aetiology is not fully understood, there have been advances in its management; surgery has a definite role to play, but the mainstay of initial treatment is non operative. The timing and type of surgery are crucially important — inappropriate surgery may worsen the problem and lead to premature amputation.

Charcot foot, or neuroarthropathy, is a rare complication of diabetes, affecting 1 to 2.5% of people with diabetes (Myerson, 1996). Some authors consider some of the radiographic changes of diabetic osteopathy to be a form of neuroarthropathy and put the incidence higher than this. It is most common in people with type 1 diabetes in the fifth and sixth decade but can occur in young patients; usually the duration of diabetes will be greater than 12 years (Laing, 1991). Mostly it occurs unilaterally but may be bilateral in up to 25% of patients (Frykberg, 2000).

Charcot foot can be defined as a relatively painless, progressive and degenerative arthropathy of single or multiple joints caused by an underlying neurological deficit. The amount of destruction and deformity seen clinically and on X-ray can be considerable which makes it a tremendous challenge to physician, surgeon and orthotist.

The association between a neurological deficit and an arthropathy was first suggested by Mitchell, an American physician, in 1831, although it was Jean-Marie Charcot in 1868 who famously attached his name to this condition (Charcot, 1868). Charcot observed the association between tabes dorsalis (due to syphilis) and arthropathy and, without the benefit of X-rays, described the natural history of the condition. Although diabetes is now the main cause of Charcot joints in the developed world, other neuropathies can cause Charcot and elsewhere leprosy may be the commonest cause.

Potential causes of Charcot foot

The aetiology is not fully understood and various neurovascular and neurotraumatic theories have been advanced. It is most likely due to repeated minor trauma in the neuropathic foot. Loss of protective sensation allows abnormal mechanical stresses which would normally be prevented due to pain. This may then lead on to spontaneous fractures, subluxations and dislocations. Autonomic neuropathy leads to increased blood flow with osteopenia, thus weakening the bone and making it more susceptible to injury (Brooks, 1986).

However, Charcot foot may occur following more major trauma, such as a fractured ankle, and has been reported during bed rest (Brower and Allman, 1981). An adequate blood supply appears to be crucial as Charcot foot is not seen in ischaemic feet, but has been reported following successful revascularisation (Edelman et al, 1987).

The patient with an acute Charcot foot usually presents with a hot, swollen, erythematous foot which may be several

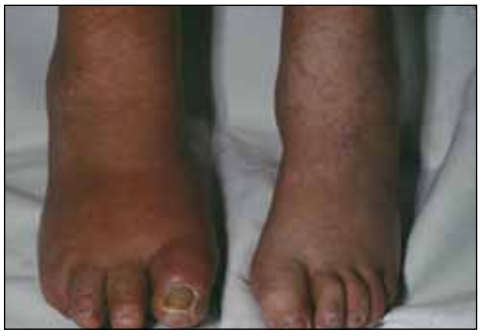

Figure 1. Acute Charcot foot.

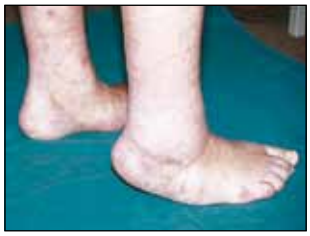

Figure 2. Rocker bottom foot.

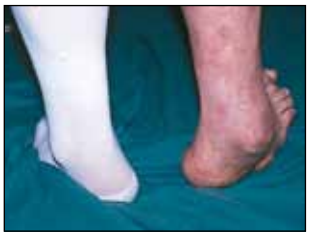

Figure 3. Charcot ankle with varus hindfoot.

Figure 4. Total contact cast.

Figure 5. Aircast walker.

degrees warmer than the contralateral foot (*Figure 1*). Typically there may be no history of trauma or the patient may recall some minor trauma perhaps weeks before. Sometimes there may be concomitant ulceration in the foot. Although the Charcot foot is often described as painless, patients will usually have some pain and discomfort. However, the level of pain reported by patients is normally considerably less than might be expected from the pathology seen.

Deformity may be evident at presentation. Depending on the timing of presentation, X-rays may show no changes, healing fractures, e.g. of the metatarsals, or subluxation and dislocation of one or more joints. The most commonly affected site is the midfoot (60% of patients), followed by the metatarsophalangeal joints (30%) and the ankle joint (10%). Because of its mode of presentation, the acute Charcot foot may be mistaken for infection with cellulitis and osteomyelitis. A useful clinical test is to adequately elevate the foot for five minutes: the erythema of a Charcot foot will recede, whereas that of a cellulitic foot will not (Brodsky, 1993).

The results of nuclear medicine and magnetic resonance imaging scans must also be interpreted with caution as it is not easy to differentiate the acute Charcot foot from infection with osteomyelitis (Brodsky, 1993). That can lead to inappropriate surgery to deal with non-existent infection. Experience is important here as occasionally patients can present with, or develop, an infected Charcot foot.

Stages of the Charcot foot

The Charcot foot develops through three stages (Eichenholtz, 1966). In stage I, the stage of development, there is acute inflammation with hyperaemia, bone softening and fragmentation and joint subluxation, dislocation and destruction. During this stage the longitudinal arch of the foot may collapse, giving rise to a rocker bottom foot (*Figure 2*), or midfoot subluxation in the transverse plane may cause a banana-shaped deformity. Subluxation at the ankle joint may lead to marked varus or valgus deformity such that the patient is no longer walking on a plantigrade foot (*Figure 3*). In stage II, the stage of coalescence, periosteal new bone formation is apparent, along with reduction of the swelling. In stage III, the stage of reconstruction, bony consolidation and healing occurs. Stage I represents the acute Charcot foot and stages II and III the reparative process. Progress through all three stages may take up to 2–3 years, although the acute phase may settle over a period of months.

Treatment

Treatment of Charcot foot is aimed at reducing the swelling, minimising residual deformity and thus reducing the risk of neuropathic foot ulceration. Some centres will aim to reduce swelling and mechanical stress by means of bed rest, elevation and non weight bearing with crutches or a wheelchair. The disadvantage of this is that the acute phase may last for six months or so and patients with neuropathy and loss of sensation may not be compliant with this. Prolonged non weight bearing may increase osteoporosis and further weaken the bone.

We therefore use either the total contact cast (*Figure 4*) (Laing et al, 1992), which is an excellent means of reducing swelling, or the diabetic Aircast walker (*Figure 5*) with inflatable air cells, which patients can sometimes tolerate better over a prolonged period of time. A total contact insole can be made for the walker. The aim of the total contact cast is to both reduce swelling and try to hold the shape of the foot while the bone is soft and deformable. In recent years, there has also been interest in the use of drugs designed to reduce bone turnover, by inhibiting osteoclastic activity, as a means of helping to settle the acute Charcot foot (Selby et al, 1994).

Surgery

In the acute stage I of a Charcot foot, surgery is almost totally contraindicated. Metalwork will not hold in soft fragmenting bone and the literature refers to many poor results from attempted arthrodeses (Sinha et al, 1972). The exception to this may be the acute, unstable but manually reducible dislocation. Under these circumstances, success has been reported in obtaining stable reduction (Myerson et al, 1994). However, any bony fragmentation or periosteal new bone formation is a contraindication.

Once the Charcot foot has reached a quiescent stage the patient is usually left with a neuropathic, deformed foot which is liable to recurrent ulceration over the bony prominences (*Figure 6*). Again, initial treatment will tend to be orthotic with footwear and insoles. However, recurrent ulceration in the presence of a fixed deformity will be an indication for surgery. Under these circumstances, surgery is indicated either to remove the bony prominences or correct deformity to produce a plantigrade foot which can be managed with orthotics.

A further indication for surgery may be deep infection with abscess formation or osteomyelitis which may necessitate drainage or removal of infected bone. In one series, half of 237 patients required surgery, including 21 major limb amputations (Pinzur, 1999).

Exostectomy

Removal of bony prominences is perhaps most common in the rocker bottom foot, where patients develop a plantar midfoot prominence which is liable to recurrent breakdown. Before any surgery, it may be preferable to heal the ulceration if practical in order to minimise infection risk; it is also important to establish that there is no underlying osteomyelitis within the bony prominence.

Diagnosing osteomyelitis in the Charcot foot is not straightforward because of the already abnormal bony architecture. It may be necessary to do a bone scan in conjunction with an Indium^{111} white cell labelled scan. If osteomyelitis is present then a wider excision of bone may be indicated.

A midfoot plantar exostosis can be excised through an incision along the medial or lateral border of the foot. The soft tissues are stripped off the underlying bone and then either an oscillating saw or osteotomes are used to remove the prominence and flatten the bony surface. The greater the surface area supporting weight the less there will be point loading with high peak pressures. A drain is normally used to reduce postoperative haematoma. Postoperatively, the wound is allowed to settle and weight bearing can then be commenced, with or without a cast, depending on whether any ulceration is still present.

Arthrodesis

Arthrodesis is usually reserved for midfoot and hindfoot deformities that are unbraceable and causing recurrent ulceration. This is considerably more complex surgery that carries the risk of amputation if it is unsuccessful; in many cases, however, it can avoid the need for amputation — and the significantly higher energy requirements of walking with an artificial leg. Once a person with diabetes has had one leg amputated there is a high incidence of contralateral amputation within a few years (Spencer et al, 1985).

A number of different classifications have been proposed for the different patterns of involvement. Brodsky et al (1987) suggested an anatomical classification into three main groups, as follows:

- Type 1 involving the tarsometatarsal and naviculocuneiform joints (*Figure 7*).
- Type 2 involving the subtalar complex including the talonavicular and calcaneocuboid joints.
- Type 3A involving the tibiotalar joint (*Figure 8*), and Type 3B involving the posterior process of the calcaneus which may develop a neuropathic fracture.

In types 1 and 2, arthrodesis, rather than exostectomy, may be indicated where midfoot deformities are unstable and the Achilles tendon is tight, which exacerbates the rocker deformity. Realignment involves open reduction of the deformity and often extensive fixation with interfragmentary screws. If the bone is too soft for solid fixation with screws the fragments may be held with pins. Lengthening of the Achilles tendon may also be necessary.

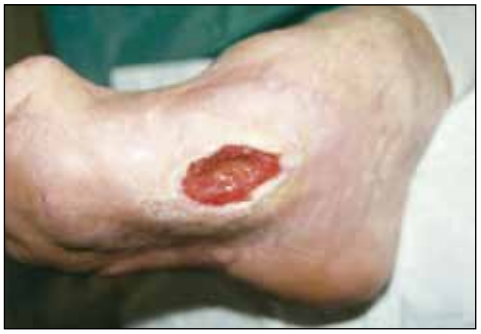

Figure 6. Charcot foot with ulceration.

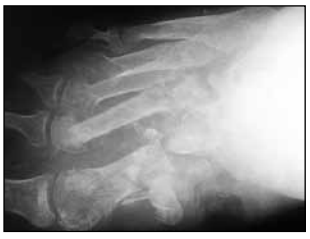

Figure 7. Type 1 Charcot foot involving tarsometatarsal joints.

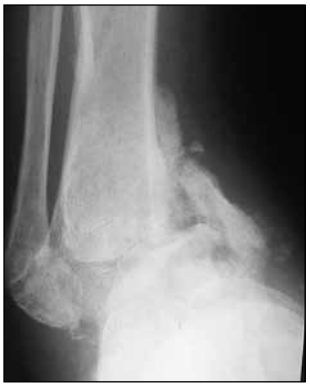

Figure 8. Charcot ankle with marked talar tilt.

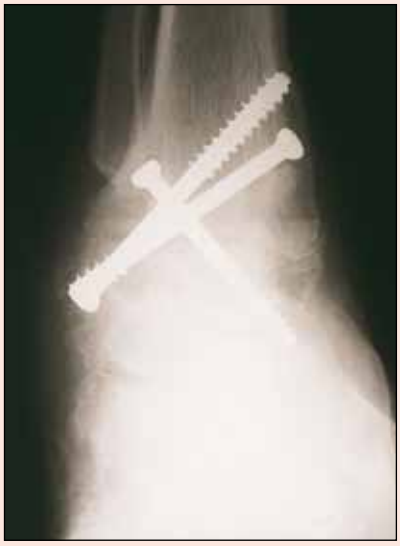

Figure 9. Ankle arthrodesis with crossed screws.

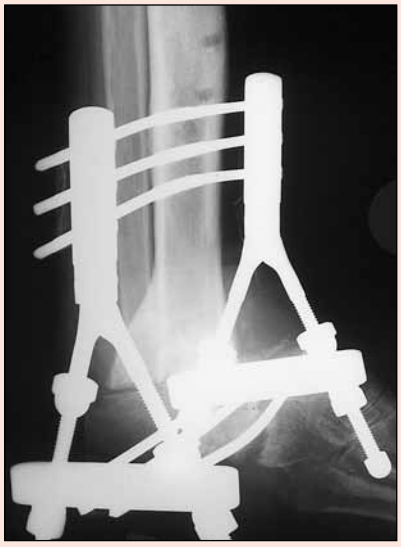

Figure 10. Arthrodesis using a Callandruccio clamp.

Type 3 ankle involvement may cause marked malalignment in the coronal plane. *Figure 3* shows a patient with gross varus of the ankle joint such that he was walking on the lateral border of the foot and had chronic ulceration there. Deformity like this is unbraceable. Arthrodesis of the ankle joint can be accomplished in a standard manner with screw fixation if the hold in the bone is sufficiently good (*Figure 9*). If the bone is soft, or a tibiocalcaneal arthrodesis is necessary, then an external fixator, such as the Calandruccio clamp (*Figure 10*), can be used or an intramedullary nail driven up through the heel. If an intramedullary nail is used then both the ankle and subtalar joints will be fixed.

Postoperative management may involve lengthy periods in plaster while bony union is taking place. Although some series have achieved good rates of union, solid bony union does not always occur. A number of these patients will stabilise though with a fibrous ankylosis and the deformity will be corrected sufficiently to adequately brace the foot and avoid recurrent ulceration. Nevertheless this is not easy surgery and most series have significant complication rates. Twenty complications in 19 patients were reported in a series of 29 patients with neuroarthropathy (Papa et al, 1993). Ten of the 29 patients had a pseudoarthrosis although seven of these were stable. One patient required a below-knee amputation. However, all of these deformities were severe and amputation would have been an alternative to arthrodesis.

Conclusion

The Charcot foot represents a tremendous problem. There is much that is still not understood about it but indications for surgery and techniques of stabilising the foot have advanced considerably over the last decade. It still remains rewarding, challenging and frustrating though, in equal measure, but is certainly not an area for the occasional foot and ankle surgeon. ■

Brodsky JW, Kwong PK, Wagner FW et al. (1987) Patterns of breakdown, natural history and treatment of the diabetic Charcot tarsus. *Orthopaedic Transactions* **11**: 484

Brodsky JW (1993) The diabetic foot. In: Mann RA, Coughlin MJ, eds. *Surgery of the Foot and Ankle.* Mosby, St. Louis: 877-958

Brooks AP (1986) The neuropathic foot in diabetes. Part II: Charcot's neuroarthropathy. *Diabetic Medicine* **3**: 116-18

Brower AC, Allman RM (1981) Pathogenesis of the neurotrophic joint: neurotraumatic vs neurovascular. *Radiology* **139**: 349–54

Charcot J-M (1868) Sur quelques arthropathies qui paraissent dependre d'une lesion du cerveau ou de la moelle epiniere. *Arch Physiol Norm Pathol* **1**: 161-78

Edelman SV, Kosofsky EM, Paul RA et al (1987) Neuro-osteoarthropathy (Charcot's Joint) in diabetes mellitus following revascularisation surgery. *Archives of Internal Medicine* **147**: 1504-8

Eichenholtz SN (1966) *Charcot Joints.* Charles C. Thomas, Springfield, Illinois

Frykberg RG (2000) Charcot foot. In: Boulton AJM, Connor H, Cavanagh PR, eds. *The Foot in Diabetes.* John Wiley, Chichester: 235-60

Laing PW (1991) The foot in diabetes. In: Klenerman L, ed. *The Foot and its Disorders*. Blackwell Scientific, London: 139-152

Laing PW, Cogley DI, Klenerman L (1992) Neuropathic foot ulceration treated by total contact casts. *Journal of Bone and Joint Surgery Br* **74**: 133-36

Myerson MS (1996) Salvage of diabetic neuropathic arthropathy with arthrodesis. In: Helal B, Rowley D, Cracchiolo III A, Myerson M, eds. *Surgery of Disorders of the Foot and Ankle.* Martin Dunitz, London: 513-522

Myerson MS, Henderson MR, Saxby T et al (1994) Management of midfoot diabetic neuroarthropathy. *Foot & Ankle International* **15**(5): 233-41

Papa J, Myerson M, Girard P (1993) Salvage, with arthrodesis, in intractable diabetic neuropathic arthropathy of the foot and ankle. *Journal of Bone and Joint Surgery Br* **75**: 1056-66

Pinzur MS (1999) Benchmark analysis of diabetic patients with neuropathic (Charcot) foot deformity. *Foot & Ankle International* **20**(9): 564-7

Selby PL, Young MJ, Boulton AJM (1994) Bisphosphonates: a new treatment for diabetic Charcot neuroarthropathy? *Diabetic Medicine* **11**(1): 28-31

Sinha S, Munichoodappa CS, Kozak GP (1972) Neuroarthropathy (Charcot joints) in diabetes mellitus. (Clinical study of 101 cases) *Medicine* **51**(3): 191-210

Spencer F, Sage R, Graner J (1985) The incidence of foot pathology in a diabetic population. *Journal of the American Podiatric Medical Association* **75**: 590-2