The Natural History of Acute Charcot’s Arthropathy in a Diabetic Foot Specialty Clinic†

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The aim of this longitudinal study was to report on the clinical characteristics and treatment course of acute Charcot’s arthropathy at a tertiary care diabetic foot clinic. Fifty-five diabetic subjects, with a mean age of 58.6 ± 8.5 years, were studied. All patients were treated with serial total contact casting until quiescence. Following casting and before transfer to prescription footwear, patients were eased into unprotected weightbearing via a removable cast walker. This cohort was followed for their entire treatment course and for a mean 92.6 ± 33.7 weeks following return to shoes. Pain was the most frequent presenting complaint in these otherwise insensitive patients (76%). The mean duration of casting was 18.5 ± 10.6 weeks. Patients returned to footwear in a mean 28.3 ± 14.5 weeks. Nine per cent of the population had bilateral arthropathy. These subjects were casted significantly longer than the unilateral group (p < 0.02). Surgery was performed on 25% of patients, with approximately two-thirds of these procedures involving plantar exostectomies and one-third fusions of affected joints. Patients receiving surgery remained casted significantly longer than non-surgical patients (p < 0.05). Additionally, men were casted longer than women (p < 0.008).

Acute Charcot’s arthropathy requires prompt, uncompromising reduction in weight-bearing stress. Our data show that the ambulatory total contact cast is very effective for this. Regardless of the specific treatment method instituted, it is imperative that appropriate and aggressive treatment be undertaken immediately following diagnosis to help prevent progression to a profoundly debilitating, limb-threatening deformity. ©1997 by John Wiley & Sons, Ltd.

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Introduction

Neuropathic osteoarthropathy, commonly referred to as Charcot’s joint or Charcot’s arthropathy was probably first described by Musgrave in 1703.1 He described it as complication secondary to venereal disease. Since then, numerous theories have been propagated regarding its pathogenesis.2–5 The current theory suggests that, following the development of autonomic neuropathy, there is increased blood flow to the extremity, resulting in osteopenia. In addition, motor neuropathies result in muscle imbalance, placing abnormal stress on the affected extremity, and sensory neuropathy renders the patient unaware of the often profound osseous destruction taking place.6–9,10

While there are a number of studies in the medical literature discussing characteristics and surgical treatment of Charcot’s Arthropathy,11–20 we have been unable to identify studies specifically reporting on long-term surgical and non-surgical outcomes of the acute form of this disease. We are also unaware of any reports of the duration of treatment of acute Charcot’s joint from inciting event to prescription footwear. Furthermore, we have been unable to identify studies reporting on the efficacy of total contact casting in the treatment of Charcot’s arthropathy in a large cohort of patients. The purpose of this longitudinal study was to report on the clinical characteristics and treatment course of acute Charcot’s arthropathy at a tertiary care diabetic foot clinic.

Patients and Methods

We abstracted data retrospectively from patient visits to a multidisciplinary tertiary care diabetic foot clinic between 1 February 1991 and 1 July 1994. The initial diagnosis of acute Charcot’s arthropathy was based on profound swelling, locally increased skin temperature, erythema, joint effusion, and bone resorption and fragmentation in an insensitive foot. All subjects studied had clinical loss of protective sensation to the 10 gramme Semmes-Weinstein monofilament wire using the method...
and criteria described by Birke. All subjects were diagnosed with diabetes mellitus based on the criteria set forth by the World Health Organization. All subjects had palpable pedal pulses on initial evaluation.

We classify Charcot’s arthropathy into two treatment-oriented phases based on radiographic, dermal thermometric, and clinical signs. The initial clinical diagnosis of acute Charcot’s arthropathy (as described above) has been well documented in the literature. Following resolution of acute neuropathic osteoarthropathy, patients are converted to the post-acute (quiescent) phase, during which time progressive weight-bearing is introduced (Figure 1).

Only those patients diagnosed with acute Charcot’s arthropathy were selected for study. Diagnosis was based on radiographic, dermal thermometric, and, if necessary, histological analysis. Histological analysis was based on bone and synovial biopsy consisting of multiple shards of bone and soft tissue embedded in the deep layers of synovium. Patients clinically and histologically diagnosed or treated prior to presentation to our facility were excluded from analysis (n = 18). Patients with concomitant osteomyelitis were also excluded (n = 8). The preliminary (clinical) diagnosis of osteomyelitis was made utilizing a sterile blunt surgical probe. This was subsequently confirmed in all cases by both microbiologic and histologic analysis via bone and synovial biopsy.

Location of Charcot’s arthropathy was described using the Sanders pattern classification. Sanders described five different patterns of Charcot’s arthropathy based on site. Pattern 1 indicates arthropathy in the forefoot. Pattern 2 refers to involvement at Lisfranc’s joint. Pattern 3 affects the bones of the lesser tarsus. Pattern 4 affects the ankle joint. Pattern 5 affects the posterior calcaneus.

All patients were initially treated with total contact casting. The total contact cast consists of an inner layer of plaster with thin felt first applied to the malleoli and tibial crest and foam to the digits for protection. The outer splints and remaining layers were made of fiberglass, with a rubber cast plug secured to the plantar aspect of the cast to increase durability. All casts were applied using the material and technique described by Kominsky. Casts were checked weekly and evaluated for proper fit.

Figure 1. Generalized treatment algorithm
Casts of patients with concomitant ulceration were changed weekly for ulcer evaluation and debridement. Cast change intervals for patients without ulceration were dependent on cast comfort and integrity (3 weeks maximum). Casting was discontinued based on clinical, radiographic, and dermal thermometric signs of quiescence. Skin temperatures were objectively monitored using a portable infrared thermometric probe (Exergen Model DT 1001, Exergen Products, Newton, MA, USA). Use of dermal thermometry has been well described in the literature.33,34 If patients presented with bilateral acute Charcot’s arthropathy, the patients remained in bilateral total contact casts until both feet/ankles normalized clinically and radiographically.

Following casting, patients progressed from removable cast walkers to accommodative footwear with ankle foot orthoses, as required. Removable cast walkers (Easystep Walker, Centec Orthopaedics, Camarillo, California, USA) were used to ease the transition from total contact casting to full, unprotected weight-bearing in prescription footwear. The transition to removable cast walker was made when skin temperature gradients were within 1°C for 2 consecutive weeks at the affected site compared with the corresponding site, contralaterally. The transition from removable cast walker to prescription footwear was based on 1 month of skin temperature equilibrium on the affected site compared to the contralateral extremity (± 1°C). This period of protected weight-bearing provided the pedorthic shoe specialist time in which to make and fit prescription footwear.

Reconstructive surgery was performed only if a deformity existed that placed the foot at risk for ulceration and could not be safely accommodated in prescription footwear. Surgery was undertaken only after radiographic signs of coalescence (trabecular bridging) or if clinically unstable pseudarthroses existed. Following surgery, patients were total contact casted until skin temperatures and postoperative oedema had returned to normal. As with those treated non-surgically, following casting, patients progressed to a removable cast walker followed by permanent prescription footwear. Following transition into footwear, patients were followed at 2-month intervals for clinical signs of recurrence. Patients were followed for a minimum of 1 year following return to prescription footwear (mean 92.6 ± 33.7 weeks).

A Mann-Whitney U-test was used to compare differences in age, duration of diabetes, length of treatment among various dichotomous groups including male and female, surgical and non-surgical, bilateral and unilateral arthropathy. A Kruskal-Wallis non-parametric test with one-way analysis of variance was used to measure the differences between location of neuropathic osteoarthropathy, duration of therapy, and skin temperature gradient. Fisher’s exact test was used to compare risk of reulceration following the acute phase of Charcot’s arthropathy.35 Values are expressed as mean ± standard deviation. For all analyses, we used an alpha level of 0.05.

Results

The study included 55 subjects, 27 male and 28 female, with a mean age of 58.6 ± 8.5 years. Fifty-four subjects (98%) were diagnosed with Type 2 (non-insulin dependent) diabetes mellitus with a mean duration of 15.9 ± 5.7 years. One subject was diagnosed with Type 1 diabetes, present for 12 years prior to presentation. There was no significant difference in age or duration of diabetes among male and female subjects. The prevalence of acute-onset Charcot’s arthropathy among all diabetic patients presenting to the Diabetic Foot Center during the study period was 12.9% (55/426).

The average initial thermometric elevation on the affected side was 5.1 ± 1.4°C. There was no significant difference between skin temperature gradient and location of neuroarthropathy, nor was there a significant difference between skin temperature gradient between males or females. Bilateral Charcot’s arthropathy was present in 9% (5/55) of the patients presenting in acute Charcot’s arthropathy. Twenty-two patients (40% of total) initially presented with a concomitant grade 1a ulceration using the University of Texas Wound Classification System.36,37 Distribution of these ulcerations correlated with location of maximum deformity as described by Sanders’ pattern classification in every case (Table 1).

Location of the deformity, based on the Sanders classification, revealed 3% forefoot, 48% at the tarsometatarsal (Lisfranc’s) joint, 34% at Chopart’s joint, 13% at the ankle joint, and 2% involving the posterior calcaneus. Nine patients presented with multiple foci of Charcot’s arthropathy. Of those 9 patients, 6 presented with a combination of type II and type III. There was no significant difference in location distribution of Charcot’s arthropathy between males and females.

When questioned, 73% could not recall a single precipitating event prior to the onset of their symptoms; 22% recalled a specific traumatic episode within 1 month of onset of symptoms; 4% related recent foot surgery. Although all subjects were clinically insensate on examination, 76% of subjects complained of pain in the foot upon presentation. There was no significant difference in those subjects who recalled a single traumatic event and duration of casting and return to footwear.

All patients were treated with serial total contact casts and remained casted for an average of 18.5 ± 10.6 weeks (range 4 to 56 weeks). All patients returned to permanent footwear. Progression to permanent footwear took 28.3 ± 14.5 weeks. All concomitant ulcerations healed during the total contact casting regimen prior to quiescence of the neuroarthropathic process. Time of casting and time to footwear is further divided by location in Figure 2. There was no significant correlation between location of acute Charcot’s arthropathy and duration cast therapy or time to footwear. Men were contact casted for a significantly longer period (21.8 ± 12.4 weeks) than women (15.2 ± 7.6 weeks) (p < 0.008). While men
Table 1. Site of ulceration on presentation with acute Charcot’s arthropathy

<table>
<thead>
<tr>
<th>Site of ulceration</th>
<th>n</th>
<th>Corresponding Sanders Classification</th>
<th>% of total ulcerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hallux interphalangeal joint</td>
<td>1</td>
<td>1</td>
<td>4.5</td>
</tr>
<tr>
<td>Cuboid metatarsal articulation</td>
<td>14</td>
<td>2</td>
<td>63.6</td>
</tr>
<tr>
<td>Talonavicular joint</td>
<td>6</td>
<td>3</td>
<td>27.3</td>
</tr>
<tr>
<td>Medial malleolus</td>
<td>1</td>
<td>4</td>
<td>4.5</td>
</tr>
</tbody>
</table>

Figure 2. Treatment duration by location of osseous destruction. The Sanders pattern classification refers to five areas of osseous destruction. These include the forefoot (pattern 1), the tarsometatarsal articulations (pattern 2), the bones and joints of the lesser tarsus (pattern 3), the ankle joint (pattern 4), and the posterior calcaneus (pattern 5).

... took longer to return to footwear (30.2 ± 14.5 weeks) than women (26.4 ± 14.8 weeks), this association did not reach significance.

Patients presenting with bilateral arthropathy remained casted for an average of 28.0 ± 14.7 weeks and returned to footwear in an average of 48.0 ± 18.2 weeks. There was a significant difference in time to return to permanent footwear between bilateral and unilateral Charcot’s arthropathy (p < 0.02). All patients with bilateral arthropathy presented with both feet involved. No patient developed contralateral Charcot’s arthropathy during the period reviewed.

Surgery was performed on 25% of patients. Of those procedures, 9 were exostectomies (i.e. midfoot planing) and 5 included fusion of affected joints with 2 of the 5 fusions receiving tendo-achilles lengthening. There were no significant differences in surgery or type of surgery performed on male versus female patients. Patients with exostectomy procedures remained casted for an average of 15.4 ± 4.2 weeks and returned to permanent footwear in an average of 27.6 ± 14.0 weeks. Patients who underwent fusions remained casted for an average of 29.0 ± 15.5 weeks and returned to permanent footwear in an average of 48.1 ± 16.5 weeks (Figure 3). Patients who underwent fusions remained casted for a longer period of time (p < 0.05) and returned to footwear later (p < 0.02) than the rest of the study population. Four of five patients undergoing fusions proceeded to bony union. One patient had an asymptomatic pseudoarthrosis of the ankle, subsequently controlled in an ankle-foot orthosis and custom moulded footwear.

Following the total contact casting regimen, 15% of patients experienced transient increases in temperature on the formerly symptomatic side. Of these patients, 88% (7/8) exhibited these elevations in the first month following return to permanent footwear. One patient experienced transient skin temperature elevation at 35 months. None of these patients showed radiographic or clinical signs of recurrent destructive changes. All of these patients were placed again on a regimen of total contact casting for an average of 2.9 ± 1.2 weeks until temperatures normalized.

Following resolution of acute Charcot’s arthropathy, four patients (7.3% of total) returned during the follow-up period with a new-onset neuropathic ulceration. These wounds appeared at a mean 10.0 ± 2.5 months following return to footwear. All recurrent wounds were grade 1a in nature. All four patients who ulcerated during the follow-up period had midfoot ulcerations on initial presentation with acute Charcot’s arthropathy, but reulcerated in the forefoot (Table 2).

Discussion

This study reports the distribution of acute Charcot’s arthropathy, prevalence of bilateral involvement and ulceration, duration to healing, and recurrence of acute arthropathy and ulceration in people with diabetes mellitus. Our results indicate that the majority of patients with Charcot’s arthropathy present with pain and a high prevalence (40%) of neuropathic ulceration at the site of joint damage. Additionally, the treatment of the disease is lengthy, spanning several months of immobilization in contact casts and cast walkers. After return to prescription footwear, relapse was infrequent with regular follow-up in a specialty foot clinic. There were no instances of fracture or refracture of the treated or of the contralateral limb. No amputations resulted as a result of Charcot’s arthropathy in this series.
While it is generally thought that people with acute Charcot’s arthropathy require a long duration of treatment until quiescence, we have been unable to find any reports in the medical literature that discuss the time period until patients could return to footwear in a cohort of patients treated with the same modality. The mean time of immobilization (casting followed by removable cast walker) prior to return to permanent footwear was approximately 6 months in our study. Myerson and coworkers reported that, following open reduction and internal fixation of acute Charcot fracture-dislocations in 8 patients, mean time of casting was 5 months. There was no report on duration to permanent prescription footwear. Similarly, Brodsky reported that patients receiving an exostectomy for plantar prominences secondary to Charcot’s arthropathy were casted for a mean 2.4 months. These exostectomies, however, were performed after the patients reached quiescence. The duration to quiescence was not reported. The results of our study indicate that time to unprotected ambulation in acute Charcot’s arthropathy was approximately two to five times longer than that of midfoot fractures in sensate patients without diabetes. The average time of total contact casting (18.5 weeks) is by no means a suggested duration of cast therapy. The range of cast duration in this study (between 1 month to over 1 year) should indicate to the clinician that individual patients with acute Charcot’s arthropathy seldom adhere to a standard treatment timetable. Clinical judgment, coupled with radiographic and thermometric data, should dictate the appropriate casting regimen and progression to prescription footwear.

The patients who underwent prophylactic surgery (25% of the total study population) fell into two categories. The first category contained patients with a relatively stable foot and a readily definable and excisable deformity. These patients underwent exostoses to remove the offending bony apex. This group was casted for far less time than the second category of surgery, which consisted of patients with clinically unstable deformities following coalescence in the post-acute period. In general, surgery in the Charcot’s arthropathy patient should be considered prophylactic in nature. If undertaken, the goal of any procedure should be to enable the patient to successfully return to accommodative footwear.

Only 22% of subjects could recall a specific traumatic
episode prior to the onset of their condition. This is in contrast to an earlier report from Pinzur et al. who reported that 53% of their patient population of Charcot's arthropathy gave a history of trauma prior to presentation. It should be considered that, in many cases of neuropathic arthropathy, a single traumatic event does not take place at all. Rather, it has been suggested that, much in the way a neuropathic plantar ulcer forms, it is repetitive minor trauma to an insensitive region that may precipitate a large percentage of acute neuroarthropathic events. Although neuropathy may have obscured sensation and thus recognition of any specific trauma, nearly three-quarters of the subjects related ill-defined foot pain.

The overall prevalence of acute Charcot's arthropathy among all patients presenting for care at the clinic reviewed during the study period (13%) was higher than in other published reports, in which prevalences ranged from 0.08% to 7.5%. Cofield et al. indicated that in diabetic patients with peripheral neuropathy, the prevalence climbs to 29%. None of these reports make a clear distinction between acute and post-acute Charcot's arthropathy. For this reason, the comparison made may not be entirely accurate. It should also be noted that the study site was a referral-based specialty diabetic foot clinic. However, we believe that our relatively high prevalence is at least partially attributable to heightened awareness of the signs and symptoms of the condition in its acute phase.

The prevalence of bilateral Charcot's arthropathy was 9% in this study population. This is substantially lower than other published studies, which report prevalence of bilateral arthropathy as high as one-third to two-thirds of cases. Perhaps more interesting than prevalence of bilateral acute arthropathy is whether contralateral events occur during treatment. In our study no patients developed contralateral arthropathy. We attribute this to the use of total contact casting. Patients who are placed in these casts are able to ambulate freely during the majority of treatment. This, coupled with reduced stride length and decreased cadence, expose the contralateral extremity to less repetitive trauma that might occur if the patient walked with crutches. These factors combined with frequent monitoring and appropriate prescription footwear may have reduced the risk of precipitation of a bilateral episode.

Following return to permanent footwear, 15% of the population exhibited unilateral elevations in skin temperatures without other objective signs of Charcot's arthropathy, mostly in the first month after returning to permanent footwear. We believe that in the majority of these cases this transient increase in temperature may have been a subclinical vascular response to the additional stress placed on the affected limb. Whether this response was a precursor to a recurrent attack of Charcot's arthropathy is not clear. If it were, the rapid reinstigation of total contact casting, frequent temperature monitoring, and slow reintroduction to footwear was probably instrumental in treating early inflammatory responses and thereby effectively eliminating refractures. This approach is part of a diabetic foot classification and treatment system that was implemented in this specialty foot clinic.

The four patients who had recurrent neuropathic ulcerations during the follow-up period all had neuropathic ulcerations present in the midfoot on initial presentation. It has been reported that, following healing of a neuropathic ulcer, 19% to 58% of patients develop another ulcer within a year. Interestingly, in our study, all recurrent ulcerations occurred in the forefoot. We attribute this to changes in foot structure and mechanics which, with progression of neuropathic osteoarthropathy (and sensorimotor neuropathy in general), may cause clawing of the digits and subsequent planarflexion of the metatarsal heads, increasing their prominence thus predisposing them to neuropathic ulceration.

The most important facet of treatment of diabetic neuropathic osteoarthropathy is prevention. Diabetic patients with sensory neuropathy should be examined regularly for increases in pedal skin temperature. Because temperature assessment by palpation is relatively insensitive and qualitative, the use of infrared dermal thermometry has been employed in our clinic to objectively and quantitatively assess subtle temperature differences. Those at greatest risk must be educated as to the appropriate signs and symptoms of acute Charcot's arthropathy, with any traumatic episode being reported as soon as possible.

With the exception of osteomyelitis, diabetic neuropathic osteoarthropathy is perhaps the most debilitating orthopaedic condition to strike the diabetic lower extremity. Its insidious onset and rapid progression makes early diagnosis and rapid institution of treatment critical to arriving at an acceptable long-term outcome. We have outlined the history and pathogenesis of diabetic neuropathic osteoarthropathy and suggested an evaluation and treatment protocol for the acute form of this devastating disease. The analysis of our treatment methods suggest they are competitively effective. The central tenet to be followed in treatment of acute Charcot's arthropathy is prompt, uncompromising reduction in weight-bearing stress, frequent monitoring, and gradual progression to unprotected weight-bearing in prescription footwear. The ambulatory total contact cast has proven very effective in this endeavour. Regardless of the specific treatment method instituted, it is imperative that appropriate and aggressive treatment be undertaken immediately following diagnosis to help prevent progression to a profoundly debilitating, limb-threatening deformity.

References


