Early Surgical Intervention for Charcot Neuroarthropathy While in the Active Stage

Here are some thoughts on the current treatment of this debilitating condition.

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Charcot neuroarthropathy (CN) is a destructive process which occurs in patients with peripheral neuropathy, most commonly due to poorly controlled diabetes mellitus. With the diabetic epidemic on the rise, Charcot foot and ankle complications such as ulceration, infection, and amputation have become increasingly more prevalent. It is currently thought that CN affects less than 1% of all diabetic patients, but up to 29% of diabetic patients with sensory peripheral neuropathy are affected by CN.[1] The increase in diabetes has led to an increase in CN research in the last several years, as the CN disease process and optimal treatment protocols are not fully understood.

CN can result in fracture, fragmentation, and dislocation to the bony architecture of the foot and ankle. This can be a devastating, limb-threatening problem. In fact, past surgeons often proposed major amputation as the primary treatment for CN with the belief that functionality could not be achieved. More recently, surgeons have found that functional limb salvage is oftentimes an obtainable goal in patients with CN. It is important to note that survival rates in the diabetic population following major amputation are staggeringly low; three-year and five-year survival rates are approximately 50% and 40%, respectively.[2] The most common cause of death following major amputation is cardiovascular disease. This statistic should encourage surgeons to attempt functional limb salvage if at all possible. Oftentimes, surgical reconstruction may be necessary to provide a stable, plantigrade foot that is wound-free.

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A Challenging Endeavor

Charcot reconstructive surgery is a challenging endeavor in the diabetic population. Factors such as uncontrolled blood glucose, peripheral vascular disease, history of wounds and/or infection, renal disease, inadequate nutrition, and obesity all can pose threats to successful reconstruction and must be optimized when possible. Non-compliance with post-operative non-weight-bearing is also a common challenge in the neuropathic patient population. These factors can all lead to wound healing and bone healing complications. A multidisciplinary approach must be taken to ensure adequate optimization of co-morbidities. Psychosocial factors also must be addressed when planning for CN reconstructive surgeries. Placement into subacute facilities is often necessary during the post-operative and rehabilitation stages.

Certain factors are non-debatable when discussing reconstructive surgery for CN. First and foremost, infection, if present, must be eradicated prior to definitive fixation. This should be accomplished by surgical debridements in combination with parenteral antibiotic therapy when necessary. Resection of all osteomyelitis must be achieved. Most often, this is accomplished through staged debridements until healthy bleeding bone is achieved and bone biopsy confirms that infection has been adequately cleared. Another non-debatable factor is arterial supply. All diabetic patients undergoing major reconstructive surgery should have adequate vascular assessment. If warranted, vascular surgery should be performed prior to any reconstructive procedures.

One factor that is commonly debated among surgeons is the timing of

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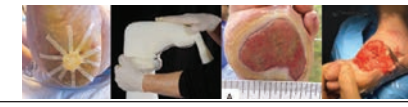

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surgical intervention for CN. The current gold standard treatment protocol is immobilization while the CN is in the active stage. Active CN is clinically identified with warmth, edema, and hyperemia as compared to the contralateral limb. Radiographically, active CN shows signs of joint effusion, fragmentation, subluxation, and/or dislocation. There are concerns for soft tissues in the presence of this active inflammatory process. This is why surgical correction in the active stage has been classically non-advisable.

Internal Fixation

Simon, et al. were the first to describe early operative arthrodesis rather than non-operative management for patients with acute, active midfoot Charcot. They stated that the standard non-operative management of active midfoot CN with total contact casting often results in malunion or nonunion of the affected area. This subsequently leads to deformity and complications

Figure 1: Radiograph of unstable active Charcot deformity *(Photo courtesy of Patrick Burns, DPM)*

such as ulceration, infection, and the need for surgical intervention.[3]

Their study included 14 patients, all with active midfoot CN classified radiographically as Eichenholtz stage I. They performed extensive debridement, open reduction of deformity, and arthrodesis with internal fixation and autologous bone graft. All 14 patients had successful arthrodesis procedures. They reported no ulcerations post-operatively. All patients regained the walking ability that they had prior to the Charcot events. They found that operating in the active stage did not lengthen the time to healing but, in fact, it seemed to speed up the healing process and more quickly reversed the destructive stage. It is hypothesized that restoring anatomic alignment and providing structural stability to the area can expedite the reversal of the destructive, active phase.[3]

Structural stability through arthrodesis is a topic that was emphasized by Sammarco in 2009. He coined the superconstruct principles when performing reconstructive surgery for CN. He believes that structural stability in CN is achieved through arthrodesis, and fusion should be extended beyond the zone of injury to unaffected joints. He also states that bony resection should be performed during fusion to allow for adequate reduction of deformity and to avoid unwanted tension to the soft tissue envelope, facilitating closure. He further states that definitive arthrodesis should be done using the strongest devices available that can be tolerated by the soft tissues.[4] These superconstruct principles, when applied to active CN, seem to reach a balance between achieving the structural stability that is desired to prevent progression of deformity while also respecting the inflamed, hyperemic soft tissue envelope.

Figure 2: Radiograph of same patient demonstrating operative reduction of deformity in the active Charcot stage with maintenance of reduction utilizing multi-plane ring external fixator and trans-articular pins *(Photo courtesy of Patrick Burns, DPM)*

External Fixation

Another method to achieve structural stability while respecting the soft tissue envelope is through the use of external fixation. Oftentimes active Charcot deformities of the foot and ankle are grossly unstable. When this is the case, immobilization with casting can still allow progression of the deformity. Active CN can also present with significant collapse and/or dislocation of joints. These scenarios make non-operative immobilization difficult. Multi-plane external fixation is one method to achieve a stable limb while limiting incisions and soft tissue dissection in the active stage.

Monaco, et al., in unpublished data, have proposed a staged surgical reconstructive protocol for patients with active CN. They reviewed 14 patients (15 reconstructions) who underwent staged reconstructions for active CN deformities. The index procedure for these patients was application of a multi-plane ring external fixator while in the active stage. Closed reduction of the deformity was performed when possible, and small accessory incisions were made as necessary to aid in reduction of the deformities. The external fixator and percutaneous, trans-articular pins were used to stabilize the reduced deformities for approximately two months.[5]

They believe that the external fixation serves several purposes. It maintains deformity reduction in a more anatomic alignment. Ulcerations, if present, are allowed to heal by complete offloading and removal of tension. Adequate debridements are able to be performed with the fixator in place if infection is present. Finally, the skeletal stability provided by the external fixator reduces inflammation, and the Charcot process calms down to an inactive stage, which in turn optimizes the soft tissue envelope. After this two-month optimization has occurred, the external fixator is removed, and the definitive arthrodesis procedure is performed using the superconstruct principles.[5]

Figure 3: Radiograph of same patient demonstrating definitive arthrodesis fixation after approximately two months in the external fixator *(Photo courtesy of Patrick Burns, DPM)*

An actual patient from the Monaco, et al, unpublished data is shown in the figures. A pre-operative radiograph of an unstable, active CN deformity (Figure 1), a radiograph with an external fixator maintaining operative reduction of deformity (Figure 2), and a radiograph after definitive arthrodesis (Figure 3) are shown.[5]

In their series of 15 staged reconstructions, they achieved a 100% limb salvage rate at a mean 25-month follow-up. Two of the 15 (13%) had an ulceration at final follow-up, which is consistent with other reports

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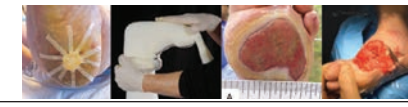

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following major reconstructive surgery for CN. This currently unpublished data was done under the direction of Patrick Burns, DPM, at the University of Pittsburgh Medical Center, and it is the largest known series of patients who underwent surgical intervention for active CN by a single surgeon.[5]

It is believed that non-operative treatment can allow an unstable injury to progress, leading to malunion and the potential for ulceration and/or infection.

Immobilization with Casting vs. Surgery

Although the current standard treatment for active CN is immobilization with casting, it is the belief of some surgeons that certain active deformities are grossly too unstable to be successfully treated with nonoperative immobilization. It is believed that non-operative treatment can allow an unstable injury to progress, leading to malunion and the potential for ulceration and/or infection. Furthermore, the skeletal stability achieved with operative reduction and fixation (internal and/or external) in a more anatomic alignment is believed to allow the soft tissue inflammatory process to quiet down more quickly. For these reasons, the timing for surgical intervention in CN is a debatable topic.

There is still much to learn regarding CN. The pathogenesis is still poorly understood, and the optimal treatment protocols are still being developed. With people living longer due to advances in medicine and the diabetes epidemic on the rise, we are sure to see more CN than ever before. The medical community has come a long way in the treatment of CN in recent years. The reasons are multifactorial. There has been a recent increase in medical literature regarding CN, which is fortunate considering that CN has been an overall under-represented topic.

There have been vast improvements in fixation technology, such as locking plate constructs and devices designed specifically for Charcot deformities. There has also been more collaboration with multidisciplinary teams taking on these challenging CN cases. Consults to internal medicine, infectious disease, nephrology, endocrinology, and vascular surgery are not uncommon to optimize the diabetic patient's comorbid conditions when faced with limb salvage reconstructive surgery for CN. Reconstructive surgery for CN is extremely challenging yet can be very rewarding. Research must continue on this topic to shed light on many unanswered questions. **PM**

References

[1] Frykberg RG, Belczyk R. Epidemiology of the Charcot Foot. Clinics in Podiatric Medicine and Surgery 2008; 25:17-28.

[2] American Diabetes Association. Diabetes 1996 Vital Statistics, American Diabetes Association, Alexandria, VA, 1996.

[3] Simon SR, Tejwani SG, Wilson DL, Santner TJ, Denniston NL. Arthrodesis as an Early Alternative to Nonoperative Management of Charcot Arthropathy of the Diabetic Foot. Journal of Bone and Joint Surgery Am 2000; 82(7):939-950.

[4] Sammarco VJ. Superconstructs in the treatment of Charcot foot deformity: plantar plating, locked plating, and axial screw fixation. Foot Ankle Clin 2009; 14(3):393-407.

[5] Monaco S, Toth A, Rettedal D, Blazek C, Burns P. (2016). Early Staged Surgical Reconstruction for Active Midfoot and Ankle Charcot Neuroarthropathy in Patients with Diabetes: A Case Series (Unpublished Manuscript).

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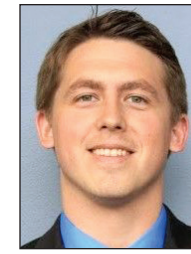

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