CME

EARN CATEGORY I CME CREDIT by reading this article and the article beginning on page 42 and successfully completing the posttest on page 47. Successful completion is defined as a cumulative score of at least 70% correct. This material has been reviewed and is approved for 1 hour of clinical Category I (Preapproved) CME credit by the AAPA. The term of approval is for 1 year from the publication date of July 2007.

LEARNING OBJECTIVES

- Describe the two major theories related to the etiology of Charcot's arthropathy
- Discuss the clinical presentation and imaging studies used in diagnosis
- Review the staging and anatomic classification systems used in the evaluation of this joint disease
- Outline conservative and surgical choices in the treatment of Charcot's arthropathy

Acute Charcot's arthropathy: A difficult diagnosis

Typically occurring in a neuropathic foot or ankle, this joint destruction must be treated properly and in a timely manner to prevent development of severe foot deformity.

Brianne Johnsen, MMSc, PA-C

In 2005, the CDC estimated that 20.8 million people in the United States, or 7% of the population, were living with diabetes.[1] Peripheral neuropathy, a major complication of diabetes found in nearly 30% of diabetic patients aged 40 years or older,[1] plays an integral role in the development of Charcot's arthropathy.

Dr. Jean-Martin Charcot first described a process of bone and joint destruction associated with syphilis-induced neuropathy in 1868 with detailed accounts of tabes dorsalis.[2] Although Charcot's arthropathy was first described as a sequella of tertiary syphilis, diabetes is now the most common cause of neuropathic arthropathy in the Western world. Charcot's arthropathy is a chronic, progressive disease of bone and joints, most commonly found in the feet and ankles of patients with diabetes and peripheral neuropathy.

All health care providers treating patients with diabetes should be aware of the potential for Charcot's arthropathy to develop. Early recognition and proper treatment can prevent progressive deformity, ulcer formation, infection, and even limb amputation.

PATHOPHYSIOLOGY

Although the exact etiology of Charcot's arthropathy is unknown, two major theories have been proposed. The *neurotraumatic theory* suggests that peripheral neuropathy leads to a lack of proprioception, which in turn causes ligament laxity. This results in increased joint range of motion, instability, and repetitive minor trauma. The inflammatory response to this microtrauma then causes increased bone resorption and weakening, leading to increased susceptibility to fractures and joint damage.

The *neurovascular theory* suggests that damage to trophic centers of the nervous system results in altered sympathetic control. This leads to increased blood flow to bones and greater bone resorption, resulting in osteopenia. The osteopenic bones are thus weakened and more susceptible to fracture and joint damage.

Despite the differences in these two theories, four factors are necessary for neuropathic arthropathy to develop: peripheral neuropathy, an unrecognized injury, continued repetitive stress on injured structures, and increased local blood flow.[3]

> "Early immobilization and joint off-loading are critical in the initial treatment of acute Charcot's arthropathy."

CLINICAL PRESENTATION

Charcot's arthropathy typically manifests in patients with long-standing diabetes and peripheral neuropathy. Patients are commonly in their fifth or sixth decade of life. The initial signs of Charcot's arthropathy are often subtle and can easily be overlooked. Unilateral foot swelling or difficulty with proper shoe fitting in a patient with diabetes should alert the health care provider to the possibility of Charcot's arthropathy.

Acute Charcot's arthropathy typically manifests as a painless, warm, erythematous, edematous foot with or without a history of trauma. In addition, the patient will usually have a bounding pedal pulse and neuropathy on examination.

These findings, however, often lead to a misdiagnosis of cellulitis or osteomyelitis.

ANATOMIC CLASSIFICATION

Multiple classification systems were developed to describe Charcot's arthropathy based on the location of the joint destruction (see Figure 1). Brodsky's system, which is based on the four most common regions affected, is perhaps the most widely used.[4]

Type 1 indicates the tarsometatarsal (Lisfranc's) region. Approximately 60% of cases of Charcot's foot occur in this region.[5] Residual deformity in this area manifests as a collapse of the longitudinal arch, resulting in a rocker-bottom foot (see Figure 2, page 24).

Type 2 indicates the hindfoot. This region is the second most common site for Charcot's arthropathy to develop.

Type 3a indicates the ankle joint. *Type 3b* indicates the posterior calcaneous.

Type 4 indicates that multiple regions are involved. Each region may be at a different stage of the Eichenholtz classification system.

Type 5 indicates the forefoot. This region is an uncommon site of Charcot's arthropathy.

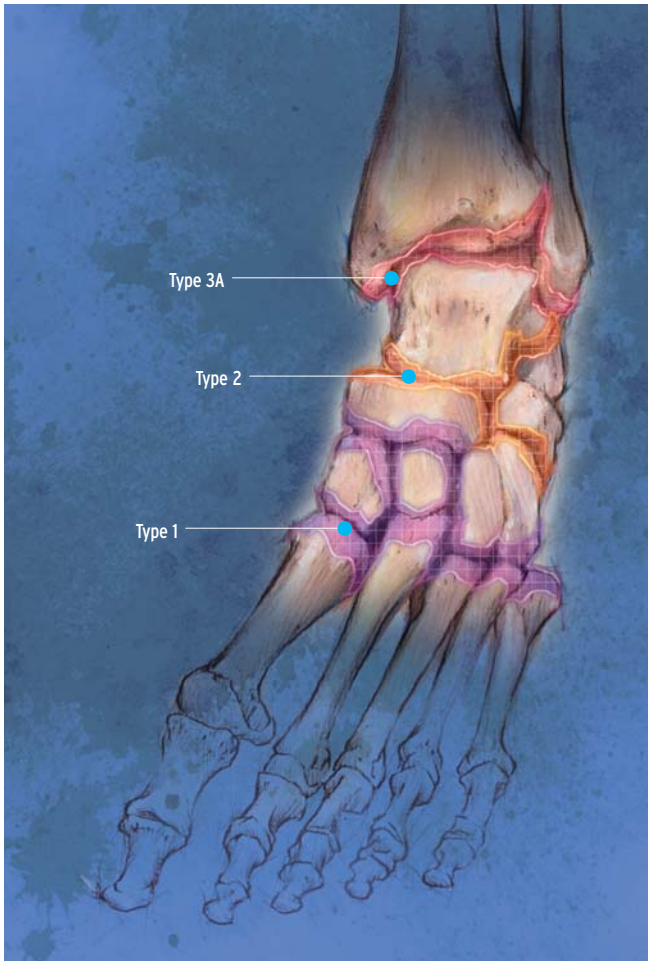
DIFFERENTIAL DIAGNOSIS

Differentiating between acute Charcot's arthropathy and osteomyelitis is challenging. The definitive diagnosis of Charcot's joint disease can be made with a synovial tissue biopsy, which will contain shards of bone and cartilage embedded deep into the synovium.[6] A bone biopsy, on the other hand, is used to make the definitive diagnosis of osteomyelitis. These tests are highly invasive, however, and involve considerable risk to the patient.

Less invasive tests are available to aid in the differentiation of the two diseases. Plain radiographs are usually ordered first because of their widespread availability and low cost. The radiographs should consist of weight-bearing anteroposterior, lateral, and oblique views (see Figure 3, page 25). Unfortunately, plain radiographs are neither sensitive nor specific for differentiating between changes due to Charcot's arthropathy and those due to an infection; instead, radiographs provide anatomic information. These images should be evaluated for disorganization related to the stage of arthropathy. Specifically, attention should be directed to the talar-first metatarsal angle, the lateral calcaneal-fifth metatarsal angle, the talonavicular coverage angle, and the alignment of the weight-bearing axis of the lower extremity.[7]

MRI provides exquisite anatomic detail of both soft tissue and bone. This imaging technique is useful in differentiating between osteomyelitis (which produces a high-intensity signal on T2 images) and Charcot's arthropathy (which produces a low-intensity signal on T2 images), but these findings have a low specificity. Higher intensity signals on T2-weighted images can also be seen with osseous tumors, trauma, and osteonecrosis.[6]

Continued on page 24



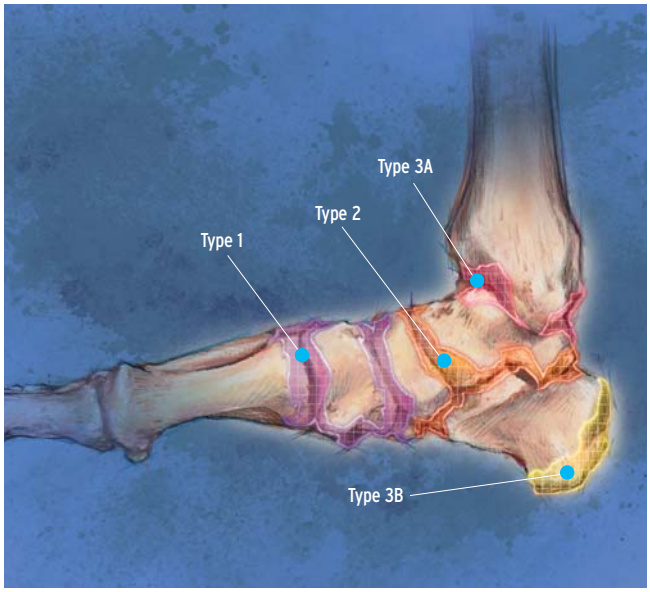


FIGURE 1. Classification system for Charcot's arthropathy based on the location of joint destruction

Scintigraphic studies may also be helpful in evaluating patients with complex presentations. Three-phase bone scans reveal osteoclast and osteoblast activity but are nonspecific for true osteomyelitis. Therefore, using indium-labeled WBC imaging (which has a higher specificity for osteomyelitis) in addition to three-phase bone scanning has been shown to be more accurate for a diagnosis of osteomyelitis, even in the setting of Charcot's arthropathy (which may also demonstrate increased metabolic activity on bone scan). This combination of imaging techniques increases the sensitivity and specificity to 80% to 90%.[8]

STAGING CHARCOT'S ARTHROPATHY

Eichenholtz was the first to develop a system for staging Charcot's arthropathy.[9] The system, originally consisting of three stages, is based on the changes seen on radiographs. The purpose of this classification system is to determine the patient's prognosis and to gauge the optimal timing for arthrodesis. Surgical intervention is most effective when performed during early stage 1 or late stage 3 disease.[9]

Stage 0 is the at-risk stage. This stage was added to the original Eichenholtz classification system to describe a patient with peripheral neuropathy who has sustained an acute sprain or fracture in the ankle or foot but does not have stage 1 disease. Unless the patient has sustained a fracture, the radiographs would be normal.

Stage 1 is the acute or developmental phase. The patient presents with an acute inflammatory process. Radiographs will show joint effusion, bone fragmentation, and joint subluxation.

Stage 2 is the subacute or coalescent phase, described as a decrease in warmth, redness, and swelling. Radiographs will show sclerotic bone surrounding the joint, resorption of intra-articular debris, and fusion of larger bony fragments.

Stage 3, or the reconstructive phase, is characterized by continued resolution of the inflammation. Radiographs will show remodeling of bone and some reformation of the joint architecture.

TREATMENT

Conservative therapy Early immobilization and joint offloading are critical in the initial treatment of acute Charcot's arthropathy. Nonoperative management strategies are historically the standard of care. The total contact cast (TCC) remains the gold standard for prolonged immobilization. This type of cast is made to conform exactly to the shape of the foot and ankle, with distribution of the pressure over a wide area. The principles of this approach are to control and decrease swelling, provide skeletal stability, and protect the soft tissues.

FIGURE 2. Severe rocker-bottom deformity with collapse of the right midfoot

The healing process in the foot and ankle of a patient with diabetes takes about twice as long as it does in a healthy person's limb; therefore, the immobilization period is lengthy. In general, treatment with nonweight-bearing immobilization is recommended for a minimum of 3 months, followed by a period of protected weight bearing.[10] During TCC treatment, frequent assessment of the skin, soft tissues, and bony structures is necessary.

Patient education regarding the diagnosis, length of treatment, and prognosis is essential. If the patient understands

KEY POINTS

- Charcot's arthropathy is a chronic and progressive disease of bone and joints most commonly found in the feet and ankles of patients with peripheral neuropathy due to diabetes.
- Four factors are necessary for neuropathic arthropathy to develop: peripheral neuropathy, an unrecognized injury, continued repetitive stress on injured structures, and increased local blood flow.
- Acute Charcot's arthropathy typically manifests as a painless, warm, erythematous, edematous foot with or without a history of trauma. In addition, the patient will usually have a bounding pedal pulse and presence of neuropathy on examination.
- The goal is to keep the patient immobilized and bearing no weight until the disease progresses to the chronic stage, which is signified by a decrease in pain and swelling and radiographic signs of resolution.

COMPETENCIES

- ●●●●● Medical knowledge
- ● Interpersonal & communication skills
- ●● Patient care
- ● Professionalism
- ● Practice-based learning and improvement
- ● Systems-based practice

the nature of this limb-threatening condition, he or she may be more motivated to adhere to the treatment plan. Emphasis on total joint off-loading, weight loss, and strict glucose control may improve the outcome of this disease.

After the plaster TCC is removed, a variety of specialized footwear options are available to continue the healing process and to prevent future deformity. The Charcot's restraint orthotic walker is designed to offload the foot and distribute plantar pressures more evenly. Patellar tendon braces have also been used to reduce plantar pressure. Custom-molded shoes and orthotics are important treatments as well, but these need to be checked regularly to ensure proper fit.

Surgical intervention Indications for surgery include chronic or recurrent ulcers associated with a bony prominence, unstable joints that are not amenable to bracing, acute displaced fractures in a patient with adequate circulation, and persistent pain. The goal of operative treatment is to restore a stable, plantigrade foot with acceptable biomechanics and to prevent a future amputation.

Arthrodesis, or joint fusion, is the most common surgical procedure used to treat a diabetes-related foot deformity. This is the procedure of choice for realigning the deformity and preventing amputation. Other surgical procedures include exostectomy of the bony prominence, osteotomy, partial tarsectomy, and Achilles tendon lengthening.

Surgical intervention in acute Charcot's arthropathy, however, is controversial. Some clinicians advocate surgery only when conservative measures have failed, whereas others propose early surgical intervention as a means for improved outcomes. Simon and colleagues demonstrated good results with early surgical intervention, measured by reulceration rates and a return to walking ability, in 14 patients with stage 1 Charcot's arthropathy who underwent midfoot arthrodesis.[3] Advocates of early surgical intervention believe TCC to be too cumbersome for treatment over an extended period of time. In addition, patients who are managed conservatively may still suffer a nonunion, which would eventually require surgery.

A 6-year study by Pinzur compared surgical interventions and nonsurgical therapy in 198 patients with Charcot's arthropathy. Surgical interventions for patients with nonplantigrade feet included osteotomy, with or without arthrodesis; debridement; simple exostectomy; and amputation. The desired endpoint in this study was long-term management with commercially available, therapeutic footwear and custom foot orthoses. This study concluded that more than half of the patients with midfoot Charcot's arthropathy could be successfully managed without surgery.[11]

Although surgical intervention for acute Charcot's arthropathy is becoming more common, the most effective treatment course should be determined through individual evaluation of each patient. Patients with diabetes often are morbidly obese and have multiple comorbidities that should be considered when contemplating surgical intervention. In addition, complications of surgery such as deep wound infection, nonunion of osteotomy or arthrodesis, malunion, frac-

FIGURE 3. Radiographs of a foot with rocker-bottom deformity (A, anteroposterior view; B, lateral view; C, oblique view)

ture, and hardware failure are possibilities that should be taken into account.

Bisphosphonates A new area of interest in the treatment of this disease is the use of bisphosphonates, which target the underlying physiologic mechanism of the disease. Bisphosphonates bind to hydroxyapatite located in the bony matrix and prevent osteoclastic resorption of bone.[12] Multiple reports have demonstrated decreased local temperature and pain, suggesting an anti-inflammatory action of pamidronate.[13,14] Although the exact mechanism of how these drugs halt bone loss and osseous destruction is not completely understood, study results using pamidronate as an adjunct to traditional treatments for Charcot's arthropathy appear to be promising.

CONCLUSION

The role of surgical intervention in the treatment of acute Charcot's arthropathy continues to be debated. Recognition of the subtle early signs of this disease process and prompt initiation of treatment are the keys to preventing permanent deformity. Conservative therapy is the standard of care. Immobility and prevention of weight bearing are used until the disease progresses to the chronic stage, which is signified by decreased pain and swelling along with radiographic signs of resolution. Too often, Charcot's arthropathy is misdiagnosed as an infection and treated improperly with devastating consequences. Perhaps the key to managing this costly disease is an increased awareness of its complications and associated long-term disability. JAAPA

Brianne Johnsen is a physician assistant fellow with Watauga Orthopaedics in Johnson City, Tennessee. She has indicated no relationships to disclose relating to the content of this article.

REFERENCES

1. Diabetes: Disabling disease to double by 2050. At a glance 2007. Centers for Disease Control and Prevention Web site. http://www.cdc.gov/nccdphp/publications/pdf/aag/ddt.htm. Accessed June 7, 2007.
2. Charcot JM. On arthropathies of cerebral or spinal origin. *Clin Orthop.* 1993;(296):4-7.
3. Simon SR, Tejwani SG, Wilson DL, et al. Arthrodesis as an early alternative to nonoperative management of Charcot arthropathy of the diabetic foot. *J Bone Joint Surg Am.* 2000;82-A(7):939-950.
4. Brodsky JW. The diabetic foot. In: Coughlin MJ, Mann RA, eds. *Surgery of the Foot and Ankle.* 7th ed. St. Louis, MO: Mosby; 1999:895-969.
5. Trepman E, Nihal A, Pinzur MS. Current topics review: Charcot neuroarthropathy of the foot and ankle. *Foot Ankle Int.* 2005;26(1):46-63.
6. Lee L, Blume P, Sumpio B. Charcot joint disease in diabetes mellitus. *Ann Vasc Surg.* 2003;17(5):571-580.
7. Schon LC, Weinfeld SB, Horton GA, Resch S. Radiographic and clinical classification of acquired midtarsus deformities. *Foot Ankle Int.* 1998;19(6):394-404.
8. Tomas MB, Patel M, Marwin SE, Palestro CJ. The diabetic foot. *Br J Radiol.* 2000;73(868):443-450.
9. Eichenholtz SN. *Charcot Joints.* Springfield, IL: Charles C. Thomas; 1966:3-8.
10. Sinacore DR. Acute Charcot's arthropathy in patients with diabetes mellitus: healing times by foot location. *J Diabetes Complications.* 1998;12(5):287-293.
11. Pinzur M. Surgical versus accommodative treatment for Charcot arthropathy of the midfoot. *Foot Ankle Int.* 2004;25(8):545-549.
12. Hughes DE, Mian M, Guilland-Cumming DF, Russell RG. The cellular mechanism of action of bisphosphonates. *Drug Exp Clin Res.* 1991;17(2):109-114.
13. Guis S, Pellissier JF, Arniaud D, et al. Healing of Charcot's joint by pamidronate infusion. *J Rheumatol.* 1999;26(80):1843-1845.
14. Stansberry KB, Moss QE, Peppard HR, et al. Intravenous bisphosphonate rapidly reverses the paradoxical increase in blood flow to the foot in Charcot neuroarthropathy. *Diabetes.* 1999;48(suppl 1):A92.