MANAGEMENT OF NEUROPATHIC FOOT AND ANKLE

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DR.ARVIND RAJAGOPALAN
Orthopaedic Surgeon

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Name : Dr.Arvind Rajagopalan
Date : 18th February 2012

4th Floor, Wing B, Gopal House, Survey No. 127/1b/1, Plot A – 1, Kothrud, Pune
Tel: + 91-20-66858109 Fax: + 91-20-41216346

MANAGEMENT OF NEUROPATHIC ANKLE

INTRODUCTION

Nueropathic Ankle also termed as Charcots Disease. Jean Martin Charcot was the first to describe arthropathies associated with tabes dorsalis (neurosyphilis). Diabetes is the leading cause of the Charcot foot today.

Charcot osteoarthropathy is an extremely destructive joint disorder affecting single or multiple joints that is almost uniformly initiated by trauma to an insensate limb or region. Those individuals acutely affected exhibit typical signs of inflammation (edema, erythema, and warmth) but generally without the protective sensation of pain. Fracture, dislocation, and instability of multiple joints within the foot or ankle are commonly seen [1, 2]. The process can potentially result in collapse of the foot and severe deformity that frequently results in gait abnormalities and ulcer formation [3, 4]. Characteristically, the entire process leading to gross deformities of the foot and/or ankle is relatively painless [5, 6].

Disorders that have the potential to produce Charcot joints -

Amyloidosis
Alcoholism
Cerebral palsy
Charcot Marie tooth
Congenital insensitivity to pain
Diabetes
Idiopathic sensorimotor neuropathy
Infection
Leprosy
Pernicious anemia
Poliomyelitis
Steroids
Syphilis
Surgery
Syringomyelia
Spina bifida
Spinal or peripheral nerve injury
Trauma

Risk factors

Epidemiological studies are helpful with identifying the risk factors for Charcot neuroarthropathy. Potential risk factors associated with DNOAP in addition to neuropathy include age, sex, weight, duration with diabetes, and osteoporosis [7]. However, the ratio of men to women with Charcot arthropathy is approximately the same and no definite sex predilection has been recorded to date [8, 9]. Age is an associated risk factor with neuropathic joint disease. When neuropathic osteoarthropathy is caused by a congenital indifference to pain or myelomeningocoele, the clinical presentation can occur in a child [7]. However, in patients with diabetes it typically presents during the fifth or sixth decade of life [10]. Other studies report the average age of diabetic neuropathic osteoarthropathy is approximately 57 years with most patients in their sixth and seventh decades [3, 12].
Weight may be a considerable risk factor since the typical patient with diabetes-related Charcot arthropathy is overweight [13]. The mean body mass index (BMI) of Charcot patients according to Pakarinen and colleagues [14] was 32.9 kg/m2 and 34.5 kg/m2 in men and women, respectively.

Duration of diabetes may be an associated risk factor for the development of DNOAP. A review of 85 patients presenting with acute Charcot arthropathy revealed that there are type differences in the demographic features of patients with type 1 and type 2 diabetes developing acute Charcot [10]. Patients with type 1 had a longer duration of diabetes than that with type 2, but developed Charcot at an earlier age. Petrova and colleagues [10] report that in patients with type 1 diabetes the most frequent decade of presentation is the fifth decade, while in type 2 it is in the sixth decade [10]. A long-standing history, at least a decade, with diabetes is common [3, 8, 15].

In type 1, the highest rate of presentation was among those with a 20- to 24-year duration of diabetes, while for type 2 the highest rate of presentation was with a 5- to 9-year duration. In another retrospective study of 36 patients with Charcot deformities, 41% of patients had type 1 diabetes and 59% had type 2 [14]. Eighty-eight percent of the patients required insulin for control of their diabetes and 12% were managed with diet or oral medication [14]. The average duration of type 1 was 28 years and type 2 was 14 years [14].

Peripheral neuropathy is associated with all disorders that produce neuroarthropathy. Severe peripheral neuropathy typically creates a loss in protective sensation. Patients with diabetes frequently have a mixed neurological deficit with components of sensory, motor and/or autonomic neuropathy.

A lack of sensory and proprioceptive awareness, sympathetic disinhibition from autonomic neuropathy, localized osteopenia, and continued weight bearing on unstable joints can lead to deterioration of the traumatic, arthritic, and/or avascular processes that occur with DNOAP [16]. In Fabrin and Holstein's study, 100% of patients had peripheral neuropathy as determined by clinical exam and biothesiometer [17]. The prevalence of peripheral polyneuropathy in the diabetic patient is estimated to be 9.00% to –32.00%, while that of Charcot neuroarthropathy in the overall diabetic population is estimated at 0.09% to 1.40% [14].

The relationship between bone mineral density (BMD) and Charcot arthropathy is unclear. It is unknown whether regional osteopenia is a risk factor for developing neuropathic joint disorders or is a result of the inflammatory process that accompanies the bone injury [18]. The osseous structures may be weakened as the result of hyperemic response, metabolic abnormalities, or periods of restricted weight bearing from other preexisting conditions.

Nonetheless, osteopenia has been shown radiographically in severe neuropathy [19] and decreased bone mineral density with diabetic neuropathic osteoarthropathy [10, 18, 20, 21]. Petrova and colleagues [21] measured the calcaneal bone density in type 1 and type 2 patients with unilateral Charcot deformities and those patients without the disorder. The calcaneal bone density in the Charcot foot was lower in both type 1 and type 2 diabetic patients [21]. It was also found that bone density was reduced in the non-Charcot foot in type 1 but not in type 2 patients [21].

Diabetic Charcot arthropathy of the foot and ankle also differs according to the pattern of initial destruction [18]. Herbst and colleagues [18] prospectively studied 55 patients with osteoarthropathy and used dual energy x-ray absorptiometry of the contralateral femoral neck or distal radius to evaluate peripheral bone density. Sixty-one Charcot feet or ankles were divided into three subtypes: fracture pattern, dislocation pattern, and combined fracturedislocation pattern. The fracture pattern was associated with a peripheral deficiency of bone mineral density (BMD), while the dislocation pattern was associated with a normal BMD.

The common underlying factor in the development of Charcot arthropathy is a loss of protective sensation with continued weight bearing and repetitive stress applied to compromised joints in the foot and ankle [3, 15]. The initial bone injury is usually subtle and unrecognized by the neuropathic patient. A history of an instigating event preceding the onset of a Charcot foot has been reported from 22% to 73% of the time [5, 14, 16]. For instance, Charcot arthropathy of the spine has occurred following traumatic paraplegia [23]. The literature also reports surgically induced Charcot arthropathy following podiatric, orthopedic, vascular, and transplantation surgery.

Darst and colleagues [24] describe a case report of osteoarthropathy following a Keller arthroplasty for a recalcitrant hallux ulceration in diabetic patient with peripheral neuropathy. Zgonis and colleagues [25] report a case study of DNOAP following partial forefoot amputation. Last, Fishco [26] provides multiple case reports of Charcot joints developing in neuropathic feet following elective podiatric surgery.

An increased rate of DNOAP has been noted in simultaneous pancreaskidney transplant patients. Flour reports 12% of 66 patients developed neuropathic jointspost-transplant; 4 of them presented within 1 year following transplantation [27]. Also in this study, four patients developed bilateral involvement at a mean of 1.4 _ 2.2 years. Interestingly, a mean pretransplant HbA1c was statistically greater in those patients who developed osteoarthropathy, and those with Charcot feet had an increased risk for rejection [27].

In short, age, weight, duration of diabetes, peripheral neuropathy, decreased BMD, and a history of transplant surgery have been proposed as risk factors for developing DNOAP. Gender does not appear to be associated with the disorder.

Distribution of involvement

Reference	Distribution
Sinha, et al (1971)	12% ankle, 47% tarsal, 34% tarso-metatarsal, 34% metatarsophalangeal
Myerson, et al (1994)	73% tarsometatarsal and naviculocuneiform, 27% talonavicular and calcanealcuboid
Schon et al (1998)	22.6% ankle, 10.0% hindfoot, 59.2% midfoot, 8.0% forefoot
Fabrin and Holstein (2000)	10/140 ankle, 26/140 forefoot,104/140 midfoot
Frykberg (2000)	10% ankle/subtalar, 5% calcaneus, 30% midtarsal, 40% tarsometatarsal, 15% forefoot
Pakarinen et al -2002	8.3% ankle, 2.7% calcaneus, 86.0% midfoot, 13.8% forefoot
Herbst (2004)	19% ankle, 28% hindfoot, 50% midfoot, 3% forefoot 23 with fracture pattern, 23 dislocation pattern and 9 patients with combination of fracture/dislocation

Morbidity and mortality are related to the disease process and the cause of the disease and its related complications. Charcot neuroarthropathy has been recognized for over 130 years and yet it remains a major cause of morbidity for patients with diabetes mellitus. Two thirds of people with Charcot foot have type 2 diabetes [8, 12, 28]. Advances in medical treatment of diabetes have resulted in both an increased lifespan and improved quality of life for diabetic patients, but many have eventual problems with their feet [14].

The major morbidity of a Charcot joint is deformity from either an osseous prominence, ‘‘rocker bottom foot,’’ or joint instability (especially noteworthy in the ankle) [29]. Deformity or instability can all to often lead to ulceration, infection, and ultimate amputation [46]. A recent benchmark analysis shows that diabetic patients with Charcot feet have especially high morbidity, rates of hospitalization, and use of medical resources [15]. Further studies are needed assess the quality of life and working capacity of patients with DNOAP.

Globally, diabetes is the fifth leading cause of death [31]. Although increased morbidity is found in patients with Charcot deformity, an increased mortality rate in these patients has also been suggested. Gazi and colleagues [32] performed a comprehensive review of patients at the diabetic foot clinic at the City Hospital in Nottingham, UK. The survival and incidence of amputations in patients with diabetic neuropathic arthropathy was compared with those diabetic patients without Charcot involvement. According to this study, the mortality of diabetic patients with Charcot foot was just as high as those with neuropathic ulceration. In those patients with osteoarthropathy, 44.7% died after a mean of 3.7 years. Of particular note, 23.4% of patients with Charcot arthropathy required a major amputation, while only 10.6% of patients without this disorder succumbed to a major amputation [32].

Causes of Charcots Syndrome

Now the hunt for the cause of what is often called ‘‘diabetic neuropathic osteoarthropathy’’ has been made difficult for several reasons. The first is that it is a condition without a definition. There are no specific pathologic markers of the disorder, and therefore no firm criteria on which the diagnosis may be based. This absence of a criterion standard means that the diagnosis rests essentially on pattern recognition, and pattern recognition itself is conditioned by the experience and beliefs of the clinician involved. Because the clinician relies on recognition of the association of nonspecific signs, the result is that some expert clinicians might make the diagnosis in instances when others might not, and this is especially true when the extent of damage is limited.

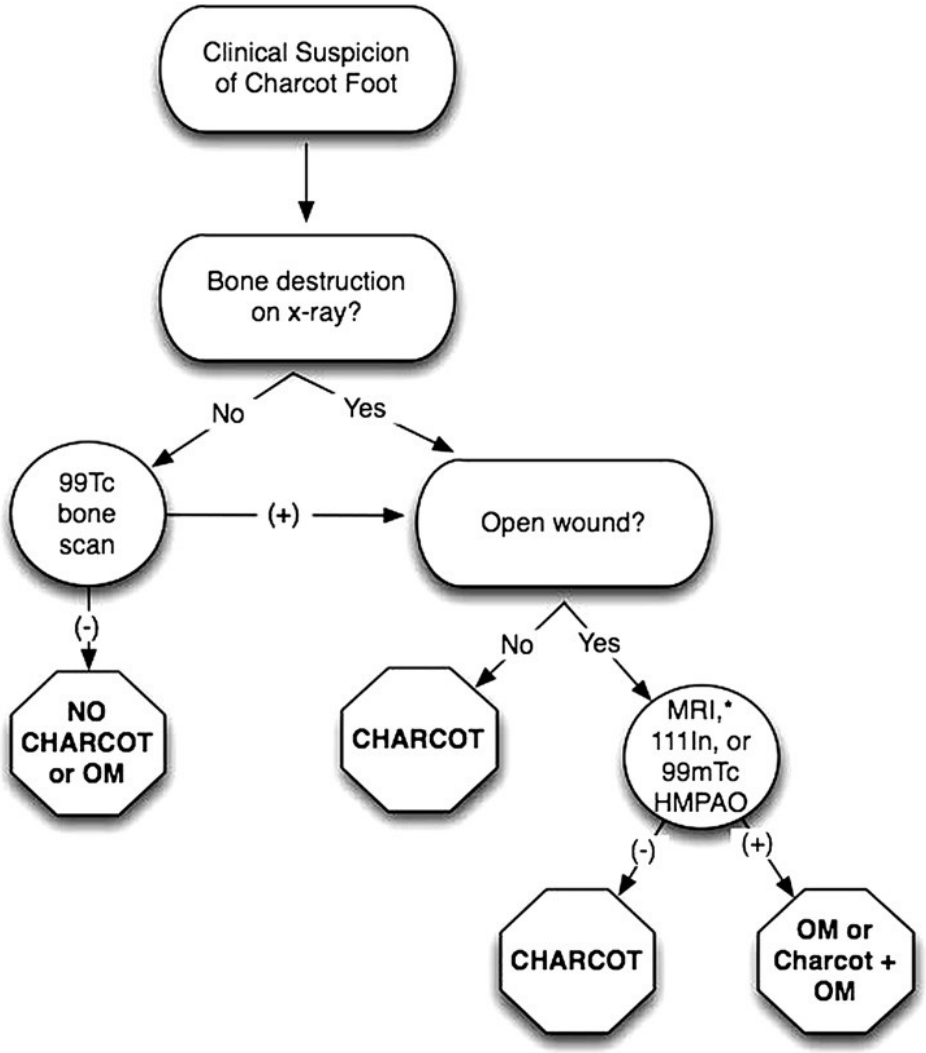
The term Charcot syndrome is suggested because the disorder is not a single disorder but a complex of changes occurring in individuals who are predisposed to its development by several different overlapping factors occurring in several different diseases. In the case of the diabetic foot, the main predisposing factors are the presence of diabetes itself, combined with neuropathy and with preservation of the peripheral circulation.

The condition only occurs when an unrelated event triggers the onset of inflammation in the affected foot, however. Instead of being short-lived, this inflammation becomes protracted as a direct result of lost protective sensation and failure to immobilize the limb. As the inflammatory phase persists, there is progressive osteolysis and damage to the bones and joints. It is further suggested that the person with diabetes and neuropathy is predisposed by increased expression of RANKL and that this expression is increased further by the advent of inflammation. The increased expression of RANKL explains the worsening osteopenia that is observed as the disease progresses and the high prevalence of vascular calcification observed in those who have had it.

The Diagnosis of Charcot Foot

The diagnosis of Charcot foot is challenging, especially in its earliest stages. It is frequently misdiagnosed as cellulitis, deep venous thrombosis, or acute gout. In the later stages, when bone destruction is visible by radiography, it is commonly misdiagnosed as osteomyelitis. This can be particularly troublesome as it may lead some physicians to place patients on long-term antibiotics unnecessarily or recommend amputation as a treatment.

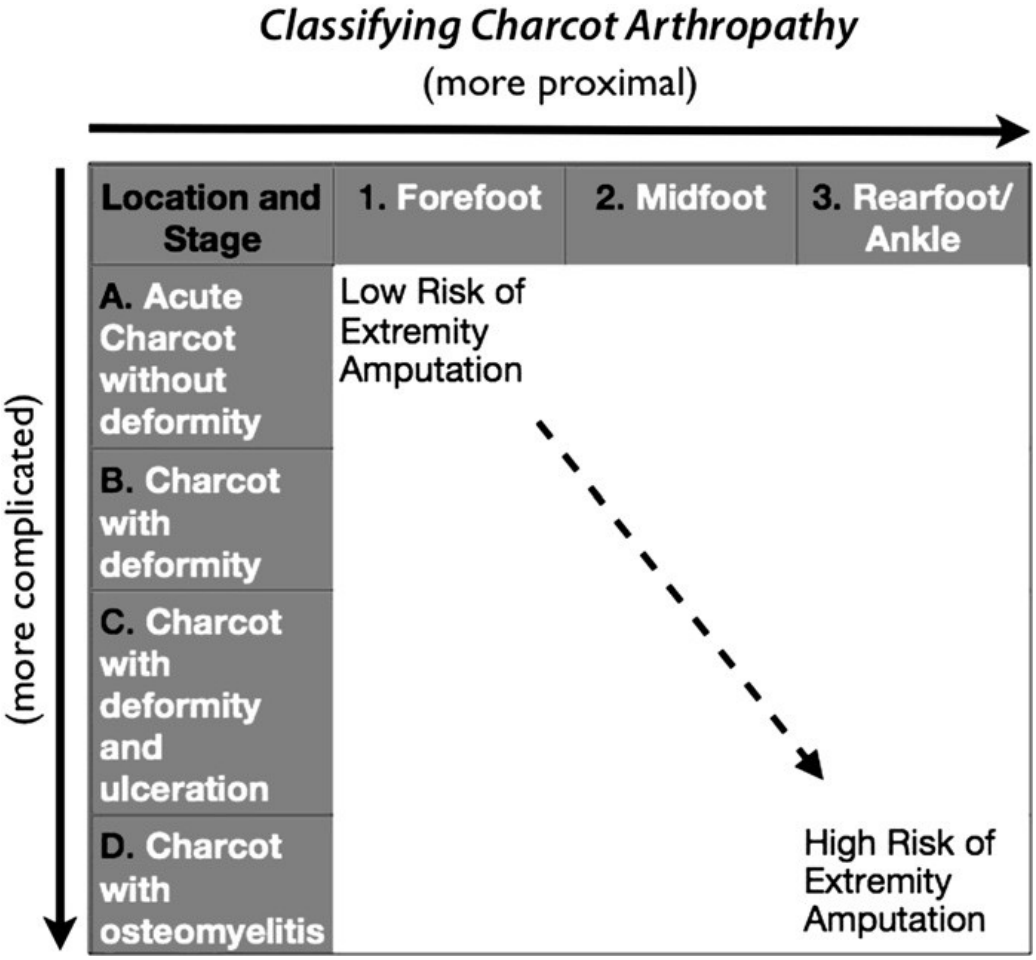
The diagnostic delay averages 29 weeks [11], allowing insensate patients to cause continued trauma to the foot, worsening the deformity. Early detection and treatment can minimize fractures and incapacitating deformities [22]. Tan and colleagues [33] proposed that acute Charcot joint disease is a ‘‘medical emergency,’’ as there are therapies available that can alter its natural history. The diagnosis of Charcot foot is made on clinical examination and imaging.



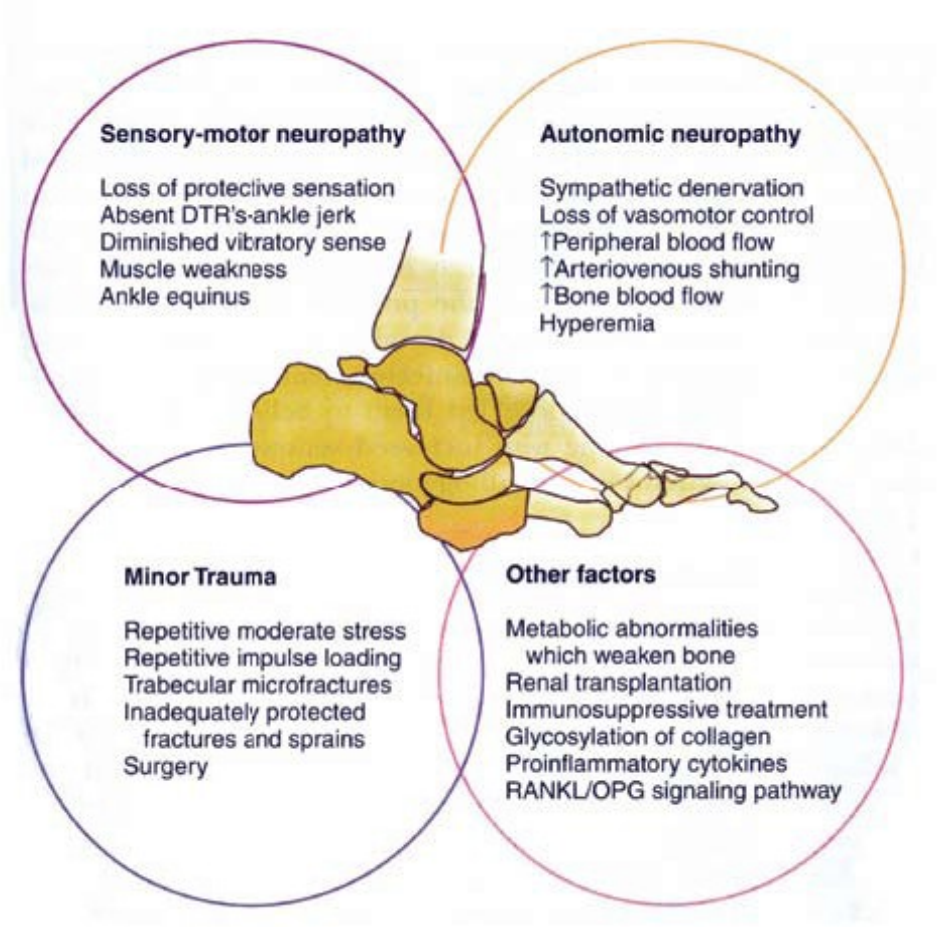
Algorithm for the differentiation of Charcot foot from osteomyelitis (OM).

While the previous classifications are useful in staging or describing the location of the joint involvement, they are not overtly prognostic. We propose a new classification that considers the complications associated with the Charcot joint, which may be a prognostic tool for amputation. This is a two-axis system with the X-axis marking the anatomy affected including 1. Forefoot; 2. Midfoot; and 3. Rearfoot / Ankle. The Y-axis describes how complicated the Charcot joint is. A is acute Charcot with no deformity, B is Charcot foot with deformity, C is Charcot foot with deformity and ulceration, and D includes osteomyelitis. It makes clinical sense that as one moves across the X-axis or down the Y-axis the Charcot foot becomes ‘‘more complicated’’ and, thus, is at greater risk for amputation.

Hence, we postulate that a 1A Charcot foot is relatively simple and at lower risk for amputation than a 3D Charcot foot. This new system combines the features of the clinical exam, radiography, and anatomy unlike the prior classifications.



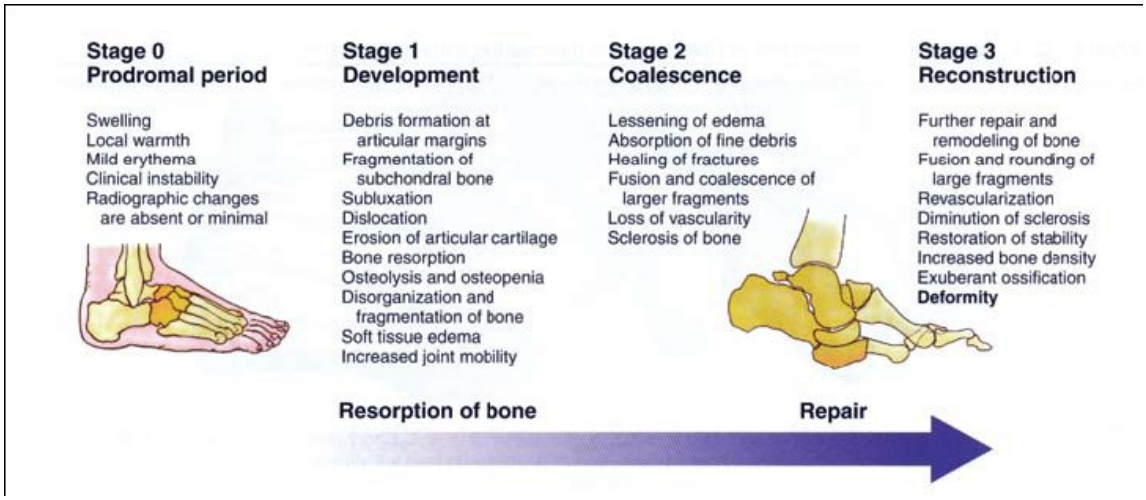
Pathogenesis of Charcot's osteoarthropathy :



The pathogenesis of the joint affected by Charcot's arthropathy may be defined as a ''vicious cycle'' of injury and repair. After initial injury, the inflammatory phase initiates an increase in localized blood flow, alongwith increased histiocytic and osteoclastic activity, removal of blood clots, and resorption of the avascular bone. In the following repair phase, new bone is laid down, producing callus around the fracture sites. Unfortunately, this normal cycle of healing is relatively inefficient, because the bone resorption, which occurred during the inflammatory phase, leaves the resultant atrophic bone easily traumatized even after walking, and the process starts again. The joints most frequently affected by the pathologic changes of Charcot's arthropathy are the weight-bearing joints, predominantly the midfoot but also the hind foot, ankle, knees, and hips.

When the midfoot / midtarsal region is affected, the deformity may result in a prolapsed arch, producing a rocker-bottom foot or a valgus or varus deviation of the forefoot. With a collapsed arch, mechanical stresses are supported during midstance, leading to progressive degeneration of osseous structures and the underlying skin. Consequently, ulcer formation, deep infection, and increased bone destruction are possible outcomes. Severe valgus or varus deformity of the foot and ankle results in large gait forces directed to small areas, such as the malleoli, that are not designed to support.

Classification of Charcot Osteoarthropathy



Charcot's disease of the joint can present in two different ways when viewed radiographically: atrophic and hypertrophic. Atrophic patterns have characteristic dissolution of bone and joint surfaces, commonly seen in the lesser metatarsal regions. The hypertrophic pattern is more common and can present anywhere within the foot. The pattern can be divided into three stages:

1. Fragmentation: the active phase of Charcot's disease; this involves destruction, whereby bony fragmentation and joint disruption are visible, leaving osseous debris surrounding the affected joint.

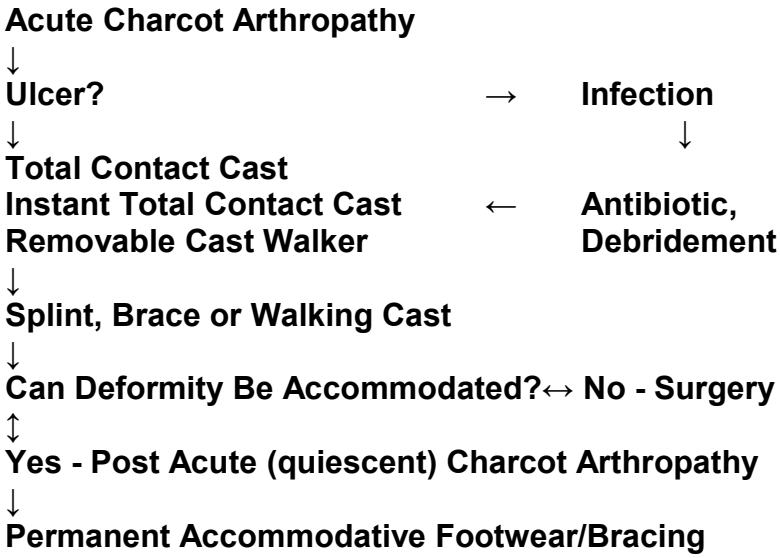
2. Coalescence: healing phase, whereby the osseous debris is resorbed and new bone is laid down. Patterns of trabeculation may be seen across the fractured margins on radiographs.

3. Reconstruction: remodeling phase, which can last from several months to years. Bone integrity is strengthened, and joints are re-established in the form of pseudoarthroses or fusions. The progression of healing during the reconstruction phase determines the outcome of the joints involved. Generally, forefoot and midfoot involvement respond favorably, whereas rearfoot and ankle involvement is more complicated with a poorer prognosis. This is attributable to the severe destruction of ligamentous structures that previously offered a large proportion of support and joint integrity in these areas. It should be noted that some investigators have added a ‘‘stage 0,’’ which is described as acute injury without radiographic evidence [34, 35].

Initial stages of acute Charcot's arthropathy

Treatment goals in the acute phase are to avoid fracture, dislocation, instability, and deformity, thereby obtaining a stable minimally deformed foot. Charcot's neuroarthropathy is one of the most debilitating progressive diseases. Charcot's neuroarthropathy is often mistaken for gout, fractures, or infection. Patients who have diabetes and neuropathy and present with a hot, swollen, erythematous foot should, in many cases, carry a diagnosis of Charcot's neuroarthropathy until proven otherwise. A high index of suspicion, coupled with aggressive intervention and protection, is likely the optimal combination to prevent the often deleterious consequences of this malady.

Treatment algorithm of Charcot's arthropathy :



Medical Treatment of Charcot Neuroosteoarthropathy

Nonpharmacological therapy –

Elimination of physical stress to the Charcot joint (‘‘offloading’’) is essential to break the vicious cycle of repeated trauma propagating the acute phase of CN. Offloading remains the cornerstone of therapy even with adjunctive pharmacological treatments, and is best achieved with a total contact cast (TCC) and reduction of weight bearing, resulting in improvement of clinical markers within 2 weeks of application [36]. Average use of a cast is approximately 12 to 18 weeks, and healing time is significantly reduced with early institution of treatment and proper adherence to partial weightbearing instructions [37, 38]. Alternatives to the TCC such as removable cast walkers have the benefit of being instantly applicable without specialist skills, but compliance with a removable device is significantly reduced [39], and making the cast irremovable with additional bands of plaster has been advocated (‘‘instant total-contact cast’’) [40].

Physical Management of the Charcot Foot

The physical treatment of Charcot arthropathy is focused on the reduction of stress application to the skeletal structure of the foot and ankle. The appropriate treatment is dependent upon the progression of the condition. During stage I the standard treatment choice is the TCC. As the bone begins healing and the clinical signs of Charcot arthropathy diminish (stage II), care may be transitioned to a removable cast walker. When patients progress to the consolidation (stage III), providers should choose the most appropriate footwear as dictated by the severity of foot deformities. Among the many options available are extra-depth shoes, AFO, CROW, and PTB.

Surgical Management of Charcot Midfoot Deformities

Charcot neuroarthropathy and the subsequent foot and ankle deformities negatively impact the lifestyle of the affected individual and may lead to permanent disability and premature retirement [41]. Early diagnosis and intervention is paramount and is associated with a significant lower incidence of deformity, in contrast to a delay in diagnosis and intervention [42]. Charcot neuroarthropathy is frequently misdiagnosed, often resulting in a delay in treatment resulting in worsening outcomes. These patients may or may not recall a traumatic event and present with erythema, warmth, and edema to the lower extremity resembling cellulites or acute septic arthritis. Frequently, they are given antibiotics and continue to

ambulate on the affected foot and cumulate mechanical trauma in the acute phase results in significant bone and joint destruction.

Treatment

In patients presenting with an acute Charcot neuroarthropathy with no apparent foot deformity, aggressive conservative treatment is the mainstay of therapy. Offloading, protection, and stabilization are the key components of therapy [43]. Midfoot deformities can be complex and may be associated with an open wound. In a structurally malaligned foot with an ulceration, the most important goal is to restore a stable, plantigrade foot with ulcer healing and elimination of infection [44]. Mitigating focal areas of increased pressure and shearing forces reduces the risk of ulcer recurrence. Foot and ankle reconstruction with external fixation has been shown to be an effective method of correcting the deformity and providing a stable, plantigrade, foot [45, 46, 47, 48].

Diagnosing Charcot neuroarthropathy requires a heightened index of suspicion. Early recognition and intervention can limit deformity. Aggressive conservative management should be initiated early in the treatment plan in an effort to minimize the devastating effects often seen with this condition. Any patient with neuropathy presenting with even a minor foot and ankle injury should be immobilized and monitored closely. Dermal thermometry and serial radiographs are useful in monitoring the course of therapy. Conservative therapy is effective if initiated early in the treatment plan; however, any delay in therapy can result in severe foot and ankle deformity in which traditional nonoperative methods alone may be inadequate. These deformities may lead to ulcerations and ultimately progress to amputation of the lower extremity. Surgical correction and stabilization is an effective method to prevent further deformity and ulcer recurrence. Numerous studies have reported success with arthrodesis of the Charcot midfoot deformity with fusion rates ranging from 78% to 100% [52]. Pinzer [45] reported a 92% favorable outcome in 26 patients who underwent reconstruction for a high- risk, non-plantigrade Charcot midfoot deformity with a neutral ring fixator. Farber and colleagues [48] reviewed 11 patients with midfoot Charcot neuroarthropathy and ulceration. The patients underwent reconstruction with external fixation and all patients progressed to therapeutic footwear at an average 24-month follow-up. If performed in the appropriate setting and for the right indications, Charcot foot reconstruction is a better alternative to lower limb amputation.

Surgical Technique

Patients are placed in a supine position on the operating room table with the leg in a neutral position. General or regional anesthesia is obtained. A pneumatic thigh tourniquet is applied and the lower extremity is prepped and draped in the usual manner. An equinus deformity at the ankle is a key contributor to the collapse at this anatomical level and the hindfoot equinus is addressed with a percutaneous triple hemisection to reestablish the calcaneal inclination angle. In cases of severe equines, the authors have performed an Achilles tenotomy to improve the calcaneal inclination angle and relieve the stress on the midfoot. If contracture still remains, a posterior ankle joint capsule release is performed.

Next, an incision is made medially at the apex of the deformity in line with the medial column. Subperiosteal dissection is carried laterally, carefully avoiding injury to the dorsalis pedis. A separate lateral incision may be placed if the planned bone resection involves the entire width of the forefoot. Malleable retractors are placed dorsal and plantar in preparation for bone resection. A biplanar wedge of bone at the apex of the deformity is planned with the apices lateral and dorsal. For accurate resection of the biplanar wedge, Kirschner wires may be placed to guide the saw cut from medial to lateral. A sagittal saw is used to begin the bone cuts and the osteotomy is completed with an osteotome and mallet. The wedge of bone is removed and any defects may be filled in with autogenous bone or a bone graft substitute. The forefoot is stabilized to the hindfoot with two 4-mm Steinman pins. Reduction is directly visualized using intraoperative fluoroscopy. Exact anatomic reduction is not required. A linear talar-first metatarsal relationship in transverse and sagittal planes with elimination of bony prominences generally result in a clinically plantigrade foot. When reduction is deemed satisfactory, the wounds are closed in layers over a drain. The tourniquet is deflated and the fixator is applied next.

At this point, the prebuilt frame consisting of two tibia rings and a foot plate are positioned on the foot and lower leg. The rings are centered on the limb ensuring adequate clearance between the skin and frame. Generally, two fingerbreadths anterior and three finger breaths posterior are sufficient to allow for postoperative edema. With placement of all wires, it is important to respect the anatomical safe zones and avoid penetration into neurovascular structures. Wires are manually advanced through the soft tissue and power instrumentation is used to advance the wire through bone. As the wire passes the far cortex, a mallet is used to advance the wire through the remaining soft tissue and skin to reduce thermal necrosis around the wire.

The calcaneus is initially stabilized with two tensioned olive wires placed at approximately 30 degrees to each other. Next, the frontal plane proximal and distal tibia wires are placed from lateral to medial perpendicular to the long axis of the tibia. It is important to ensure bicortical purchase to prevent fracture through the bone. The wires are secured to the frame and tensioned accordingly. Next, wires are placed medial to lateral across the medial face of the proximal and distal tibia at approximately 45 degrees to their respective frontal plane wire. These wires are secured to the ring and tensioned. At this point, one or two wires are placed proximal to the arthrodesis site and fastened to the foot plate and tensioned. Next, with the assistance of intraoperative fluoroscopy, a wire is placed in the forefoot distal to the osteotomy and ‘‘walked back’’ and secured to the foot plate one or two holes proximal to where it exited the foot. As the wire is tensioned to the frame, the forefoot segment compresses against the hindfoot. This ‘‘bent-wire’’ technique allows for uniform compression at the arthrodesis site.

Complications

Complications with Charcot reconstruction using external fixation are common and could be divided into minor and major complications. Minor complications do not alter the postoperative course and include superficial wound dehiscence, wire irritation, or loss of wire stability. Major complications alter the postoperative course and, at times, require return trips to the operating room. These include soft tissue infections, wire breakage, and, most commonly, pin tract infections. The reported incidence of pin tract infection ranges between 5% and 100%, with most studies reporting in the range of 10% to 20% [49]. Erythema and drainage around pin sites are usually the result of micromotion or unstable wires and this may require wires to be tensioned further during the postoperative course to prevent further irritation and the development of a pin tract infection. Risk of postoperative infection is higher when reconstruction is performed in the presence of an open ulceration [50].

Risk of delayed union or nonunion is elevated in this high-risk population. The risk may be decreased with the use of an implantable bone stimulation device in these patients [51]. Smoking has been associated with an increased rate of nonunion and patients are offered assistance in quitting preoperatively.

Charcot deformity affects the rearfoot and ankle less often than the midfoot, but the resultant deformities typically are more severe and difficult to stabilize conservatively. The resultant instability in the ankle leads to a limb-threatening deformity, and surgical intervention and salvage are more common. To date, few data on the best course of treatment are available, but the use of limb-salvage techniques is on the rise. With increasing knowledge of the disease and with technological advances in internal and external fixation, limb salvage is becoming more consistent. This article discusses basic techniques in deformity planning and current uses of internal and external fixation techniques for rearfoot and ankle limb salvage.

Rear foot

Preventing further deformity is a key element in the treatment of patients who have Charcot deformity. In many cases the acute Charcot event, if treated appropriately, can maintain reasonable alignment, and surgery or ulceration from pressure areas in the midfoot can be avoided [53]. This experience has not been described in rearfoot and ankle literature.

Acute or chronic deformity with instability often requires surgical stabilization. Acute Charcot deformity in the hindfoot and ankle leads to greater instability than in the midfoot; therefore the potential for major complication is higher. In the acute patient who has severe deformity and collapse, external fixation is used commonly, whether or not there is concomitant ulceration. Stability will aid the repair of soft tissues, much as in open trauma situations, and the external fixator allows access for local care. The fixator also may allow the correction and maintenance of deformity during the initial stages.

Management of the chronic stage

Once conservative measures have failed to control the deformity, heal ulcerations, or provide a stable extremity amenable to bracing, surgical intervention is warranted. Osseous intervention in the rearfoot and ankle is challenging. Corrective osteotomies or tendon work alone will not give the needed long-term stability. As in other joints with Charcot deformities, arthrodesis is the treatment of choice, but achieving a solid fusion can be challenging. In a review of arthrodesis of the Charcot knee deformity, Drennan and colleagues [54] found that important factors for success include (1) careful removal of all cartilage and debris, (2) debridement to bleeding subchondral bone, (3) meticulous fashioning of bone surfaces for contact, (4) complete debridement of all synovial and scarred capsule, and (5) stable internal fixation.

There are many options for rearfoot and ankle fusions. Once the joints are prepared, unhealthy bone has been debrided, and the deformity has been corrected, the area is stabilized. Internal fixation uses large screws, plates, and intramedullary fixation. Increased stability can be achieved with locked plates, reconstruction plates, or blade plates.

Correction of osseous deformities requires preoperative and intraoperative planning and often requires templates. In the operating room, the patient must be positioned properly. With rearfoot and ankle procedures, a bump under the ipsilateral hip with the patient in a supine position is most common. This position allows the leg to be in a more neutral position for deformity correction and also makes the lateral side more accessible for surgical approach to the ankle. The leg should be prepped and draped above the knee. Fixation for these deformities requires access to the entire leg. The knee is also a landmark for rotation of the lower extremity. To reduce the chance of malunion during arthrodesis, external rotation should align the second toe and the tibial crest. A thigh tourniquet commonly is used as well. In many cases, the tourniquet is elevated during the dissection and deformity correction to aid in visualization and is released once temporary fixation is in place.

Incisions are large. For the rearfoot and ankle, a utilitarian lateral incision often is used. It begins approximately 6 cm from the tip of the lateral malleolus, courses along the lateral border of the fibula, and then makes a gentle curve over the sinus tarsi and calcaneal cuboid joint. This incision allows access to much of the rearfoot and ankle complex. The fibula, if present, is removed 5 cm proximal to the ankle joint and can be used as graft if healthy. The ankle and subtalar joints are visualized easily, and talectomy can be performed if needed.

A second medial incision may be used. It typically is positioned just anterior to the medial malleolus and courses between the tibialis anterior and posterior tendons. This incision allows access to the talonavicular joint and medial gutter of the ankle for medialization of the talus for intramedullary rod fixation. Both incisions can communicate anteriorly, with dissection carried across the distal tibia. A malleable retractor can be used here to protect tissues during corrective osteotomies.

After more ‘‘normal’’ relationships are established and deformity has been corrected, Steinman pins are used for temporary fixation across the rearfoot and ankle. Calcaneal inclination is one important key. Once the rearfoot and ankle are reduced, it gives a building block for the remaining midfoot, and ankle deformities can be addressed. Depending on final fixation, a half-pin is inserted into the calcaneus using fluoroscopy. It enters posteriorly following the normal inclination angle. After the Achilles

lengthening, the half-pin can be used to pull the Achilles into a more anatomic position. At this time a transfixation pin is placed and aids in holding the rearfoot. It enters the inferior calcaneus and is driven proximally through the talus, if present, and into the anterior cortex of the tibia. These pins can be incorporated later into an external fixator, if used. Together these pins provide stability of the rearfoot during further correction. From this point the remaining fixation depends on surgeon preference, clinical circumstances, available healthy bone, and comfort level. Internal fixation is common. External fixation also can be used alone, especially in the face of open ulceration or the acute Charcot process, where fixation is needed away from the unhealthy neuropathic bone.

Whatever the type of fixation, the use of larger, sturdier, and even doubled hardware is common. For internal fixation, locking plates are available. With these plates, all components are locked together at a fixed angle, to disperse force better. For failure to occur, the entire construct must fail, not just one screw. Extra wires, half-pins, or full rings are some of the easiest ways to increase the strength of external fixation.

MATERIALS AND METHODS

Patients with chronic charcot disease of the foot and ankle with instability who had undergone surgical correction at MV Hospital for Diabetes during the period June 2009 to November 2011, were taken up for the study.
13 patients satisfied the above criteria.

All patients were diabetics, with deformity of foot and ankle. All patients had exhausted their preventive managements and presented with long standing deformities, ulcer of foot and ankle.

Patient No.1. : S.R, 69/m, Diabetic since 22 yrs diagnosed with Charcots of Ankle.Patient initially treated with TCC. He developed a ulcer of his foot. The ulcer was treated and correction footwear given. The Deformity started to develop at the ankle and footwear was discontinued. Initially treated with AO external fixation.

Patient No.2 : L.M, 65 year old Male with 18 years diabetic and charcot of ankle and foot. Patient had severe instability of the ankle. Patient had a non healing ulcer in the medial aspect of ankle. Patient was taken in for AO external fixator with intra medullary nail. Patient went in for non union of the Arthrodeisis site. Surgery had to be done twice for metal breakage. Finally developed only fibrous ankylosis.

Patient No.3 : D.K, 55 years old Male with 16 years of Diabetes with Charcot ankle. Patient had 3 months old injury to right ankle, native treatment. Went in for deformity and instability of ankle. Tibio talar fusion done using plate osteosynthesis.

Patient No.4 : K.N , 79 year old Male with 25 years diabetic and charcot of ankle – This patient was treated with AO External Fixator for Tibio Calcaneal Fusion. Patient had ulcer over the heel. The ulcer healed and went in for union after three months.

Patient No.5 : P.R, 70 year old Male with 20 years diabetic and charcot of ankle – This patient had a ulcer measuring about 8-10 cms over the antero lateral aspect of the ankle. There was no osteomyelitis of the distal tibia. The calcaneum was completely destroyed. Talectomy was done. Patient was taken up for Ilizarov ring fixator for tibio - calcaneum fusion.

Patient No.6 : A.R, 58 year old Male with 18 years diabetic and charcot of ankle with untreated trimalleolar fracture of the ankle. The ankle was unstable and grossly deformed with severe callus formation. Patient was treated by Tibio Talar Fusion using Ilizarov ring fixator. Arthrodeisis site went in for bony union.

Patient No.7 : G.B, 58 year old Female with 23 years diabetic and charcot of ankle with trimalleolar fracture ankle treated by open reduction and internal fixation of the fracture. Six months post op, patient developed deformity at the ankle. Has been advised fusion.

Patient No.8 : N.M, 78 year old Male with 30 years diabetic and charcot of ankle with mid foot amputation and non healing ulcer of the foot. The foot was inverted and internally rotated. The non healing ulcer of the hind foot was more than a year old. The patient was taken up for ankle arthrodesis and correction of inverted stump.

Patient No.9 : N.H, 58 year old Male with 18 years diabetic and charcot of ankle with non healing ulcer over the lateral aspect of ankle with partially destroyed talus. Talectomy was done. Tibio Calcaneum Fusion using Ilizarov ring fixator. Went in for bony fusion.

Patient No.10 : R.J, 52 year old Female with 20 years diabetic and early charcot of ankle with trimalleolar fracture treated with open reduction and internal fixation of fracture.

Patient No.11 : D.N, 69 year old Female with 25 years diabetic with Nephropathy and charcot of ankle, morbidly obese patient, discharging cinus medial aspect of ankle. Ankle Arothrodesis of Tibio Talar Fusion. Good progression of fusion was seen in the third post op month. Patient expired due to Nephropathy.

Patient No.12 : M.N, 69 year old Female with 24 years diabetic and charcot of mid foot. Non healing ulcer in the medial aspect of foot. Wedge resection and internal fixation of mid foot deformity done. AO external fixator additional given.

Patient No.13 : S.M, 75 year old Male with 30 years diabetic and charcot of ankle with non healing ulcer over the foot and ankle. The patient was stabilized with a AO external Fixator for two months followed by Ilizarov Fixator for six months. Went in for good fusion.

The above were followed and their outcome were studied.

OUTCOMES

Patient No.1. : S.R, 69/m, - The AO external fixator was used for fusion of the Tibio Calcaneal bones as the patient had a discharging sinus of the medial aspect of the ankle. The tallus was partially destroyed. Tallectomy was performed and the distal tibia and calcaneum were freshened and fused. Patient went in for fibrous ankylosis only.

Patient No.2 : L.M, 65 year old Male – Patient was taken in for AO external fixator with intra medullary nail. Patient went in for non union of the Arthrodeisis site. Surgery had to be done twice for metal breakage. Finally developed only fibrous ankylosis. Deformity corrected with plantigrade foot.

Patient No.3 : D.K, 55 years old Male – Patient had a fibrous union of the fusion site. Patient had instability. Post native treatment for bimalleolar fracture of ankle with severe callus formation was taken up for tibio talar fusion, as instability was only in the tibio talar joint with gross deformity ankle. Plate osteo synthesis was done using locking distal tibial plate. Patient went in for fibrous ankylosis. Lost to follow up.

Patient No.4 : K.N , 79 year old Male – Patient had non healing ulcer of the heel and medial side of the ankle. The ulcer was not healing with correction footwear. Debridement of the ulcer with AO external fixator was done. Ulcer healed in four weeks time. AO fixator was strengthened, went in for plantigrade foot with 50 % union at tibio calcaneal site.

Patient No.5 : P.R, 70 year old Male - This patient had non healing ulcer of the foot and lateral aspect of ankle. Deformity was severe in the ankle. Investigation revealed that the tallus was destroyed. Tallectomy was done and Ilizarov ring fixator was used for tibio - calcaneum fusion. Patient went in for good fusion at tibio calcaneal site. Patient had plantigrade foot and was walking full weight bearing.

Patient No.6 : A.R, 58 year old Male – The patient was having deformity of the ankle with the destruction of the tallus. Partial Tallectomy was done and Ilizarov ring fixator with one foot plate and two rings for the distal leg was used. Patient went in for solid bony union of the arthrodeisis site.

Patient No.7 : G.B, 58 year old Female – This patient had a trimalleolar fracture with chatcot ankle. Patient was taken up for open reduction and internal fixation with plates and screws of the fracture. Six months post op, patient developed deformity of the ankle. Patient was advised metal exit with tibio talar fusion. Patient is walking with partial weight bearing with correction footwear.

Patient No.8 : N.M, 78 year old Male – This patient had an old mid foot charcot which went in for infection of the toes and was amputated at the mid foot level. Patient developed in the ulcer as deformity in the ankle started and foot getting inverted. Non healing ulcer at the lateral aspect of the stump. Ilizarov ring fixator was used and foot was brought to neutral. Ulcer healed, deformity corrected at the ankle.

Patient No.9 : N.H, 58 year old Male – Patient was treated for ankle instability and deformity with correction footwear. Due to non compliance from the patient's side, he underwent a tibio calcaneal fusion with tallectomy. Patient was six months on fixator with good fusion.

Patient No.10 : R.J, 52 year old Female – Patient had a fall and sustained injury to ankle - trimalleolar fracture. Fixed with plates and screws. Six months post op, no instability of ankle. Result – Good.

Patient No.11 : D.N, 69 year old Female – Taken up for Ilizarov ring fixator. Patient did well at arthrodeisis site – tibio talar fusion. Patient was walking non weight bearing from the fourth month. Patient expired due to complications following diabetic nephropathy.

Patient No.12 : M.N, 69 year old Female – Mid foot deformity healed well with internal fixation. Patient had plantigrade stable foot.

Patient No.13 : S.M, 75 year old Male – The non healing ulcer of the medial aspect of the ankle was treated initially. Patient had complications due to diabetes. Sixty days after the initial debridement of the ulcer, patient was taken up for the Ilizarov ring fixator – tibio calcaneal fusion. Six month post op, patient had a stable plantigrade foot with a healed skin grafted ulcer.

ANALYSIS

Of the thirteen patients operated, two patient's required re-surgery for breakage / hardware failure. Two post traumatic patients - trimalleolar fracture, one went in for deformity of the ankle and the other healed well. Patients treated with Ilizarov ring fixator went in for optimum arthrodeisis of ankle. One patient had fibrous ankylosis with osteolysis, one patient expired due to complications of diabetic nephropathy. Of the remaining seven patients, all went in for stable plantigrade foot and ankle.

DISCUSSION

All the patients were operated under sciatic nerve blocks in supine position. The affected limb was cleaned and draped. We used a medial and a lateral incision over the ankle exposing the tibio talar and the talo calcaneal joints. The sub talar joints were exposed when disease had affected them. The osteophytes were exicised, the joints exposed. The joint surface were shaved and freshened. Any remnant's of tallus was used as bone graft after removing the articular surface. The foot and ankle were aligned to see whether the deformity had been corrected, if not, further alignment done. The tibio talar bones were brought together and stabilized with two stemen pin or terminally threaded 2.5 mm – k wires. The implant decided on was used. Assembled ring fixator was used. Good compression at arthrodeisis site was achieved, intra-op., bone grafting was used additionally, wherever required. The wound was closed with a drain-in-situ. IV antibiotics was given for a period of 10 days and converted to oral for another 10 days. Sutures were removed alternately on the 15th and the 20th day.

CONCLUSIONS AND RECOMMENDATIONS

Charcot foot is becoming a larger clinical problem due to the increased incidence of diabetes and morbid obesity, and the improved longevity of affected patients. As the problem has become more aware to Orthopaedic Foot and Ankle surgeons, the interest of the Orthopaedic device industry has provided improved implants for the surgical treatment of this disorder. Increasing incidence and awareness, combined with increased interest by Orthopaedic Foot and Ankle surgeons and improved implants appears to predict a more favorable future for this very complex patient population.

In our study, we find that use of Ilizarov ring fixator in treating charcots rear foot and ankle gives a better surgical outcome when compared to other modalities of fixation.

We recommend from our study of 13 patients that Ilizarov ring fixator to be used in the management of charcot foot and ankle in patients with severe deformity and instability.

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