CHAPTER **36**

EVALUATION AND TREATMENT OF ACUTE CHARCOT PROCESSES IN THE FOOT AND ANKLE

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INTRODUCTION

Nearly every physician dedicated to the diagnosis and treatment of disorders affecting the lower extremity has been witness to the dramatic effects of Charcot neuroarthropathy on the foot and ankle. This is especially true for those physicians who specialize in diabetic foot care and/or practice in tertiary care facilities, teaching hospitals or wound care centers. Yet, even for those physicians who encounter this disorder with frequency, its initial recognition can, at times, be quite challenging. Early diagnosis and prompt treatment of this disorder are absolute requisites if severe deformity and subsequent ulceration are to be prevented.

Approximately 15% of the diabetic population will develop a lower extremity ulcer during the course of the disease process and nearly 14-20% of these patients will ultimately require an amputation as a result of the ulceration.[1-3] Costs for ulcer care in the US have been reported to average nearly $4,600 per ulcer episode, while the direct and indirect costs of a single lower extremity amputation ranges from $20,000 to $40,000.[1,4] Following lower extremity amputation of one limb, there is a 50% incidence of amputation of the contralateral limb within 2-5 years.[1,2] Additionally, the 5 year mortality rate has been reported to be as high as 68% following a single lower extremity amputation. A quick review of these statistics reinforces the importance of prompt recognition and treatment of Charcot processes affecting the foot and ankle. The goal of this article is to familiarize the reader with the etiology of Charcot neuroarthropathy and provide information that will assist in the recognition of the condition in its earliest stage so that proper treatment might be instituted.

Table 1

COMMON CONDITIONS ASSOCIATED WITH THE DEVELOPMENT OF CHARCOT JOINTS

Diabetes mellitus	Leprosy
Syphilis	Alcoholism
Spina bifida	Syringomyelia
Poliomyelitis	Pernicious anemia
Charcot-Marie-Tooth disease	Cerebral vascular accident
Spinal cord injury	Peripheral nerve injury
CNS/PNS tumors	Lead poisoning

PATHOPHYSIOLOGY

The common denominator among all of the disorders in which the Charcot process develops is peripheral neuropathy (Table 1). When the condition was first described, most neuropathic joints occurred as a result of tabes dorsalis related to tertiary syphilis. However, during the middle part of the twentieth century, the number of cases of Charcot joints secondary to diabetes mellitus surpassed those secondary to tabes dorsalis and continues to be the number one etiology today. Although neuropathy plays a central role in the development of Charcot neuroarthropathy, it should be noted that there is at least one etiology of the Charcot process that does not involve peripheral neuropathy; neuropathic joints have also been known to occur in joints that have undergone multiple repeat injections with corticosteroids. This article will focus on the development of neuropathic arthropathy in patients with diabetes mellitus, as it is this patient population who most commonly present to the foot and ankle specialist for the evaluation and treatment of this disorder.

Unfortunately, when many physicians consider the presence of neuropathy in patients, they often consider it to be an entity in which there is a singular deficiency in sensory perception within the

affected nerves. As a result, they will focus their neurologic exam on the evaluation of sensory deficits such as light touch, vibration, proprioception, and temperature. More often than not, evaluation and assessment of the patient for motor and/or autonomic neuropathy is not performed. Evaluation of each of these types of nerve impairment is essential, as dysfunction in each of these nerve fiber types, in all likelihood, plays a significant role in the development of the Charcot foot and ankle.

Although sensory impairment is known to play a dominant role in the development of diabetic foot ulcerations, its role in the developing Charcot foot or ankle has not been fully established. The prevailing school of thought proposes that the insensate foot is more susceptible to Charcot changes because of a loss of deep pain and proprioceptive sensations in the affected joints. This may set the stage for ligamentous and capsular laxity and potential subluxation of joints as they are subjected to the normal stresses of ambulation. Ultimately, this may lead to altered articulations between bones upon weightbearing with resultant cartilage erosion, osteochondral fragmentation and intraarticular fracture or dislocations.

Motor neuropathy of the lower extremity also plays a role in the pathogenesis of the Charcot foot and ankle. During clinical exam, decreased or absent deep tendon reflexes will often be present. Typically, the motor nerves to the intrinsic muscles are affected first. The loss of intrinsic muscle function interferes with the normal biomechanical function of the affected foot and leads to classic digital deformities. The resultant retrograde force on the metatarsal heads causes them to be displaced plantarly, subjecting this area to increased pressure during gait and possible hyperkeratotic tissue formation with or without ulceration.

Atrophy of the anterior leg muscles secondary to motor neuropathy will also cause an alteration of the normal biomechanical forces within the foot. Weakened extensors provide the posterior leg muscles with a mechanical advantage and creates a functional equinus deformity at the ankle joint. The resultant limitation of dorsiflexion at the ankle joint during the propulsive period of gait is usually compensated by subtalar and midtarsal joint pronation. The result of this compensation is a pronated foot that has lost its ability to sustain the normal shock forces that occur during gait. In the Charcot prone patient, this compensation may lead to the development of progressive subluxation/dislocation of the midtarsal and tarsometatarsal joints, resulting in a classic rocker-bottom foot deformity.[5]

Detection of deficiencies in the sympathetic nervous system during routine physical examination can be challenging due to the absence of abundant clinical findings. In the feet, autonomic neuropathy is typically diagnosed by an absence of sweating combined with the presence of dry cracked skin. Distention of superficial veins may also be present. Questioning the patient for commonly associated manifestations of autonomic neuropathy can be beneficial in establishing this diagnosis (Table 2).

Table 2

COMMON MANIFESTATIONS OF AUTONOMIC NEUROPATHY

Impotence	Gastic hypomobility/ hypermobility
Diarrhea	Postural hypotension
Resting tachycardia	Fecal incontinence

The sympathetic nervous system is distributed throughout the foot via blood vessels and regulates blood flow by actively vasoconstricting or passively vasodilating arterioles. In the neuropathic foot with autonomic neuropathy, the influence of the sympathetic nervous is characteristically absent and blood vessels undergo unopposed dilation. Following a trauma in the neuropathic patient, the normal inflammatory response brings increased blood flow into the injured area in the days following the trauma. However, unlike the patient with a normal sympathetic nervous system, patients with impairment of the sympathetic nervous system may experience a prolonged inflammatory response. The resulting profound increase in blood flow to the area is known as neuropathic edema and has been associated with generalized osteopenia in the affected extremity. The weakened neuropathic bone is now further predisposed to further fracture or dislocation, especially in the presence of abnormal biomechanical forces.

Neurovascular Versus Neurotraumatic Theory of the Charcot Process

Although there is no debate that neuropathy plays a role in the development of the Charcot joint, the precise neural mechanism responsible for the development of this clinical entity has remained a source of considerable controversy, debate and speculation over the years. Historically, two main theories have been advanced to explain the underlying mechanisms responsible for the development of the neuropathic joint.

The first theory proposed to explain the development of the Charcot joint focused on damage to the "trophic" centers present in the anterior horn cells of the spinal cord.[6] Charcot, the originator of this theory, believed that these centers played an essential role in protecting joints by maintaining their nutrition.[7] Damage to these "trophic" centers as a consequence of trauma or systemic disease resulted in what Charcot termed ataxic neuropathy. Although the existence of Charcot's "trophic" centers have never been proven, the idea that the joint nutrition is directly related to blood flow has since lead many investigators to examine the effect of hypervascular states on the development of neuropathic joints. Today, this theory has become known as the neurovascular or French theory and is based upon the idea that bone and joint destruction occurs as a result of osteoclast activation that develops from a neurally initiated increase in blood flow in the affected part.[8]

The second theory was advanced and supported by Volkmann and Virchow,[8] who adamantly opposed Charcot's theory. They argued that the neuropathic joint develops as a consequence of the repeated trauma that occurs in the insensate joint. Accordingly, they believed that progressive fracture or dislocations in Charcot-prone patients develop as a result of the abnormal mechanical stresses that are sustained by neuropathic patients during ambulation. This theory has since become known as the neurotraumatic or German theory.

In an attempt to better define the pathogenesis of neuropathic joints, Eloesser, in 1917, performed a series of experiments in cats.[9] In one group of 38 cats, he cut the posterior roots of the spinal cord leading to the extremity being studied. In this group, 27 of the cats developed destructive changes in the bones and joints of the insensate extremity. In another study, he first induced a destructive arthritis in the hip joint and later severed the posterior roots. In this group of 6, only one neuropathic joint developed. In the final study group of three cats, he simultaneously cut the posterior roots and created a destructive lesion in the hip joint. All 3 cats in this group developed a neuropathic joint. From this experiment, Eloesser concluded that trauma is the causative factor in the development of the Charcot joint.

In 1967, Johnson repeated this experiment utilizing 18 cats and 6 dogs.[10] Some of the animals were kept in wide mesh cages after the procedure, which allowed the insensate limbs to become caught and entangled. In the process of trying to free themselves, many animals developed fractures and dislocations. Another group of animals were kept in small, narrow mesh cages after the procedure. Protected from the trauma of a possible entangled limb, none of these animals developed pathologic changes in their bones or joints. Finally, in a group of 6 animals, Johnson produced a trauma within the knee joint of an insensate limb. Five animals had an uneventful recovery while 1 continued to have an inflammatory response in the knee and ultimately developed a dislocation of the joint without fracture. From his experiments, Johnson, like Eloesser, concluded that trauma is the causative factor in the development of the Charcot joint. According to Johnson, the most beneficial finding of his studies was the realization that protection from trauma can prevent neuropathic joints from occurring in at risk individuals.

Although the scientific deductions made by Eloesser and Johnson are somewhat hard to follow, they were convinced that their studies supported the neurotraumatic theory by demonstrating that repetitive trauma in the insensate limb may lead to the development of the neuropathic joint changes. However, other investigators have questioned the ability of the neurotraumatic theory to explain the development of Charcot arthropathy in neuropathic patients who have suffered an acute fracture or dislocation. In 1980, Kristiansen reported 3 cases of acute fractures in diabetics that ultimately developed neuropathic joint changes.[11] Prior to this report, the incidence of Charcot changes secondary to acute fractures had received little to no attention in the literature. Since Kristiansen's report, numerous other authors have documented the development of Charcot changes in neuropathic patients who had sustained acute fractures.[12-14]

Although a few of the patients in these retrospective reviews received prompt evaluation and

treatment for their fractures, the vast majority of the patients did not present for several days to weeks following their fractures and were ambulatory prior to initial evaluation. Presumably, the combined delay in treatment and weightbearing prior to the institution of immobilization and non-weightbearing was enough repetitive trauma at the fracture sites for Charcot changes to develop. But the question still begs to be answered, how does the neurotraumatic theory explain the development of a Charcot process in the neuropathic patient who has sustained an acute fracture and received timely, appropriate treatment?

The rapidity in which many Charcot joints develop led Brower and Allman to argue that the neurotraumatic theory cannot fully explain the pathogenesis of the Charcot joint.[15] In their study of 91 patients with neuropathic joints, 6 of the patients developed destructive changes within 6 weeks. Furthermore, they found that 23% of the patients in their study developed Charcot joints as a result of spontaneous or stress fractures that developed without any history of trauma or increased activity. These findings led Brower and Allman to conclude that Charcot changes occurred in a majority of their patients only after a neurally initiated vascular reflex had led to increased blood flow and subsequent weakening of bone in the injured extremity.

The concept of a hypervascular diabetic limb may seem contradictory to the thinking of some physicians. After all, we are traditionally taught that the diabetic lower extremity is predisposed to severe "peripheral vascular disease" that occurs as a result of both microvascular and macrovascular disease. Yet, adequate blood flow is generally considered to be a prerequisite for the development of a Charcot joint.

The explanation for the increased blood flow in the neuropathic foot has been linked to increased arteriovenous shunting that occurs as a result of sympathetic denervation of the lower extremity vasculature.[7,8] Loss of sympathetic tone allows arteriovenous shunts to open and creates venous distension, lower extremity edema and an increase in skin temperature. Boulton et al measured the venous PO_2 in both patients with diabetes and control groups.[16] They found that the venous PO_2 in diabetic patients with neuropathy and active foot ulcers averaged 63.0 mmHg. In contrast, nondiabetic controls had an average venous PO^2 of 45.5 mmHg. They also examined diabetic patients with neuropathy but no history of foot ulcers and diabetic patients without neuropathy. The average venous PO_2 was 53.8 and 52.8 mmHg, respectively two groups. The results of this study clearly indicate that increased lower extremity perfusion is present in diabetic patients compared to nondiabetic patients and that lower extremity perfusion is dramatically increased neuropathic diabetic patients with active foot ulcerations.

Edmonds et al used non-invasive Doppler studies to compare blood flow in the feet of diabetic patients with neuropathy, without neuropathy, and control subjects.[17] They found markedly abnormal blood velocity profiles in diabetic patients with severe neuropathy as demonstrated by a dramatic increase in diastolic flow when compared to the diabetics without neuropathy and the control group. This finding led the researchers to conclude that diabetic neuropathy was associated with increased peripheral blood flow and arteriovenous shunting.

There seems to be overwhelming evidence that patients with Charcot neuroarthropathy have increased blood flow and pooling in the feet. Furthermore, the result of this pathologic derangement can be directly correlated to decreased bone density in the Charcot foot (and, to a lesser degree, diabetic patients in general). Edmonds et al used radionucleotide imaging with Tc-99 MDP to study the feet of acute Charcot patients and found increased bone blood flow and increased osteoclastic activity.[18] The authors hypothesized that these changes were the result of autonomic neuropathy. Gough et al provided direct evidence for an increase in osteoclastic activity in the acute Charcot foot when they measured markers for both osteoclastic and osteoblastic activity in patients with acute and chronic Charcot.[19]

Young et al used bone densitometry to compare patients with Charcot joints and matched neuropathic non-Charcot patients.[20] They found a statistically significant decrease in the bone density of patients with Charcot joints. The bone mineral density in the feet of neuropathic non-Charcot patients and Charcot patients averaged 1.27 g/cm2 and 1.09 g/cm2 respectively. Interestingly, the authors also found that peroneal nerve conduction velocities were directly correlated to bone mineral density. For instance, those patients with higher nerve conduction velocities had a higher bone density. Other authors have also confirmed the presence of decreased bone mineral density in Charcot patients.[21]

CLINICAL STAGING OF THE CHARCOT PROCESS

Clinical staging is an essential step in the evaluation of the Charcot process because the stage of the deformity will often dictate the course of treatment. In his classic treatise on Charcot joints, Eichenholz outlined three distinct stages of neuroarthropathy based upon radiographic findings.[22] Eicheholz believed that each stage could last from weeks to years before progressing to the next stage. He also noted that the Charcot process might halt after the first or second stage and remain unchanged. Alternatively, the completed process may repeat and go through all of the stages again.

Eichenholz's stage I, the stage of development, is characterized by capsular distention, fragmentation of the subchondral bone and attached articular cartilage and debris formation at the articular margins. Eichenholz's stage II is known as the stage of coalescence. During this stage there is absorption of fine debris as well as fusion of large fragments, which eventually adhere to the adjacent bones. Sclerosis of the end of bones may be present and occurs as a result of a loss of vascularization that occurs during the first stage. Stage III, the stage of reconstruction is characterized by a reformation of joint architecture. The bone ends and major fragments become rounded and there is a decrease in sclerosis as a result of revascularization.

One of the weaknesses of Eichenholtz's staging of the Charcot process is the lack of correlation between radiographic findings and the associated clinical findings. In reality, staging of the deformity based solely on radiographic findings is often difficult even for the most experienced practitioner. However, more accurate staging may be accomplished when the associated clinical findings are correlated with the radiographic findings.

In the initial stage of the deformity, the Charcot patient will exhibit increased skin temperature, edema, and redness. The pedal pulses are often bounding and an increase in joint mobility will also be noted on examination. As the patient progresses through Eichenholz's stages II and III there is a progressive decrease in skin temperature, edema, and redness. The pedal pulses will often return to normal with the cessation of the inflammatory cycle. A progressive decrease in joint mobility and increased stabilization occurs as reconstruction progresses. More often than not, the patient who has progressed through all three stages will have some type of significant residual deformity and/or joint instability.

Several authors have added a fourth stage (stage 0) to the Eichenholz classification in order to represent the high risk associated with the neuropathic patient who has sustained an acute fracture, dislocation or sprain.[23-25] Radiographic evaluation of the Stage 0 patient may show either simple or comminuted fracture, widening of joint spaces, dislocation, or normal anatomy. Clinical evaluation of the patient will demonstrate swelling, warmth, and possible joint instability resulting from ligamentous laxity.

Acute fracture/dislocations in neuropathic patients are treated according to established fracture management guidelines.[24] Open reduction and internal fixation should be employed as warranted in those patients who are medically stable to undergo the procedure. However, during the preoperative evaluation the surgeon must recognize the potential for the development of a Charcot process following acute fracture or dislocation in neuropathic patients. Postoperatively it is recommended that these patients be immobilized in a non-weightbearing short leg cast for a minimum of 8-10 weeks until bony healing occurs. Patients are frequently treated with up to 6-12 months of protected weightbearing and are monitored closely for the development of malunions or Charcot changes.[24] Additionally, we recommend that protective off-weight loading of the contralateral limb be performed with a MAFO or similar device.

The authors regard any acute injury in a neuropathic patient as having the potential to develop into a full-blown Charcot process. Furthermore, we believe that many in the medical community, especially primary care providers, fail to recognize many Stage 0 Charcot patients. This is especially true for those individuals who sustain injury without obvious radiographic findings of fracture or dislocation. As a result, this population is at risk for progressive deformity that could ultimately lead to ulceration, infection and possible lower extremity amputation. The authors would like to bring a heightened awareness to the Stage 0 Charcot patient and describe the criteria by which these patients are identified and treated at our facility.

THE STAGE 0 CHARCOT PATIENT

The most common initial presenting complaint is the sudden onset of swelling of the foot, ankle and at least the lower leg segment; it may be profound or moderate. Rarely is this edema mild. It may be accompanied by varying degrees of erythema and calor and is most commonly misdiagnosed as a deep vein thrombosis by physicians not experienced in the condition. When subsequent testing to rule out a thromboembolic phenomenon proves negative, a diagnosis of gout or cellulitis may be made and treatment for the same initiated.

Pain may or may not be present and deformity is not appreciated. Patients frequently complain of the inability to wear conventional shoes and may have developed some areas of superficial irritation from shoe pressure due to the edema. Not uncommonly a history of a minor injury such as a misstep or minor ankle sprain injury is recalled; in some cases the history of an injury is remote or nonexistent. Some patients are unable to recall an inciting event and indicate they simply awoke one morning and noticed significant swelling.

The physical examination confirms the presence of moderate to severe pitting edema mimicking severe venous insufficiency; unless the patient had a prior history of venous insufficiency the classic skin changes associated with the disease are absent. The temperature may be quite significant in contrast to the contralateral extremity; the authors have noted temperature variations as much as 10-12° F based upon objective measurements. Normal skin surface temperature varies between 85-87° F; objective measurements can be obtained using surface monitors such as those employed in anesthesia. Erythema is less commonly present but can at times mimic cellulitis. Pain is usually minimal and unimpressive even with manipulation of the major joints (i.e. ankle, subtalar, midtarsal, tarsometatarsal, etc.)

The pedal pulses may be difficult to palpate but are readily confirmed via simple Doppler testing or other noninvasive testing modalities. Capillary rebound is usually instantaneous or normal; no appreciable delay is present. Epicritic sensation testing is usually decreased due to the edema of the limb; a more accurate assessment can be made by examination of the contralateral extremity. Deformity of the foot is not appreciated but can be difficult to assess without pedal radiographs if the edema is severe, obscuring visualization of normal anatomic landmarks such as the fifth metatarsal base, navicular tuberosity or even the malleoli. Passive range of motion of the major joints may be normal or reveal a perceived laxity or increased mobility compared to the opposite foot. Accurate joint assessment can be difficult due to the edema obscuring normal anatomic landmarks.

In many cases, conventional weightbearing or non-weightbearing radiographs are not impressive; no fractures or dislocations are noted. Significant increase in the soft tissue volume and density are usually the most and only impressive finding. A few small flecks of bone adjacent to one or more joints may be appreciated but are commonly overlooked and discarded as being incidental. A subtle change in the overall alignment of the foot might be appreciated, especially when compared to previous films, if available.

Confirmation of a clinically suspected Charcot arthropathy begins with confident exclusion of other differential diagnosis including deep vein thrombosis, cellulitis or other infectious process and inflammatory arthritis such as gout. Laboratory studies such as a complete blood count with differential, sedimentation rate, and C-reactive protein might be helpful but are not a substitute for an accurate and complete history and physical examination.

The most reliable specialized test during this earliest stage of the disease is a conventional triphasic bone scan (Tc-99 MDP); the scan will reveal significant and impressive uptake in all three phases with a progressive concentration of the isotope within the affected bone(s) by 3-6 hours following the initial injection. In cases of suspected osteomyelitis, a labeled white blood cell scan (In-111 or Tc-99 HMPAO) will demonstrate positive uptake at 3-6 hours post injection but should not be considered as positive until a 24-hour follow-up reading is obtained. In the authors' experience, a positive In-111 or Tc-99 HMPAO scan should be considered a false positive for osteomyelitis unless there is uptake demonstrated at 24 hours.

Once the conventional bone scan provides convincing evidence of an osseous process in spite of the lack of obvious bone disease on conventional radiographs, appropriate treatment can be initiated with confidence. If further assessment of the bone process is desired, a CT scan or MRI can be obtained and will clearly delineate the extent of bone stress or

micro fracture. Subtle tarsal or tarsometatarsal subluxation might also be appreciated. An MRI will often demonstrate the stress phenomenon of the bone as well as marrow edema, which is consistent with the clinical presentation as well as the conventional bone scan. MRI is likely to prove as sensitive as a conventional bone scan in diagnosing the underlying osseous pathology, however, is subject to greater misinterpretation when reviewed by an inexperienced radiologist with minimal knowledge of the disease process. For this reason, along with the increased cost differential, the authors do not routinely employ any MRI to establish or confirm the initial diagnosis.

Primary treatment consists of patient and family education regarding the disease process and its sequela. Ensuring tight control of the patient's blood sugar cannot be overemphasized; patients should be encouraged to pursue such control through their primary care physician or endocrinologist. Other coexisting morbid conditions should also be given appropriate medical attention. The importance of family and spousal support should be emphasized.

The most important local treatment is the off-loading of all weight from the affected extremity. Prior to immobilization of the lower extremity in a cast or splint, serial Jones compression bandages are applied at weekly intervals until all edema has subsided. This process usually takes no more than 2-3 weeks unless patients are noncompliant. Diuretic agents may also be helpful. Patients are encouraged to maintain elevation of the limb and avoid periods of dependency to accelerate resolution of the edema.

Once the edema has resolved, a short-leg non-weightbearing synthetic cast is applied. Multiple layers of compression material and one or two elastic bandages are applied beneath the synthetic rolls that maintain the desired position of the foot; the position is usually one of a neutral attitude with the ankle at 90 degrees. We typically maintain off-loading for 12-16 weeks; cast changes are infrequent and rarely done more than once during this period. When the skin surface temperature has reached near normal compared to the opposite limb, gradual protective weightbearing is initiated. Removable cast braces, often with inflatable air bladders are used for this transition from non-weightbearing to full weightbearing for several weeks to months. Serial radiographs taken 5-8 weeks are taken to monitor changes in the architectural alignment and configuration of the foot.

Although definitive information is lacking regarding the efficacy of noninvasive electrical bone stimulation for any stage of Charcot arthropathy it is the authors' contention that such treatment can only be a useful and helpful adjunct to healing. We attempt to employ this therapy whenever possible, especially in patients we consider to be at high risk for further breakdown. The financial implications continue to be the single biggest barrier to its routine use.

Custom molded functional bracing is employed in some patients, especially if subtle changes in alignment are observed clinically and/or on pedal radiographs. The specific bracing technique and device are left to the expertise of a certified orthotist or prosthetist skilled and knowledgeable about this disease entity. In higher risk patients functional bracing of the contralateral extremity is prescribed at the onset of treatment to help minimize breakdown of the foot, which is subjected to increased stress during the prolonged course of treatment. Custom orthotic devices may be sufficient once the process has completely stabilized and ankle joint alignment has been maintained.

When difficulty is encountered and the edema, warmth, and overall inflammation of the foot persist, strong consideration is given to the administration of a bisphosphonate such as Aredia® (pamidronate, Novartis, East Hanover, NJ) to help arrest the process.[26-28] Although not approved by the FDA for the treatment of Charcot neuroarthropathy, the authors have found this drug to be a safe and efficacious means of decreasing the pain and edema associated with this process. In addition, the authors have noted this drug to aid in the overall consolidation process. We typically employ an intravenous infusion of 30 or 60 mg of Aredia given at two-week intervals; a maximum of six doses are administered.

If the Charcot process continues despite appropriate treatment, then the authors will typically employ early surgical intervention in an effort to prevent further breakdown of the foot and ankle complex. Although the specific details of surgical reconstruction of the acute Charcot foot and ankle are beyond the scope of this text, the authors would give consideration to either percutaneous stabilization and/or single, double or triple joint fusions combined with tendoAchilles lengthening in patients who have failed to respond to conservative therapy.

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