The Diabetic Charcot Foot

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Lee C. Rogers and Robert G. Frykberg

Abstract

The diabetic Charcot foot is a potentially limb-threatening deformity associated with peripheral neuropathy and concomitant injury. Often the precipitating injury is fairly minor, but unrecognized due to the underlying peripheral sensory neuropathy. With existing loss of protective sensation the neuropathic individual continues to walk on the injured extremity causing progressive inflammation with varying degrees of bone and joint pathology. Severe deformity can ensue that predisposes to ulceration, infection, and potential amputation. It is therefore critical to diagnose this condition early in its natural history to prevent progressive foot or ankle deformity and instability.

This chapter reviews the etiology, diagnostic methods, and various treatment options for the active and inactive Charcot arthropathy of the foot and ankle.

Introduction

The Charcot foot is a devastating but oftentimes preventable complication of diabetes with peripheral neuropathy. The condition has several synonyms including Charcot's arthropathy, Charcot joint disease, Charcot syndrome, neuroarthropathy, osteoarthropathy, and many derivations or combinations thereof. It is named after Jean-Martin Charcot (1825–1893), a French neurologist who first described the joint disease associated with tabes dorsalis and named it the "arthropathy of locomotor ataxia." In 1881, J.-M. Charcot presented his findings at the 7th International Medical Congress in London which was attended by many acclaimed physicians of the era. During this meeting the eponym "Charcot's Disease" was designated by Sir James Paget to these degenerative neuropathic changes in bones and joints [1, 2]. Although W. Musgrave in 1703 and later J.K. Mitchell in 1831 ostensibly described osteoarthropathy associated with venereal disease and spinal cord lesions, respectively, Charcot's name remains synonymous with neuropathic arthropathies regardless of etiology [3].

W.R. Jordan in 1936 was the first to fully recognize and report on the association of neuropathic arthropathy with diabetes mellitus [4, 5]. In that comprehensive review of the neuritic manifestations of diabetes, he described a 56-year-old woman with diabetes duration of approximately 14 years who presented with "a rather typical, painless Charcot joint of the ankle." His description typifies the classic presentation we now commonly recognize in patients with long-standing diabetes and neuropathy. Subsequently, Bailey and Root in their 1947 series noted that 1 in 1100 patients with diabetes mellitus developed neurogenic osteoarthropathy [5]. In the classic 1972 Joslin Clinic review of 68,000 patients by Sinha et al., 101 patients were encountered with diabetic Charcot feet [6]. This ratio of 1 case in 680 patients with diabetes brought greater attention to this disorder and characterized the affected patients' clinical and radiographic presentations. In the subsequent 30 years there has been a significant increase in the number of reports on diabetic neuroarthropathy, its complications, and management [4–8]. The prevalence of this condition is highly variable, ranging from 0.15% of all diabetic patients to as high as 29% in a population of only neuropathic diabetic subjects [2, 6, 8, 9]. A prospective study of a large group of patients with diabetes from Texas reported an incidence of 8.5 per thousand per year. Neuroarthropathy was significantly more common in Caucasians than in Mexican Americans (11.7/1000 vs. 6.4/1000) [10]. While this study may give us better insight into the true frequency of neuroarthropathy in diabetes, much of the data we currently rely upon is based upon retrospective studies of small single center cohorts. Nonetheless, the incidence of Charcot foot cases reported is very likely an

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underestimation since many cases go undetected, especially in the early states and cases that receive early appropriate treatment may never be formally diagnosed if the natural history is interrupted [2, 7, 9]. The frequency of diagnosis of the diabetic Charcot foot appears to be increasing as a result of increased awareness of its signs and symptoms [11]. Although the original descriptions of neuropathic osteoarthropathy were attributed to patients with tertiary syphilis, diabetes mellitus has now become the disease most often associated with this severe foot disorder. Not only are patients with Charcot foot deformities at greater risk of amputation than those with neuropathic ulcers but without Charcot foot, a study from the UK has also found them to have a higher mortality [12, 13]. While the power of this study did not allow for significant differences to emerge, it does confirm the need for larger population-based studies to fully elucidate the epidemiology of this limb-threatening complication. Overall, the 4- or 5-year relative mortality rate is 28–45% in those with Charcot foot and diabetes [12, 13]. van Baal reported the life expectancy of someone diagnosed with Charcot foot is 7.9 years in the UK [14].

Etiology and Pathogenesis

Charcot foot can be defined as a noninfectious and progressive condition of single or multiple joints characterized by joint dislocation, pathologic fractures, and severe destruction of the pedal architecture which is closely associated with peripheral neuropathy [2, 7]. Almost uniformly, trauma of some degree when superimposed on the neuropathic extremity precipitates the cascade of events leading to the joint destruction. Neuroarthropathy, therefore, may result in debilitating deformity with subsequent ulceration and even amputation [15, 16]. Charcot foot can result from various disorders which have the potential to cause a peripheral neuropathy. With the decline in numbers of patients with tertiary syphilis since Charcot's time and the concomitant rise in prevalence of diabetes mellitus, the latter disease has now become the primary condition associated with the Charcot foot.

There are several conditions producing radiographic changes similar to Charcot joints. These include acute arthritides, psoriatic arthritis, osteoarthritis, osteomyelitis, osseous tumors, and gout. These joint affectations, in the presence of neuropathy, make the correct diagnosis even more difficult to ascertain [6]. Nonetheless, the characteristics of the joint changes, site for predilection, and clinical correlation assist in determining the true underlying diagnosis.

The primary risk factors for this potentially limb-threatening deformity are the presence of dense peripheral neuropathy, normal circulation, and a history of preceding trauma, often minor in nature and may be unnoticed [15, 17]. There is no apparent predilection for either sex [2]. Trauma is not necessarily limited to typical injuries such as sprains, contusions, or fractures. Foot deformities, prior amputations, and joint infections may result in sufficient stress that can lead to neuroathropathy. Likewise, foot surgery in a patient with neuropathy can result in enough trauma and spark a Charcot event [18]. Renal and/or pancreatic transplantation have also been implicated as an inciting event leading to the development of a Charcot foot [19, 20].

Although the exact pathogenesis may vary from patient to patient, it is undoubtedly multifactorial in nature [17, 21]. The *neurotraumatic* (German) theory has traditionally been proposed as the primary etiology of osteoarthropathy in which neuropathy and repeated trauma produce eventual joint destruction. The loss or diminution of protective sensation allows repetitive micro- or macrotrauma producing intracapsular effusions, ligamentous laxity, and joint instability. With continued use of the injured extremity further degeneration ensues that eventually results in a Charcot joint. Underlying sensory neuropathy resulting from any disorder is therefore a prerequisite under this theory of pathogenesis. However, the neurotraumatic theory does not explain all accounts of Charcot arthropathy, especially its occurrence in bedridden patients [2, 7, 15].

The *neurovascular reflex* (French) theory, in contrast, proposes that increased peripheral blood flow due to autonomic neuropathy leads to hyperemic bone resorption [22]. This theory might indeed correspond to Charcot's original hypothesis of a central "nutritional" defect, although we now recognize this process as a *peripheral* nerve disorder. Autonomic neuropathy (and endothelial dysfunction) results in an impairment of vascular smooth muscle tone and consequently produces a vasodilatory condition in the small arteries of the distal extremities [23, 24]. Impairment of neurogenic vascular responses in patients with diabetic neuropathy has been supported by one study that consequently also showed preserved maximal hyperemic responses to skin heating in patients with Charcot arthropathy [23, 25]. In concert with associated arteriovenous shunting there is a demonstrable increase in bone blood flow in the neuropathic limb. The resultant osteolysis, demineralization, and weakening of bone can predispose to the development of Charcot foot [2, 17, 22, 25–27]. Several studies have demonstrated reduced bone mineral density with an apparent imbalance between the normally linked bone resorption and production in patients with osteoarthropathy [27–29]. Specifically, greater osteoclastic than osteoblastic activity has been noted in acute neuroarthropathy, suggesting an explanation for the excessive bone resorption during the acutely active stage [23, 27].

The actual pathogenesis of Charcot arthropathy most likely is a combined effect of both the neurovascular and neurotraumatic theories [17, 26, 30]. It is generally accepted that trauma superimposed on a well-perfused, but severely

neuropathic, extremity can precipitate the development of an acute Charcot foot. Approximately 50% of those with Charcot foot recall some incipient trauma [31]. But the presence of sensory neuropathy can render the patient unaware of the initial precipitating trauma and often profound osseous destruction takes place during continued ambulation. The concomitant autonomic neuropathy with its associated osteopenia and relative weakness of the bone predisposes it to fracture [23, 28]. A vicious cycle then ensues where the insensate patient continues to walk on the injured foot, thereby allowing further damage to occur [7]. With added trauma and fractures in the face of an abundant hyperemic response to injury, marked inflammation and edema soon follows. Capsular and ligamentous distension or rupture is also a part of this process and leads to the typical joint subluxations and loss of normal pedal architecture culminating in the classic rocker-bottom Charcot foot. The amount of joint destruction and deformity which results is highly dependent upon the time at which the proper diagnosis is made and when non-weight-bearing immobilization is begun [7]. A simplified cycle of the pathogenesis of Charcot joints is illustrated in Fig. 22.1.

Tightening of the posterior leg muscle complex (equinus) may play a special role in the development of the Charcot midfoot deformity. Achilles tendons of those with Charcot foot are morphologically different than disease-matched controls [32, 33]. The pull of the tendon on the calcaneus increases the forces resulting in subluxation or dislocation at the midfoot joints (Fig. 22.2).

Often it is a fracture, either intra-articular or extra-articular, which initiates the destructive process. This had not been fully appreciated until Johnson presented a series of cases in which diabetic patients developed typical Charcot joints after sustaining neuropathic fractures [34]. Additionally, amputation of the great toe or first ray, often a consequence of infection or gangrene in the diabetic patient, may lead to neuropathic joint changes in the lesser metatarsophalangeal (MTP) joints and tarsometatarsal (TMT) joints. Presumably, this is a stress-related factor secondary to an acquired biomechanical imbalance. Intra-articular infection can also be implicated as an inciting event leading to this endpoint. In effect, almost any inflammatory or destructive process introduced to a neuropathic joint has the potential for creating a Charcot joint. Herbst et al. have recently reported

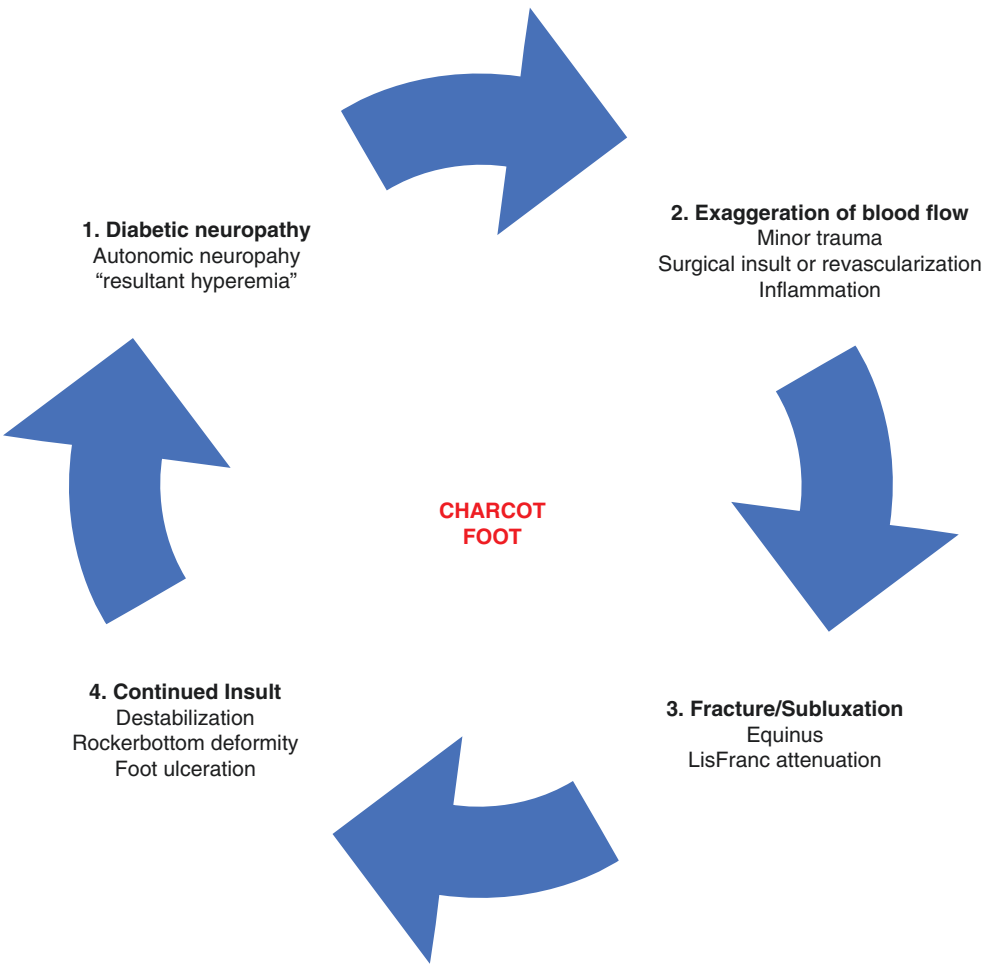

Fig. 22.1 Pathogenic cycle of diabetic neuroarthropathy. (From Lee C. Rogers, with permission)

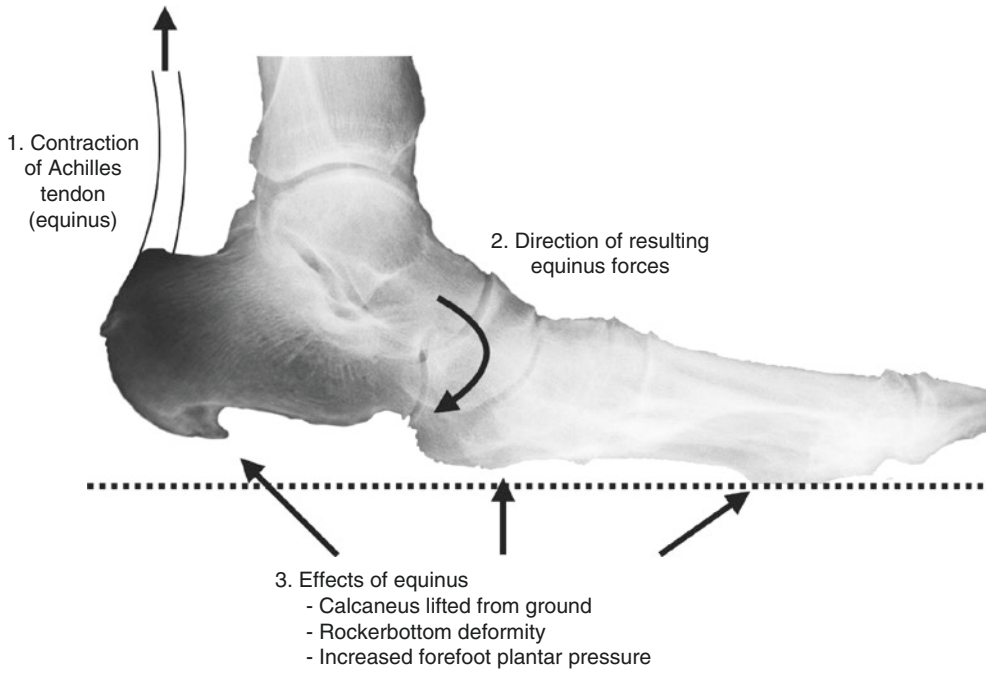

Fig. 22.2 The contribution of the Achilles tendon and equinus to Charcot foot deformity. (From Lee C. Rogers, with permission)

their findings concerning the type of presentation as related to patients' bone mineral density (BMD) [35]. They found that patients with normal BMD had typical changes in the midfoot primarily comprised of joint dislocations. However, in those patients with reduced BMD, fracture patterns predominated in the ankle and forefoot.

Several authors have noted the similarities between the acute destructive phase in Charcot arthropathy and reflex sympathetic dystrophy (complex regional pain syndrome) [23, 24, 36]. Both conditions are associated with an exaggerated vascular response as well as with the development of osteopenia. Both can also be related to previous acute trauma. While the underlying pathophysiological processes are not yet firmly established, both are marked by excessive osteoclastic activity and seem to respond well to treatment with bisphosphonates [36]. Jeffcoate has also suggested that a dysregulation of the RANK-L (receptor activator of nuclear factor kappa B ligand)/OPG (osteoprotegerin) signaling pathway and attendant effects on blood flow and bone turnover might also play a role in this regard [37, 38]. Further study is required, however, to determine how these pathways interact in patients with neuropathy to cause increased vascularity and subsequent osteopenia.

Clinical Presentation

The classic presentation for acute osteoarthropathy includes several characteristic clinical findings which are summarized in Table 22.1. Typically, the patient with a Charcot foot will have had a long duration of diabetes, usually in excess of 12 years. Although all age groups can be affected, a review of the literature in this regard indicates that the majority of patients are in their sixth decade (mid-fifties) [2, 17]. A more recent report, however, indicates that there is an apparent age difference in onset between type 1 and type 2 diabetic patients [39]. Whereas the average age at presentation for the entire cohort and type 2 patients is indeed in the sixth decade, for type 1 patients the age at onset was in the fifth decade (forties). Patients with type 1 diabetes also demonstrated a longer duration of the disease than in type 2 diabetic patients with osteoarthropathy (24 vs. 13 years) [39]. This has also been corroborated by an earlier report from Finland [40]. While unilateral involvement is the most frequent presentation, bilateral Charcot feet can be found in 9–18% of patients [6, 15].

Table 22.1 Clinical features of active Charcot foot

Vascular	Neuropathic	Skeletal	Cutaneous
Bounding pedal pulse	Absent or diminished:	Rocker-bottom deformity	Neuropathic ulcer
Erythema	Pain	Medial tarsal subluxation	Hyperkeratoses
Edema	Vibration	Digital subluxation	Infection
Warmth	Deep tendon reflexes	Rearfoot equinovarus	Gangrene
	Light touch	Hypermobility, crepitus	
	Anhidrosis		

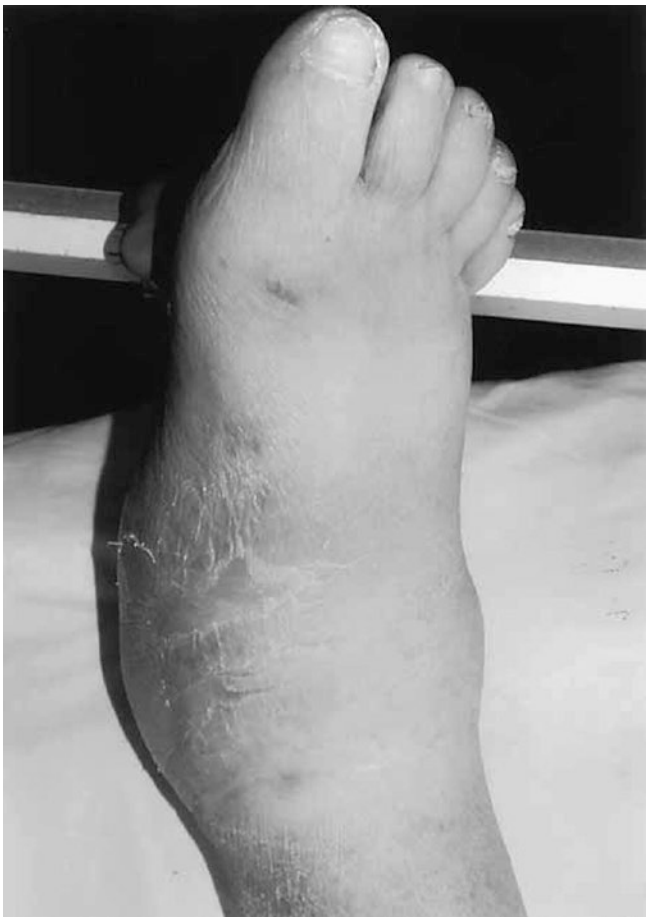

Fig. 22.3 Acute Charcot ankle with profound foot and leg edema

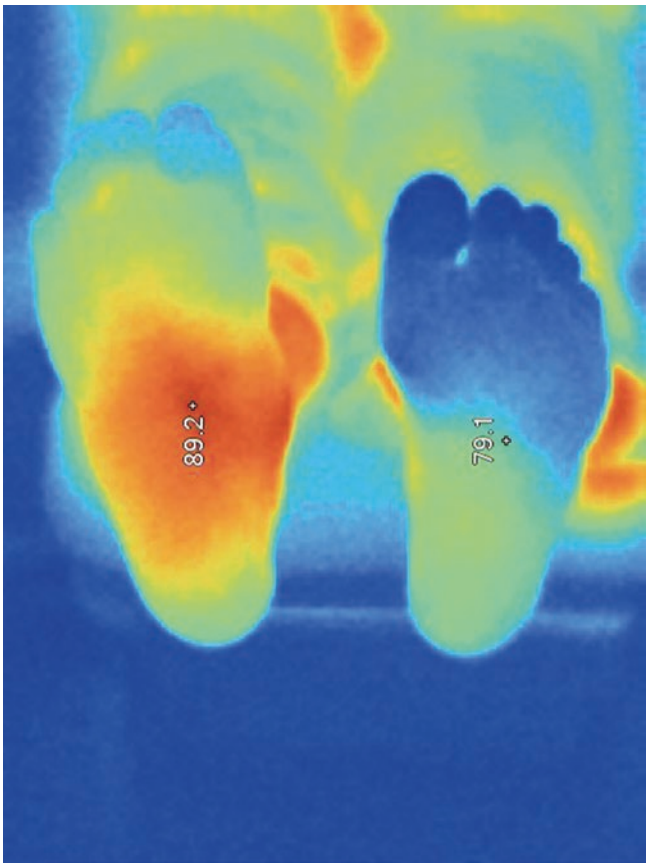

Fig. 22.4 Thermograph of the plantar feet with a significant temperature difference, indicating an active right Charcot foot. (From Lee C. Rogers, with permission)

The initial presentation for acute active Charcot arthropathy is usually quite distinct in that a diabetic patient will seek attention for a profoundly swollen foot that is difficult to fit into a shoe (Fig. 22.3). Although classically described as painless, 75% of these patients will complain of pain or aching in an otherwise insensate foot [15]. Frequently, an antecedent history of some type of injury can be elicited from the patient [31]. When no such history is available, the precipitating event might simply have gone unrecognized in the neuropathic limb.

On examination, the pulses will be characteristically bounding even through the grossly edematous foot [17, 41]. Occasionally, however, the swelling will obscure one or both pedal pulses. In concert with the hyperemic response to injury, the foot will also be somewhat erythematous and warm or hot. The skin temperature elevation can be ascertained by dermal infrared thermometry or thermography and will contrast with the unaffected side by 3–8 °C (Fig. 22.4) [2, 15, 40, 42, 43]. There is always some degree of sensory neuropathy in which reflexes, vibratory sense, proprioception, light touch, and/or pain (pin prick) are either diminished or absent. As mentioned, the patients will most often relate some localized pain although often mild in comparison to the deformity present. Motor neuropathy can present as a foot drop deformity or with intrinsic muscle atrophy. Ankle equinus can sometimes be ascertained initially, but may be difficult to perceive if there is gross osseous deformity and laxity in the midfoot. Autonomic neuropathy, which coexists with somatosensory neuropathy, can be clinically appreciated by the presence of anhidrosis with very dry skin and/or thick callus or by measuring heart rate variability with deep breathing [23, 24]. Another fairly frequent cutaneous finding is a plantar neuropathic ulceration, especially in an active Charcot foot of long duration. A concomitant ulceration will therefore raise questions of potential contiguous osteomyelitis [17, 30, 41].

The skeletal changes frequently manifest as obvious deformity of the medial midfoot with collapse of the arch and/or rocker-bottom deformity (Fig. 22.5) [2, 30]. Associated findings might often include hypermobility with crepitus, significant instability, and ankle deformity.

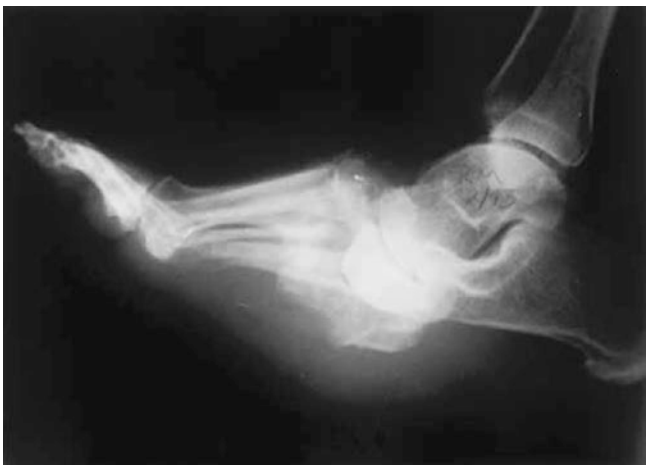

Fig. 22.5 Radiograph of rocker-bottom Charcot foot with collapse of the midfoot

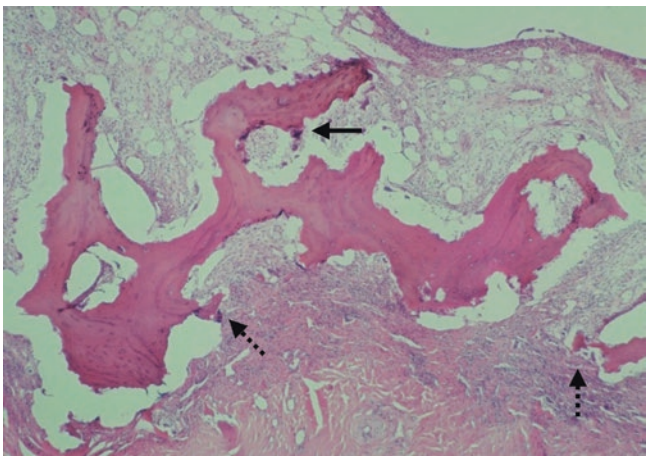

Fig. 22.6 Light micrograph of a pathology slide of bone from a foot with active neuroarthropathy (100×, decalcified, H&E stain). Note the center trabeculum has incongruous edges with osteoclasts (solid arrow), many inflammatory cells, and trabecular fragmentation (broken arrows). (From Lee C. Rogers, with permission)

Diagnosis of Active Charcot Foot

The diagnosis of active Charcot foot is primarily based on history and clinical findings, but should be confirmed with imaging. Inflammation plays a key role in the pathophysiology and is the earliest exam finding [44]. When presented with a warm, swollen, insensate foot, plain radiographs are invaluable in ascertaining the presence of osteoarthropathy [17, 45]. In most cases, no further imaging studies will be required to make the correct diagnosis. However, in the active, prodromal "stage 0" there may be primarily soft tissue changes noted without evidence of distinct bone or joint pathology [46, 47]. Further investigation with scintigraphy, MRI, or serial radiographs should be considered when suspicion is high for osteoarthropathy [48–50]. With a concomitant wound, it may initially be difficult to differentiate between acute Charcot arthropathy and osteomyelitis solely based on plain radiographs [51]. Additional laboratory studies may prove useful in determining the appropriate diagnosis. Leukocytosis can often suggest acute osteomyelitis; however, this normal response to infection can be blunted in persons with diabetes [51, 52]. While the erythrocyte sedimentation rate (ESR) may also be elevated in the case of acute infection, it often responds similarly to any inflammatory process and is therefore nonspecific. When the ulcer probes to bone, a bone biopsy may be helpful in distinguishing between osteomyelitis and osteoarthropathy [17]. A biopsy consisting of multiple shards of bone and soft tissue embedded in the deep layers of synovium is pathognomonic for neuroarthropathy (Fig. 22.6) [53].

Radiographic Imaging

Radiographically, osteoarthropathy takes on the appearance of a severely destructive form of degenerative arthritis. Serial X-rays will customarily demonstrate multiple changes occurring throughout the process and can assist in monitoring disease activity. Rarely will nucleotide scanning, CT, or MRI be necessary to establish the diagnosis. The acute or developmental stage is marked by an abundance of soft tissue edema, osteopenia, multiple fractures, loose bodies, dislocations, or subluxations [30, 54]. These radiographic findings are fairly typical of noninfective bone changes associated with diabetes and have been described well by Newman [55]. In addition to alterations in the normal pedal architecture, the metatarsal heads and phalanges will frequently demonstrate atrophic changes often called diabetic *osteolysis*. Synonyms for this phenomenon include a "sucked candy" appearance, "pencil pointing," "hour glass" deformities of the phalanges, or mortar and pestle deformity of the MTP joints. Massive osteolysis can also occur in the rearfoot during the acute stage, especially in the ankle and subtalar joints (Fig. 22.7). These

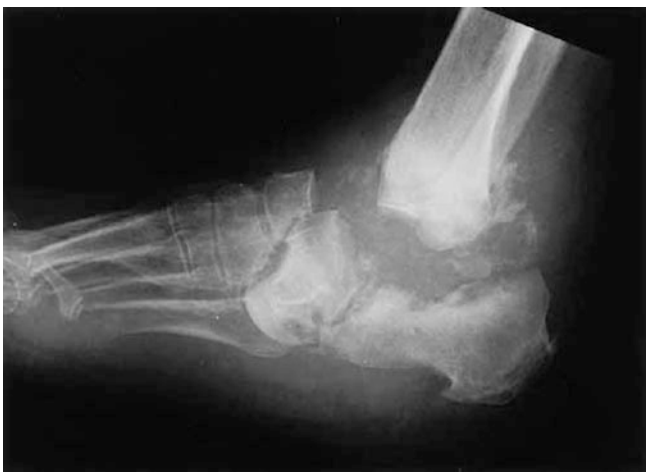

Fig. 22.7 Osteolysis of the talus and disintegration of the ankle and Subtalar joints

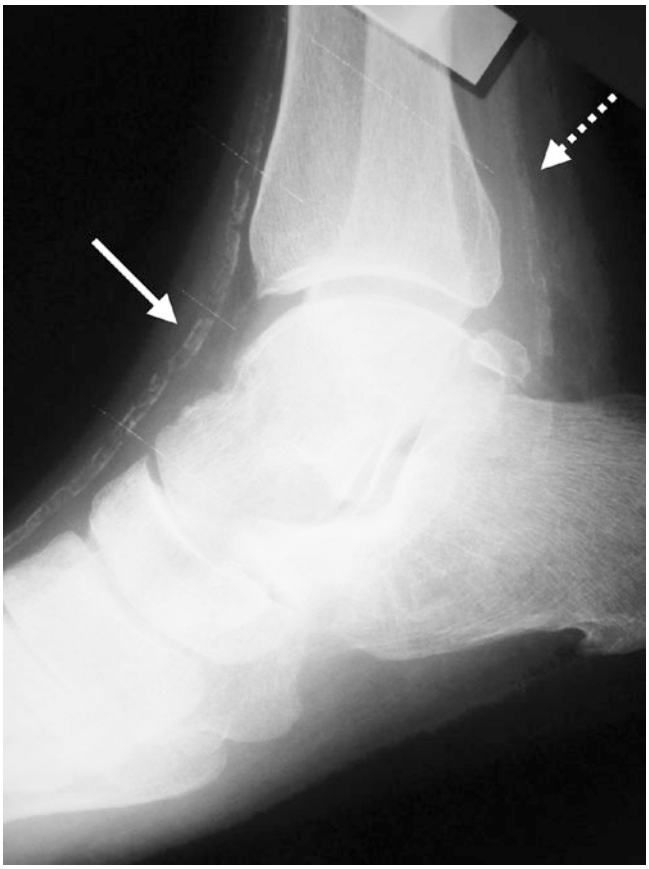

Fig. 22.8 Calcification of the vascular intima media (Monckeberg's sclerosis) can be seen in many patients with Charcot foot. In this lateral ankle radiograph the anterior tibial/dorsalis pedis (solid arrow) and the posterior tibial (broken arrow) arteries are visible. (From Lee C. Rogers, with permission)

changes will often coexist with the obvious fractures that initiated the destructive process. Medial arterial calcification is another associated finding in Charcot arthropathy (Fig. 22.8) [23].

Chronic reparative or quiescent radiographic changes include hypertrophic changes such as periosteal new bone formation, coalescence of fractures and bony fragments, sclerosis, remineralization, and a reduction in soft tissue edema [2, 17, 53]. Rocker-bottom deformities, calcaneal equinus, dropped cuboid, or other deformities not previously appreciated may also become visible, especially when taking weight-bearing images. Lateral weight-bearing foot radiographs are invaluable since they show two important radiographic features of Charcot foot deformities, the calcaneal inclination angle and the talo-first metatarsal relationship. The calcaneal inclination angle (normally 20°) is often reduced or in declination (negative angle). The lateral talo-first metatarsal relationship (a line bisecting the talus and the first metatarsal) should be unbroken (Fig. 22.9). Table 22.2 summarizes the varieties of radiographic changes found in neuroarthropathy.

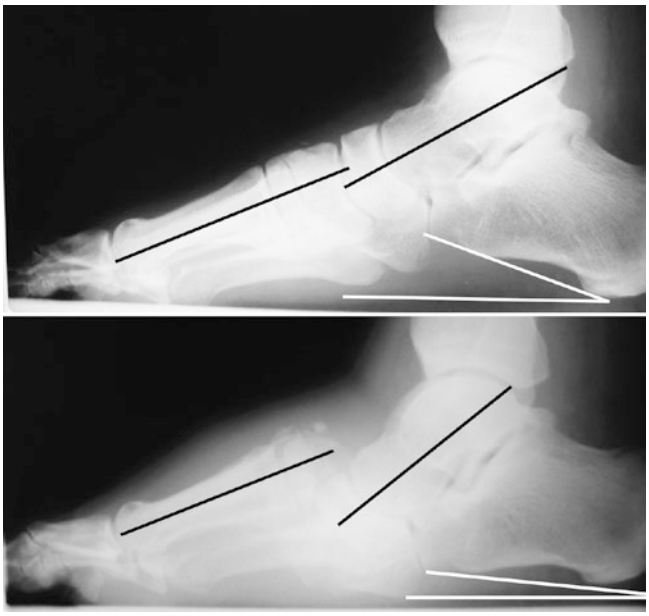

Fig. 22.9 Lateral weight-bearing radiograph of the foot in the same patient before (top) and after (bottom) the development of a rocker-bottom deformity. The calcaneal inclination angle (white line) has decreased and the talo-first metatarsal relationship (black line) is broken in the bottom image. (From Lee C. Rogers, with permission)

Table 22.2 Radiographic changes in neuroarthropathy

Stage	Atrophic changes	Hypertrophic changes	Miscellaneous
Active	Osteolysis – Resorption of bone	Periosteal new bone Intra-articular debris, Joint mice, fragments	Joint effusions Subluxations Fractures
	Metatarsal heads, Phalangeal diaphyses, MTP, subtalar, ankle	Osteophytes, Architectural collapse, Deformity	Soft tissue edema Medial arterial calcification Ulceration
	Osteopenia		
Inactive	Distal metatarsal and rearfoot osteolysis, Bone loss	Periosteal new bone, Marginal osteophytes, Fracture bone callus	Resorption of debris Diminished edema Sclerosis
		Rocker bottom, Midfoot or ankle deformity	Ulceration
		Ankylosis	

Sanders and Frykberg described radiographic patterns of joint involvement based upon joint location in diabetic patients [2]. These patterns may exist independently or in combination with each other as determined through clinical and radiographic findings. They are illustrated in Fig. 22.10 and described as follows: Pattern

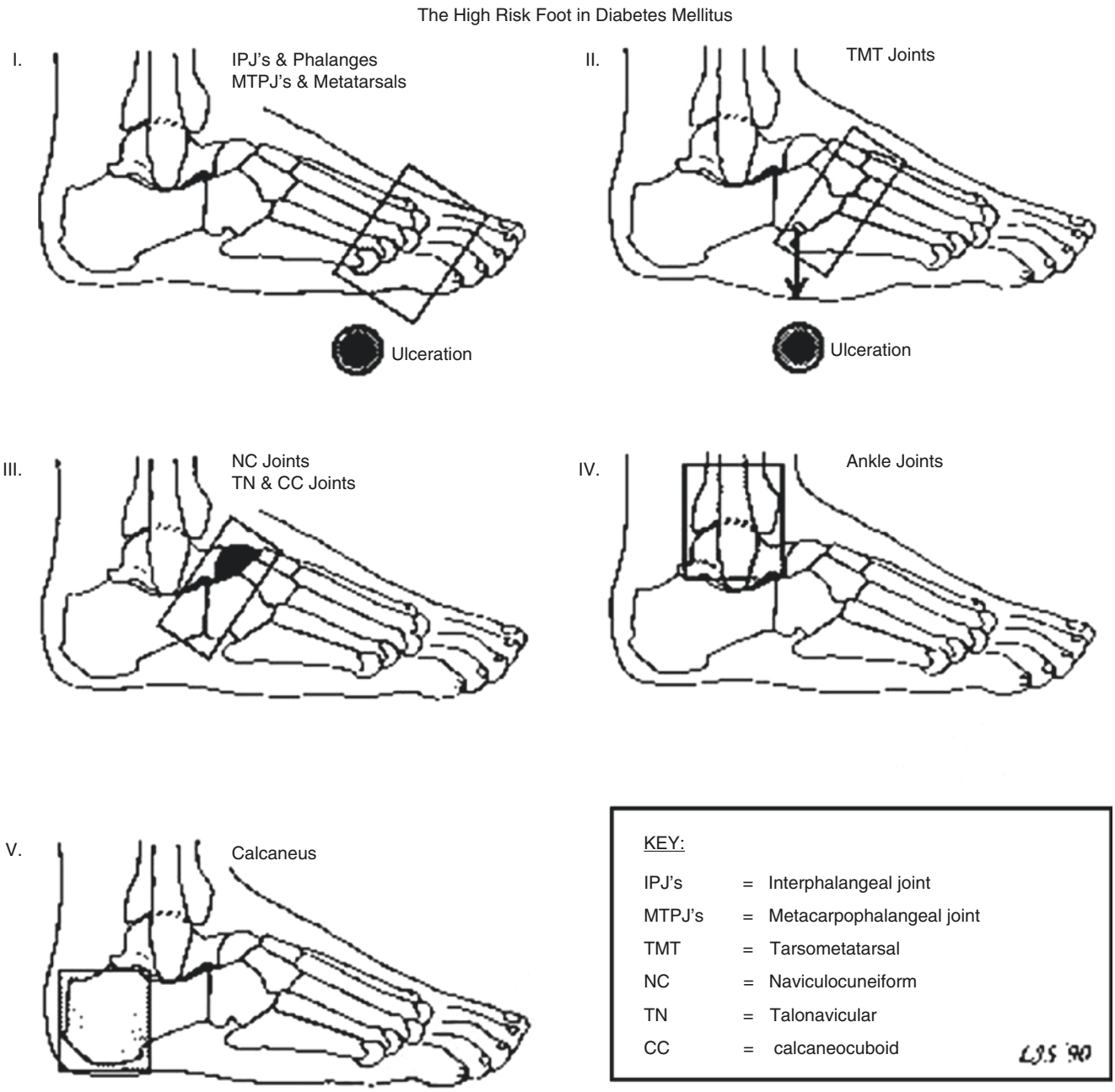

Fig. 22.10 Patterns of diabetic osteoarthropathy based on anatomic sites of involvements. (from Sanders LJ, Frykberg RG. The Charcot foot. In: Frykberg RG, editor. The high risk foot in diabetes mellitus. New York: Churchill Linvingston; 1991. p. 325–35,with permission)

I—Forefoot—Metatarsal-phalangeal joints, Pattern II—Tarsometatarsal (Lisfranc's) joint, Pattern III—Midtarsal and navicular-cuneiform joints, Pattern IV—Ankle and subtalar joints, and Pattern V—Calcaneus (Calcaneal Insufficiency Avulsion Fracture) [2, 29, 30].

Pattern I: Forefoot

Pattern I encompasses atrophic changes or osteolysis of the metatarsophalangeal and interphalangeal joints with the characteristic sucked candy appearance of the distal metatarsals

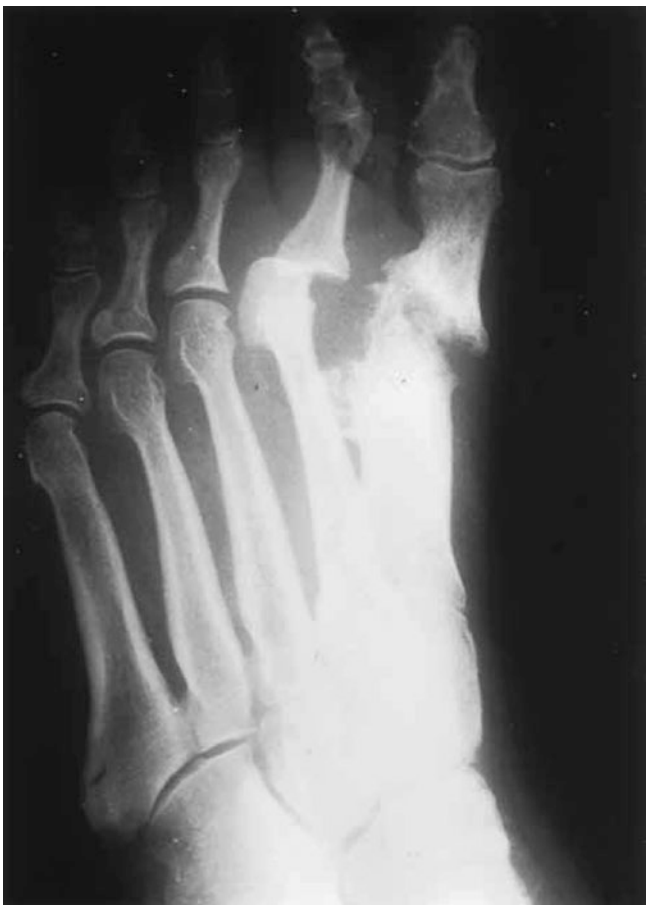

Fig. 22.11 Pattern I: osteolytic changes involving the first metatarsals and phalanx are evident without any current infection documented

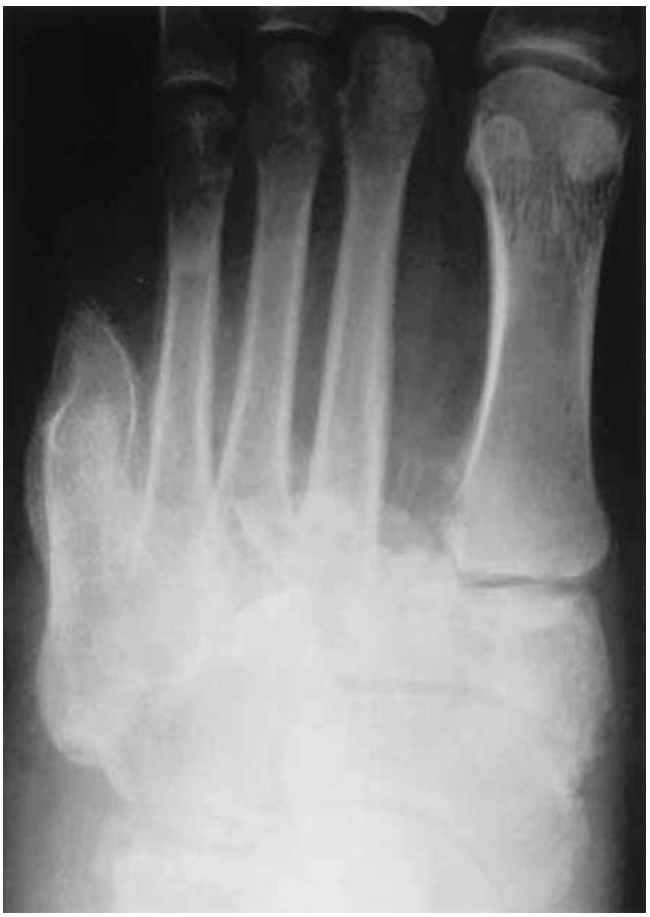

Fig. 22.12 Pattern II: Lisfranc's joint dislocation with associated fractures is evident in this common presentation of the Charcot foot. (Fifth ray had previously been amputated)

(Fig. 22.11) [41]. Frequently, atrophic bone resorption of the distal metatarsals and phalanges accompanies other changes found in the midfoot and rearfoot. An infectious etiology has been proposed for these findings although osteolysis can occur without any prior history of joint sepsis. Reports of 10–30% of the neuroarthropathies have been categorized as Pattern I [6, 22].

Pattern II: Tarsometatarsal (Lisfranc's) Joint

Pattern II involves Lisfranc's joint, typically with the earliest clue being a very subtle lateral deviation of the base of the second metatarsal at the cuneiform joint. Once the stability of this "keystone" is lost, the Lisfranc's joint complex will often subluxate dorsolaterally.

Fracture of the second metatarsal base allows for greater mobility in which subluxation of the metatarsal bases will occur. The rupture of intermetatarsal and tarsometatarsal ligaments plantarly will also allow a collapse of the arch during normal weight-bearing, leading to the classic rocker-bottom deformity. Compensatory contracture of the gastrocnemius muscle will frequently follow and create a further plantarflexory moment to accentuate the inverted arch. This pattern also is commonly associated with plantar ulcerations at the apex of the collapse, which typically involves the cuboid or cuneiforms [2, 17]. This was the most frequent pattern of presentation for diabetic Charcot feet in the Sinha series and represents the most common presentation in clinical practice (Fig. 22.12) [6].

Pattern III: Midtarsal and Naviculocuneiform Joints

Pattern III incorporates changes within the midtarsal (Chopart's) joint with the frequent addition of the naviculocuneiform joint. As described by Newman [55] and Lesko and Maurer [55, 56], spontaneous dislocation of the talona-

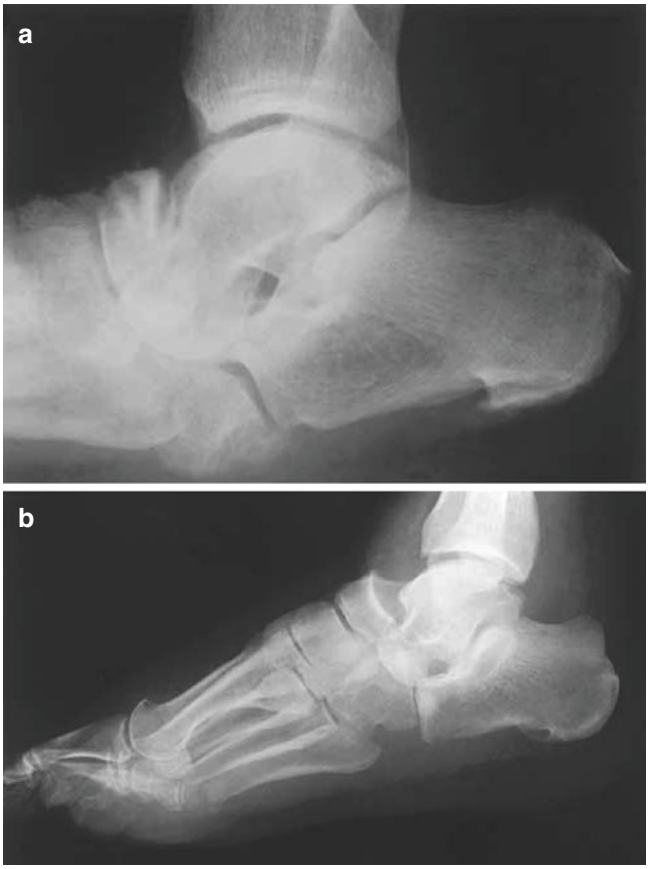

Fig. 22.13 Pattern III: (**a**) Talonavicular dislocation with "dropped cuboid" and plantarflexed calcaneus. (**b**) Talonavicular dislocation with early subtalar and calcaneal-cuboid subluxation. Note absence of fractures or osteochondral defects

vicular joint with or without fragmentation characterizes this pattern. Newman further suggests that isolated talonavicular joint subluxation might even be considered as an entity separate from osteoarthropathy, although still an important element of noninfective neuropathic bone disease [55]. Lisfranc's joint changes (Pattern II) are often seen in combination with Pattern III deformities of the lesser tarsus (Fig. 22.13).

Pattern IV: Ankle and Subtalar Joint

Pattern IV involves the ankle joint, including the subtalar joint and body of the talus (Fig. 22.14). Disintegration of the talar body is equivalent to the central tarsal disintegration of Harris and Brand [57]. The destructive forces are created by joint incongruity and continued mechanical stress which eventually erodes the talus. Massive osteolysis is frequently observed in this pattern with attendant ankle or subtalar subluxation and angular deformity. As noted, tibial or fibular malleolar fractures frequently are seen in association with neuroarthropathy in this location and most likely precipitated

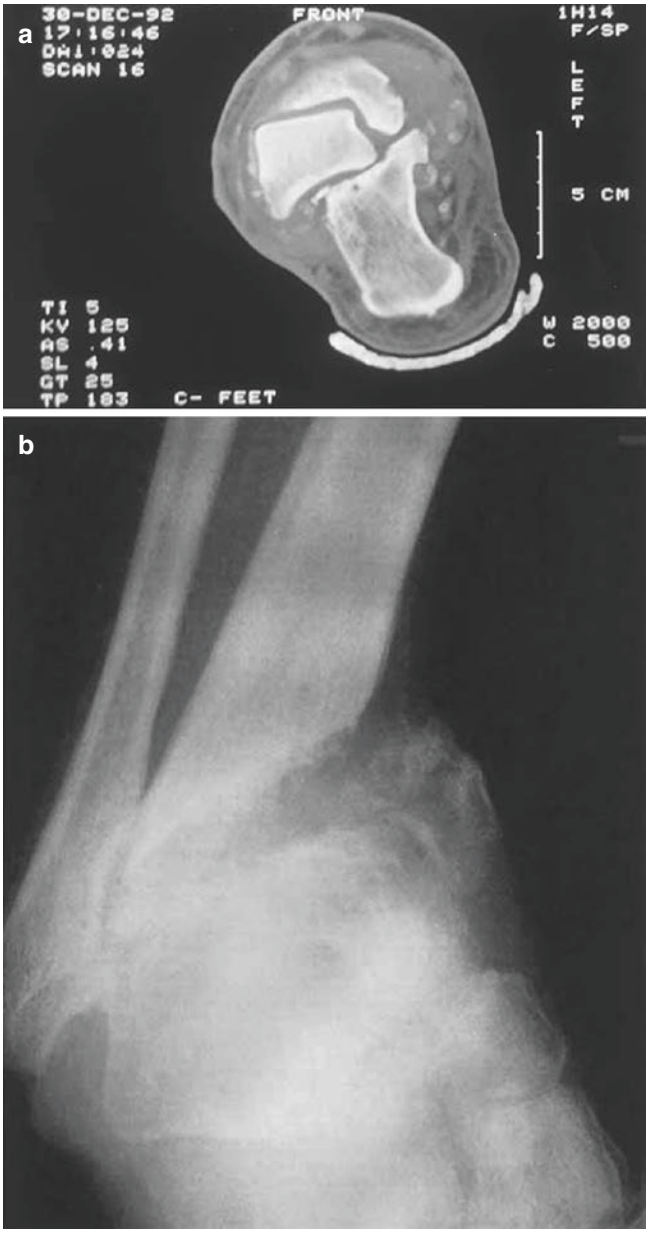

Fig. 22.14 Pattern IV (**a**) Subtalar joint dislocation diagnosed on CT Scan. (**b**) Acute ankle Charcot with medial malleolar fracture and medial displacement of foot

the development of the joint dissolution. Pattern IV Charcot is found in approximately 10% of reported cases [2, 6].

Pattern V: Calcaneus (Calcaneal Insufficiency Avulsion Fracture).

Pattern V, the least common presentation (~2%), is characterized by extra-articular fractures of the calcaneus (posterior pillar). This extra-articular fracture is included in the neuropathic osteoarthropathy classification; however, there is no joint involvement (Fig. 22.15). This is more appropriately

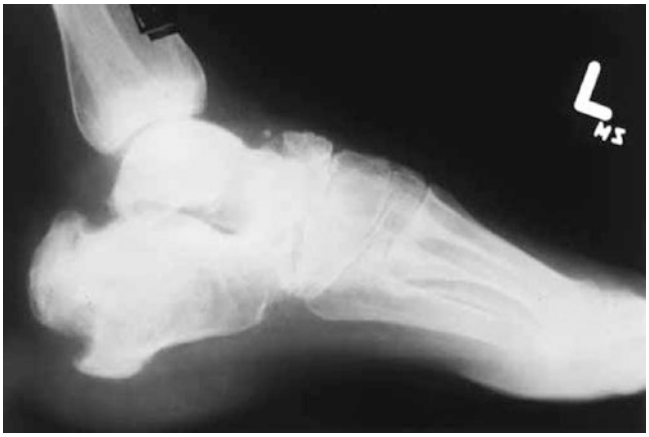

Fig. 22.15 Pattern V: Calcaneal insufficiency avulsion fracture of the calcaneus

considered as a neuropathic fracture of the body or, more commonly, the posterior tuberosity of the calcaneus. El-Khoury and Kathol [58, 59] have termed this entity the "calcaneal insufficiency avulsion fracture."

Advanced Imaging

Technetium (Tc^{99}) bone scans are exquisitely sensitive for detecting Charcot arthropathy but are generally nonspecific in assisting in the differentiation between osteomyelitis and acute neuroarthropathy [48, 60, 61]. Indium (In^{111}) scanning has been shown to be more specific for infection [50, 61–63]. However, false-positive scans can frequently be found in a rapidly evolving acute osteoarthropathy without associated osteomyelitis. Additional studies helpful in differentiating Charcot arthropathy from osteomyelitis include Tc-HMPAO labeled white blood cell scans and magnetic resonance imaging [49, 60, 64, 65].

MRI examination can also be very sensitive to the earliest changes in neuroarthropathy, but again, it is difficult to reliably detect bone infection superimposed upon the gross changes noted surrounding a Charcot joint [49, 51, 60]. Morrison suggests the consideration of "secondary signs" of osteomyelitis may help the clinician discern between Charcot foot and osteomyelitis on MRI [66]. Table 22.3 lists the secondary signs of Charcot foot and osteomyelitis.

Another imaging modality that may show some promise in this regard is positron emission tomography (PET). Hopfner and colleagues recently reported that this modality could not only detect early osteoarthropathy with 95% sensitivity, but could also reliably distinguish between Charcot lesions and osteomyelitis even in the presence of implanted hardware [67]. However, no study is 100% accurate in distinguishing neuropathic bone lesions from infectious entities. Therefore, clinical acumen is necessary for detecting Charcot

Table 22.3 "Secondary signs" of Charcot foot or osteomyelitis on MRI

	Charcot foot	Osteomyelitis
Characteristic	No visible track to bone Primarily affects midfoot	Visible track from skin to bone Primarily affect forefoot and rearfoot
	Multiple bones involved Deformity is common	Usually only one bone affected Deformity is uncommon

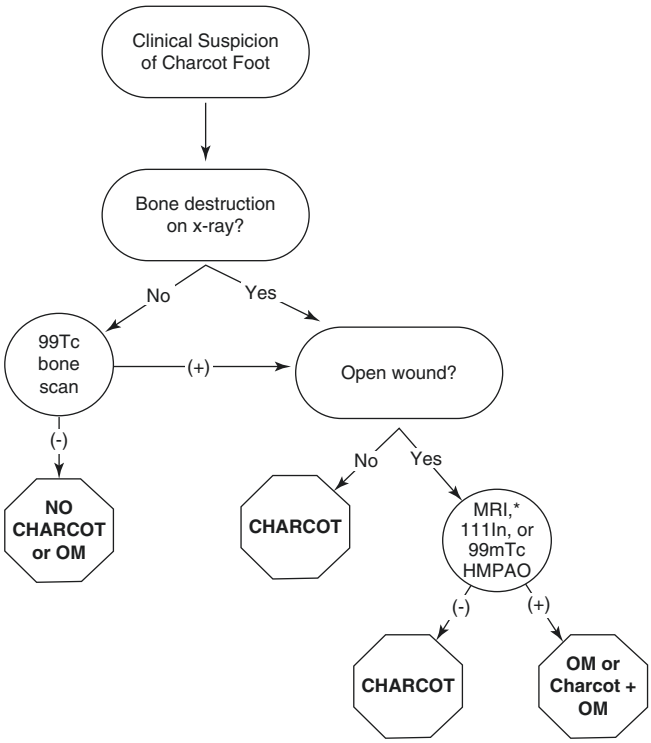

Fig. 22.16 Standard imaging studies to aid in differentiating Charcot foot from osteomyelitis. (From Rogers LC, Bevilacqua NJ. Imaging of the Charcot foot. *Clin Podiatr Med Surg*. 2008;25(2):263–274, vii., with permission)

arthropathy at its onset, and clinical judgment remains of paramount importance in properly assessing and managing these patients. Rogers and Bevilaqua presented a simplified algorithm based on imaging study to help differentiate Charcot foot from osteomyelitis (Fig. 22.16) [51].

Classification of Charcot Arthropathy

The most common classification system of Charcot arthropathy is based on the radiographic appearance as well as physiologic stages of the process. The *Eichenholtz classification* divides osteoarthropathy into developmental, coalescence, and reconstructive stages [53]. Several other authors have subsequently proposed an earlier *Stage 0* that corresponds to

the initial inflammatory period following injury but prior to the development of characteristic bony radiographic changes [46, 68, 69]. This prodromal period might be considered as an "osteoarthropathy in situ" stage. The traditional *developmental* stage is characterized by fractures, debris formation, and fragmentation of cartilage and subchondral bone. This is followed by capsular distension, ligamentous laxity, and varying degrees of subluxation and marked soft tissue swelling. Synovial biopsy at this time will show osseous and cartilaginous debris embedded in a thickened synovium, which is pathognomonic for the disease [53]. The *coalescence* stage is marked by the absorption of much of the fine debris, a reduction in soft tissue swelling, bone callus proliferation, and consolidation of fractures. Finally, the *reconstructive* stage is denoted by bony ankylosis and hypertrophic proliferation with some restoration of stability when this stage is reached. In certain cases, however, severe osseous disintegration occurs due to prolonged activity. In these situations the condition may be referred to as chronically active and little healing, if any, takes place. While the system is radiologically very descriptive and useful, its practical clinical applicability is less so. In clinical practice, the initial developmental stage is considered *active* or *acute*, while the coalescent and reconstructive stages are considered to be the *inactive* or *quiescent* stages. Other classification systems have been described based upon anatomic sites of involvement but do not describe the activity of the disease [46, 57, 70–72]. Rogers and Bevilacqua described a prognostic staging system based on anatomic location and complicating factors of the Charcot joint (Fig. 22.17) [72, 73], which was later validated by Viswanathan et al. in a group of 53 patients [74]. The Sanders and Frykberg classification is descriptive, based on the site of involvement and was described in detail above [2].

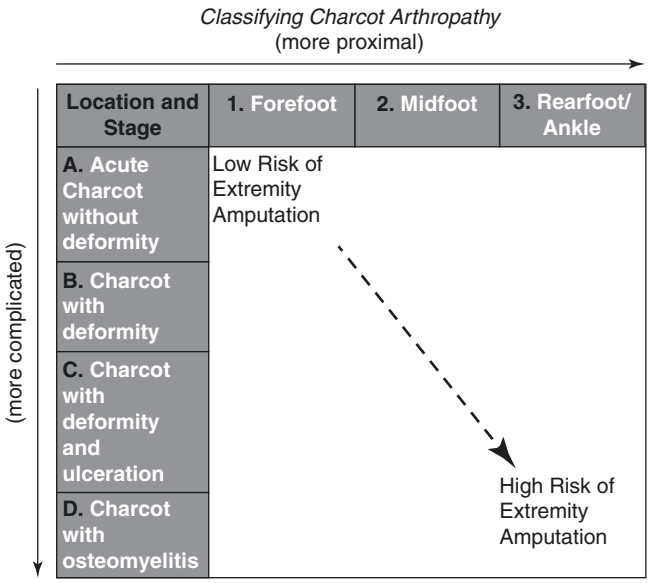

Fig. 22.17 Rogers and Bevilacqua dual axis classification of Charcot foot based upon location and complications. (From Lee C. Rogers, with permission)

The American Diabetes Association and American Podiatric Medical Association created the Joint Task Force on the Charcot Foot comprised of a multinational group of Charcot foot experts in 2010. Given the confusion about classifications, their limited prognostic value, and inability to direct treatment, the Joint Task Force recommended simplifying the clinical classification of the Charcot foot to *active* or *inactive* based on the presence of inflammation [44].

Medical Management

Offloading

Immobilization and reduction of stress are considered the most important treatment for active Charcot arthropathy [17, 44]. Effective offloading or complete non-weight-bearing on the affected limb removes the continual trauma and should promote conversion of the active Charcot joint to the inactive quiescent phase [17, 40, 54]. Non-weight-bearing is an accepted form of offloading for most foot and ankle injuries; however, three point crutch gait may increase pressure to the contralateral limb, thereby predisposing it to repetitive stress and ulceration or an active Charcot episode [56]. Additionally, those with diabetes and neuropathy tend to be older and overweight and do not have the cardiovascular reserve for the additional energy required to use crutches effectively. A patient with neuropathy severe enough to lead to a Charcot foot is likely to have proprioceptive impairment and had an increased risk of falling with offloading treatment, especially crutches. Since total non-weight-bearing is frequently unattainable for many patients in this category, total contact casts (TCC) may serve as a useful alternative and is the most effective form of offloading while bearing weight [75]. TCC can be applied safely in those with Charcot foot, but should be changed frequently at first since edema tends to reduce greatly with immobilization and offloading which will lead to a poorly fitting cast [44]. Figure 22.18 shows the major mechanisms of action of a total contact cast in relieving plantar pressure and immobilizing the foot and ankle. In those patients where a TCC cannot be used, a soft compressive dressing or Unna's Boot in concert with a removable cast walker or pneumatic walking brace can also be used secondarily in this regard [30]. However, a large study in the United Kingdom found that in patients using removable devices took significantly longer to heal versus the TCC group [18]. In the presence of ulcers or infections, frequent debridements and careful observation are required.

Offloading and immobilization should be anticipated for approximately for 6 months or more, depending on the severity of joint destruction. Conversion to the inactive/reparative phase is deduced by a reduction in pedal temperature to

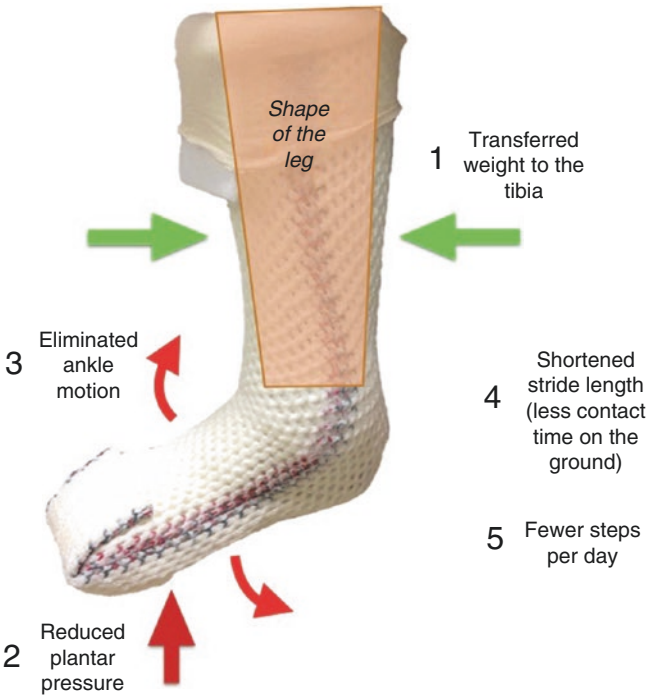

Fig. 22.18 An image of a total contact cast (TCC) depicting the major mechanisms of action in offloading and immobilization. (From Lee C. Rogers, with permission)

Table 22.4 Offloading/immobilizing devices used in the management of Charcot feet

• Total contact cast (TCC)
• Wheelchair
• Crutches
• Rolling knee walker
• Removable cast walker (RCW)
• Patellar tendon-bearing orthosis (PTBO)
• Charcot restraint orthotic walker (CROW)

within 4 °F (2.5 °C) of that of the unaffected side and a sustained reduction in edema [15]. This should be corroborated with serial radiographs indicating consolidation of osseous debris, union of fractures, and a reduction in soft tissue edema. McGill et al. have found a reduction in skin temperature and bone scan activity that mirrors activity of Charcot neuroarthropathy, both of which improve as the condition achieves the inactive stage or quiescence [43].

When the patient enters the inactive stage, management is directed at a gradual resumption of weight-bearing with prolonged or permanent bracing [15, 17, 44]. Care must be taken to gradually wean the patient from non-weight-bearing (or TCC) to partial to full weight-bearing with the use of assistive devices (i.e., crutches, cane, or walker). Progression to *protected* weight-bearing is permitted, usually with the aid of some type of ambulatory, immobilizing device (Table 22.4) [76]. Charcot restraint orthotic walkers ("CROW") or other similar total contact prosthetic walkers have gained acceptance as useful protective modalities for the initial period of weight-bearing after TCC [77]. These custom-made braces usually incorporate some degree of patellar tendon bearing as well as a custom footbed with a rocker sole. A more readily available option is a pneumatic walking brace or similar removable cast walker that might incorporate a cushioned footbed or insole. These can be made less-removable or non-removable by simply applying adhesive tape or cast bandaging around the body of the brace to help encourage compliance (Fig. 22.19) [78].

The mean time of rest and immobilization (casting followed by removable cast walker) prior to return to permanent protective footwear is approximately 4–12 months [15, 18, 40]. Feet must be closely monitored during the time of transition to permanent footwear to insure that the acute inflammatory process does not recur. Forefoot and midfoot deformities often do well with custom full-length inserts and comfort or extra-depth shoes once bracing is no longer

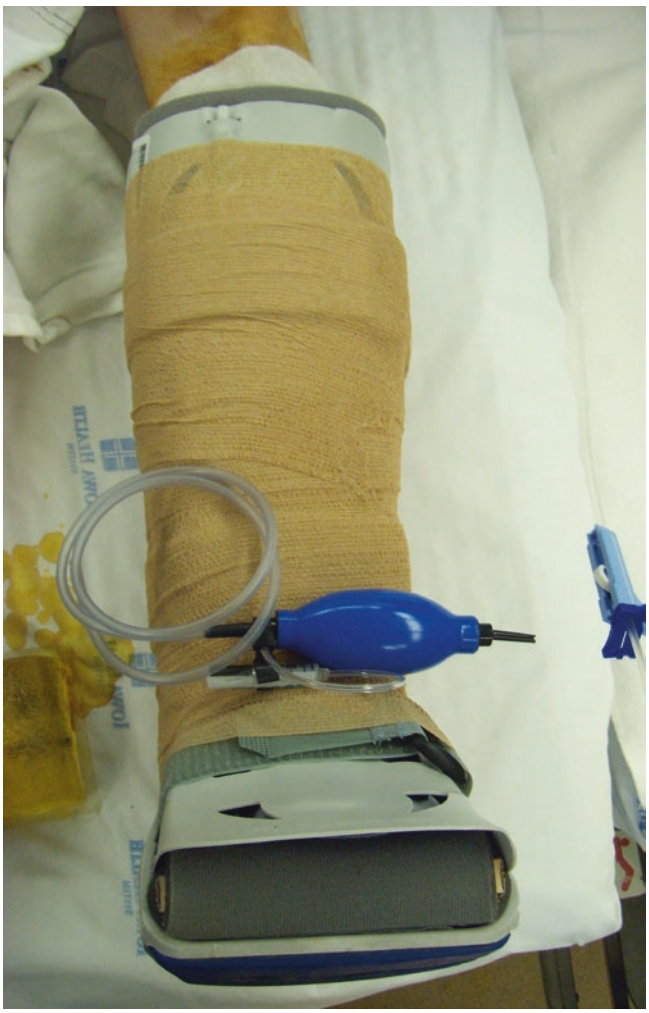

Fig. 22.19 A removable cast walker (RCW) rendered "less removable" with an external layer of cohesive bandage. (From Lee C. Rogers, with permission)

required [17]. Continuing effective offloading with non-custom bracing or TCC often serve as interim footwear prior to obtaining permanent custom-made footwear. Severe midfoot deformities will often require the fabrication of custom shoes to accommodate the misshapen foot. Rearfoot neuroarthropathy with minimal deformity may require only a deep, well-cushioned shoe with a full-length orthotic device. For mildly unstable ankles without severe deformity or joint dissolution, high-top custom shoes can sometimes provide adequate stability against transverse plane rotational forces. The moderately unstable ankle will benefit from an ankle foot orthosis (AFO) and a high-top therapeutic shoe. The severely unstable or maligned rearfoot will require a patellar tendon bearing (PTB) brace incorporated into a custom shoe [79, 80]. The PTB brace has reportedly decreased the rearfoot mean peak forces by at least 32% [80].

Anti-resorptive Therapy

In the setting of altered bone mineral density (BMD) in patients with diabetes and neuropathy, there has been recent interest in the adjunctive use of bisphosphonate therapy in acute Charcot arthropathy [28, 36, 81, 82]. However, further study has cast a negative shadow on their routine use for Charcot foot [18] and it is not recommended by the Joint Task Force [44]. These pyrophosphate analogs are potent inhibitors of osteoclastic bone resorption and are widely used in the treatment of osteoporosis, Paget's disease, and reflex sympathetic dystrophy syndrome. Although one uncontrolled study of six patients found significant reductions in foot temperature and alkaline phosphatase levels as compared to baseline, its small size and lack of a control group preclude making any meaningful conclusions from the treatment [82]. A subsequent multicenter randomized trial in the UK from this same group was performed using a single intravenous infusion of pamidronate compared to saline infusion [36, 82]. The treatment group had significant falls in temperature and markers of bone turnover (deoxypyridinoline crosslinks and bone specific alkaline phosphatase) in subsequent weeks as contrasted to the control subjects. However, no differences in clinical or radiographic outcomes were reported. Trials of oral bisphosphonates with alendronate have been done but the effects of the treatment take up to 6 months which is not likely sufficient in this limb-threatening disorder requiring more urgent action [83]. Until definitive controlled outcome studies are performed which concurrently measure serum markers of osteoclastic activity and attempt to assess improvements in clinical and radiological healing, and based on further clinical outcomes study, the routine use of bisphosphonate therapy should be avoided.

Another pharmacologic agent interrupting the bone resorptive pathway which has been investigated in Charcot foot is intranasal calcitonin. It is often used for osteoporosis has been shown to reduce markers of bone turnover and foot temperature differences in Charcot foot [84]. Some have theorized that it has a direct effect on RANK-L and may interrupt the deposition of calcium from the bone to the intima media of the blood vessels [37].

Bone Stimulators

Another modality which has been applied to the management of acute neuroarthropathy is the use of bone stimulation [85–87]. In one study of 31 subjects randomized to either casting alone or cast with Combined Magnetic Field (CMF) electrical bone stimulation, there was a significant reduction in time to consolidation of the Charcot joints in the study group (11 vs. 24 weeks) [86]. Low intensity pulsed ultrasound (LIPUS) has also been suggested as a useful adjunct in promoting healing of Charcot fractures, although this report only presented two cases of patients successfully treated after undergoing revisional surgery for recalcitrant deformities [88]. While both types of modalities have been proven successful in healing chronic nonunions or even fresh fractures (in the case of LIPUS), their efficacy in promoting prompt healing of acute Charcot fractures or union of surgical arthrodeses has yet to be proven by large, well-controlled randomized clinical trials. Direct current implantable bone stimulators have shown benefit in Charcot foot reconstruction with arthrodesis [89].

Surgical Treatment

The Charcot foot should not be considered as primarily a surgical disorder, with a few exceptions. There is an abundance of support in the literature confirming the need for initial attempts at medical treatment, including offloading, to arrest the destructive process by converting the active Charcot joint to its inactive state [17, 40, 44, 79]. The Joint Task Force produced a treatment algorithm when considering nonsurgical versus surgical treatment (Fig. 22.20). As indicated by Johnson in 1967, the three keys to treatment of this disorder should be prevention first, followed by early recognition, and once diagnosed, protection from further injury until all signs of "reaction" have subsided [34]. Surgery should be contemplated when attempts at medical treatment as previously outlined have failed to provide a stable, plantigrade foot or in cases of gross dislocation. Additionally, when uncontrollable shearing forces result in recurrent plantar ulcerations or in those unusual cases that demonstrate continued destruction despite non-weight-bearing, procedures such as simple bone resections, osteotomy, midfoot or major tarsal reconstruction, and ankle

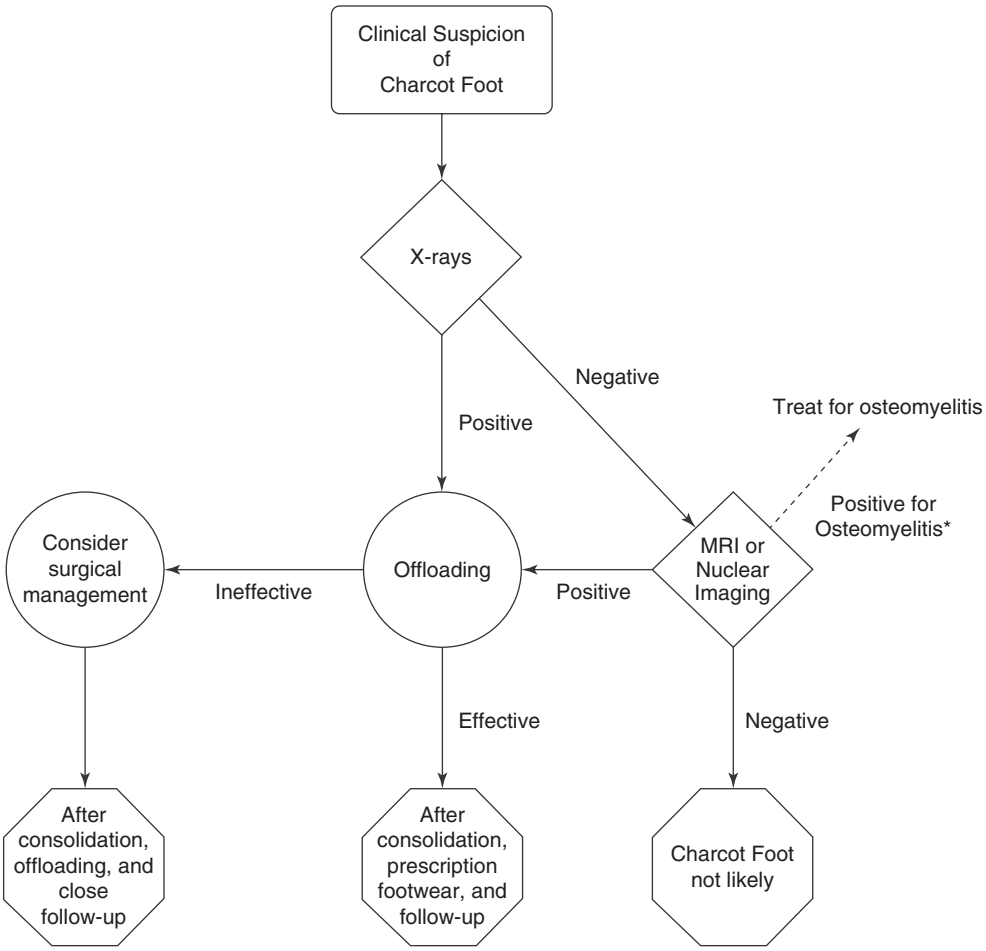

Fig. 22.20 The American Diabetes Association/ American Podiatric Medical Association Charcot Foot Joint Task Force treatment algorithm. (From Rogers LC, et al. The Charcot foot in diabetes. J Am Podiatr Med Assoc 2011;101:437–46, with permission)

arthrodesis might become necessary [44]. However, a recent review of one center's experience with midfoot neuroarthropathy in 198 patients (201 feet) indicated that more than half of these patients could be successfully managed without the need for surgery [76].

Although becoming more common in clinical practice, surgery on the Charcot foot is not a new concept, but there is still little good quality evidence to show its value [90]. Steindler, in 1931, first reviewed his series of operative results in tabetic patients including one subtalar arthrodesis [91]. He, like Samilson [92], Harris and Brand [57], and Johnson [34] many years later, recommended early recognition of the arthropathy, immediate protection from external deforming forces, and early operative stabilization when significant malalignment and instability precluded further conservative treatment. Samilson in 1959 [92] and Heiple in 1966 [93] were early to recognize the necessity for compressive internal fixation and prolonged immobilization in effectuating a solid bony fusion.

Harris and Brand in 1966 provided insight into this disorder associated with leprosy and described their five patterns of "disintegration of the tarsus" [57]. Full immobilization was always deemed imperative as an initial treatment; however, when progression continued or an unsatisfactory result was obtained, early surgical fusion was advocated. One year later Johnson published his large series which established the need for early recognition and protection to allow the acute inflammatory response to subside prior to surgical intervention [34]. As he stated, "Appropriate surgery on neuropathic joints, performed according to these principles, should be undertaken with great respect for the magnitude of the problem but not with dread." Johnson clearly favored osteotomy or arthrodesis in selected patients with quiescent Charcot joints and deformity in order to restore more normal alignment. Since the trauma of surgery could result in further absorption of bone during the acute stage, great emphasis was placed on resting the part until there was clinical and radiographic evidence of repair. Only then could surgery be attempted with a favorable chance for success [34].

Indications and Criteria

Instability, gross deformity, and progressive destruction despite immobilization are the primary indications for surgical intervention in neuroarthropathy [17, 34, 44, 94].

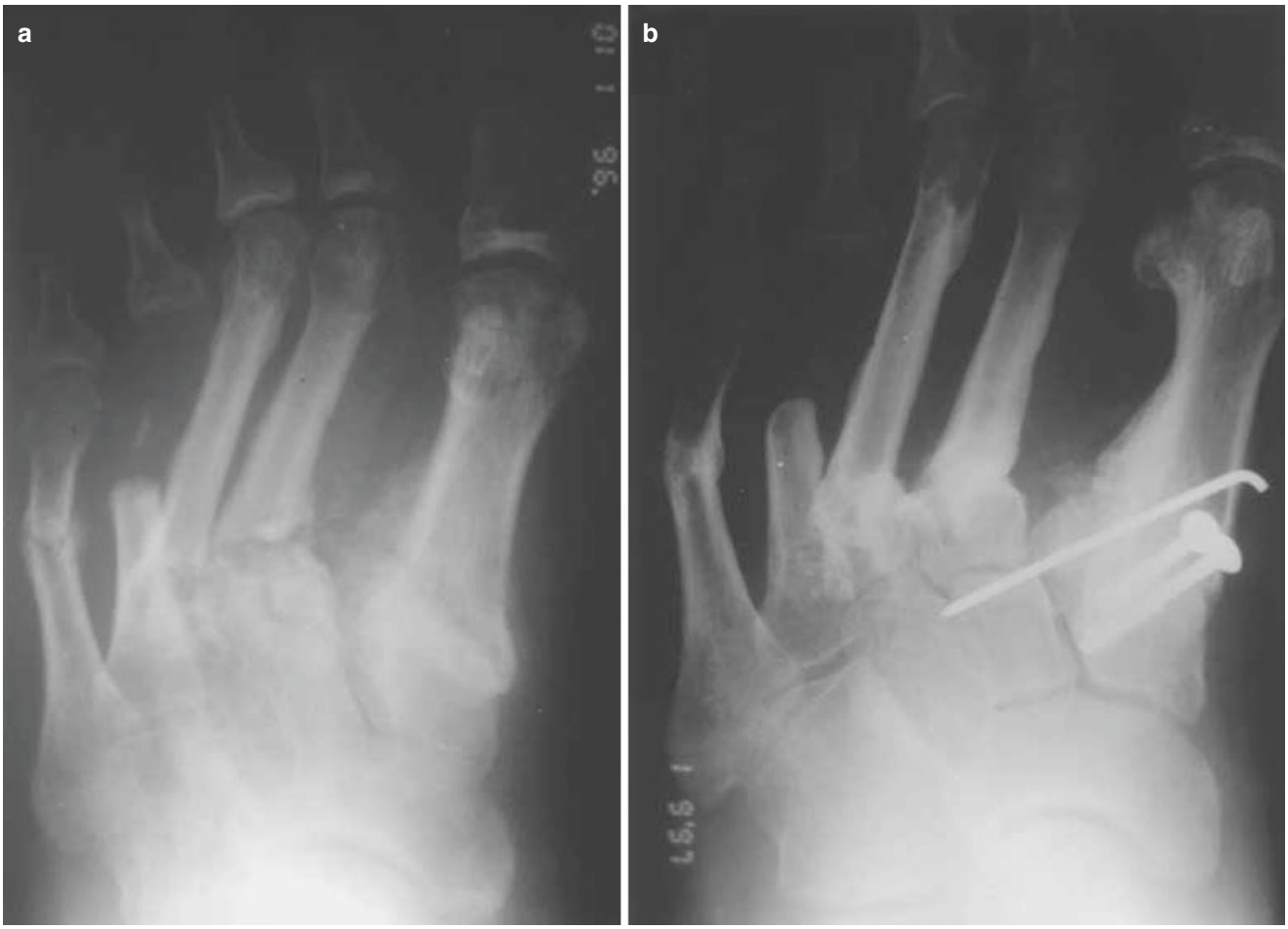

Fig. 22.21 (**a**) Preoperative X-ray of patient with dorsally dislocated first metatarsal-cuneiform joint and several metatarsal fractures. (**b**) Stability, resolution of symptoms, and complete healing was achieved with a limited arthrodesis of the first ray

Additionally, recurrent ulceration overlying resultant bony prominences of the collapsed rear, mid, and forefoot may require partial ostectomy to effect final healing when performed in conjunction with appropriate footwear therapy [95, 96]. Pain or varying degrees of discomfort will frequently accompany the deformity and may be refractory to medical treatment in some patients. Attributable to chronic instability, this can be effectively eliminated by limited arthrodeses at the primary focus of the neuroarthropathy (Fig. 22.21).

Lesko and Maurer[56] and Newman [55, 97] in their considerations of spontaneous peritalar dislocations advocate primary arthrodesis in those acute cases where there is a reducible luxation in the absence of significant osseous destruction. Since these luxations may be the initial event in the sequence leading to typical osteoarthropathy, early intervention following a period of non-weight-bearing has been recommended to counteract forces which would most likely lead to further progression of the deformity.

Age and overall medical status should also weigh heavily in the decision regarding suitability for surgery. Recognizing that arthrodesis and major reconstructions will require cast immobilization and non-weight-bearing for 6 months or more, selection of the appropriate patient is critical to a successful outcome [98–100]. Since the majority of patients with osteoarthropathy are in their sixth to seventh decades and may likely have coexistent cardiovascular or renal disease, careful consideration must be given to the risk versus benefit of lengthy operative procedures and the attendant prolonged recuperation [42]. As mentioned, a simple bone resection or limited arthrodesis might suffice in an older patient with a rocker-bottom deformity prone to ulceration as opposed to a complete reconstruction of the midfoot [96, 101]. The former procedures can be done under local anesthesia relatively quickly, require a shorter convalescence, are prone to fewer complications, and can provide a stable, ulcer-free foot when maintained in protective footwear. Nevertheless, major foot reconstructions and arthrodeses are certainly indicated in those healthier patients with severe deformity, instability, or recurrent ulcerations who have not satisfactorily responded to medical treatment [44, 100] (Fig. 22.22). In all cases, however, the patient must be well

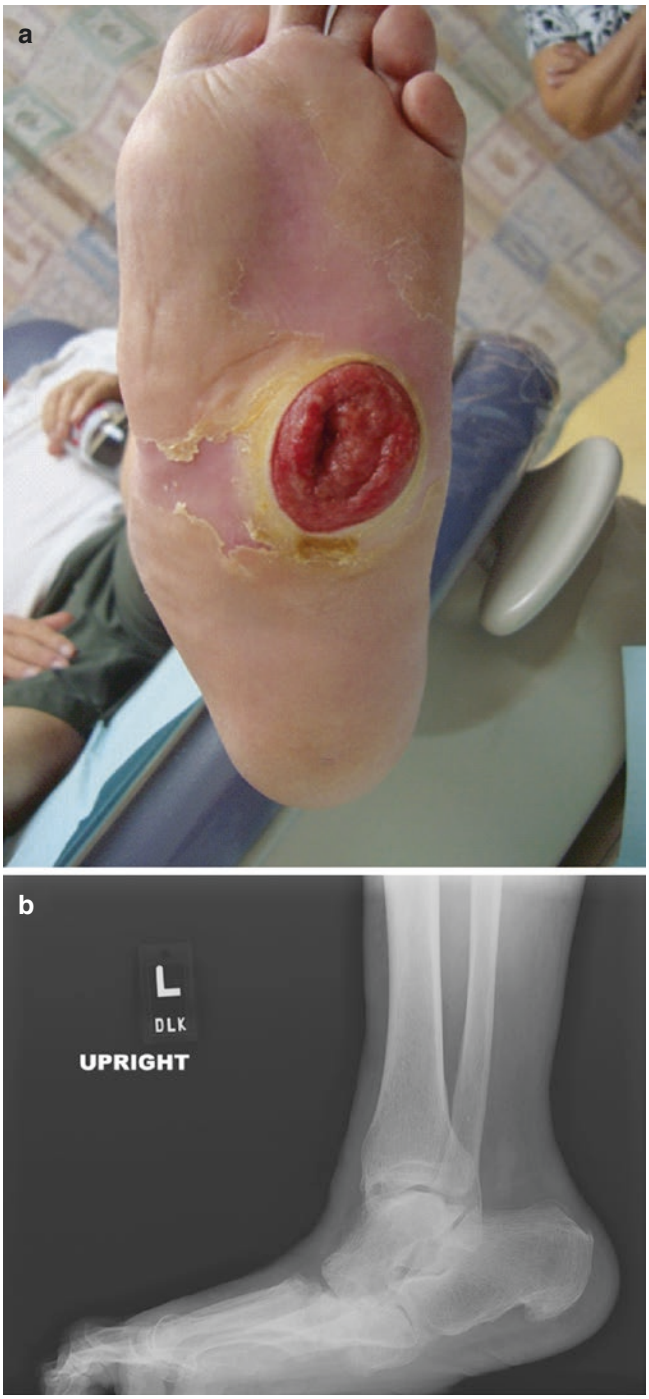

Fig. 22.22 (**a**) This patient has a chronic midfoot ulcer associated with a rocker-bottom deformity. (**b**) Radiograph of same patient showing severe rearfoot equinus and midfoot deformity

educated as to the necessity for strict compliance with postoperative immobilization and non-weight-bearing or partial weight-bearing for as long as 6–12 months.

An acute deformity, either a spontaneous dislocation or the more advanced fracture-dislocation paradigmatic of neuroarthropathy, is generally rested and immobilized prior to any attempted surgery. Surgery during the active stage has the potential to compound and exacerbate the bone atrophy indicative of this inflammatory stage of destruction. Hence, it may be counterproductive as well as detrimental to operate on these feet until they have been converted to the quiescent, reparative stage. One small series, however, indicates successful arthrodesis rates with preserved foot function in patients with acute arthropathy of the midfoot [102]. Others have also advocated early operative repair with arthrodesis during stage 0 or stage 1, especially when nonoperative treatment has failed to prevent further deformity or arrest the destructive process [103–105]. Notwithstanding, this aggressive surgical approach needs confirmation through larger comparative trials prior to its adoption in the routine management of the acute Charcot foot.

Surgical Procedures

Surgery performed primarily on chronic Charcot feet has met with increased success in recent years as experience develops and improvements in fixation are made. With an average union rate of 70% and improved alignment with stability, surgery on the Charcot foot has the potential not only to save limbs but also to improve quality of life [45]. Surgical correction of the Charcot foot can be segregated based on complexity, with the simpler surgeries having fewer complications (Fig. 22.23).

Ostectomy of plantar prominences in the face of recalcitrant or recurrent neuropathic ulceration is perhaps the most frequent procedure performed on Charcot feet [96]. Such operations are fairly easy to perform and do not generally require lengthy periods of immobilization beyond attaining wound closure. Surgical approaches are varied, with direct excision of ulcers by ellipse or rotational local flaps predominating. Alternative incisions are performed adjacent to ulcers or prominences, either through a medial or lateral approach. One report suggests that excision of medial plantar prominences fare better and with fewer complications

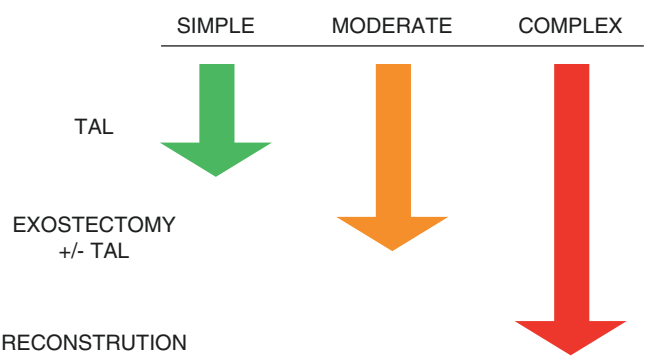

Fig. 22.23 A chart of the scaling complexity of Charcot foot surgeries. *TAL* tendo-Achilles lengthening. (From Lee C. Rogers, with permission)

than those under the lateral midfoot [96]. However, an earlier study reviewing experience with only lateral column ulcers reported an 89% overall healing rate [101]. A flexible approach to both incision and soft tissue coverage, including tissue transfer, is therefore required for optimal outcomes in cases of midfoot plantar ulceration.

Arthrodesis of unstable Charcot joints of the midfoot and rearfoot frequently becomes necessary to provide a useful, plantigrade foot in those situations where bracing or footwear therapy have been unsuccessful [17, 44, 100, 106]. Major foot reconstruction is also an attractive alternative to amputation in patients with chronic or recurrent ulceration. Thompson et al. recommend reconstructive surgery for Charcot deformities unable to function with load sharing orthoses [107, 108]. Commonly, a tendo-Achilles lengthening precedes the fusion to ultimately diminish the plantarflexory forces contributing to pedal destruction [17, 109]. The traditional method for arthrodesis has been open reduction with solid internal fixation for uninfected Charcot joints, while external fixation is utilized when there is suspected infection of the joint fusion site [100]. In recent years, however, there has been greater interest in using external fixation and circular (Ilizarov) frames for stabilization in the Charcot foot of acute and chronic durations, and for maintenance of correction for major reconstructions (Fig. 22.24) [110–112]. Proposed benefits of circular frames include their ability to maintain fixation even in osteopenic bone, early weight-bearing ability, avoidance of fixation devices at sites of ulceration and potential bone infection, the ability to correct severe deformities, and the capability for gradual adjustments in position and compression throughout the reparative process [110]. For ankle deformities requiring arthrodesis, some prefer to use retrograde intramedullary nails alone or in concert with external fixators to provide stability and enhanced rates of fusion [113–115].

Operative fusion techniques vary by site, but generally require meticulous excision of the synovium, resection of sclerotic bone down to a healthy bleeding bed, open manipulation, and precise osteotomies prior to rigid fixation. Tissue handling must be gentle to avoid undue trauma and dissection must be mindful of underlying neurovascular structures. After reduction of deformity temporary fixation is achieved with large Steinman pins, K-wires, or guide pins when cannulated screw systems are to be used [109]. After copious lavage, a surgical drain is placed before primary wound closure. External circular frames are generally constructed preoperatively and then applied with appropriate technique after wound closure.

Newer research studying dynamic peak plantar pressures pre- and postoperatively shows promise in proving that surgical reconstruction of the Charcot foot is beneficial (Fig. 22.25) [116].

Postoperative to internal fixation procedures, the patient immediately undergoes immobilization of the foot with a posterior splint or bivalve cast. The patient must adhere to strict bed rest and prevent lower extremity dependency for several days until the soft tissue swelling subsides and serial below knee casting begins. The patient will remain non-weight-bearing for a minimum of 2–3 months prior to considering partial weight-bearing. In general, protected weight-bearing should be the rule for 6–12 months in order to avoid nonunion or late deformity in these difficult patients. After external fixation weight-bearing status is variable. Some surgeons allow limited or full weight-bearing while others choose to keep patients non-weight-bearing while the frame is in place. The contralateral extremity should be protected from the components of the external fixator which could cause injury. This can be accomplished by covering the external fixator or the contralateral extremity [117]. Advancement to weight-bearing cast, total contact cast, or walking brace will follow after evidence of consolidation. One reasonable approach is to remove the fixator after 2 months with subsequent application of an ambulatory total contact cast for several more months until there is evidence of radiographic consolidation [106]. Once healed, therapeutic footwear with or without bracing is necessary to prevent recurrent foot lesions.

Complications

Traditionally, surgery on neuropathic joints had been met with a good deal of failure including high rates of nonunion, pseudoarthrosis, and infection. Most such occurrences can now be attributed to a failure of appreciation of the natural history of osteoarthropathy and lack of attention to the necessary criteria and the basic tenets of surgery on Charcot joints as previously discussed. Even with this knowledge, however, complications can ensue in these high-risk feet during the immediate postoperative period and beyond.

Infection can be a major sequel of surgery and of course can threaten the success of an attempted arthrodesis site as well as the limb itself. Most longitudinal studies and reports of surgery on the Charcot foot indicate a certain percentage of patients in whom osteomyelitis or severe infection developed that necessitated major amputation [15, 42]. Therefore, caution must constantly be exercised in these patients to ensure that infection or osteomyelitis is controlled and eradicated prior to reconstructive surgery. Perioperative antibiotic therapy is certainly indicated in these compromised patients and once present, infection must be aggressively treated. With the use of external fixators comes the risk of pin tract infections or wire breakages requiring further surgery [118, 119]. But if complications are managed on a proper and

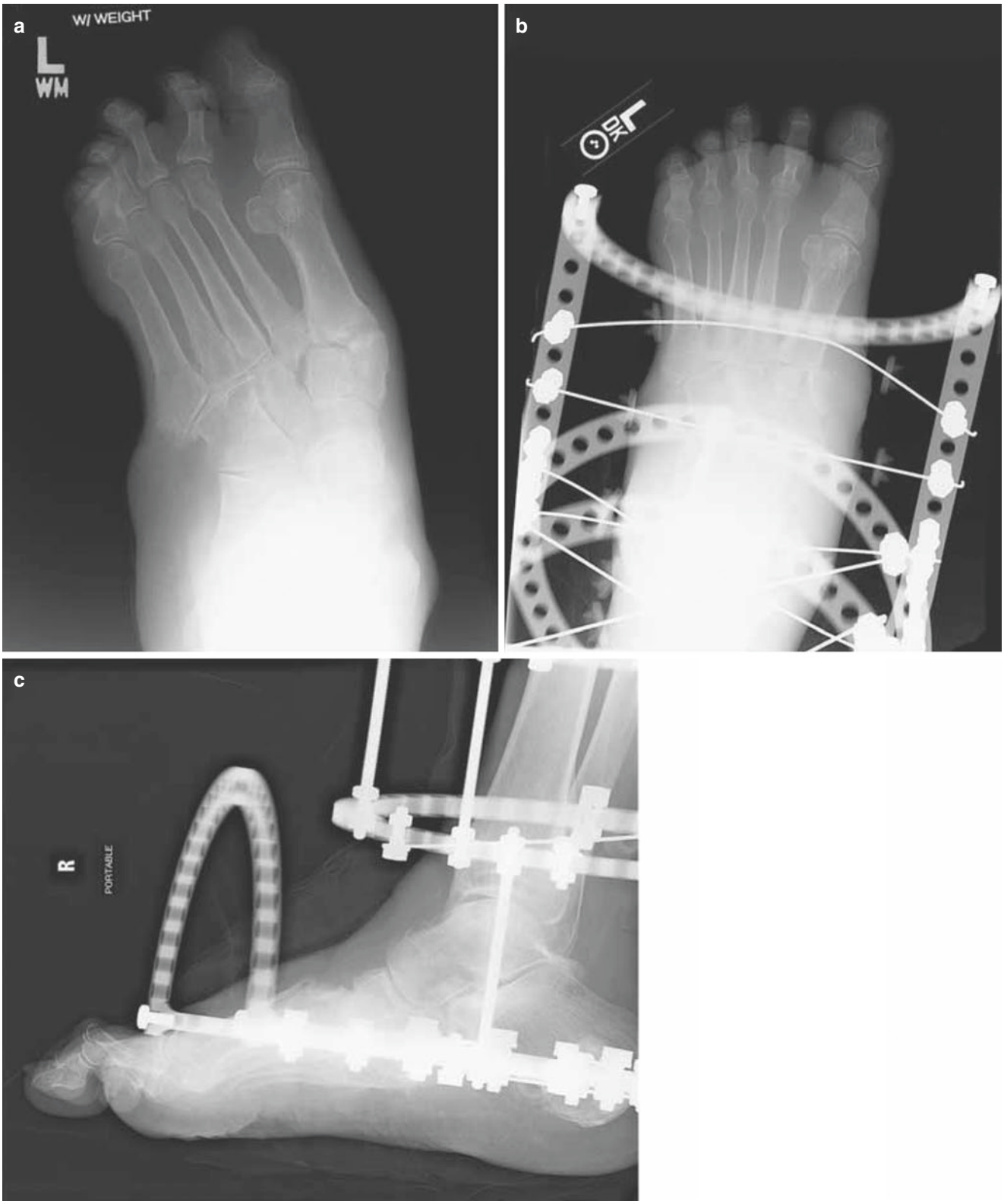

Fig. 22.24 Midfoot Charcot deformity corrected with circular external fixation. (**a**) Preoperative AP view showing midfoot deformity. (**b**) Postoperative AP view showing correction and frame in place. (**c**) Lateral postoperative X-ray with circular frame in place

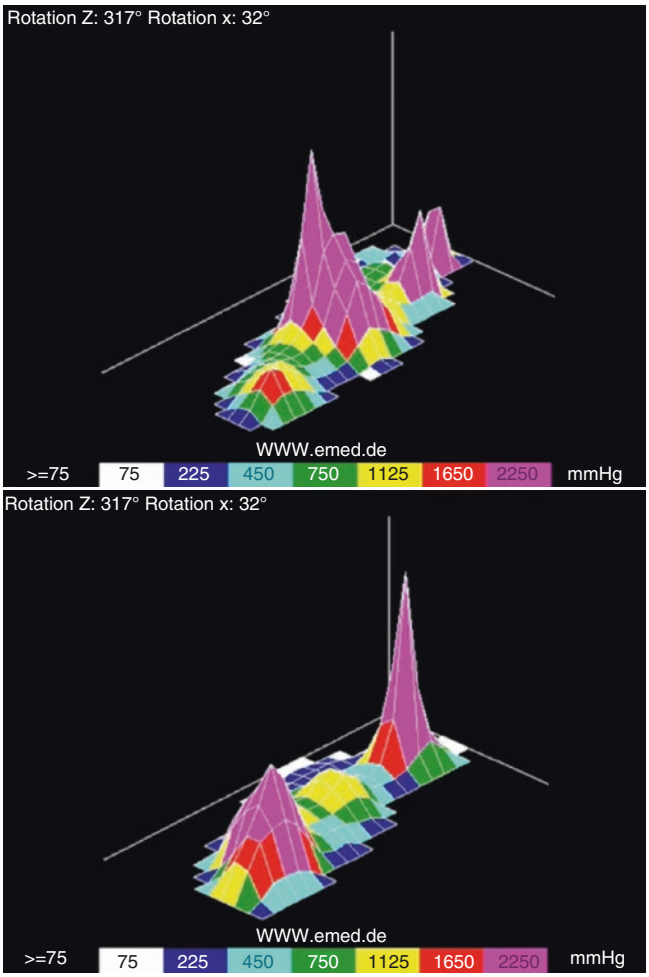

Fig. 22.25 Illustration of dynamic peak plantar measurements in a patient before (top) and 6 months after (bottom) Charcot foot reconstruction. Note the resolution of high plantar midfoot pressures postoperatively and the return of a more normal pattern, which includes higher pressure under the heel and forefoot. (From Lee C. Rogers, with permission)

timely basis, their presence does not change the outcome of the surgery.

Pseudoarthrosis and nonunion are very troublesome complications in non-neuropathic patients undergoing arthrodesis or osteotomy. However, this is not always the case in neuropathic patients undergoing the same type of reconstructive procedures. As long as stability and satisfactory alignment are achieved, a failure of complete arthrodesis or union is not necessarily considered to be a failure of surgery [109, 110]. Just as they will not sense the discomfort of post-traumatic arthritis in unreduced fracture-dislocations, these patients will have no symptoms from a stable, well-aligned nonunion. Nonetheless, the surgical principles for achieving solid union as previously discussed must always be followed when operating on these patients.

Since the trauma of surgery in itself can potentially incite an acute active reaction in a chronic inactive neuropathic joint, one must always treat the newly operated foot as an active Charcot joint. Furthermore, Clohisy makes a strong argument for prophylactic immobilization of the contralateral extremity to prevent the development of an acute deformity on the supporting foot [120]. Ablative or corrective procedures of the forefoot can also have detrimental effects on adjacent structures as well as on the midfoot and rearfoot. Biomechanical alterations will result in increased areas of vertical and shear stress in new sites which will then be predisposed to ulceration and neuroarthropathy. Therefore, surgery of any kind on the neuropathic foot must be performed with discretion and with attention to proper postoperative care to obviate the occurrence of these potentially destructive sequelae.

Amputation should usually be regarded as a procedure of last resort in neuropathic patients and not as a normal consequence of osteoarthropathy. While this outcome can sometimes represent a failure in early recognition and management, amputation usually results from overwhelming postoperative infection or late stage ulcerations. Unfortunately, amputation will always be a necessary consideration in this complicated group of patients [121]. In certain situations, amputation might be the *best* alternative to a difficult reconstruction in an unstable patient or in those patients who do not wish to engage in the lengthy recuperative period that follows a major arthrodesis. However, this is generally reserved for those extremities beyond salvage after all other attempts at medical and reconstructive care have failed.

Conclusion

The Charcot foot is a very serious limb-threatening complication of diabetes that can be attributed to preexisting peripheral neuropathy compounded by some degree of trauma. Oftentimes the diagnosis is missed which can lead to further destruction [122, 123]. The attendant hypervascular response coupled with osteopenia, fractures, and dislocations can rapidly evolve into severe foot deformities as a consequence of continued weight-bearing. It is therefore incumbent upon both the patient to seek early consultation and the practitioner to diagnose the process early in order to arrest the progression of the destructive phase and institute appropriate treatment. While non-weight-bearing and immobilization remain the most effective treatment in the active stage, over the last decade there has been greater interest in surgical solutions for the severe deformities, recurrent ulcers, or instability. As our knowledge and experience have grown, long-term outcomes have improved. As of yet, however, many questions remain unanswered pertaining to the precise mechanisms involved in the etiology of neuroarthropathy as well as those concerning optimal early and late stage treatments. With a heightened suspicion for the disorder, further prospective research, and an evidence-based approach to treatment, the future holds even greater promise for these patients.

References

1. Charcot JM. Sur quelaques arthropathies qui paraissent depender d'une lesion du cerveau ou de la moele epiniere. Arch Des Physiol Norm et Path. 1868;1:161–71.
2. Sanders LJ, Frykberg RG. The Charcot foot. In: Frykberg RG, editor. The high risk foot in diabetes mellitus. New York: Churchill Linvingston; 1991. p. 325–35.
3. Sanders LJ. What lessons can history teach us about the Charcot foot? Clin Podiatr Med Surg. 2008;25(1):1–15, v.
4. Jordan WR. Neuritic manifestations in diabetes mellitus. Arch Intern Med. 1936;57(2):307–66.
5. Bailey CC, Root HF. Neuropathic foot lesions in diabetes mellitus. N Engl J Med. 1947;236(11):397–401.
6. Sinha S, Munichoodappa CS, Kozak GP. Neuro-arthropathy (Charcot joints) in diabetes mellitus. Medicine. 1972;51(3):191.
7. Frykberg RG, Kozak GP. Neuropathic arthropathy in the diabetic foot. Am Fam Physician. 1978;17(5):105–13.
8. Sanders LJ, Frykberg RG. Charcot neuroarthropathy of the foot. In: Bowker JH, Pfeifer MA, editors. Levin and O'Neals The diabetic foot. 6th ed. St. Louis: Mosby; 2001.
9. Cofield RH, Morrison MJ, Beabout JW. Diabetic neuroarthropathy in the foot: patient characteristics and patterns of radiographic change. Foot Ankle. 1983;4(1):15–22.
10. Lavery LA, Armstrong DG, Wunderlich RP, Tredwell J, Boulton AJM. Diabetic foot syndrome evaluating the prevalence and incidence of foot pathology in Mexican Americans and non-Hispanic whites from a diabetes disease management cohort. Diabetes Care. 2003;26(5):1435–8.
11. Frykberg RG. Charcot foot: an update on pathogenesis and management. In: Boulton A, Connor H, Cavanagh PR, editors. The foot in diabetes. 3rd ed. Chichester: John Wiley and Sons; 2000.
12. Gazis A, Pound N, Macfarlane R, Treece K, Game F, Jeffcoate W. Mortality in patients with diabetic neuropathic osteoarthropathy (Charcot foot). Diabet Med. 2004;21(11):1243–6.
13. Sohn M-W, Lee TA, Stuck RM, Frykberg RG, Budiman-Mak E. Mortality risk of Charcot arthropathy compared with that of diabetic foot ulcer and diabetes alone. Diabetes Care. 2009;32(5):816–21.
14. van Baal J, Hubbard R, Game F, Jeffcoate W. Mortality associated with acute Charcot foot and neuropathic foot ulceration. Diabetes Care. 2010;33(5):1086–9.
15. Armstrong DG, Todd WF, Lavery LA, Harkless LB, Bushman TR. The natural history of acute Charcot's arthropathy in a diabetic foot specialty clinic. J Am Podiatr Med Assoc. 1997;87(6):272–8.
16. Kozak GP, Campbell DR, Frykberg RG, Cavanagh PR. The diabetic Charcot foot. In: Management of diabetic foot problems. 2nd ed. Philadelphia: WB Sauners; 1995.
17. Frykberg RG, Armstrong DG, Giurini J, et al. Diabetic foot disorders: a clinical practice guideline. American College of Foot and Ankle Surgeons. J Foot Ankle Surg. 2000;39(5 Suppl):S1–S60.
18. Game FL, Catlow R, Jones GR, et al. Audit of acute Charcot's disease in the UK: the CDUK study. Diabetologia. 2012;55(1):32–5.
19. Del Vecchio JJ, Raimondi N, Rivarola H, Autorino C. Charcot neuroarthropathy in simultaneous kidney-pancreas transplantation: report of two cases. Diabet Foot Ankle. 2013;4. https://doi.org/10.3402/dfa.v4i0.21819.
20. García Barrado F, Kuypers DR, Matricali GA. Charcot neuroarthropathy after simultaneous pancreas-kidney transplantation: risk factors, prevalence, and outcome. Clin Transplant. 2015;29(8):712–9.
21. Bevilacqua NJ, Bowling F, Armstrong DG. The natural history of Charcot neuroarthropathy. In: Frykberg RG, editor. The diabetic Charcot foot: principles and management. Brooklandville, MD: Data Trace Publishing Company; 2010. p. 13–28.
22. Brower AC, Allman RM. Pathogenesis of the neurotrophic joint: neurotraumatic vs. neurovascular. Radiology. 1981;139(2):349–54.
23. Jeffcoate W, Lima J, Nobrega L. The Charcot foot. Diabet Med. 2000;17(4):253–8.
24. Boulton AJM. The diabetic foot: from art to science. The 18th Camillo Golgi lecture. Diabetologia. 2004;47(8):1343–53.
25. Veves A, Akbari CM, Primavera J, et al. Endothelial dysfunction and the expression of endothelial nitric oxide synthetase in diabetic neuropathy, vascular disease, and foot ulceration. Diabetes. 1998;47(3):457–63.
26. Edelman SV, Kosofsky EM, Paul RA, Kozak GP. Neuro-osteoarthropathy (Charcot's joint) in diabetes mellitus following revascularization surgery: three case reports and a review of the literature. Arch Intern Med. 1987;147(8):1504–8.
27. Gough A, Abraha H, Li F, et al. Measurement of markers of osteoclast and osteoblast activity in patients with acute and chronic diabetic Charcot neuroarthropathy. Diabet Med. 1997;14(7):527–31.
28. Young MJ, Marshall A, Adams JE, Selby PL, Boulton AJ. Osteopenia, neurological dysfunction, and the development of Charcot neuroarthropathy. Diabetes Care. 1995;18(1):34–8.
29. Jirkovská A, Kasalický P, Boucek P, Hosová J, Skibová J. Calcaneal ultrasonometry in patients with Charcot osteoarthropathy and its relationship with densitometry in the lumbar spine and femoral neck and with markers of bone turnover. Diabet Med. 2001;18(6):495–500.
30. Rajbhandari SM, Jenkins RC, Davies C, Tesfaye S. Charcot neuroarthropathy in diabetes mellitus. Diabetologia. 2002;45(8):1085–96.
31. Foltz KD, Fallat LM, Schwartz S. Usefulness of a brief assessment battery for early detection of Charcot foot deformity in patients with diabetes. J Foot Ankle Surg. 2004;43(2):87–92.
32. Grant WP, Foreman EJ, Wilson AS, Jacobus DA, Kukla RM. Evaluation of Young's modulus in Achilles tendons with diabetic neuroarthropathy. J Am Podiatr Med Assoc. 2005;95(3):242–6.
33. Grant WP, Sullivan R, Sonenshine DE, et al. Electron microscopic investigation of the effects of diabetes mellitus on the Achilles tendon. J Foot Ankle Surg. 1997;36(4):272–8. discussion 330
34. Johnson JT. Neuropathic fractures and joint injuries Pathogenesis and rationale of prevention and treatment. J Bone Joint Surg Am. 1967;49(1):1–30.
35. Herbst SA, Jones KB, Saltzman CL. Pattern of diabetic neuropathic arthropathy associated with the peripheral bone mineral density. J Bone Joint Surg Br. 2004;86(3):378–83.
36. Jude EB, Selby PL, Burgess J, et al. Bisphosphonates in the treatment of Charcot neuroarthropathy: a double-blind randomised controlled trial. Diabetologia. 2001;44(11):2032–7.
37. Jeffcoate W. Vascular calcification and osteolysis in diabetic neuropathy-is RANK-L the missing link? Diabetologia. 2004;47(9):1488–92.
38. Jeffcoate WJ, Game FL. New theories on the causes of the Charcot foot in diabetes. In: Frykberg RG, editor. The diabetic Charcot foot: principles and management. Brooklandville, MD: Data Trace Publishing Company; 2010. p. 29–44.
39. Petrova NL, Foster AVM, Edmonds ME. Difference in presentation of Charcot osteoarthropathy in type 1 compared with type 2 diabetes. Diabetes Care. 2004;27(5):1235–6.
40. Pakarinen TK, Laine HJ, Honkonen SE, Peltonen J, Oksala H, Lahtela J. Charcot arthropathy of the diabetic foot. Current concepts and review of 36 cases. Scand J Surg. 2002;91(2):195–201.
41. Banks AS, McGlamry ED. Charcot foot. J Am Podiatr Med Assoc. 1989;79(5):213–35.
42. Fabrin J, Larsen K, Holstein PE. Long-term follow-up in diabetic Charcot feet with spontaneous onset. Diabetes Care. 2000;23(6):796–800.

43. McGill M, Molyneaux L, Bolton T, Ioannou K, Uren R, Yue DK. Response of Charcot's arthropathy to contact casting: assessment by quantitative techniques. Diabetologia. 2000;43(4):481–4.
44. Rogers LC, Frykberg RG, Armstrong DG, et al. The Charcot foot in diabetes. J Am Podiatr Med Assoc. 2011;101(5):437–46.
45. Trepman E, Nihal A, Pinzur MS. Current topics review: Charcot neuroarthropathy of the foot and ankle. Foot Ankle Int. 2005;26(1):46–63.
46. Sella EJ, Barrette C. Staging of Charcot neuroarthropathy along the medial column of the foot in the diabetic patient. J Foot Ankle Surg. 1999;38(1):34–40.
47. Wukich DK, Belczyk RJ. Silent neuropathic bone injuries and dislocations – stage 0. In: Frykberg RG, editor. The diabetic Charcot foot: principles and management. Brooklandville, MD: Data Trace Publishing Company; 2010. p. 117–30.
48. Keenan AM, Tindel NL, Alavi A. Diagnosis of pedal osteomyelitis in diabetic patients using current scintigraphic techniques. Arch Intern Med. 1989;149(10):2262–6.
49. Schweitzer ME, Morrison WB. MR imaging of the diabetic foot. Radiol Clin North Am. 2004;42(1):61–71.
50. Palestro CJ, Mehta HH, Patel M, et al. Marrow versus infection in the Charcot joint: indium-111 leukocyte and technetium-99m sulfur colloid scintigraphy. J Nucl Med. 1998;39(2):346–50.
51. Rogers LC, Bevilacqua NJ. Imaging of the Charcot foot. Clin Podiatr Med Surg. 2008;25(2):263–274, vii.
52. Armstrong DG, Lavery LA, Sariaya M, Ashry H. Leukocytosis is a poor indicator of acute osteomyelitis of the foot in diabetes mellitus. J Foot Ankle Surg. 1996;35(4):280–3.
53. Eichenholtz SN. Charcot joints. Springfield, IL: Charles C. Thomas; 1966.
54. Frykberg RG, Mendeszoon ER. Charcot arthropathy: pathogenesis and management. Wounds. 2000;12(6; SUPP/B):35B–42B.
55. Newman JH. Spontaneous dislocation in diabetic neuropathy. A report of six cases. J Bone Joint Surg Br. 1979;61-B(4):484–8.
56. Lesko P, Maurer RC. Talonavicular dislocations and midfoot arthropathy in neuropathic diabetic feet. Natural course and principles of treatment. Clin Orthop Relat Res. 1989;240:226–31.
57. Harris JR, Brand PW. Patterns of disintegration of the tarsus in the anaesthetic foot. J Bone Joint Surg Br. 1966;48(1):4–16.
58. Kathol MH, El-Khoury GY, Moore TE, Marsh JL. Calcaneal insufficiency avulsion fractures in patients with diabetes mellitus. Radiology. 1991;180(3):725–9.
59. El-Khoury GY, Kathol MH. Neuropathic fractures in patients with diabetes mellitus. Radiology. 1980;134(2):313–6.
60. Sella EJ, Grosser DM. Imaging modalities of the diabetic foot. Clin Podiatr Med Surg. 2003;20(4):729–40.
61. Seabold JE, Flickinger FW, Kao SC, et al. Indium-111-leukocyte/technetium-99m-MDP bone and magnetic resonance imaging: difficulty of diagnosing osteomyelitis in patients with neuropathic osteoarthropathy. J Nucl Med. 1990;31(5):549–56.
62. Schauwecker DS, Park HM, Burt RW, Mock BH, Wellman HN. Combined bone scintigraphy and indium-111 leukocyte scans in neuropathic foot disease. J Nucl Med. 1988;29(10):1651–5.
63. Johnson JE, Kennedy EJ, Shereff MJ, Patel NC, Collier BD. Prospective study of bone, indium-111-labeled white blood cell, and gallium-67 scanning for the evaluation of osteomyelitis in the diabetic foot. Foot Ankle Int. 1996;17(1):10–6.
64. Blume PA, Dey HM, Daley LJ, Arrighi JA, Soufer R, Gorecki GA. Diagnosis of pedal osteomyelitis with Tc-99m HMPAO labeled leukocytes. J Foot Ankle Surg. 1997;36(2):120–6. discussion 160
65. Croll SD, Nicholas GG, Osborne MA, Wasser TE, Jones S. Role of magnetic resonance imaging in the diagnosis of osteomyelitis in diabetic foot infections. J Vasc Surg. 1996;24(2):266–70.
66. Morrison WB, Schweitzer ME, Batte WG, Radack DP, Russel KM. Osteomyelitis of the foot: relative importance of primary and secondary MR imaging signs. Radiology. 1998;207(3):625–32.
67. Höpfner S, Krolak C, Kessler S, et al. Preoperative imaging of Charcot neuroarthropathy in diabetic patients: comparison of ring PET, hybrid PET, and magnetic resonance imaging. Foot Ankle Int. 2004;25(12):890–5.
68. Schon LC, Marks RM. The management of neuroarthropathic fracture-dislocations in the diabetic patient. Orthop Clin North Am. 1995;26(2):375–92.
69. Yu GV, Hudson JR. Evaluation and treatment of stage 0 Charcot's neuroarthropathy of the foot and ankle. J Am Podiatr Med Assoc. 2002;92(4):210–20.
70. Brodsky JW, Rouse AM. Exostectomy for symptomatic bony prominences in diabetic Charcot feet. Clin Orthop Relat Res. 1993;296:21–6.
71. Schon LC, Weinfeld SB, Horton GA, Resch S. Radiographic and clinical classification of acquired midtarsus deformities. Foot Ankle Int. 1998;19(6):394–404.
72. Frykberg RG, Rogers LC. Classification of the Charcot foot. In: Frykberg RG, editor. The diabetic Charcot foot: principles and management. Brooklandville, MD: Data Trace Publishing Company; 2010. p. 55–64.
73. Rogers LC, Bevilacqua NJ. The diagnosis of Charcot foot. Clin Podiatr Med Surg. 2008;25(1):43–51, vi.
74. Viswanathan V, Kesavan R, Kavitha KV, Kumpatla S. Evaluation of Rogers Charcot foot classification system in south Indian diabetic subjects with Charcot foot. J Diabet Foot Complication. 2010;4(3):67–70.
75. Snyder RJ, Frykberg RG, Rogers LC, et al. The management of diabetic foot ulcers through optimal off-loading: building consensus guidelines and practical recommendations to improve outcomes. J Am Podiatr Med Assoc. 2014;104(6):555–67.
76. Pinzur M. Surgical versus accommodative treatment for Charcot arthropathy of the midfoot. Foot Ankle Int. 2004;25(8):545–9.
77. Morgan JM, Biehl WC 3rd, Wagner FW Jr. Management of neuropathic arthropathy with the Charcot restraint orthotic Walker. Clin Orthop Relat Res. 1993;296:58–63.
78. Armstrong DG, Short B, Espensen EH, Abu-Rumman PL, Nixon BP, Boulton AJM. Technique for fabrication of an "Instant Total-Contact Cast" for treatment of neuropathic diabetic foot ulcers. J Am Podiatr Med Assoc. 2002;92(7):405–8.
79. Caputo GM, Ulbrecht J, Cavanagh PR, Juliano P. The Charcot foot in diabetes: six key points. Am Fam Physician. 1998;57(11):2705–10.
80. Saltzman CL, Johnson KA, Goldstein RH, Donnelly RE. The patellar tendon-bearing brace as treatment for neurotrophic arthropathy: a dynamic force monitoring study. Foot Ankle. 1992;13(1):14–21.
81. Anderson JJ, Woelffer KE, Holtzman JJ, Jacobs AM. Bisphosphonates for the treatment of Charcot neuroarthropathy. J Foot Ankle Surg. 2004;43(5):285–9.
82. Selby PL, Young MJ, Boulton AJ. Bisphosphonates: a new treatment for diabetic Charcot neuroarthropathy? Diabet Med. 1994;11(1):28–31.
83. Pitocco D, Ruotolo V, Caputo S, et al. Six-month treatment with alendronate in acute Charcot neuroarthropathy a randomized controlled trial. Diabetes Care. 2005;28(5):1214–5.
84. Bem R, Jirkovská A, Fejfarová V, Skibová J, Jude EB. Intranasal calcitonin in the treatment of acute Charcot neuroosteoarthropathy: a randomized controlled trial. Diabetes Care. 2006;29(6):1392–4.
85. Bier RR, Estersohn HS. A new treatment for Charcot joint in the diabetic foot. J Am Podiatr Med Assoc. 1987;77(2):63–9.
86. Hanft JR, Goggin JP, Landsman A, Surprenant M. The role of combined magnetic field bone growth stimulation as an adjunct in the treatment of neuroarthropathy/Charcot joint: an expanded pilot study. J Foot Ankle Surg. 1998;37(6):510–5. discussion 550–551
87. Grady JF, O'Connor KJ, Axe TM, Zager EJ, Dennis LM, Brenner LA. Use of electrostimulation in the treatment of diabetic neuroarthropathy. J Am Podiatr Med Assoc. 2000;90(6):287–94.

88. Strauss E, Gonya G. Adjunct low intensity ultrasound in Charcot neuroarthropathy. Clin Orthop Relat Res. 1998;349:132–8.
89. Hockenbury RT, Gruttadauria M, McKinney I. Use of implantable bone growth stimulation in Charcot ankle arthrodesis. Foot Ankle Int. 2007;28(9):971–6.
90. Lowery NJ, Woods JB, Armstrong DG, Wukich DK. Surgical management of Charcot neuroarthropathy of the foot and ankle: a systematic review. Foot Ankle Int. 2012;33(2):113–21.
91. Steindler A. The tabetic arthropathies. JAMA. 1931;96(4):250–6.
92. Samilson RL, Sankaran B, Bersani FA, Smith AD. Orthopedic management of neuropathic joints. AMA Arch Surg. 1959;78(1):115–21.
93. Heiple KG, Cammarn MR. Diabetic neuroarthropathy with spontaneous peritalar fracture-dislocation. JBJS Case Connector. 1966;48(6):1177–81.
94. Baravarian B, Van Gils CC. Arthrodesis of the Charcot foot and ankle. Clin Podiatr Med Surg. 2004;21(2):271–89.
95. Leventen EO. Charcot foot—a technique for treatment of chronic plantar ulcer by saucerization and primary closure. Foot Ankle. 1986;6(6):295–9.
96. Catanzariti AR, Mendicino R, Haverstock B. Ostectomy for diabetic neuroarthropathy involving the midfoot. J Foot Ankle Surg. 2000;39(5):291–300.
97. Newman JH. Non-infective disease of the diabetic foot. J Bone Joint Surg Br. 1981;63B(4):593–6.
98. Pinzur MS. Benchmark analysis of diabetic patients with neuropathic (Charcot) foot deformity. Foot Ankle Int. 1999;20(9):564–7.
99. Myerson MS, Alvarez RG, Lam PW. Tibiocalcaneal arthrodesis for the management of severe ankle and hindfoot deformities. Foot Ankle Int. 2000;21(8):643–50.
100. Pinzur MS. Surgical management – history and general principles. In: Frykberg RG, editor. The diabetic Charcot foot: principles and management. Brooklandville, MD: Data Trace Publishing Company; 2010. p. 165–88.
101. Rosenblum BI, Giurini JM, Miller LB, Chrzan JS, Habershaw GM. Neuropathic ulcerations plantar to the lateral column in patients with Charcot foot deformity: a flexible approach to limb salvage. J Foot Ankle Surg. 1997;36(5):360–3.
102. Simon SR, Tejwani SG, Wilson DL, Santner TJ, Denniston NL. Arthrodesis as an early alternative to nonoperative management of Charcot arthropathy of the diabetic foot. J Bone Joint Surg Am. 2000;82-A(7):939–50.
103. Wang JC, Le AW, Tsukuda RK. A new technique for Charcot's foot reconstruction. J Am Podiatr Med Assoc. 2002;92(8):429–36.
104. Jolly GP, Zgonis T, Polyzois V. External fixation in the management of Charcot neuroarthropathy. Clin Podiatr Med Surg. 2003;20(4):741–56.
105. Pinzur MS, Sammarco VJ, Wukich DK. Charcot foot: a surgical algorithm. Instr Course Lect. 2012;61:423–38.
106. Farber DC, Juliano PJ, Cavanagh PR, Ulbrecht J, Caputo G. Single stage correction with external fixation of the ulcerated foot in individuals with Charcot neuroarthropathy. Foot Ankle Int. 2002;23(2):130–4.
107. Thompson RC Jr, Clohisy DR. Deformity following fracture in diabetic neuropathic osteoarthropathy. Operative management of adults who have type-I diabetes. J Bone Joint Surg Am. 1993;75(12):1765–73.
108. Marks RM, Parks BG, Schon LC. Midfoot fusion technique for neuroarthropathic feet: biomechanical analysis and rationale. Foot Ankle Int. 1998;19(8):507–10.
109. Bevilacqua NJ, Rogers LC. Surgical management of Charcot midfoot deformities. Clin Podiatr Med Surg. 2008;25(1):81–94, vii.
110. Cooper PS. Application of external fixators for management of Charcot deformities of the foot and ankle. Semin Vasc Surg. 2003;16(1):67–78.
111. Lamm BM, Paley D. Reduction of neuropathic foot deformity with gradual external fixation distraction and midfoot fusion. In: Frykberg RG, editor. The Diabetic Charcot Foot: Principles and Management. Brooklandville, MD: Data Trace Publishing Company; 2010. p. 197–210.
112. Wukich DK, Zgonis T. Circular external fixation in Charcot neuroarthropathy. In: Frykberg RG, editor. The diabetic charcot foot: principles and management. Brooklandville, MD: Data Trace Publishing Company; 2010. p. 233–44.
113. Mendicino RW, Catanzariti AR, Saltrick KR, et al. Tibiotalocalcaneal arthrodesis with retrograde intramedullary nailing. J Foot Ankle Surg. 2004;43(2):82–6.
114. Pinzur MS, Kelikian A. Charcot ankle fusion with a retrograde locked intramedullary nail. Foot Ankle Int. 1997;18(11):699–704.
115. Moore TJ, Prince R, Pochatko D, Smith JW, Fleming S. Retrograde intramedullary nailing for ankle arthrodesis. Foot Ankle Int. 1995;16(7):433–6.
116. Najafi B, Crews RT, Armstrong DG, Rogers LC, Aminian K, Wrobel J. Can we predict outcome of surgical reconstruction of Charcot neuroarthropathy by dynamic plantar pressure assessment?—a proof of concept study. Gait Posture. 2010;31(1):87–92.
117. Bevilacqua NJ, Dankert JP, Rogers LC, Armstrong DG. A technique to protect external fixation devices. J Foot Ankle Surg. 2008;47(2):172–4.
118. Rogers LC, Bevilacqua NJ, Frykberg RG, Armstrong DG. Predictors of postoperative complications of Ilizarov external ring fixators in the foot and ankle. J Foot Ankle Surg. 2007;46(5):372–5.
119. Wukich DK, Belczyk RJ, Burns PR, Frykberg RG. Complications encountered with circular ring fixation in persons with diabetes mellitus. Foot Ankle Int. 2008;29(10):994–1000.
120. Clohisy DR, Thompson RC Jr. Fractures associated with neuropathic arthropathy in adults who have juvenile-onset diabetes. J Bone Joint Surg Am. 1988;70(8):1192–200.
121. Eckardt A, Schöllner C, Decking J, et al. The impact of Syme amputation in surgical treatment of patients with diabetic foot syndrome and Charcot-neuro-osteoarthropathy. Arch Orthop Trauma Surg. 2004;124(3):145–50.
122. Tan AL, Greenstein A, Jarrett SJ, McGonagle D. Acute neuropathic joint disease: a medical emergency? Diabetes Care. 2005;28(12):2962–4.
123. Chantelau E. The perils of procrastination: effects of early vs. delayed detection and treatment of incipient Charcot fracture. Diabet Med. 2005;22(12):1707–12.