Charcot Neuroarthropathy of the Foot and Ankle

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Keywords
Charcot neuropathic arthropathy • Etiology • Polyneuropathy • Charcot deformity • Charcot arthropathy classification • Conservative treatment • Surgical treatment

Introduction

Charcot arthropathy of the foot was first described in detail in the nineteenth century by Jean-Martin Charcot (1825–1893), one of the most celebrated French physicians of the nineteenth century, who accurately and systemically described the findings on patients with various underlying diseases which lead to common symptoms regarding the foot and ankle [19, 66]. However, William Musgrave of Extair (1651–1721), a British physician and antiquary, had described similar symptoms before Jean-Martin Charcot; in his work, "Antiquitates Britanno-Belgicae," he published four volumes on arthritis including descriptions of arthritis due to venereal disease [39]. Additional authors also reported their findings around this time [30, 45]. Charcot himself gave John Kearsley Mitchell (1793–1858) from Philadelphia credit for his publication 37 years before he did [48]. However, despite the numerous reports describing Charcot arthropathy from this time period, Jean-Martin Charcot was the physician who documented his findings in the most exact and systematic manner [18].

Etiology

Charcot arthropathy is defined as aseptic destruction of the foot, which may ultimately lead to massive and complete destruction of bones and joints of the foot and ankle. To date, Charcot arthropathy is not fully understood in detail; numerous diseases have been found to be associated with the

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Table 17.1 Etiology of Charcot arthropathy

Diabetes mellitus
Alcohol abuse
Syphilis
Lepra
Hemodialysis
Cerebral palsy
Myelomeningocele
Syringomyelia
Neurologic disorders (e.g., Charcot-Marie-Tooth disease, peroneal muscular atrophy)
Intra-articular steroid injections
Idiopathic

Charcot arthropathy (Table 17.1), but the etiologic origin often remains unclear in particular cases. In contrast to the twentieth and twenty-first century, leprosy and lues have been described as the most important etiologic factors associated with Charcot arthropathy in the nineteenth century. Currently, diabetes is the most common risk factor leading to Charcot arthropathy followed by others like alcohol abuse, neuropathy, syringomyelia, amyloidosis, and hereditary neuropathies as well as patients with spinal trauma [46].

The differentiation between diabetic foot syndrome and diabetic neuroosteoarthropathy is important; diabetic foot syndrome is one of the most devastating and serious complications of diabetes and is defined as a foot affected by ulceration that is associated with neuropathy and/or peripheral arterial disease of the lower leg in patient with diabetes [2, 54]. Diabetic foot syndrome does not inevitably lead to Charcot arthropathy [44]. The high risk in diabetics for kidney failure is another associated cause of polyneuropathies [7, 11, 49, 89].

Charcot arthropathy often ends in devastating deformities, which often need to be addressed surgically. The clinical differentiation between septic and aseptic Charcot arthropathy is of crucial importance, since infection is a common complication of Charcot foot disease, but not a trigger. The compounding factors of reduced or even absent sensitivity of the foot and the substantial deformity of prominent bones may provoke high shear stresses of the skin. This can lead to ulcerations and, furthermore, to subsequent infection of Charcot ulcers [2].

Pathomechanic Theory

There are two main pathomechanic theories of Charcot arthropathy: the "German theory" and the "French theory." The German theory relies on the absence of sensation and multiple microtraumas resulting in fractures and deformities, and this theory with underlying mechanical theory was supported by Volkman and Virchow [16, 39, 112]. In contrast, the neurovascular theory, or "French theory," was advocated by Charcot. This theory is based on neural impairment, and autosympathectomy is responsible for excessive blood flow which results in poor bone quality and high fracture susceptibility [16, 39].

Classification Systems

Different classification systems of Charcot arthropathy have been described in the current literature. One of the most common and widely used classifications is the classification proposed by orthopedic surgeon Sidney N. Eichenholtz (1909–2000) [94]. In his monograph with the title "Charcot Joints," he described clinical and radiographic data of 68 patients with Charcot arthropathy [25]. In this work, Eichenholtz described and defined three stages of Charcot arthropathy: (I) development, (II) coalescence, and (III) reconstruction and reconstitution (Table 17.2). In 1990, Shibata et al. [105] modified Eichenholtz classification by adding stage 0, because clinical signs of Charcot arthropathy were found to precede radiographic changes. The Eichenholtz classification has some limitations; it is characterized by high subjectivity and the validity of the classification remains unknown. Furthermore, this classification is based on a temporal staging system without description of anatomic locations. Finally, the Eichenholtz classification does not count for symptoms and comorbidities in patients with Charcot arthropathy, as it relates only to radiographic findings with focused physical examination [94].

Table 17.2 The modified Eichenholtz classification [25, 105]

Stage	Radiographic findings	Clinical findings	Treatment
0 (prodromal)	Normal radiographs	Swelling, erythema, warmth	Patient education, serial radiographs to monitor progression, protected weight bearing
I (development)	Osteopenia, fragmentation, joint subluxation or dislocation	Swelling, erythema, warmth, ligamentous laxity	Protected weight bearing with total contact cast or prefabricated pneumatic brace. Cast or brace should be used until radiographic resolution of fragmentation and presence of normal skin temperature (usually needed for 2–4 months)
II (coalescence)	Absorption of debris, sclerosis, fusion of larger fragments	Decreased warmth, decreased swelling, decreased erythema	Total contact cast, prefabricated pneumatic brace, Charcot restraint orthotic walker, or clamshell ankle-foot orthosis
III (reconstruction)	Consolidation of deformity, joint arthrosis, fibrous ankyloses, rounding and smoothing of bone fragments	Absence of warmth, absence of swelling, absence of erythema, stable joint ± fixed deformity	Plantigrade foot: custom shoe insoles with rigid shank and rocker bottom sole Nonplantigrade foot with/without ulceration: debridement, exostectomy, deformity correction, or fusion with internal fixation

In the last decade, categories based on magnetic resonance imaging (MRI) have been described (Table 17.3) [15]. MRI is more sensitive than conventional radiographs to detect acute bony injuries, and therefore, classification based on MRI can more accurately describe the evolution of Charcot arthropathy. Additionally, MRI can better explain the histopathological findings in patients with Charcot arthropathy (Table 17.3) [50, 75].

The classification of Charcot arthropathy of the foot and ankle into anatomic categories has been described in numerous studies [6, 9, 34, 40, 46, 98, 102]. The Sanders and Frykberg classification describes five deformity location patterns including the (I) forefoot, (II) tarsometatarsal (Lisfranc) joint, (III) midtarsal and naviculocuneiform joints, (IV) ankle and subtalar joints, and (V) calcaneus (Fig. 17.1) [34].

The Brodsky classification is based on four anatomic areas as assessed using conventional radiographs (Fig. 17.2) [46]. Type 1 involves the Lisfranc joint line including the metatarsocuneiform and naviculocuneiform joints. Type 1 Charcot arthropathy is the most common and represents about 60 % of all cases. Type 2 is the second most common type of Charcot arthropathy with 30–35 % of all cases and includes any or all of the triple joint complexes of the hindfoot: the subtalar, the talonavicular, and the calcaneocuboid joints. Types 3A and 3B are relatively minor groups; type 3A involves the ankle joint and type 3B presents with a pathologic fracture of the tubercle of the calcaneus.

In 1998, Schon et al. [101] published a very detailed classification of Charcot midfoot arthropathy. Midfoot area is the most commonly affected by Charcot arthropathy [64, 102], and therefore, only midfoot Charcot arthropathy was described in this classification system. The authors considered the anatomical aspects, clinical manifestations, and different severity stages (Table 17.4) [101].

Prevalence of Charcot Arthropathy

The reported prevalence of Charcot arthropathy is relatively low ranging from 0.08 to 7.5 % in the current literature [122]. However, only a limited number of studies address the incidence and prevalence of Charcot arthropathy and the numbers they report are inconsistent [23, 86]. For example, in 1972, Sinha et al. [107] reported a

Table 17.3 Clinical and CT/MRI findings of different stages of the Charcot arthropathy based on MRI [16]

Stage	Clinical symptoms	CT/MRI findings	Histopathology
Active stage, grade 0	Mild inflammation (swelling, warmth, pain, increased by unprotected walking); no gross deformity	Obligatory: diffuse BMO and STO (Kiuru grade I–III), no cortical disruption Facultative: subchondral trabecular microfractures (bone bruise), ligament damage	Lamellar bone with active surface Remodeling of trabeculae associated with microfractures. Marrow space replaced by loose spindle cells
Active stage, grade 1	Severe inflammation (swelling, warmth, pain, increased by unprotected walking); gross deformity, increased by unprotected walking	Obligatory: fractures with cortical disruption, BMO, and STO (Kiuru grade IV) Facultative: osteoarthritis, cysts, cartilage damage, osteochondrosis, joint effusion, fluid collection, bone erosion/necrosis, bone lysis, debris, bone destruction, joint luxation/subluxation, ligament damage, tenosynovitis, bone dislocation	Increased vascularity of the marrow space, active remodeling of woven bone Compatible with response to (impaction) fracture Osteonecrosis. Thickened synovium, fragmented cartilage and subchondral bone, invasion of inflammatory cells and vascular elements
Inactive stage, grade 0	No inflammation, no gross deformity	No abnormal findings or minimal residual BMO, subchondral sclerosis, bone cysts, osteoarthrosis, ligament damage	Sclerosis of the bone characterized by broad lamellar trabeculae with collagenous replacement and a low vascularity of the marrow space
Inactive stage, grade 1	No inflammation; persistent gross deformity, ankylosis	Residual BMO, cortical callus (Kiuru grade IV); joint effusion, subchondral cysts, joint destruction, joint dislocation, fibrosis, osteophyte formation, bone remodeling, cartilage damage, ligament damage, bone sclerosis, ankyloses, pseudoarthrosis	Woven bone, immature and structurally disorganized fibrosis

BMO bone marrow edema, *CT* computed tomography, *MRI* magnetic resonance imaging, *STO* soft tissue edema

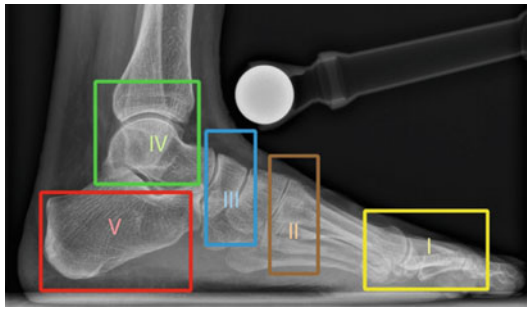


Fig. 17.1 Sanders and Frykberg classification of diabetic neuropathic osteoarthropathy [34]: (*I*) forefoot, (*II*) tarsometatarsal (Lisfranc) joint, (*III*) midtarsal and naviculocuneiform joints, (*IV*) ankle and subtalar joints, and (*V*) calcaneus

prevalence rate of 1:680 in a total population of 68,000 subjects. Smith et al. [108] specifically addressed the prevalence of Charcot arthropathy in 428 patients with diabetes. The observed prevalence of Charcot changes was 1.4 % and all 6 patients had midfoot Charcot arthropathy [108]. Fabrin et al. [29] from Denmark reported substantially lower incidence of Charcot deformity of 0.3 %/year in a diabetic population including 5000 patients with type 1 or type 2 diabetes. Leung et al. [57] reported the incidence of Charcot arthropathy as 0.041 cases per 1000 diabetic patients per year in Hong Kong. Stuck et al. [110] investigated the incidence of Charcot arthropathy using the Department of Veterans Affairs inpatient and outpatient administrative datasets. In 2003, 0.12 % of all patients with diabetes were newly diagnosed with Charcot arthropathy. Obese patients were 59 % more likely to develop Charcot arthropathy [110]. Regarding the fact that the diabetes is the most important etiology for development of Charcot arthropathy, the increasing overall incidence of Charcot arthropathy is not surprising: the World Health Organization expects an increase in

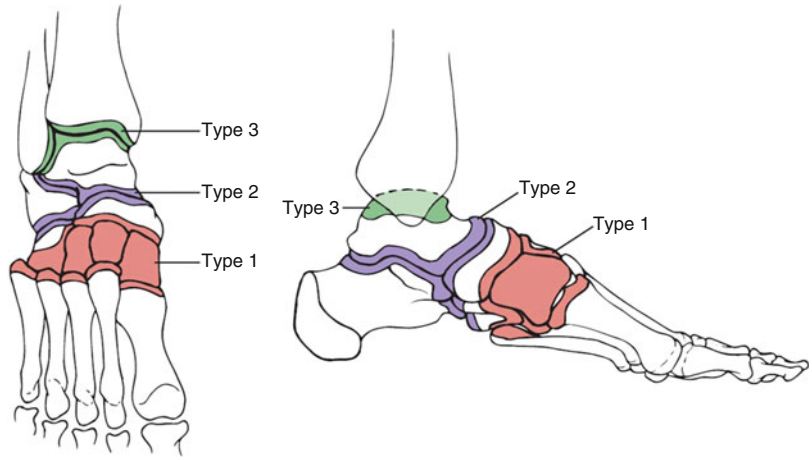


Fig. 17.2 Brodsky classification of Charcot arthropathy of the tarsus [46]: *type 1*, tarsometatarsal and naviculocuneiform joints; *type 2*, subtalar, talonavicular, or calcaneocuboid joints; and *type 3*, tibiotalar joint

prevalence of diabetes worldwide from 2.8 to 4.4 % until 2030 [119].

Diagnosis

The diagnosis of Charcot arthropathy starts with general and orthopedic physical examination at the first presentation of the patient. Both lower extremities from the hip to the tiptoes should be examined together. Both feet and ankles should be inspected to detect any deformities while the patient is standing followed by examination of the patient's gait. Patients with end-stage Charcot arthropathy usually present with gross deformity including substantial loss of longitudinal and transversal arch with so-called rocker bottom foot [51, 109]. However, the exact diagnosis of Charcot arthropathy and its stage is often very challenging; there is no exact literature data; the rate of misdiagnosis is quite high [7]. The red, swollen, and insensate foot is often misinterpreted as infection (e.g., erysipelas) or edema. Some patients may present with both entities including Charcot arthropathy and infection which is definitely the worst-case scenario. Therefore, infection should be ruled by accurate and complete patient history, clinical examination, and taking blood samples (white blood cell count, C-reactive protein, procalcitonin, etc.) [28].

The "position test" is used to evaluate the swelling of the lower leg [37, 113]. The patient is asked to lift the affected leg while lying supine. The leg should remain lifted for 3–4 min, with help from the examiner if needed. In patients with Charcot arthropathy, the swelling usually declines, while in patients with infection, with or without osteomyelitis, the swelling persists over the time [37, 113].

The careful clinical evaluation includes the neurologic assessment which assesses clinical effects of sensory, motor, and autonomic neuropathy. Neurologic evaluation starts with sensation testing which may include Semmes-Weinstein monofilament, temperature and vibratory testing, and motor nerve conduction velocity studies [8, 14, 31, 36, 70, 81]. In active Charcot arthropathy, the affected lower leg is usually 8 °C warmer than the contralateral side [29]. However, in patients with infection, cellulitis, and/or osteomyelitis, temperature difference are also often observed.

Another crucial step of the clinical assessment is the vascular evaluation to determine whether local circulation is sufficient for primary healing [103]. The measurement of the ankle-brachial index is important to assess the micro- and macrovascular status. Values between 0.9 and 1.2 are appropriate, while an index less than 0.9 indicates impaired blood flow [74, 84]. Doppler analysis is another useful diagnostic tool to assess circulation in patients with Charcot arthropathy [88, 121]. Wu et al. [121] demonstrated in 15 patients with acute diabetic Charcot arthropathy that the Doppler

Table 17.4 The Schon classification system describing Charcot neuroarthropathy of the midfoot [101]

Type	Descriptions
I	*Lisfranc pattern*
IA	Breakdown along the medial column of Lisfranc joints, primarily in the first, second, and third metatarsocuneiform joints. Increased pressures on the first metatarsal and perhaps a hallux valgus. Foot may be slightly abducted and arch may have slight drop, but no rocker bottom deformity
IB	The foot has a medial rocker or a medial prominence from excessive abduction of the foot. There may be a slight fullness underneath the fourth and fifth metatarsocuboid joint but no complete rocker bottom deformity on the plantar lateral side. Pedobarographic examination shows a medial prominence underneath the first metatarsocuneiform joint and sometimes a slight plantar or lateral prominence in the more advanced type IB cases
IC	Extension plantarly of the medial rocker toward the plantar lateral side of the midfoot underneath the fourth and fifth metatarsocuboid joint. Central rocker often ulcerates and is at risk for infection
II	*Naviculocuneiform pattern*
IIA	Instability or arthritis of the naviculocuneiform joint causes lowering of the medial arch and results in fullness underneath the sagging fourth and fifth metatarsocuboid joints
IIB	The medial arch lowers further, but because the deformity is occurring more proximally in the medial foot, there is no medial rocker. A lateral rocker develops underneath the progressively collapsing fourth and fifth metatarsocuboid joints
IIC	Extension plantarly of the lateral rocker toward the central and medial plantar aspect of the foot. This prominence often ulcerates and is at risk for infection
III	*Perinavicular pattern*
IIIA	Early avascular necrosis of the navicular or minimally displaced fracture of the navicular. Very mild lowering of the medial arch and fullness plantarly underneath the fourth and fifth metatarsocuboid joint from a decrease in lateral arch height
IIIB	Progressive fragmentation of the navicular with dorsal subluxation of the navicular on the talus and shortening of the medial column. A lateral rocker bottom deformity develops under the fourth and fifth metatarsocuboid joint
IIIC	Clinically, the rocker bottom deformity shifts from underneath the fourth metatarsocuboid joint to slightly more proximally under the cuboid itself toward the central aspect of the foot. Typically, the talus is severely plantar flexed within the ankle mortise and the navicular sits dorsally on the neck of the talus. There is loss of length of the medial column and some secondary changes may develop between the talus and cuneiform joints. There may be complete dorsal translation of the medial column with the remnants of the cuneiform metatarsals sitting dorsally on the neck of the talus. Ulceration and infection are likely
IV	*Transverse tarsal pattern*
IVA	Lateral subluxation of the navicular on the talus with resultant abduction of the foot and valgus of the calcaneus. The calcaneus begins to lose its pitch and there may be a dorsal translation of the cuboid relative to the axis of the calcaneus. Patients begin to have a more central-lateral fullness over the calcaneocuboid joint. This fullness is more proximal and more central than that which occurs at the fourth and fifth metatarsal calcaneocuboid joints
IVB	Progressive adduction of the foot on the head of the talus; decrease in the medial arch height; calcaneal pitch is more parallel to the ground. A plantar central rocker develops underneath the calcaneocuboid joint
IVC	There may be severe crushing of the calcaneocuboid articulation or dorsal translation of the cuboid relative to the plantar aspect of the calcaneus. The calcaneus and the talus are in progressive equinus. There often is extreme abduction of the navicular on the talus and, at times, complete dislocation of this joint. Clinically, there is a central proximal rocker because the posterior calcaneal tuberosity is non-weight bearing and all the weight is borne at the distal end of the calcaneus and cuboid. There may be a medial rocker underneath the navicular and the central rocker underneath the calcaneus plantarly. These cases have osteomyelitis of the distal calcaneus or, occasionally, develop in the talus because it is uncovered by the navicular

spectrum analysis may reflect the activity of Charcot arthropathy. Doppler sonography should also be used as screening tool to diagnose deep vein thrombosis [111].

Pedobarography is a diagnostic tool for assessing very precisely static and dynamic foot pressure [20, 69]. Pedobarography can be also used to assess the efficacy of surgical treatment

Table 17.5 Wagner classification for diabetic foot ulcers

Wagner grade	Clinical characteristics
0	Preulcerative or postulcerative lesion
1	Partial or full-thickness superficial ulceration
2	Ulceration that probes to tendon or capsule
3	Deep ulceration to the bone
4	Partial foot gangrene
5	Whole foot gangrene

Table 17.6 University of Texas classification of diabetic foot ulcers

Grade	Clinical characteristics
0	Preulcerative or postulcerative lesion
1	Partial or full-thickness superficial ulceration
2	Deep wound that involves tendon or capsule
3	Wound penetrating the bone or joint
Stage	
A	Clean wound
B	Nonischemic infected wound
C	Ischemic noninfected wound
D	Ischemic infected wound

[69]. In patients with Charcot arthropathy, two pressure peaks are often observed: one originating from the heel and the other from the forefoot. However, in patients with progressive rocker bottom deformity, pressure distribution changes toward a large peak at the midfoot [69].

During the clinical assessment, special attention should be given to patients with ulcerations and wounds. Different staging classifications for diabetic ulcers have been described in the literature; the Wagner classification is the most widely used and accepted grading system for diabetic foot ulcers (Table 17.5) [13, 118]. However, the University of Texas classification should also be considered as it is more advanced and considers parameters, depth, and ischemia (Table 17.6) [53].

Radiographic evaluation in patients with Charcot arthropathy includes first the conventional weight-bearing radiographs: anteroposterior and lateral views of the foot, mortise view of the ankle, and Saltzman view (hindfoot alignment view) (Fig. 17.3) [96]. Only weight-bearing radiographs should be used for radiographic assessment because non-weight-bearing radiographs are often misleading regarding the evaluation of deformities [104]. Typical radiographic findings in patients with Charcot arthropathy are dependent on the arthropathy stage and can include fragmentation, subluxation, luxation, and bone fractures (Table 17.2). Computed tomography (CT) is an imaging modality for precise evaluation of bone anatomy, especially the cortical bone (Fig. 17.4). Magnetic resonance imaging (MRI) is another advanced imaging tool to assess pathologies of bone and soft tissues (Fig. 17.5). Zampa et al. [125] used dynamic MRI to assess the activity level of acute Charcot arthropathy in 40 diabetic patients and demonstrated its reliability for predicting and monitoring treatment outcome. MRI is especially helpful in the early stages of Charcot arthropathy as it can evaluate bone pathologies with more sensitivity than conventional radiographs [17]. However, the differentiation between Charcot arthropathy and osteomyelitis is also difficult using MRI [21].

Conservative Treatment

Diabetes is known as a multisystem disease requiring a multidisciplinary approach which may include orthopedic surgery, vascular surgery, internal medicine, endocrinology, neurology, infectious disease, physical therapy, rehabilitation, orthotics, prosthetics, and other fields [46]. A multidisciplinary team approach and patient education are crucial to obtain favorable results following treatment in patients with Charcot arthropathy. It has been demonstrated that appropriate patient education may help to decrease the incidence of ulceration in diabetic patients [24].

In most patients with Charcot arthropathy, conservative treatment is the primary therapy. One of the most important aims of the conservative treatment is to off-load and to immobilize the affected limb (Case 17.1) [46, 93]. The patients are immobilized using various methods such as total contact casts (TCC), walking boots, or other orthotic devices [65]. Especially in patients with Charcot arthropathy stage I, early diagnosis and starting the conservative treatment is important.

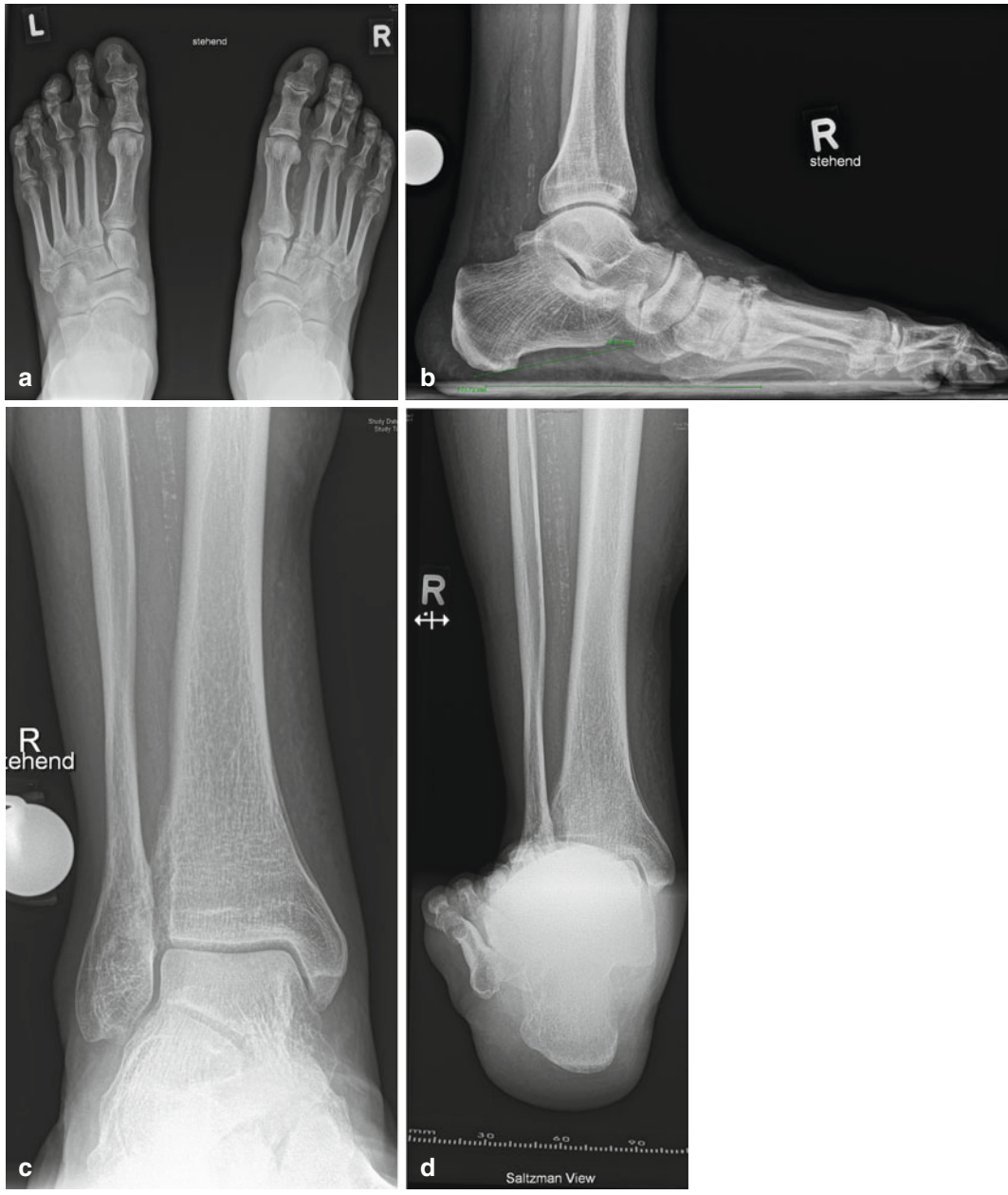


Fig. 17.3 Conventional weight-bearing radiographs of the right foot/ankle including (**a**) anteroposterior and (**b**) lateral views of the foot, (**c**) mortise view of the ankle, and (**d**) Saltzman view of the hindfoot. Radiographs are of a 69-year-old male patient with progressive Charcot arthropathy of the right midfoot: type II according to the Sanders and Frykberg classification or type 1 according to the Brodsky classification. Anteroposterior view of the ankle demonstrates progressive destructive Charcot arthropathy of the 1st–3rd tarsometatarsal joint with consecutive abductus deformity of the forefoot. Lateral view also demonstrates Lisfranc joint arthropathy with Lisfranc joint sack and consecutive loss of the medial arch. Mortise view of the ankle shows physiological alignment of the tibiotalar joint without evidence of osteoarthritis. Saltzman view of the hindfoot demonstrates valgus malalignment of the hindfoot with abductus deformity of the forefoot ("too-many-toes sign"). Note the calcification of the main vessels around the ankle joint

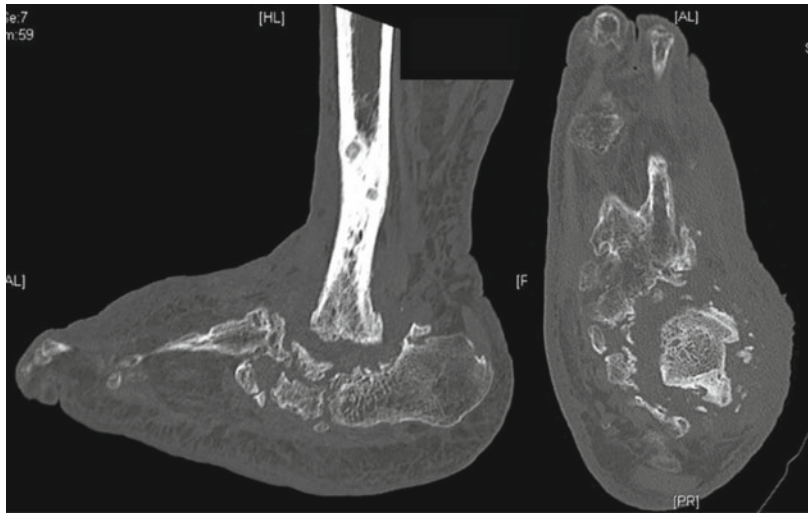


Fig. 17.4 Computed tomography of a 63-year-old male patient presented with Charcot arthropathy (Case 17.4)

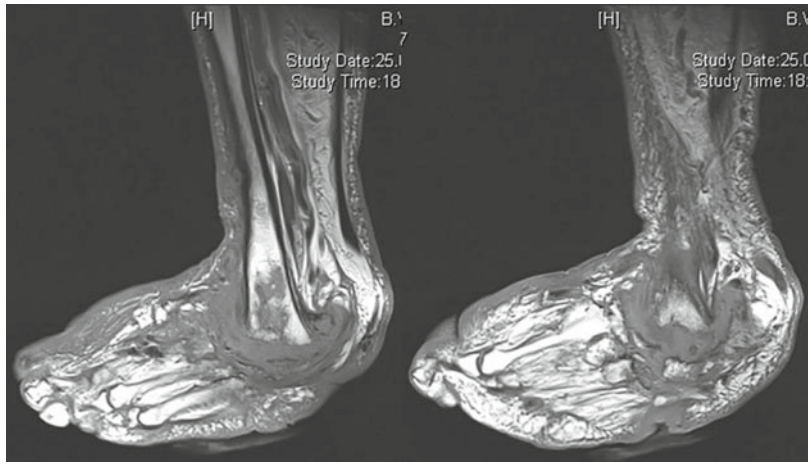


Fig. 17.5 Magnetic resonance imaging of a 63-year-old male patient presented with Charcot arthropathy (Case 17.4)

Stage I usually lasts for 5–6 weeks [44, 81], and following this period of time, the deformities may become permanent. Non-weight bearing is usually recommended for 8–12 weeks to help avoid the destructive phase of Charcot arthropathy [34].

Especially in patients with diabetic ulcers, the immobilization using the TCC has been reported to have promising outcomes. Mueller et al. [67] compared the treatment of TTC with traditional dressing treatment in patients with diabetic plantar ulcers; in patients treated with TTC, 19 of 21 ulcers healed in 42 ± 29 days, while traditional dressing treatment resulted in healing of 6 of 19 ulcers in 65 ± 29 days [67]. Frigg et al. [33] analyzed outcomes of TTC in 28 patients with 34 diabetic ulcers, and effective healing was observed in 85 % of all patients. However, the recurrence rate was 57 % [33].

Appropriate shoe choice is an important part of conservative care of patients with Charcot arthropathy [46]. Numerous publications have demonstrated the effectiveness of therapeutic footwear in reducing plantar pressure peaks and subsequent decrease in rate of plantar ulcerations [12, 22, 60, 91, 115].

Currently, there is no evidence-based literature demonstrating clear efficacy of the pharmacological management of acute Charcot arthropathy [3]. There is limited data suggesting that pamidronate, alendronate, and calcitonin provide some clinical and biomechanical improvement [3].

Surgical Treatment

Surgical armamentarium for treating Charcot arthropathy is diverse, ranging from surgical debridement of ulcerations to the realignment of deformities associated with Charcot arthropathy. As already mentioned, first, the conservative treatment should be performed first, and in cases with conservative treatment failure, the underlying problem should be addressed surgically.

The timing of surgical treatment in patients with Charcot arthropathy remains a controversial clinical dilemma because surgical treatment can be highly complicated [61]. Hastings et al. [41] investigated the progression of foot deformity in 15 subjects with Charcot arthropathy at one and two years after initial assessment. In all patients significant worsening of foot alignment was observed [41]. These findings support the need for aggressive surgical intervention in cases with dislocation and/or deformities to prevent limb-threatening complications.

Lawall [55] described the initial surgical treatment in patients with Charcot arthropathy as "IRAS:" "Infection control," followed by "Revascularizing procedures" (if necessary), "minor Amputation" (if necessary), and adequate "Shoe wear."

In patients with diabetic foot ulcers, the first step of treatment or planning of treatment is to classify the lesions (Tables 17.5 and 17.6). The following questions should first be addressed: extent of lesion, perfusion of the foot, and possible infection [46]. It is especially important for patients with infected diabetic ulcers to be admitted to the hospital for appropriate treatment which commonly utilizes a multidisciplinary approach with consultations for metabolic management and infectious diseases. IV antibiotics and surgical debridement of the wound may also be indicated [46, 59].

The differentiation between Charcot arthropathy and osteomyelitis is often challenging. Dual-isotope single-photon emission computed tomography-computed tomography (SPECT-CT) is a helpful diagnostic tool with higher accuracy in diagnosing and localizing infection compared with conventional imaging [42, 43].

According to general recommendation, vascular evaluation should be performed in patients with Charcot arthropathy. If necessary, further vascular surgical intervention should be performed to optimize the vascular status [5, 72, 73].

The next step in surgical treatment of Charcot arthropathy is the "minor amputation." This procedure includes exostectomies for symptomatic bony prominencies [61, 80]. Brodsky and Rouse [10] reported the midterm results in 12 patients who underwent exostectomy for symptomatic bony prominencies due to type I Charcot arthropathy. In total, 25 % of patients experienced complications, most commonly due to soft tissue healing. In general, this procedure has been demonstrated to be satisfactory, with significantly less morbidity and faster healing than more major reconstructive procedures including midfoot arthrodesis [10]. Similar well-promising results have been demonstrated in a retrospective study by Rosenblum et al. [95] which assessed 31 patients with a mean follow-up of 2.5 years.

The last and the most demanding step in surgical treatment of Charcot arthropathy is the correction of underlying deformities. The surgical treatment of deformities associated with Charcot arthropathy still remains a subject of controversial debate. However, the main aim of surgical treatment of Charcot arthropathy is undisputedly a plantigrade and stable foot without any bony prominences resulting in no risk of diabetic ulceration and the ability to wear normal and conventional shoes [92]. There is no overall "perfect" surgical treatment of Charcot arthropathy to achieve the aforementioned aims. Numerous studies have been published in the last decades describing surgical techniques

Table 17.7 Complications associated with surgical procedures in patients with Charcot arthropathy

Surgical complications
Wound healing problems, delayed wound healing
Superficial/deep infection with/without osteomyelitis
Recurrent or new ulcerations
Recurrent or new deformities
Malunion/delayed union/nonunion with or without loosening/failure of hardware
Necrosis (especially talus necrosis)
Amputation

involving the use of internal or external fixation devices including solid or cannulated screws, plates, blade plates, intramedullary nails, and external fixators [101, 114]. For preoperative planning, the exact localization of Charcot arthropathy should first be assessed. We recommend the aforementioned Sanders and Frykberg classification (Fig. 17.1) [34]. Preoperative imaging including weight-bearing radiographs and CT and/or MRI if necessary should be used for meticulous preoperative planning including assessment of degenerative changes and concomitant deformities. The list of possible complications associated with any surgical procedures in patients with Charcot arthropathy is extensive (Table 17.7), and informed consent should include all of them. Patients should be appropriately informed before the surgery that the risk of postoperative complications is higher than in non-diabetic patients and that several complications can occur at once.

In the Sanders and Frykberg type I area, Charcot arthropathy is not a commonly observed problem. Ulcerations are probably the most common problem of the forefoot in diabetic patients; approximately 75 % of all diabetic foot ulcers are localized under the metatarsal heads [35]. Patients with diabetic forefoot ulcers often present with an Achilles tendon contracture [78]. It has been demonstrated that the patients with persistent diabetic ulcers after TCC treatment had a mean lack of ankle dorsiflexion of −10.5°, while patients with successful treatment had some dorsiflexion with 1.9° [58]. Mueller et al. [68] performed a randomized clinical trial comparing outcomes for patients with diabetes mellitus and a neuropathic plantar ulcer treated with a TCC with and without an Achilles tendon lengthening. All diabetic ulcerations healed in the Achilles tendon lengthening group, and the risk for ulcer recurrence was significantly lower in this group with 75 % and 52 % less at 7 months and 2 years, respectively [68]. In patients with forefoot ulcerations but without osseous deformities of the forefoot, pressure can be substantially reduced by metatarsal shortening osteotomies or plantar condylectomy [35]. In some cases, the distal resection of all four lesser toe metatarsals is necessary (e.g., Clayton procedure). In those cases, the first ray should be stabilized first with metatarsophalangeal arthrodesis (Fig. 17.6) [47].

The midfoot including Sanders and Frykberg types II and III areas is the most common area for Charcot arthropathy [10, 46]. The surgical reconstruction and stabilization of the midfoot with Charcot arthropathy is often technically demanding because of complex anatomy and biomechanics in this area of the foot. All present deformities including luxations and subluxation (Fig. 17.7) should be carefully analyzed preoperatively. In the last decades, different surgical procedures have been described to treat different stages of Charcot arthropathy with and without concomitant deformities.

In patients with stable midfoot deformity who present with bony prominencies causing the midfoot ulceration, exostectomy is indicated as the first-choice surgical treatment [10, 38]. In general, the incision for the exostectomy should be performed on the non-weight-bearing plantar surface of the foot. The use of a separate incision and not through the ulcer may reduce the risk of bony contamination [10].

In patients with unstable Charcot arthropathy and/or in patients with failed exostectomy, midfoot reconstruction with open reduction and internal fixation and arthrodesis is recommended. In general, midfoot reconstruction can be performed using a medial and/or lateral longitudinal approach [35]. The surrounding soft tissue dissection should be performed carefully and minimized to ensure postopera-

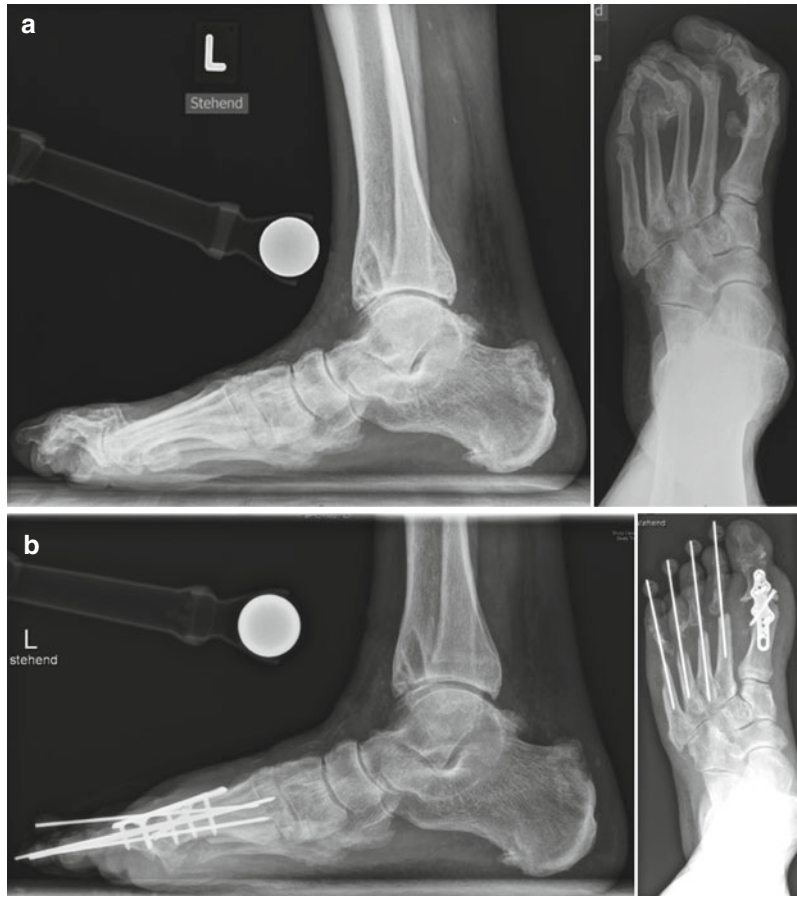


Fig. 17.6 Surgical treatment of Charcot arthropathy of the Sanders and Frykberg type I. (**a**) A 77-year-old male patient with progressive Charcot arthropathy of the forefoot including all five metatarsophalangeal joints with substantial deviation deformity. (**b**) Surgical treatment including the arthrodesis of the 1st metatarsophalangeal joint and resection of all four lesser toe metatarsals

tive wound healing. After the cartilage is debrided in joints with Charcot degenerative changes, often, one or more osteotomies are required to realign the midfoot. Marks et al. [62], in their paper which won the Roger A. Mann award, performed the biomechanical analysis of different fixation techniques in neuroarthropathic feet. They demonstrated that a plate applied to the plantar aspect of the medial midfoot provides significantly better stability than fixation with screws alone [62]. Pope et al. [83] established in their biomechanical cadaver study that midfoot fixation using plantar planting has a stiffer construct than fixation using intramedullary screws.

Recently, Sammarco [97] presented research that also sought to treat Charcot midfoot deformity. Sammarco used the term "superconstruct" to describe the surgical technique he used to stabilize the midfoot deformity. A superconstruct was defined by four following factors: (1) fusion is extended beyond the zone of injury to include joints that are not affected to improve the stability of fixation; (2) bone resection is performed to shorten the extremity for adequate reduction of deformity without compromising the soft tissue envelope; (3) the strongest fixation hardware is used dependent on soft tissue status; and (4) the fixation hardware should be applied in a manner that maximizes mechanical function and stability

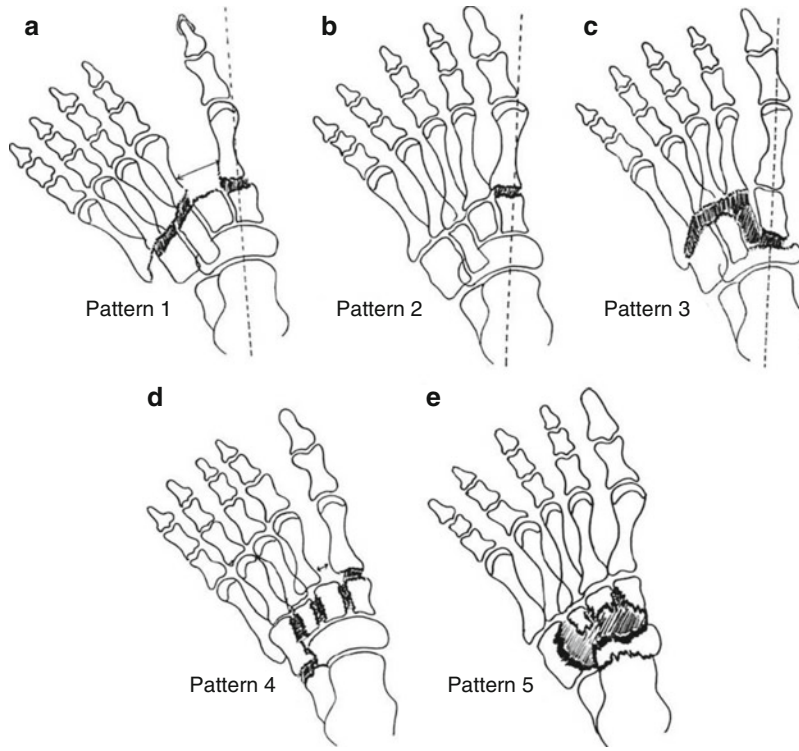


Fig. 17.7 Sammarco and Conti classification of pattern of Charcot midfoot dislocations [98, 99]. (**a**) Diastasis between the 1st and 2nd metatarsals, with middle and lateral column dislocation/dissolution at the tarsometatarsal joint. (**b**) 1st tarsometatarsal joint involvement only. (**c**) Medial column dislocation at the naviculocuneiform joint, with tarsometatarsal joint dislocation of the middle and lateral columns. (**d**) 1st tarsometatarsal joint dislocation with 1st–2nd metatarsal diastasis, intercuneiform fragmentation, and extension to the calcaneocuboid joint. (**e**) Perinavicular arthropathy with distal intertarsal fragmentation and extension

[97]. Early reports using this novel technique are promising [64, 99, 100]. Sammarco et al. [99] treated 22 patients with Charcot midfoot deformity using this technique. Complete osseous union was observed in 16 of 22 patients, while five other patients had a partial union and only one patient presented with nonunion. All radiographic alignment parameters of the midfoot significantly improved following the reconstructive procedure and remained stable over time [64]. The intramedullary fixation devices should not be used alone due to low rigidity and inefficient rotational instability. However, Wurm et al. [123] reported favorable results with high fusion rate of 92 % in 17 ft.

Sanders and Frykberg types IV and V areas together make up the hindfoot which can also be affected by Charcot arthropathy and diabetic ulcerations. The main goal of surgical treatment in these patients is to maintain a stable hindfoot with neutral alignment allowing for ambulation with or without minimal restrictions. In patients with isolated Charcot arthropathy of the subtalar joint, subtalar arthrodesis should be performed [82]. However, in most cases the entire hindfoot complex and especially the Chopart joint is involved in the Charcot arthropathy. In this patient cohort, a triple arthrodesis involving subtalar, talonavicular, and calcaneocuboid joints should be performed [71, 76, 126]. In cases with a well-preserved calcaneocuboid joint, the double fusion including subtalar and talonavicular joints can be performed as an alternative [126]. In patients with end-stage degenerative change of the tibiotalar joint due to Charcot arthropathy, the treatment of choice is the ankle arthrodesis [1, 32, 79, 124]. Lee et al. [56] published a case report on a 45-year-old female patient with unilateral ankle Charcot

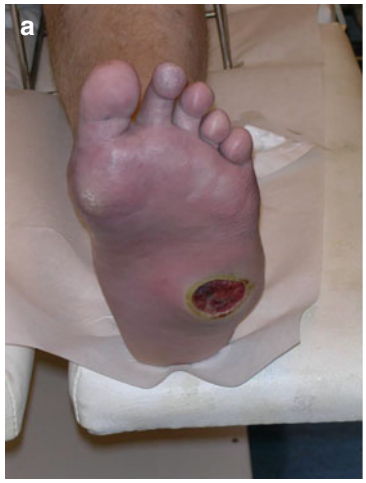
arthropathy treated by total ankle replacement using a three-component prosthesis design. However, we do not recommend total ankle replacement in patients with Charcot ankle arthropathy due to lack of evidence regarding the midterm and long-term results following this procedure [4].

Different fixation types have been described in the current literature; however, the use of external fixators including Ilizarov ring systems may provide an acceptable postoperative outcome [26, 32, 79, 116, 120]. Fragomen et al. [32] presented midterm results in 101 patients who underwent complex ankle arthrodesis using the Ilizarov fixation. In 15 of 101 patients, the ankle arthrodesis was performed due to Charcot ankle arthropathy with a complete osseous union in 73 % [32]. El-Gafary et al. [26] used Ilizarov frame to perform the ankle arthrodesis in patients with Charcot arthropathy. In all patients a solid fusion occurred at the mean time of 18 weeks [26]. Hockenbury et al. [27] used implantable bone growth stimulation in 10 patients with Charcot ankle arthropathy. In 9 of 10 patients, a solid union was achieved [27].

In patients with the Charcot arthropathy involving tibiotalar and subtalar joints and/or substantial ankle instability (e.g., "floppy hindfoot"), the tibiotalocalcaneal corrective arthrodesis should be performed [52, 63, 77, 85, 87, 106, 117]. Von Recum [117] published a detailed surgical technique of the tibiotalocalcaneal corrective arthrodesis using a curved intramedullary nail. This technique has been used in 13 patients, and in all patients osseous healing was completed between four and seven months [117]. Similar encouraging results have been observed in the study by Pyrc et al. [85] with 21 patients treated using the same intramedullary implant. Siebachmeyer et al. [106] treated 20 patients with Charcot arthropathy of the hindfoot with a retrograde intramedullary nail. At a mean follow-up of 26 months, limb salvage was achieved in 12 patients (80 %) [106]. Richter et al. [90] performed a biomechanical study comparing two different intramedullary retrograde nails: straight and curved fixation devices. Comparable biomechanical findings were observed in both groups [90].

Clinical Cases

Case 17.1 A 54-year-old male patient suffering from Charcot arthropathy was diagnosed with a lateral plantar ulceration several months ago (a). The conservative therapy including TCC for a total of 4 months was initiated (b). During the treatment a superficial infection occurred requiring minor excision of the ulceration and debridement. The ulceration healed completely after a total of 27 weeks of conservative treatment (c). At the two-year follow-up, no ulceration was recurrent.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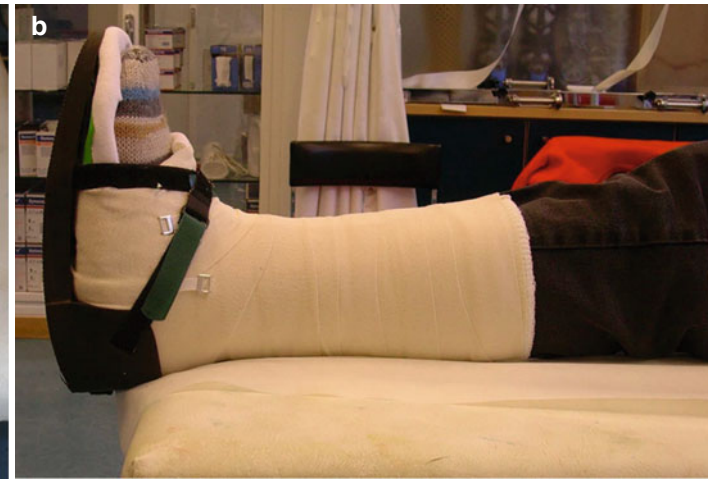
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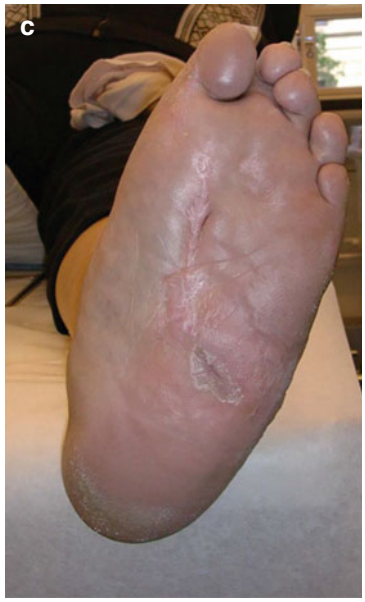
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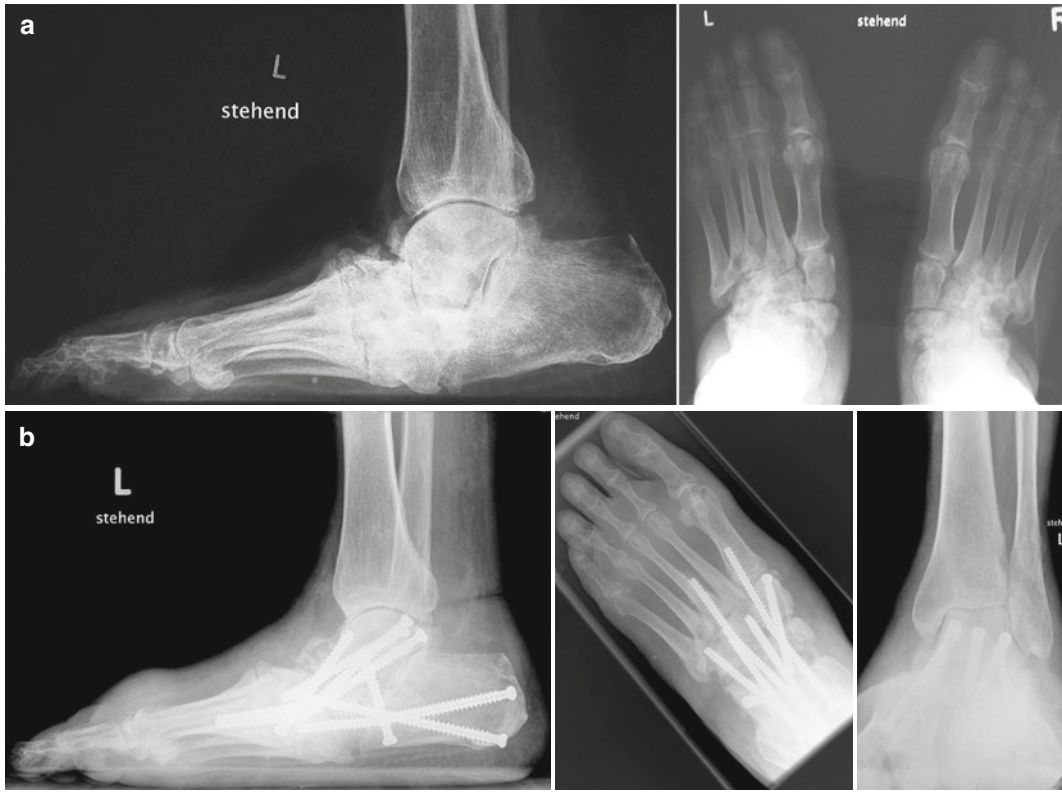
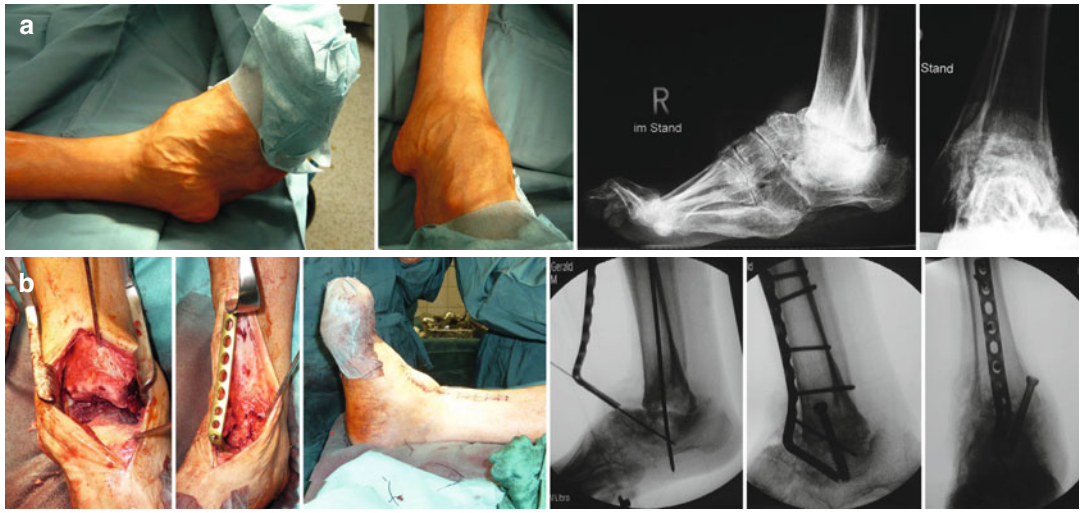
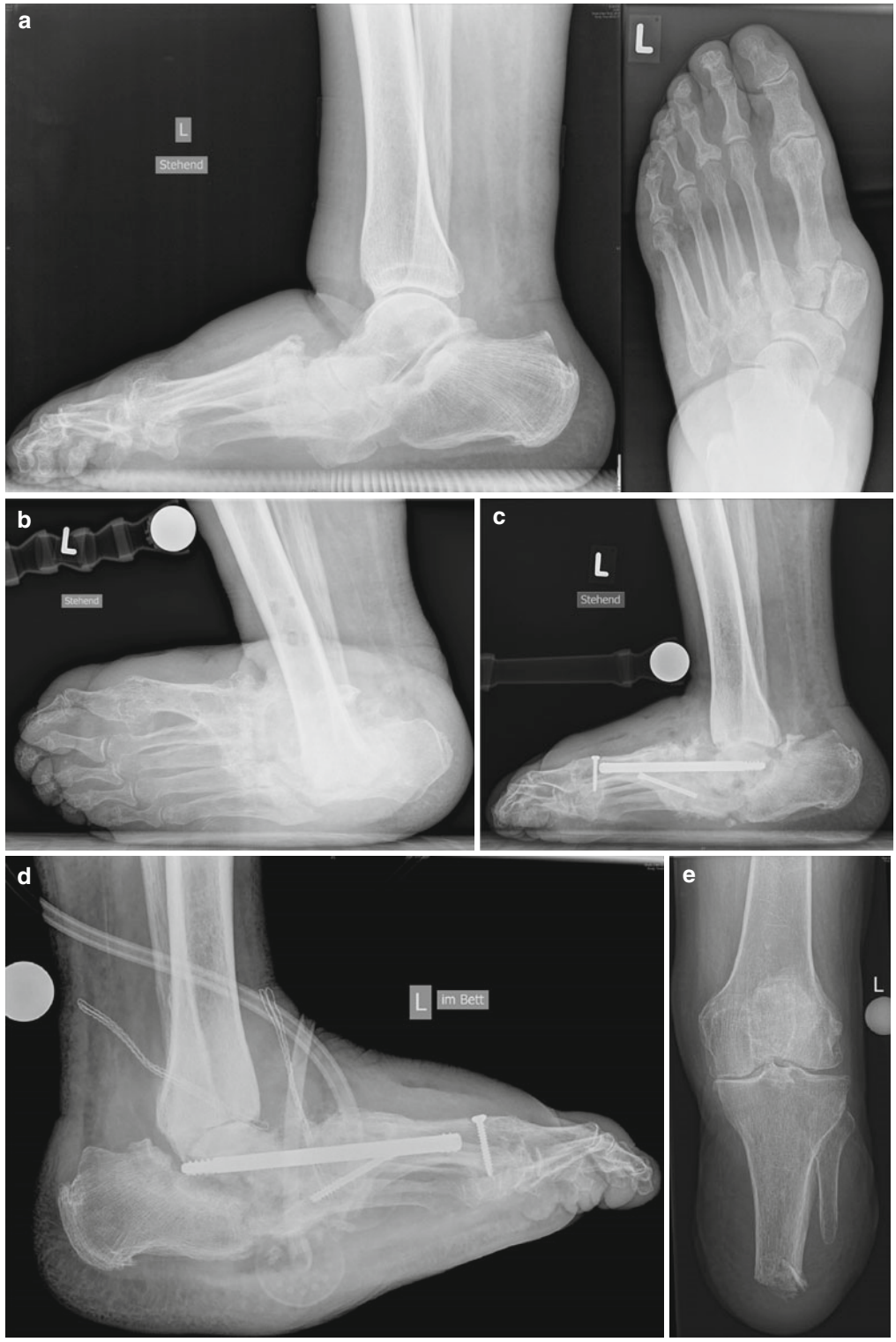
Case 17.2 A 57-year-old male patient presented with Charcot midfoot arthropathy of the Sanders and Frykberg type II. Previously, the Charcot arthropathy in Eichenholtz stage I was misdiagnosed and no specific therapy was initiated. Due to substantial deformity, the patient was no longer able to mobilize using regular shoes (a). Surgical treatment was performed to stabilize the midfoot (b). Although the underlying deformity was not fully corrected, a stable and plantigrade foot was obtained allowing for mobilization in normal shoes.



Case 17.3 A 67-year-old male patient presented with Charcot midfoot arthropathy of the Sanders and Frykberg type IV in his right ankle. The Charcot arthropathy was diagnosed as the Eichenholtz stage II. In the subsequent months, the patient experienced a progression of Charcot arthropathy of the hindfoot with recurrent small lateral plantar ulceration (a). An ankle arthrodesis through the anterior approach using a blade plate fixation was then performed (b).



Case 17.4 A 63-year-old male patient presented with Charcot midfoot arthropathy of the Sanders and Frykberg type II of his left foot. Due to the collapsing of the medial arch, the patient showed a typical "rocker bottom" deformity (a). The conservative treatment with a total contact cast was recommended; however, the patient declined treatment, indicating that his symptoms were not debilitating. The subdued pain level can be explained by progressive diabetic neuropathy. Two years later, the patient presented with grotesque deformity of the entire foot and hindfoot (b) with consecutive ulcerations on the lateral foot border due to irregular plantar loading. The conservative treatment with a total contact cast for three months resulted in complete healing of ulcerations. Afterward, midfoot stabilization using a solid bolt was performed (c). During the postoperative rehabilitation, the patient refused mobilization in the boot and ignored the follow-ups in our clinic. Seven months following the reconstructive surgery, he presented with infected ulceration on the plantar side and was treated by debridement and vacuum-assisted closure therapy (d). Several surgical debridements have been performed; however, the deep infection progressed to osteomyelitis, and a below-knee amputation was performed (e).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Postoperative Rehabilitation

Following surgical reconstruction, patients should be non-weight-bearing or partial weight-bearing for 6–12 weeks. However, as many patients with Charcot arthropathy present with several comorbidities, it is not possible to follow these recommendations in many cases. Therefore, it is important to use immobilization devices including casts or boots to decrease the loading of the foot. All patients should be seen regularly in the outpatient clinic every 7–14 days. Physiotherapeutic support is important to improve the patient's gait and to maintain partial weight bearing if possible.

Conclusions

The best results for patients with Charcot arthropathy can be achieved by orthopedic surgeons with experience in treating diabetic patients, who use a multidisciplinary approach to treatment. In patients with acute phase Charcot arthropathy, the conservative treatment with immobilization of the affected lower limb is the primary recommended approach. Reconstruction of Charcot arthropathy is a technically demanding procedure, and different approaches have been described in the current literature with more or less promising results.

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