

Charcot Osteoarthropathy

Here's a review of the history, incidence, pathophysiology, diagnostic presentation, and current trends in treatment.

Goals and Objectives

After completing this CME, the reader will:

- 1) Understand the history of Charcot osteoarthropathy from the time of the initial description to currently in the medical community.
- 2) Understand the pathophysiology of Charcot osteoarthropathy.
- 3) Understand classification systems designed to help identify the stage of Charcot osteoarthropathy along with the location.
- 4) Understand the clinical, radiographic, and advanced imaging presentations of Charcot osteoarthropathy.
- 5) Understand the current standard of care for both conservative and surgical treatment of Charcot osteoarthropathy.
- 6) Understand the techniques for reconstruction of the Charcot osteoarthropathy.
- 7) Understand medical treatments available for the low bone mineral density associated with Charcot osteoarthropathy.

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Following this article, an answer sheet and full set of instructions are provided (p. 212).—**Editor**

By Linnie Rabjohn, DPM, Dan Yarmel, DPM, Kevin Roberts, DPM, and Harold Schoenhaus, DPM

Introduction

Charcot osteoarthropathy is a secondary manifestation of various

medical conditions that share a combination of profound sensory, motor, and autonomic neuropathy. Diabetes mellitus is the leading disease causing this destructive process in the current medical community. There is no cure for this pro-

cess after it has been activated; only standard of care and conservative or surgical treatment prevents further breakdown and minimizes risk for ulceration and limb loss.

Current trends for advanced

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treatment of Charcot osteoarthropathy include stabilization with external fixation, use of intramedullary nails for an attempt at limb salvage, and medical advancements linked to increasing peripheral bone mineral density. The presentation and treatment strategies vary according to each individual patient. All share a treatment goal of a plantigrade foot able to weight-bear with weight evenly distributed along the plantar aspect of the foot, minimizing long-term complications.

History and Incidence

A French neurologist, Jean Martin Charcot, gave the first description of neuropathic osteoarthropathy in the mid-19th century.^{1,2} During the time period of the initial description, the disease process was linked directly to syphilitic posterior column degeneration of the spinal cord. Over the last century, Charcot osteoarthropathy has been found to be a secondary manifestation of a multitude of medical conditions, some of which are listed in Table 1.^{3,4,5} The presentation of Charcot osteoarthropathy cannot be distinguished based on the disease associated with its development.³

Impairment, or a loss of the ability to input sensory data from joints and muscles, and the loss of autonomic control of vasculature links all of these medical conditions to neuropathic osteoarthropathy. In the current medical community of the western world, diabetes mellitus (DM) has emerged as the leading cause of lower extremity Charcot osteoarthropathy.

Medical advancements since the time of Charcot himself have allowed for an increase in life expectancy of people suffering from DM. Therefore, as the duration of time that neuropathy associated with DM increases, the incidence of related Charcot osteoarthropathy

also increases. In the United States alone, approximately 4% of the population is diagnosed with DM.⁶ Within the DM population, the incidence of Charcot has been reported to be as low as 0.1% and as high as 29%.^{2,7,8}

Pathophysiology

An exact account of the pathophysiology leading to Charcot osteoarthropathy has yet to be developed and described within medical literature. Since the initial description of this disease process, two potential and well known theories have become known to the medical community. The "French Theory," described by Jean Charcot, relates that the arthritic changes are caused by disturbances in nutrition to the joints and bone surfaces due to damage of the central nervous systems.¹ An expansion of this theory links autonomic neuropathy to loss of sympathetic tone and constrictive control of blood vessels leading to arterial vasodilatation, and ultimately an increase in blood flow.

Volkman and Virchow described what is known as the "German Theory," relating the breakdown of the joint to repetitive trauma in an insensate foot.¹ A significant increase in forefoot pres-

TABLE 1
Medical Conditions Associated with the Development of Charcot Osteoarthropathy

Diabetes mellitus	Paraplegia
Tabes dorsalis	Peripheral nerve lesions
Syringomyelia	Leprosy
Poliomyelitis	Congenital pain insensitivity
Multiple sclerosis	Pernicious anemia
Myelodysplasia	Spinal cord lesions
Alcoholism	Lyme disease
Rheumatoid Arthritis	

sure in new onset midfoot Charcot osteoarthropathy, when compared to pressures in neuropathic patients without arthropathy, lends a suggestion that this increased repetitive trauma to the forefoot can cause this common form of Charcot osteoarthropathy.² While these two main theories for development are described distinctly, a combination of the basis of the theories, along with other risk factors associated with Charcot osteoarthropathy, may more fully encompass the pathophysiologic process.

Risk Factors

The most notable and important risk factors associated with the development are diabetes mellitus and neuropathy, but other associated risk factors should not be ignored and include presence of deformity with increased plantar pressures, nephropathy, chronic inflammation, and metabolic abnormalities.

Neuropathy, by itself, encompasses three main areas, all relating to Charcot osteoarthropathy. The type of sensory neuropathy associated with DM is both distal and symmetric in a stocking-glove pattern.⁹ Sensory neuropathy allows for the breakdown of bone and joints without the recognition of the patient. This can lead to a very disastrous cycle where there is no protective response to the dislocations and fractures and therefore full weight-bearing causes a micro-trauma that remains ongoing.

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Sensory neuropathy is present to some degree in all patients afflicted with Charcot osteoarthropathy.

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Motor Neuropathy

Motor neuropathy causes wasting of mainly the intrinsic muscles within the foot. In later stages of this form of neuropathy, extrinsic muscle groups may also become involved. This lack of muscle tone can lead to skeletal deformities. With the presence of deformity within the foot, pressure will be increased to the abnormal areas of prominence, which can lead to further mechanical breakdown.

Autonomic neuropathy instigates a loss of sympathetic tone of arterial vessels leading to a vasodilatation and an ultimate increase in blood flow. This increase in blood flow causes an arterio-venous shunting leading to rapid-forward flowing blood in neuropathic patients.^{3,10}

Osteolysis and bone resorption occur in an environment with adequate blood flow, like that with Charcot osteoarthropathy.³ This increase in blood flow has been documented in Charcot osteoarthropathy patients.

A series of three cases of patients with severe arteriosclerosis obliterans has been documented in which the patients underwent vascularization and subsequently developed Charcot osteoarthropathy after the return of blood flow.³ Doppler ultrasonograms have also demonstrated an increase in velocity of forward flowing blood in the arteries of neuropathic limbs.^{11,12}

Increased blood flow was also documented in neuropathic limbs, with a resultant increased uptake in the first and second phases of Technetium Tc99m medronate bone scans.¹⁰ Venous occlusion plethysmog-



Figure 1: Rocker-bottom Charcot deformity (chronic) with subluxed plantar cuboid, causing an ulceration to develop with possible underlying osteomyelitis.

raphy has also shown higher resting peripheral blood flow rates in insensate neuropathic feet.¹³

Pathologic Fractures

Bone may be predisposed to pathologic fractures and dislocations due to results of a chronic inflammatory cycle.¹⁴ Inflammation of bone leads to recruitment of tissue macrophages and osteoclasts. The role of the osteoclasts in this scenario is to remove injured osteoblasts and chondrocytes, allowing for the replacement of bone. By doing so, there is a change in the micro-architec-

The most common site for breakdown is at the tarso-metatarsal joint, also known as Lisfranc's joint.

ture of the bone described as a demineralization. The increased susceptibility to fracture is the result of this weakening of bone substance.

Metabolic Abnormalities

Metabolic abnormalities associated with severe renal disease can further weaken bone, making diabetic nephropathy a risk factor for Charcot osteoarthropathy.¹⁵ This subset of the DM population can have secondary manifestations of uremia, such as hyperparathyroidism, osteomalacia, and renal osteodystrophy, all of which weaken bone and render the patient more susceptible to Charcot osteoarthropathy.

The cause of Charcot osteoarthropathy should not be considered the result of one theory, either vascular or sensory-based. In actuality, the exact mechanism may vary from one individual to another, but share the same basic principles of neuropathy, increased pressures, metabolic abnormalities,

and inflammation. This combination of increased blood flow, loss of protective sensation, gross and micro architectural bone deformity more likely helps composite the larger factors associated with development.

Diagnosis

A timely diagnosis of Charcot osteoarthropathy is essential in order to halt the destructive process within the lower extremity. As with treatment of this disease, presentation may vary from individual to individual. There are general concepts, however, that should make the diagnosis apparent to physicians who specialize in the foot and ankle.

Classically, the presentation of an acute Charcot process has been described as a foot and/or ankle with gross deformity, edema, erythema, increased temperature, bounding pedal pulses, possibly complicated with the presence of ulceration (Figure 1). These basic principles should always cue a physician to a differential diagnosis of Charcot osteoarthropathy.

Sensory Neuropathy

Sensory neuropathy is present to some degree in all patients afflicted with Charcot osteoarthropathy; however, the presence of pain should in no way defer from this diagnosis. A loss of protective sensation allows for the cyclic nature of the disease in which a patient may continue weight-bearing during the acute stage even with recognition of discomfort. There is usually not a lot of pain associated with this condition.

To assist in the diagnosis of sensory neuropathy, the Semmes-Weinstein 5.07 monofilament is accurate and very applicable in any patient with DM.^{16,27}

Loss of protective sensation can be diagnosed if the patient is unable to perceive the monofilament on the skin of the foot and/or ankle. Different dermatomes are present within the lower extremity; therefore care should be taken to adequately assess all sensory nerves entering the foot.

It is essential that a physician use this test to determine a baseline

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sensory diagram of all diabetic patients, since the risk of developing Charcot osteoarthropathy and/or ulceration increases with the loss of protective sensation.

A benchmark study performed to look at the diagnosis and treatment of Charcot osteoarthropathy by the American Association of Orthopedic Surgeons showed that a large percentage of foot and ankle specialists were not utilizing this simple screening test in their practices.⁶

Measurement of Plantar Pressures

The measurement of plantar pressures may also serve as a guideline for the risk of development of

Charcot osteoarthropathy in those patients with diabetes mellitus. A study performed on 146 patients found a significant increase in the plantar forefoot pressures of those patients with mid-foot Charcot osteoarthropathy and those with neuropathic ulcers when compared to those with no neuropathy or those with neuropathy but no process.²

The presence of bounding pedal pulses may or may not be evident in a patient with Charcot os-

teoarthropathy. While it is well-recognized that adequate and often increased amount of blood within the involved area is necessary for the development of Charcot osteoarthropathy, bounding pedal pulses should not be used as a definitive guide.

Absence of Pulses

Pulses may be absent in these patients due to several factors. The gross deformities associated with this process may alter the normal anatomic

A temperature gradient of greater than two degrees Celsius between the affected and unaffected side is indicative of an active Charcot process.

TABLE 2
Eichenholtz Radiographic Classification for Charcot Osteoarthropathy

Stage 1: hypervascular (acute)	joint laxity, subluxation, osteochondral fragmentation, debris formation,
Stage 2: coalescent (subacute)	absorption of the debris, fusion of the larger fragments of bone, sclerotic bone margins
Stage 3: reparative (chronic)	loss of sclerosis, further fusion of bone segments

TABLE 3
Sanders and Frykberg Anatomic Classification for Charcot Osteoarthropathy

Zone 1	distal and proximal interphalangeal joints metatarsophalangeal joints
Zone 2	tarsometatarsal joints (Lisfrancs)
Zone 3	naviculo-cunieform joints talo-navicular joint calcaneocuboid joint
Zone 4	ankle joint subtalar joint
Zone 5	calcaneus

route for the posterior tibial artery and the dorsalis pedis artery. This may make it difficult for the physician to palpate the vessel. Also, the presence of severe edema may disguise the presence of the pulse due to the large amount of fluid within the soft tissues. For this reason, if one is unable to palpate pulses, other methods of vascular assessment may be used in the office or hospital setting.

Hand-held Dopplers

Hand-held Dopplers are indispensable in an office and hospital setting. This allows for assessment of blood flow to the ankle and foot, even when pulses cannot be palpated. The clinician should hear a biphasic sound wave when placed over the artery being evaluated.

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Other non-invasive studies, such as pulse volume recordings and arterio-brachial indexes, allow for a vascular evaluation. Consultation of a vascular surgeon may be necessary if there is any suggestion of vascular compromise.

Other Signs

Erythema and calor may compromise the ability to definitively diagnose a process of Charcot osteoarthropathy from that of cellulitis occurring secondary to an infection. Both can occur simultaneously and infection must be ruled out if an ulceration or suspected abscess is present. Inflammation alone, however, can cause the erythema and calor when the Charcot process is occurring without infection. A temperature gradient of greater than two degrees Celsius between the affected and unaffected side is indicative of an active Charcot process.¹⁷ This has been used by physicians to gauge if the process has moved into an inactive state.

Classifications and Staging

Eichenholtz, in 1966, developed a classification for Charcot osteoarthropathy based on plain radiographs (Table 2).¹⁸ The importance of this classification lies in the ability of a physician to evaluate radiographs and, based on the findings, determine the stage of the disease process. Being able to distinguish both clinically and radiographically the stage of the process will help determine treatment options.

Sanders and Frykberg's Classification

Sanders and Frykberg's classification is based on the predominant joint location of the Charcot process (Table 3).¹⁹ The most common site for breakdown is at the tarsometatarsal joint, also known as Lisfranc's joint. Subtle subluxation/dislocation often can be seen at the earliest initiation of the process at the second metatarsal base and medial cuneiform articulation.

This breakdown at the midfoot has been associated with the presence of equinus. The gastroc-soleal

complex can pull the hindfoot into plantar-flexion, not only limiting available dorsiflexion, but also increasing the amount of pressure occurring at the forefoot (Figure 2).²

At the initial diagnosis of Charcot osteoarthropathy, baseline weight-bearing radiographs of both lower extremities are necessary.

This leads to the subluxations and fractures at the midfoot and a "rocker-bottom" foot type.

Plantar Ulcerations

Plantar ulcerations are common with this midfoot breakdown secondary to the bony prominences that develop in this region. Plantar-

lateral ulcers may indicate a plantarly subluxed cuboid.

Advanced Diagnostic Techniques

Plain radiographs are used to determine the stage of the Charcot process, based on the classification of Eichenholtz, discussed previously. Radiographically, Charcot osteoarthropathy can be identified in two forms, hypertrophic and atrophic. Resorption of bone ends occurs in the atrophic form, mainly found in the upper extremity.

The lower extremity mainly accounts for the hypertrophic form, with which foot and ankle specialists are more familiar, with evident joint space narrowing, bone fragmentation, and osseous deformity (Figure 3).^{3,10} This form has both the destructive and reparative characteristics which can be further specified with Eichenholtz staging.

Charcot Osteoarthropathy Vs. Osteomyelitis

A controversy that surrounds the diagnosis of Charcot osteoarthropathy is the inability to distinguish this disease process clinically and with imaging modalities from osteomyelitis. An exact determination between these two conditions can be made only through biopsy of the bone and synovial tissue involved. A pathologic specimen of bone with OM would reveal suppurative inflammation with presence of bacteria or other microorganisms within the bone, whereas Charcot osteoarthropathy would reveal bone fragments within synovial tissue.

In the event that a deep ulceration is present with a Charcot process, it would be beneficial

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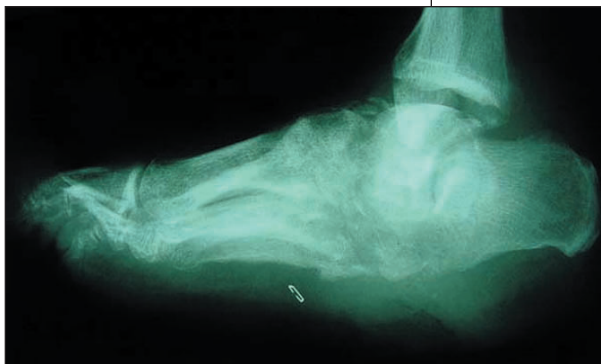


Figure 2: Chronic Charcot osteoarthropathy of the midfoot with rocker-bottom appearance. Note the equinus deformity of the calcaneus due to the gastroc-soleal complex pulling the calcaneus into plantar-flexion.

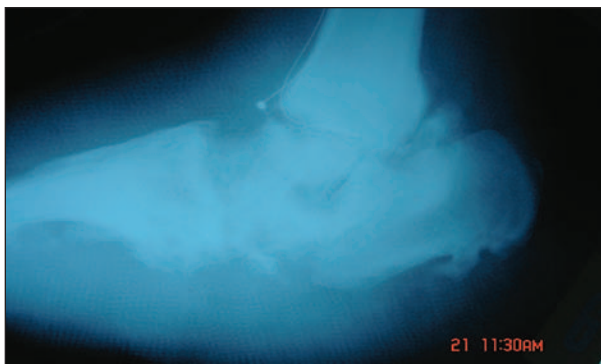


Figure 3: Acute Charcot osteoarthropathy with evident bone erosion and fragmentation. Severe midfoot and rearfoot deformity is present along with soft tissue edema noted.

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for the physician to rule out any presence of bone infection by biopsy.

Plain radiographs, technetium-99m-methylene diphosphonate bone scans, indium-111-leukocyte bone scans, and magnetic resonance imaging cannot distinguish between these two processes with reliable accuracy. Radiographs of both Charcot osteoarthropathy and OM show similar qualities of bone erosion, fragmentation, lucency, and new bone formation.²⁰ Technetium-99 bone scans do not allow for a diagnosis with satisfactory specificity.¹⁰ An increase in uptake of all three phases is present with both OM and Charcot osteoarthropathy (Figure 4).

A study evaluating the efficacy of indium-111-leukocyte bone scans combined with technetium-99 bone scans for the diagnosis of suspected OM with underlying Charcot determined that too high of a number of false positive indium-111-leukocyte bone scans made Charcot osteoarthropathy indistinguishable from OM by this imaging alone.¹⁰

The inflammatory nature of Charcot osteoarthropathy can lead to a large number of leukocytes at the involved area, which corresponds to the uptake seen in the indium-111-leukocyte bone scans in patients with only a Charcot process and no OM.

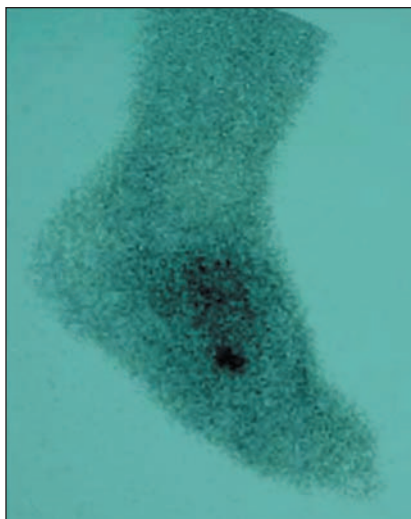


Figure 4: Representation of an Indium-111 bone scan of a patient with acute Charcot osteoarthropathy and osteomyelitis (determined by bone biopsy).

MRI

Magnetic resonance imaging can be utilized for the diagnosis of both Charcot osteoarthropathy and OM in the foot and ankle. With both processes, however, change in signal would be seen within bone marrow, making this modality unable to distinguish between Charcot osteoarthropathy and OM (Figure 5).¹⁰ The above-mentioned imaging modalities can be beneficial for the diagnosis of Charcot osteoarthropathy and OM when they occur separately. When there is a need to distinguish between bone infection and bone breakdown, only a biopsy can make a sufficient determination.

Standard of Care

The Diabetes Mellitus Committee of the American Orthopedic Foot and Ankle Society set out to determine current treatment of Charcot osteoarthropathy among specialists after recognition that this process is one of the most controversial issues facing foot and ankle physicians.⁶ This benchmark study determined that current practice patterns are very inconsistent.²⁵

There is a base of knowledge and treatment that must be obtained and followed for appropriate standard of care to be given during and after the Charcot process. First, it is essential for a multi-specialty approach for the care of a diabetic patient with Charcot osteoarthropathy. Attention to the medical, orthopedic, surgical, and prosthetic need of a patient must be met by a team approach of doctors familiar with this disorder.

Standard of care begins with assessing all diabetic patients for the presence of neuropathy and other risk factors associated with Charcot osteoarthropathy, as discussed previously. At the initial diagnosis of Charcot osteoarthropathy, baseline weight-bearing radiographs of both lower extremities are necessary. This is even more essential in those patients with juvenile onset diabetes since this subset of patients

have been reported to have a higher incidence of bilateral occurrence Charcot osteoarthropathy.¹⁵

Contralateral Films

Contralateral films will be beneficial for several reasons. Not only can they be used to compare pathologic anatomy to that of an unaffected foot and ankle, but they also allow for baseline radiographs of

both sides so a physician can follow the effectiveness of treatment and watch for any breakdown that may occur on the unaffected lower extremity. Further films of the uninvolved extremity should

be taken throughout the treatment cycle so as to identify earlier any Charcot changes in that extremity.

Since the initial description of this disorder, immobilization has been deemed a valuable treatment for those patients who are in the acute stages. Even though this is noted to be part of a standard approach, the technique for immobilization can vary among specialists. It becomes important to realize the technique which would best serve your patient and limit future risk of bone breakdown and ulcer formation.

Casting

Casting is an option for those patients who currently do not have ulcerations. If ulceration is present, daily wound care is essential. Windowing of a cast would be the only efficacious method for casting when wound care is also needed. Total contact casting has become the gold standard for immobilization by cast for diabetic patients. The study performed by the American Orthopedic Foot and Ankle Society, however, found that less than a quarter of specialists were using total contact casts as part of their treatment.⁶

While total contact casting is an effective way of distributing weight evenly during the weight-bearing process to minimize risk of soft tissue breakdown, the expense and

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*Small-wire
ring fixators can be
used for stabilization
and immobilization.*

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time associated with this technique could discourage some physicians from its use.

External Fixation

External fixation is an alternative for immobilization of the Charcot process, especially if there is a concern that the patient would be at great risk for ulcerating in a cast or if the patient currently has an ulcer. Small-wire ring fixators can be used for stabilization and immobilization. Manipulation prior and during the ring fixator application allows the physician to place the foot in a more plantigrade attitude for coalescence. A physician may also choose to walk the wires of the frame in order to provide compression to the involved area. While the frame is on the patient, daily wound care can be provided to ulcerations on the lower extremity.

Non-Weight-Bearing

In order to halt the cycle of continued breakdown of bone and joints by mechanical trauma, non-weight-bearing (NWB) is essential in providing standard of care in the acute phase. In an ideal world where the compliant patient is in good enough physical health along with having adequate cardiac function, NWB would not be an issue; however, many DM patients have impaired cardiac function or are obese and unable to be NWB.

In addition to this, it is difficult for a patient to comprehend the extent of damage that they are causing by continuing to walk on the affected foot and ankle since they are not in pain from the injury. This leads to a very high rate of non-compliance in this group of patients. So then, how does a physician provide standard of care if the patient is unable to be NWB by crutches or walker?

In this scenario, an option is a wheelchair for the duration of the

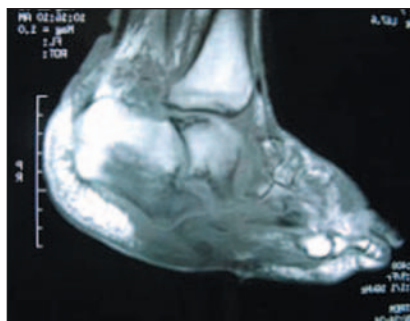


Figure 5: MRI (sagittal view) of a T2-weighted image of a patient with acute Charcot osteoarthropathy.

acute phase, which could take up to two to six months. If the patient is not amendable to this, then adequately document that you have explained the consequences of WB and continued destruction.

Conservative Treatment

Treatment for Charcot osteoarthropathy is on-going throughout the patient's life. The ultimate goal of treatment, once the chronic stage has been reached, is not to achieve normal anatomic structure, but to achieve and maintain a plantigrade foot able to weight-bear without significant risk for ulceration or reactivation. Conservative care is ideal if the foot and ankle can be maintained in a chronic state without risks through accommodation.²¹

As the patient enters the time of healing when weight-bearing is allowed, accommodative bracing with custom-molded orthotics, custom-molded added-depth shoes, and/or an ankle foot orthosis will be needed to distribute weight evenly along the plantar aspect of the foot and provide stability if needed to the subtalar and ankle joints.

Advanced Treatment

Conservative accommodation may not be a viable option due to the presence of inherent instability or marked deformity. If stability can be maintained, but osseous prominences pose a threat of future

soft tissue breakdown, simple exostectomies or plantar planings will be necessary. Ulcerations typically are located directly plantar to the apex of deformity.¹⁶ If an ulcer is present and superficial (does not probe to bone), the exostectomy incision can be made either medially or laterally dependent on location, so the bone is not approached through the ulceration. This will help minimize risk of infection by avoiding colonized soft tissue. Need for reconstructive surgery in Charcot patients should be limited to those who cannot be controlled

Bony prominences will occur if the weight falls medially or laterally, resulting in a valgus or varus malalignment, respectively.

with accommodative devices and simple surgical procedures.⁴

Reconstruction

The term reconstruction can carry with it the meaning of attempting to construct damaged anatomy back to what was the normal anatomic structure. This is not the case for reconstruction of Charcot deformities. For this disorder, reconstruction should be defined as providing stability through arthrodesis in a manner so as the structure of the foot/ankle is made plantigrade, but not by restructuring the foot to a normal anatomic state.²⁰ The components that defined normal anatomy do not exist after breakdown and therefore this is not an obtainable goal.

Reconstructive and post-operative needs vary with the location of Charcot osteoarthropathy. Charcot of the midfoot can occur at either the tarso-metatarsal joint complex or through the lesser tarsal joints. Either site often leads to a rocker-bottom midfoot where one or more

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Conservative accommodation may not be a viable option due to the presence of inherent instability or marked deformity.

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of the tarsal bones become prominent plantarly and an equinus deformity of the calcaneus occurs due to the pull of the gastrossoleal complex.

For this reason, an Achilles tendon lengthening is often necessary in order to achieve a plantigrade midfoot through arthrodesis. A subluxing plantar cuboid is often associated with soft tissue breakdown on the plantar-lateral aspect of the foot.

Correction by exostectomy alone may require the removal of a large amount of bone leading to instability and collapse of this area. For this reason, a triple arthrodesis in which the cuboid can be relocated dorsally is an acceptable option and maintains needed bone laterally.

Although the ankle is less frequently involved at 3-10% of all Charcot cases, involvement at this site can cause severe instability and has a higher risk of amputation.⁸ In order for a reconstruction of the ankle to be successful, the weight-bearing axis of the lower extremity must be centered over the ankle and subtalar joints.²⁰

Bony Prominences

Bony prominences will occur if the weight falls medially or laterally, resulting in a valgus or varus malalignment, respectfully. Once the ankle becomes unstable secondary to Charcot, there is a significant risk of tibia and fibula collapse causing destruction of the talus. If this does occur, the reconstructive option is a tibio-calcaneal fusion using either internal or external fixation.

A physician must obtain good apposition of the bone fusion site with adequate internal fixation of a blade plate, a combination of screws and various plates, or an intramedullary nail. Several studies have looked at the success of inter-

nal fixation for arthrodesis in patients with Charcot osteoarthropathy. An overall success rate with multiple internal fixation methods was 95%.¹² This success rate includes pseudoarthrosis, which can be considered a success if the fusion site is stable, thus maintaining alignment.

Intramedullary Nails

Intramedullary (IM) nails are being used in Charcot osteoarthropathy for ankle or tibiocalcaneal arthrodesis. The effectiveness and safety of using these devices in diabetic patients have been evaluated and compared to other forms of internal fixation.⁸ A 92.8% rate of successful limb salvage was found in 14 patients when used for an ankle arthrodesis.

The patients who had complications and needed hardware removed or the entire nail removed were those with a current ulceration at the time of application. Therefore, it is recommended that another form of fixation be utilized when there is any thought of infection of soft tissue or bone, or if there is a portal for a future infectious process.

Also, in these studies, all the patients involved had a recommendation of proximal amputation prior to the IM nail surgery. This implies that IM nails are considered mainly for cases of salvage, not for pure reconstruction.

Complications with use of IM nails include

osteomyelitis, malunion, nonunion, delayed union, pseudoarthrosis, and below-knee amputations when the IM nail fails.²²

External Fixation

External fixation is available in many different forms and can be utilized in patients with Charcot

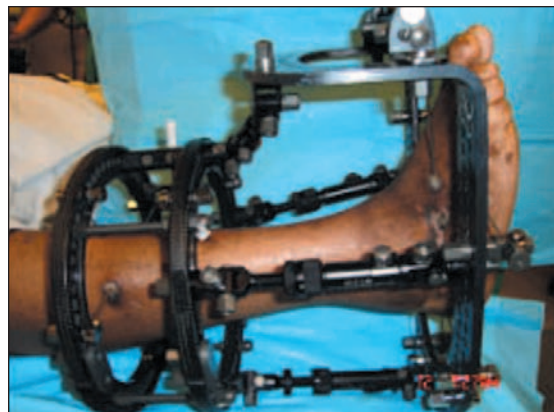


Figure 6: Small wire external ring fixator placed on the extremity of a patient with midfoot acute Charcot osteoarthropathy.

osteoarthropathy in any stage of the process.²³ If used during the acute stage for mainly immobilization, there can be manipulation to allow for some correction of deformity with the initial application (Figures 4 and 5).

This is a very logical approach to a staged set of procedures to achieve a plantigrade foot. Obviously, an external fixator is applicable to those patients who have ulcerations in which internal fixation would be contraindicated. A number of external fixation devices may be considered based on the location of the Charcot process and include small rail fixators, small wire external ring fixators, and unilateral fixators.

Small wire fixators offer the most immobilization because not only do they immobilize the forefoot to the rearfoot, but they also immobilize the smaller segments of the forefoot. They are also beneficial for ankle, subtalar, and/or midfoot arthrodesis and Achilles tendon lengthening. Small rail fixators may be used when only a portion of the foot needs immobilization such as the medial column at Lisfranc's joint. These are also less cumbersome for patients and do not carry with them the same degree of social impact as a full ring fixator.

Medical Treatment

A foot affected with Charcot osteoarthropathy demonstrates excessive osteoclastic activity without concomitant increase in osteoblastic activity. Recently, re-

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External fixation is available in many different forms and can be utilized in patients with Charcot osteoarthropathy in any stage of the process.

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search has focused on the use of supplemental bisphosphonates to determine if decreasing the osteoclastic activity of the involved bones will slow the progression of the deforming process.

The exact mechanism of bisphosphonates is not known, but what is apparent is that the bisphosphonate is absorbed by bone and attaches to the bone matrix. This prevents osteoclasts from attaching to the bone, inhibiting their activity. Bisphosphonates also directly affect the osteoclasts on the cellular level by apoptosis.

There is an indirect effect of bisphosphonates through effects on osteoblasts, which in turn, inhibit osteoclastic activity. Only a few limited studies are available that are not completely conclusive about the effect of bisphosphonates.

In a retrospective study looking at the effects of one infusion of pamidronate in 13 patients, a significant difference was found in decrease of limb temperature when compared to control subjects.¹⁴

There was also a significant decrease at two weeks after infusion of the alkaline phosphatase levels. A decrease in alkaline phosphatase was also seen in six patients given six infusions of pamidronate, but was not seen until after the third infusion.²³

The temperature gradient in these patients also decreased from an average initial gradient of 3.4 degrees Celsius to 1 degree Celsius after only one infusion. A larger study involving 39 patients showed that in the 20 patients receiving pamidronate, there was a 32% decrease in alkaline phosphatase and in control patients, there was no change in the levels of alkaline phosphatase.²⁴

Decreased Bone Density

Charcot osteoarthropathy has been linked to a decreased peripheral

bone mineral density in patients suffering from fractures and dislocations. Based on the World Health Organization (WHO) criteria for osteoporosis, Charcot patients with fracture patterns had either t-scores below -2.5 signaling osteoporosis or below -1 defining osteopenia.

Forteo

Forteo, a synthetic terapatide, offers a different mechanism from bisphosphonates in which osteoblasts are stimulated, increasing both the peripheral and axial bone mineral densities and decreasing the risk of future fractures. Bone mineral density has been shown to be exponentially associated with fracture risk in trials of subjects with osteoporosis.

If by using Forteo, peripheral bone mineral density can be increased, decreasing the risk of future fractures/dislocations of the lower extremity and halting the progression of Charcot osteoarthropathy, a new limb-salvage modality would be available for this condition.²⁰ To date, no clinical research has been reported on the use of Forteo in Charcot patients. The link of Charcot osteoarthropathy to decreased peripheral bone mineral density has been well developed and cited.

Conclusion

Charcot osteoarthropathy is a limb-threatening destructive process that occurs in patients who suffer from sensory, motor, and autonomic neuropathy associated with medical diseases such as DM. Diagnosis begins at the physician

level with monitoring protective sensation of DM patients along with strong suspicion of an acute Charcot process when the patient presents with classical signs. Treatment must then be determined on an individual patient basis in which it must be determined whether or not a patient can be treated conservatively or will require surgical intervention when entering the chronic phase. A firm understanding of accommodation devices and goals of reconstruction must be achieved by both the physician and patient in order to obtain success. ■

Bone mineral density has been shown to be exponentially associated with fracture risk in trials of subjects with osteoporosis.

A foot affected with Charcot osteoarthropathy demonstrates excessive osteoclastic activity without concomitant increase in osteoblastic activity.

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Charcot...

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See answer sheet on page 213.

1) What is the possible cause of increased blood flow found in neuropathic patients:

- A) A motor neuropathy that leads to intrinsic muscle wasting
- B) A sensory neuropathy that leads to mechanical micro-trauma without recognition of the patient
- C) An autonomic neuropathy that leads to loss of sympathetic control of arterial vessels causing a vasodilation
- D) All of the above

2) Which Eichenholtz stage is described by sclerosis at joint margins, the beginning of fusion of large bone fragments, and absorption of fine debris:

- A) Hypertrophic (acute)
- B) Coalescent (subacute)
- C) Reconstructive (chronic)
- D) None of the above

3) According to Sanders' and Frykberg's classification of Charcot osteoarthropathy, which location is considered zone 2 and also the most occurrent:

- A) Calcaneus
- B) Ankle joint
- C) Tarso-metatarsal joint
- D) First metatarsal-phalangeal joint

4) What location of ulceration is predominant when there is a plantar subluxed cuboid:

- A) Plantar-medial
- B) Calcaneal
- C) Laterally at the fibular margin
- D) Plantar-lateral

5) Which form of Charcot osteoarthropathy is most commonly seen in the lower extremity and properly described below:

- A) Hypertrophic; bone fragmentation, joint narrowing, osseous deformity
- B) Hypertrophic; resorption of the bone ends
- C) Atrophic; bone fragmentation, joint narrowing, osseous deformity
- D) Atrophic; resorption of the bone ends

6) Which of the following diagnostic modalities is the most specific and sensitive when determining a diagnosis of Charcot osteoarthropathy versus osteomyelitis:

- A) MRI
- B) Pathologic biopsy
- C) Bone Scans
- D) Plain radiographs

7) In which phase of a Technetium-99 bone scan is there an increased uptake for acute Charcot osteoarthropathy:

- A) Phase 1 (blood flow)
- B) Phase 2 (blood pool)
- C) Phase 3 (delayed)
- D) All of the above

8) Which of the following medical conditions have been associated with the development of Charcot osteoarthropathy:

- A) Alcoholism
- B) Lyme Disease
- C) Syphilis
- D) All of the above

9) The type of neuropathy that is commonly associated with diabetes mellitus can be described as:

- A) Proximal and symmetric
- B) Distal and symmetric
- C) Proximal and asymmetric
- D) Distal and asymmetric

10) The temperature gradient that can be considered significant when comparing an affected foot with an unaffected foot is:

- A) greater than 2 degrees Celsius difference on the affected side
- B) greater than 1 degree Celsius difference on the affected side
- C) greater than 2 degrees Fahrenheit difference on the affected side
- D) greater than 1 degree Fahrenheit difference on the affected side

11) Which of the following can clinically rule out the differential diagnosis of Charcot osteoarthropathy:

- A) Loss of protective sensation

- B) Presence of discomfort at the suspected site
- C) Presence of a red, hot, swollen foot with an infected ulceration
- D) None of the above

12) Which of the following Eichenholtz stage would be described as joint subluxation, joint laxity, bone fragmentation, and bone debris within soft tissues:

- A) Hypertrophic (acute)
- B) Coalescent (subacute)
- C) Reconstructive (chronic)
- D) None of the above

13) Which of the following is an advantage of using a small wire external ring fixator for a patient with acute Charcot osteoarthropathy:

- A) An external ring fixator is cheaper than applying a total contact cast.
- B) An external ring fixator will allow access to ulcerations in order to continue wound care.
- C) An external fixator is less cumbersome for the patient and is more socially acceptable.
- D) All of the above.

14) What is the reasoning behind using bisphosphonates and other drugs used to treat osteoporosis, like Forteo, in patients with acute Charcot osteoarthropathy:

- A) Charcot osteoarthropathy has been associated with a peripheral low bone mineral density.
- B) These medications have been shown to affect the breakdown of bone by osteoclasts and/or the build-up of bone by osteoblasts.
- C) This would treat an underlying cause of Charcot osteoarthropathy and help minimize further break-down of bone.
- D) All of the above.

Continued on page 212

(cont'd)

15) When considering surgical reconstruction of a Charcot osteoarthropathy deformity, which stage is the most stable stage to perform the surgery:

- A) Acute
- B) Coalescent
- C) Chronic
- D) Both A and B

16) If a physician suspects underlying osteomyelitis with a Charcot deformity, which modality can be used to confirm the presence of osteomyelitis:

- A) Tec-99 bone scan
- B) Indium-111 bone scan
- C) MRI
- D) Bone culture and pathology

17) What would one find in the pathology report for a patient with Charcot osteoarthropathy regarding the tissue and bone specimens?

- A) Suppurative matter within the bone fragments
- B) Bacterial colonies
- C) Bone fragments within synovial tissue
- D) Any specimen sent would come back as normal bone and soft tissue

18) Which of the following are considered helpful when determining if a patient is at risk for development of Charcot osteoarthropathy:

- A) PET scans
- B) Forefoot pressure measurements
- C) Semmes-Weinstein assessment
- D) Both B and C

19) What is considered the goal of surgical reconstruction of a foot and/or ankle deformity caused by Charcot osteoarthropathy:

- A) Providing the minimal risk of future reactivation of the Charcot process and developing ulcerations
- B) Reconstructing the foot structure so that it most closely resembles the normal foot structure prior to surgery
- C) Providing a plantigrade foot that is able to weight bear and function with weight distributed along the plantar aspect of the foot
- D) Both A and C

20) When using an IM nail to arthrodesis the ankle joint, which of the following would be considered a reason to delay the surgery or consider a different form of fixation:

- A) Charcot osteoarthropathy of the ankle and subtalar joint
- B) Open ulceration present at the time of surgery
- C) Presence of suspected osteomyelitis within the tibia distally
- D) Both B and C

See answer sheet on page 213.

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Charcot Osteoarthropathy
(Rabjohn, Yarmel, Roberts,
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