The Susceptible Insensate Foot

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Introduction

Patients with limbs which are both insensate and functionless often are best treated with amputation to improve hygiene, functional potential with prosthetics, and often cosmesis. There exists, however, a large population of patients whose lower extremities are insensate, but remain functional. Because of continued functional demands, and the loss of important protective mechanisms, breakdown of the delicate articulations occurs resulting in neuropathic arthropathy.

While there are a multiplicity of disease states associated with neuropathic arthropathy, there are certain general principles and characteristics inherent in the final common pathway of the Charcot joint. In years past, neurosyphillis was the major cause. Nowadays, diabetes mellitus is by far the most common cause.

This article will explore some of the historical aspects, causes, pathophysiology, clinical manifestations, and principles of treatment as they relate to neuropathic arthropathy of the susceptible insensate foot.

Historical Aspects

Jean Martin Charcot, at La Salpetriere in 1868, first called attention to "ataxic" forms of arthropathy associated with neurological diseases, the most commonly recognized cause being tabes dorsalis.[1,2,4] Charcot attributed the acute and destructive arthropathy to the loss of certain "neurotrophic influences" ncessary to support the normal joints.[6]

Charcot's contemporaries, Volkmann and Virchow, disagreed with this "trophic," or what was known as the "French" theory.[2] They argued that the arthropathy was due to continued mechanical stress and trauma on an insensitive biological structure.[2] These stresses continued in the absence of normal protective reflexes, which inevitably lead to a cycle of injury, inflammation, further injury, and finally instability and joint destruction. The end result, now the "Charcot joint."

This basic process was gradually recognized in an ever broadening horizon of disease entities. Myelitis and syringomyelia were recognized as causes in 1875 and 1892 respectively.[1] It was not until 1936 that Jordan described neuropathic arthropathy in the diabetic,[5] now the most common cause of Charcot joints.[4]

Etiologic Factors

The myriad of conditions which can produce Charcot joints is well outlined elsewhere.[2,6] The three most common causes are diabetes mellitus, tabes dorsalis, and syringomyelia.[4] The prevalence of neuropathic arthropathy in diabetes is only 0.1% to 0.5%, as compared to tabes dorsalis and syringomyelia which are 5% to 10% and 25%, respectively.[4] The almost epidemic numbers of diabetics makes them the largest group seen clinically, however.

Various theories have been espoused, such as Charcot's "neurotrophic" theory, Volkmann's "mechanistic" theory, and "neurovascular" theories.[4] Each stresses some aspect of the observations made in the neuropathic ar-

thropathy process. Certainly, "trophic" nerves have never been proven.[2] Mechanical trauma most certainly has a major role in the process, as is noted by many authors.[1,2,3,6,7,8]

The basic concept of the mechanical theory is the blunting or eliminating of pain and proprioceptive information received from the involved body part. This dampens the afferent input for both conscious and nociflexive response patterns which have evolved to protect the extremity from intolerable mechanical stresses, and thus avoid injury.[8] The loss of proprioceptive and fine sensory input leads to ataxic gait patterns which further increase mechanical stresses.

The spectrum of sensory deficit can be from an apparently normal sensory examination, to complete anesthesia.[4] Patients can experience pain, but it is invariably much less than expected for the degree of trauma and distortion of bone and soft tissues.[2,4,5,7] When pain does occur, it is usually secondary to severe post-traumatic inflammation of richly innervated synovial and pericapsular structures.[4,6] Joint proprioception, which normally inhibits hypermobility, is diminished, or absent, allowing instability to develop and progress.[8]

Attempts to explain the rapidity of the process and bony reabsorption, seen especially in the diabetic patient,[6,10] have been made with the "neurovascular" theory.[4] This theory states that an abnormal "neurovascular reflex"[4] increases blood flow, resulting in bony washout, and hyperemic distensible soft tissue supports, all of which predispose the joint to a destructive process with normal stresses. The high incidence of objective autonomic dysfunction in diabetics lends some support to this theory.[4]

As stated by Hurzwurm and Barja,[4] ". . . a more plausible explanation is that all of the above theories play a role . . . ," but to different degrees in each patient.

Simply, relatively minor fractures in an otherwise normal foot or ankle can lead to rapid Charcot arthropathy if neuropathy is present.[7]

One can think about the insensate foot like the insensate mouth after our friendly dentist mercifully relieves pain. If we insist on eating before the anesthetic wears off, despite his instructions, we can induce a "Charcot mouth." We will have pain for our indiscretion within several hours. The patient with neuropathy will continue to "chew away," oblivious of the damage he creates.

Clinical Features

The foot is the most commonly affected part of the appendicular skeleton.[4] However, it should be noted that different distributions of skeletal involvement can be seen, such as primarily upper extremity involvement with syringomyelia. The spine, knee, and hip may also be involved.[11] Why one joint in an insensate extremity is involved, while other joints remain normal, has remained unanswered.[1]

Patients commonly present with the chief complaint of swelling, deformity, or mal perforant ulcers.[4,5] Pain may or may not be present, but is usually dependent upon presence of acute inflammation.[4,5]

As described by Charcot and Volkmann,[2] the process of joint disruption begins with a period of swelling, erythema, local hyperemia, and effusion. This acute phase presentation is a manifestation of a normal acute inflammatory response to injury. If the injury is not perceived, the already edematous and hyperemic tissues receive continued trauma, recurrent inflammation, and poor, inadequate healing occurs. This eventually, if unchecked, leads to progressive soft tissue and bony deformity,[5,6] more characteristic of the chronic phase. An important distinction must be made between acute inflammation and infection, as both can present with the same local findings of swelling, erythema, and increased skin temperature. In the Charcot joint, however, laboratory studies, such as the white blood and differential counts and sedimentation rate, are normal; and importantly, there are no systemic manifestations such as fever or signs of sepsis.[5]

Usual deformities include increasing flat foot to complete arch collapse, ankle and hindfoot valgus (or varus), and forefoot external rotation and eversion.[5,6,8] Mal perforans ulcers are formed intradermally, under heavy callous, caused by abnormal weight bearing.[3,5] A 50% association of diabetic mal perforans with neuroarthropathy has been described,[5] usually occurring at the metatarsophylangeal joint level.

Patterns of joint involvement have been described in the diabetic. Primary ankle and subtalar joint patterns are frequent, with mid-tarsal

joints most frequently involved.[6] Tarsometatarsal and metatarsophylangeal involvement have each been described in up to 30% of cases[4] (Figures 1 and 2).

Radiological characteristics of neuropathic arthropathy progress from debris at the articular margins and periarticular calcifications, to diffuse bony fragmentation which can coalesce to larger fragments and large osteophytes.[1] Later changes include bony marginal sclerosis in attempts to reform articulations[1] (Figure 2).

Pathologic examination reveals bone and cartilage fragments in the synovial tissues, and fibroblastic reaction with some round cell infiltrates in ligamentous and capsular soft tissues.[4,6]

Circulatory status may be good in the Charcot foot,[4] but it is crucial to establish the diagnosis of vascular compromise on first evaluation as this can drastically affect treatment and outcome, especially in the diabetic.[5]

Neuropathic arthropathy can be the presenting problem with previously undiagnosed diabetics.[7]

Figure 1A. Initial evaluation of a 54 year old female diabetic. Normal AP, lateral, and oblique views of the left foot.

Complicating factors in the clinical course are spontaneous fractures, which can hasten the degenerative process; deformity, which can be quite rapid in syringomyelia, tabes dorsalis, and with varus deformities; and soft tissue injury, predominantly neurotrophic plantar ulcers.[6]

Treatment

Treatment follows from the recognition that the extremity is injured; and is likely to have continued trauma because of the neuropathy. Early recognition should allow curtailment of the progression, but because of the 'nature of the beast', there is often significant arthropathy at presentation.

Control of neuropathy, if this is possible, should be a primary consideration. This should be followed by attention to soft tissue injuries, or skin ulcerations which may require local debridement.[6] Evaluation of circulation is also part of the initial evaluation,[6,9] with necessary vascular intervention performed if this is a concomitant problem.

Cast immobilization to decrease edema, allow bony and soft tissue healing, and avoid or correct deformity, has been advocated by many authors.[1,4,6,7,9,10] Prolonged immobilization is essential to allow healing and stabilization.[4,6,9] Casting should continue until the local temperature has returned to that of the uninvolved or inactive side. It can then be assumed that the acute repair process has abated, and progression to supportive and protective orthoses is possible.[4,6,9]

Because of the potential for rapid progression, periodic x-rays must be obtained to assess progression which may alter therapy[5] (Compare Figures 1B and 1C).

The indications for orthopaedic surgical intervention include unacceptable deformity, making shoeing difficult; bony prominences, causing ulceration; concomitant infection, requiring debridement and drainage; and deformities with a high likelihood of progression (i.e. varus).[6] "Bumpectomies," decompressive fusions of digits, Keller bunionectomies, and subtalar or ankle debridements and fusions are some of the more commonly indicated procedures.[5,6] Total joint arthroplasty has no place in the neuropathic patient as it will inevitably

Figure 1B. At age 59 years, the lateral view is still normal.

Figure 1C. Only ten months later, lateral view of same foot shows advanced Charcot changes of the ankle, subtalar, and metatarsalphylangeal joints.
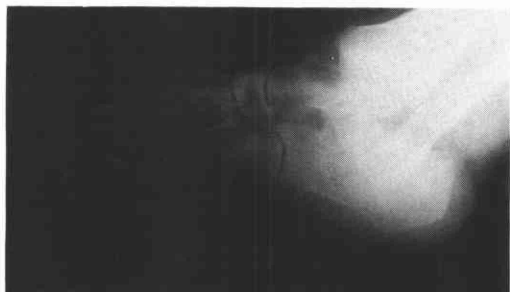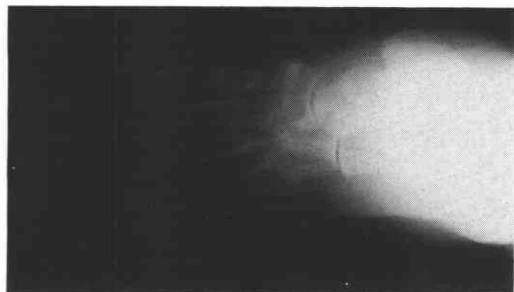

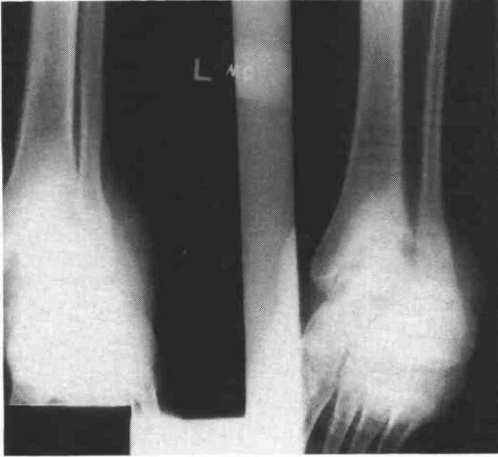**Figure 1D.** AP view.

Figure 1D. Oblique view.

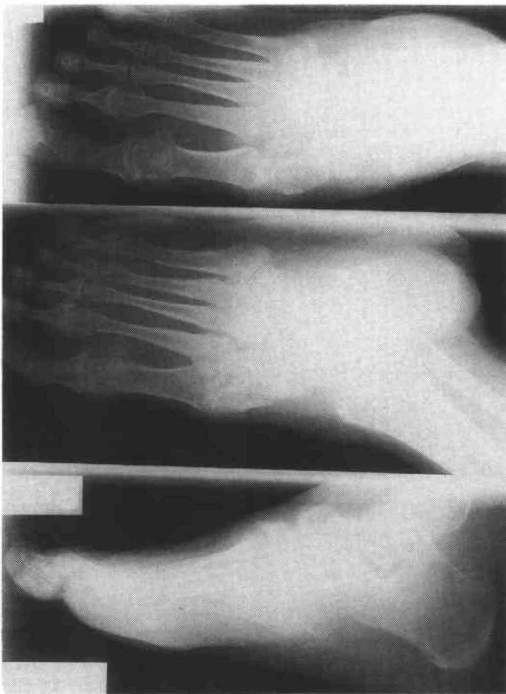**Figure 1E. AP and mortise views of the ankle at the same time as 1C and 1D.**

Figure 2. The right foot of same patient in Figure 1. Lateral, oblique, and AP views show midtarsal, tarsal-metatarsal, as well as interphylangeal Charcot joint changes—a different pattern of joint involvement in the same patient. Elements of bone fragmentation, joint subluxation and dislocation and bone formation are represented.

be disrupted by the same process that destroyed the natural joint.[6]

Conclusions

The major problem of the insensate foot is its susceptibility. Ataxia, secondary to neuropathy, imparts abnormal stresses and trauma to an extremity no longer able to detect injury. The neuropathy is usually irreversible, so defensive measures must be taken to control the process of joint destruction. Well fit ankle and foot orthoses to support unstable joints and redistribute weight bearing forces more evenly are the next line of defense once cast immobilization has controlled the injury reaction and allowed healing. Surgery is useful to correct unacceptable or unstable deformities and relieve skin pressures.

By understanding the patient's perceptions, and the pathophysiology of the Charcot foot, we can provide treatment to prolong the functional life and avoid the complications of the insensate foot.

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