Long-Term Follow-Up in Diabetic Charcot Feet With Spontaneous Onset

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OBJECTIVE — To assess the long-term results after Charcot breakdown with spontaneous onset in diabetic feet.

RESEARCH DESIGN AND METHODS — This study was retrospective. A total of 115 patients (140 feet), 107 with acute deformity and 8 with chronic Charcot deformity, were followed for a median of 48 months (range 6–114). The routine treatment for acute cases was a weight-off regimen with crutches and foot protection with therapeutic shoes until skin temperature had normalized followed by increased weightbearing and the use of bespoke shoes or modification of conventional shoes.

RESULTS — The incidence of Charcot deformity was 0.3%/year in the diabetic population investigated. About half of the patients were active in their jobs. Major complications were encountered in 5 (4%) of the patients that required surgical intervention: arthrodesis for unstable malaligned ankles in 3 subjects (1 bilaterally) and major amputation in 2 subjects for unstable ankle and pressure sores. Minor complications were recorded in 43% of subjects: new attacks of Charcot breakdown in 41 patients (36%) and/or foot ulceration in 43 patients (37%) that required minor surgical procedures for 11 patients. All healed except in 2 patients: 1 patient died before the Charcot fractures had healed, and 1 patient died with an unhealed ulcer. No patient lost the ability to walk independently.

CONCLUSIONS — Major surgical procedures in only 4% were particularly related to patients with Charcot deformities in the ankle. Minor complications were recorded in about half of the patients. Lifelong foot care is required for diabetic patients with Charcot feet.

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Osteoarthropathy (i.e., Charcot foot breakdown) in diabetic patients is considered a risk factor for foot ulceration and for leg amputation. Numerous reports exist regarding small series of selected patients, but only a few larger consecutive series have appeared in recent literature. Moreover, information on late prognosis is sparse. The present study describes early and long-term results in a consecutive series of patients in a diabetic population.

RESEARCH DESIGN AND METHODS

— A retrospective study was carried out with 115 consecutive patients who had 140 feet with evidence of osteoarthropathy and who were treated during a 10-year period (1 January 1984 to 1 January 1994) in the Steno Diabetes Center, which is a hospital specializing in diabetes with a population of 3,000 patients that increased during the study period to 5,000 patients. A total of 94 subjects had type 1 diabetes, and 21 subjects had type 2 diabetes (56 women and 59 men). The age at the time of the Charcot event was a median of 54 years (range 27–80) (Fig. 1). The duration of diabetes for patients at the first Charcot attack was a median of 22 years (0–50) for type 1 diabetes and a median of 8 years (0–19) for type 2 diabetes (Fig. 2). Included were 107 patients presenting a red warm swollen foot with spontaneous onset who exhibited radiological evidence of osteoarthropathy (i.e., fragmentation and osteolysis of bones followed by new bone formation and ankylosis of the small joints involved). Of these 107 patients, 90 had symptoms during the previous 3 months. Moreover, 8 patients with typical Charcot rocker bottom deformity that had developed over a period of some months in adult life and with radiological evidence of Charcot deformity were included in the series. Deformities caused by bone fractures related to accidents were not included.

Treatment occurred in the outpatient clinic. In the case of excessive swelling, a few days of immobilization in bed or in a wheelchair (sometimes in the hospital) was necessary to reduce the edema. The routine treatment was a weight-off regimen involving 2 crutches and foot protection involving therapeutic footwear with a rigid bottom and pedal arch supports (Rathgeber sandals; Rathgeber Bioform GmbH, Heilbronn, Germany), fitted individually with soft insoles molded from functional imprints when necessary. Control of edema was managed with an elastic bandage followed by compression stockings and sometimes assisted by diuretics. The healing of fractures was controlled primarily by measuring the skin temperature at intervals of 2–6 weeks (Thermocouples medical precision thermometer DM 852; Thermocouples, Ellab, Copenhagen). In the acute stage, the temperature of the skin was often 6–8°C higher than the corresponding skin area on the contralateral foot. When the temperature difference during the weight-off period had decreased to about 1–2°C and the edema had subsided, increased weightbearing was allowed. In the case of a recurrent increase in skin temperature and edema, a new period of restricted weightbearing was prescribed. Sequential X-rays at intervals of 6–12 weeks

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A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

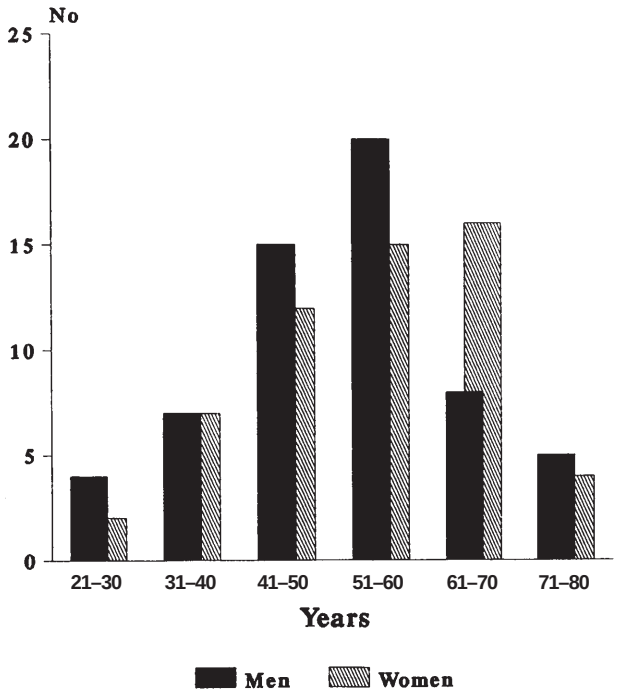

Figure 1—*Age distribution at the initial Charcot attack.*

were also used but were less helpful than temperature measurements in judging progression in healing, deterioration, or new attacks. The weight-off regimen was maintained in most cases for 4–6 months.

New Charcot attacks were defined by the following clinical criteria: a warm swollen foot appearing after at least 6 months of normal weightbearing after a previous attack and subsiding slowly during a period of at least 3 months of off-load. X rays were used to identify whether new skeleton regions were involved.

When full weightbearing in protective shoes had been tolerated for 2 or 3 months without the recurrence of symptoms, bespoke footwear or corrections of conventional shoes were initiated. The shoes had buffer heels, stiffened soles with soft insoles molded from functional imprints, and pedal arch supports. Total-contact cradles were not used. Regular examinations by chiropodists were prescribed for all patients, and the patients were followed twice a year in the multidisciplinary diabetic foot clinic.

During the early period of the study, 3 cases were treated with weightbearing in a total-contact plaster cast. One of these patients developed gangrene from pressure necrosis caused by the cast and required transfemoral amputation. Since then, plaster casts have not been used.

RESULTS

Patient characteristics

The series was evaluated 6 months after the end of the study period and yielded a median observation period of 48 months (6–114).

A total of 53 patients (46%) (30 men, 23 women) were employed, and 62 patients (29 men, 33 women) were pensioners. All patients had peripheral neuropathy determined by clinical examination and biothesiometry. A total of 104 patients had strong pulsations in the pedal arteries. In 11 patients, no pulsations could be detected. Segmental blood pressure measurements demonstrated that the arterial perfusion was normal or only slightly decreased in 9 of those patients (i.e., patients with systolic ankle blood pressures of 120–220 mmHg and toe blood pressures of 75–180 mmHg). In 1 patient only, ischemia was evident with a systolic ankle blood pressure of 70 mmHg and a toe blood pressure of <10 mmHg. The patient's history suggested that arterial occlusion had occurred later than the Charcot attack. In the remaining patient, no measurements were made, but no clinical signs of decreased arterial perfusion were evident.

HbA_{1c} measured at the time of the Charcot attack was a median of 9.4% (5.6–14), which indicates poor glycemic control. Only 14 patients had values <7.5%, which corresponds to average blood glucose values <9 mmol/l.

A total of 31 patients (27%) previously had toe amputations performed, 24 in the

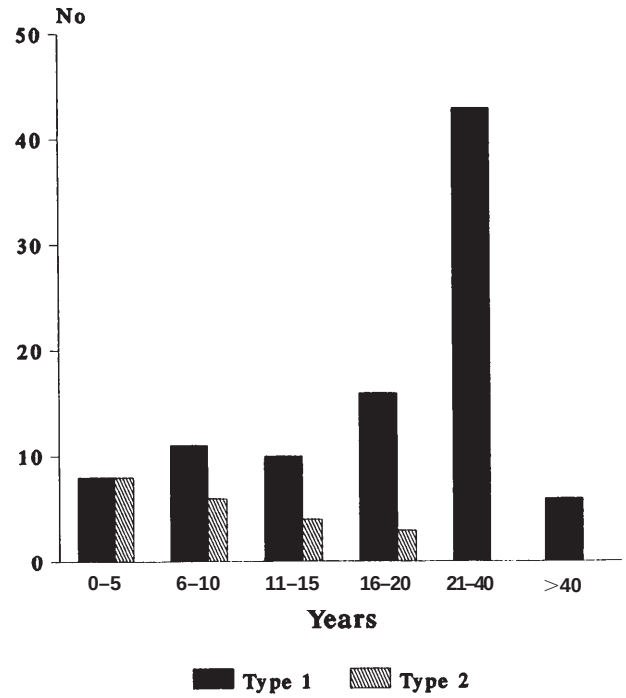

Figure 2—*Duration of diabetes at the initial Charcot attack.*

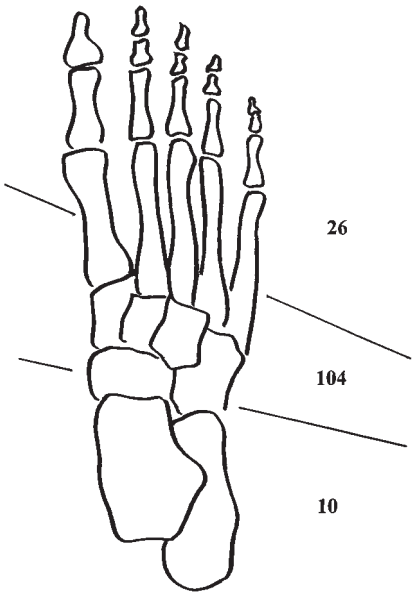

Figure 3—*Localization of first-time Charcot attacks in 140 feet. In 9 feet, the ankle joint was involved in the first attack.*

Charcot foot and 7 in the contralateral foot. A total of 2 patients previously had a below-knee amputation.

Incidence and localization

The diabetes center followed an average of 4,000 patients during the 10-year study period, and the number of first-time Charcot patients was ~11/year, which yields an incidence rate of 0.3%/year.

Figure 3 demonstrates the localization of the fractures. The bones most frequently involved were those forming the top of the pedal arch. Involvement of the ankle joint was found in 9 patients (8%) (10 feet). One of these patients had had multiple midfoot Charcot events throughout a 9-year period. A total of 3 feet had traumatic ankle fractures from 3 to 9 years previously. In 2 patients, the ankle Charcot deformity occurred in relation to mobilization after treatment for hip fractures or tibial fractures.

New Charcot attacks

Only 1 patient had simultaneous bilateral Charcot breakdown that required the use of a wheelchair for 4 months. In 19 patients with bilateral Charcot deformity, the interval between the attacks was a median of 2 years (1–6), and in 5 patients, the interval could not be determined.

More than 1 attack was recorded in 34 patients (30%) (i.e., 24 with nonsimultaneous bilateral Charcot deformity and 10 with new attacks in the previously affected foot). A total of 10 subjects with bilateral and 1 subject with unilateral Charcot deformity (10% total) experienced further attacks (i.e., 3 attacks in 8 patients and multiple attacks in 3 patients). A total of 2 patients with multiple attacks had rheumatoid arthritis.

Among the 20 patients who had more than 1 attack in the same foot, the primary lesion was localized to the forefoot in 5 patients, to the midfoot in 14 patients, and to the hindfoot in 3 patients. Thus, no particular localization was likely to indicate repeated Charcot attacks.

Treatment of Charcot deformity and walking function

The overall results are shown in Tables 1–3. A total of 2 patients died before the fractures healed; 3 patients with severe instability and malalignment of the ankle had tibio-talar fusion by extensive bone resection, realignment, and compression arthrodesis (1 bilaterally and 1 for a first-time attack and 1 for a late new attack). These stabilization procedures were performed between 4 and 17 months after the initiation of the Charcot breakdown. One patient with a painful unstable ankle refused arthrodesis and preferred a transtibial amputation performed 4 months after onset. A total of 5 other patients with ankle involvement were treated either with a leather brace (1 patient) or with bespoke shoes (4 patients). The patient with gangrene resulting from a plaster cast had an above-knee amputation 2 months after the onset of the Charcot attack and was rehabilitated with a prosthesis.

A total of 2 patients with an extreme propensity to develop foot ulcers despite adequate footwear had a wheelchair prescribed for intermittent use, but all surviving 113 patients retained their ability to walk independently (Table 3).

Foot ulcers

A total of 7 patients (6%) developed foot ulcers during a Charcot attack; as previously described, major amputation was required for pressure ulceration resulting from a plaster cast in 1 patient. In 3 patients with malaligned unstable ankles, the ulcerations occurred at the malleoles, and these ankles were treated as described above. Exostectomy was required for healing in 1 patient, and in 2 patients, the ulcers healed during off-loading.

Late ulceration caused by a mismatch between the footwear and the progressive deformity of the foot (often in relation to delivery of new shoes or to poor compliance) was found in 36 patients (31%) in 45 feet after a median of 36 months (6–120 months). One of these ulcers healed after arthrodesis of an unstable ankle. Minor surgical procedures for ulcers were performed in 10 patients (toe amputation in 7 patients and other revisions in 3 patients). The late ulcers healed in all patients except 1 who died before healing.

Table 1—*Healing in 115 patients with Charcot feet*

	Patients	Feet
Charcot foot healed during off-load	108	132
Tibio-talar fusion	3	4
Major amputation	2	2
Deceased	2	2

Data are *n*.

CONCLUSIONS

Pathomechanics

The pathogenesis of the Charcot foot is multifactorial (1,2). Attention has predominantly been paid to the joints as reflected by the terms "osteoarthropathy," "neuropathic joints," or "Charcot's neuroarthropathy." However, stress bone fractures are more likely the essential lesions initiating the Charcot breakdown. Some investigators have described small fractures (cracks) in the bones before complete disintegration (3), and radiographic examinations of diabetic neuropathic feet demonstrate a high frequency of healed fractures, most of which were previously unrecognized by the patients (4,5). Stress bone fractures and sometimes rupture of ligaments are likely to occur because of repetitive stress, possibly in combination with demineralization of bones, foot deformities, and rigidity of the joints. The following recent studies elucidate these factors in the pathomechanism.

Increased stress

Repetitive stress is related to the walking pattern of diabetic individuals. This first came to our attention when, for a short time, total-contact plaster casts were used to treat Charcot feet and neuropathic ulcers. The casts, if not particularly reinforced, broke down after a few days, which suggests abnormally high foot-to-ground forces. Concordantly, increased peak forces

Table 2—*Early and late complications in 115 patients with Charcot foot*

	Patients	Feet
Early complications		
Bilateral simultaneous acute Charcot attack	1	2
Foot ulcer	2	2
Foot ulcer requiring exostectomy	1	1
Unstable ankle (with ulcer)		
Healed after tibio-talar fusion	2	3
Major amputation	1	1
Foot ulcer from a plaster cast with major amputation	1	1
Death before healing	1	1
Late complications		
Contralateral Charcot attack	14	14
Contralateral Charcot attack and recurrence	10	10
Ipsilateral new Charcot attack	9	9
Ipsilateral new Charcot attack requiring ankle arthrodesis	1	1
Foot ulcer	35	44
Deceased with foot ulcer	1	1

Data are *n*.

during heel strike in diabetic patients with peripheral neuropathy have recently been documented (6). The stress is increased by the presence of foot deformities and by rigidity of the joints (7,8) as evidenced by abnormally high peak plantar pressures (9). Finally, several patients reported a period of overuse immediately before the Charcot attack (10), and traumatic bone fractures may also initiate Charcot breakdown (11).

Decreased strength of the skeleton

Osteopenia has been shown radiographically in severe neuropathy (4), and decreased bone density has been demonstrated in patients with Charcot feet (12). Weakening of the bones may be the result of increased blood flow resulting from peripheral neuropathy (13) or may be because of metabolic abnormalities such as the common finding of poorly controlled glycemia in the period preceding the attack. Periods of restricted weightbearing for other conditions such as corrective foot surgery (14), toe amputations, or ulcerations (10) may also weaken the bones.

Thus, ample evidence exists of increased stress and of decreased skeleton strength to explain why diabetic individuals are prone to fatigue bone fractures in the feet.

Table 3—*Footwear, orthoses, and prostheses in 115 patients with Charcot foot*

	Patients
Modified conventional shoes	26
Bespoke shoes or boots	81
Ankle–foot orthosis after an ankle arthrodesis	3
Ankle–foot orthosis for an unstable ankle	1
Leg prosthesis	2
Deceased	2
Total	115

Data are *n*.

Patient characteristics

The localization of the Charcot lesions in this series and the demographic data are in accordance with other studies (1), and our incidence rate of Charcot deformity of 0.3% is within the range previously reported (0.08–0.5%) (1). Normal arterial blood supply in combination with severe neuropathy as well as hyperglycemia preceding the attack are characteristic findings. The high number of younger patients and patients active in their jobs demonstrates that Charcot feet together with other diabetic complications threaten working capacity and quality of life.

Treatment

The total-contact plaster cast is considered by some to be the "gold standard" of treatment of the acute Charcot foot (15–17), but crutches and therapeutic protective shoes (possibly after a few days of using a wheelchair) are adequate in most cases. This is evidenced by the finding that major surgical correction was required in <5% of the patients vs. 25% in the series of Armstrong et al. (16) and 33% in the study of Schon et al. (17). Although deformities were frequent in our series, nearly all cases could be accommodated by corrections with footwear or bespoke shoes. However, in Charcot deformity of the ankle (as found in 8% of the patients), the limb is often seriously threatened and requires major surgical procedures or extensive bracing.

Late complications

In the literature on Charcot feet in diabetic individuals, only a few series have had a significant follow-up time (a median of 3.6 years for 47 cases [15] and a median of 1.8 years for 55 cases [16]). The present study is, to our knowledge, the only consecutive large series with a follow-up period extending to 10 years. Despite careful attention to footwear and follow-up, 54 patients (47%) had new attacks of Charcot deformity and/or foot ulcerations. These complications healed but demonstrate the need for lifelong foot care.

A weight-off regimen with crutches and protective shoes was satisfactory with severe complications in only 4% of patients (i.e., limb loss in 2 patients and tibio-talar fusion in 3 patients mostly related to Charcot deformity in the ankle). New attacks of Charcot deformity and foot ulcerations were frequent but healed. No patient lost the ability to walk, but morbidity is a threat to working capacity and quality of life together with other complications of diabetes.

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