

The Use of a Novel Suture Retention Device to Prevent Surgical Wound Dehiscence (SWD)

The HEMIGARD® is a simple, inexpensive device that can reduce SWD.

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Introduction

Diabetes increases the risk for many serious health problems. One of the more devastating complications of long-standing diabetes is the development of a pattern of osseous destruction known as Charcot neuroarthropathy (CN). This is a progressive condition characterized by acute fracture, dislocation and joint destruction in the neuropathic foot. This process can often be painless due to the loss of sensory innervation present in patients with

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diabetes. As the disease advances, it is common to see bone destruction, joint subluxation, structural abnormalities, rocker-bottom plantar deformities, and ulcerations that can lead to infection and amputations. Patients with diabetes and Charcot neuroarthropathy have a significant decrease in quality of life and increase in mortality and morbidity when compared to patients with diabetes alone.¹

CN was originally described by the French neurologist Jean-Martin Charcot in his famous 1868 publication², yet to date the pathophysiology of the disease is still debated. Currently, there are two prevailing theories. The first is based on the original French theory that the condition is neurovascular in nature. This hypothesis suggests that vasomotor changes related to autonomic nerve dysfunction compounded by sensory nerve damage leads to vasodilation and increased peripheral blood flow causing bone destruction and degradation.³ Bone resorption, fracture, and subsequent

subluxation are thought to be direct results of this heightened sympathetic activity.³ A second neurotraumatic theory postulates that it is in fact repetitive micro-traumatic events occurring in the neuropathic foot contributing to subclinical fractures that go untreated and lead to the hallmark bone destruction and joint subluxation.³ The central idea of this theory is that there is an amplified inflammatory response caused by local trauma.³ Both of these theories have merit. CN is most likely caused by a combination of sensory and autonomic neuropathy contributing to an increased vulnerability to trauma resulting in structural changes over time.

Although the incidence of CN is relatively low, this condition causes significant morbidity and has a mortality rate estimated to be as high as 28%.¹ Severe structural abnormalities such as midfoot collapse frequently result in gait abnormalities and skin breakdown leading to ulceration. One study found as many as 34% of CN patients developed foot ulcerations as a direct result of CN joint dislocation.¹ In a study by Sohn, et al., the amputation risks were 12 times higher in patients with both CN and ulcer versus CN alone.⁴ It is often difficult to successfully brace and stabilize the foot in cases of advanced structural deformities. Thus, surgical reconstruction to correct CN

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New Concepts and Studies

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Figure 1: Areas of tissue necrosis were clinically noted at the second post-op visit.



Figure 2: The medial and lateral dehiscent surgical sites

deformities can mitigate the morbidity and mortality seen in these patients.

The primary goal of CN surgery is to restore functional activity through structural stabilization and alignment of the foot, thus allowing the patient to ambulate in appropriate footwear or pedorthic devices without com-

intervention occurred on the right limb. Post-operatively the patient developed a surgical site dehiscence that required months of aggressive wound care. To prevent a similar occurrence on the left limb, a novel adhesive suture retention device (HEMIGARD® ARS; SUTURE-GARD Medical, Inc.; Corvallis, OR) was used to augment skin closure. The addition of this device allowed for uneventful healing of the skin structures of the left limb post-operatively.

Case Report

A 60-year-old male patient, with a 14-year history of type 2 diabetes mellitus, presented to the clinic following a recent acute Charcot event on the right foot. There was no history of previous trauma. He had suffered for

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plications. Indications for surgical intervention include non-braceable deformities, gross instability, progressive joint destruction, impending ulceration, non-healing ulceration, recurrent ulceration, osteomyelitis, or limitations of gait. Surgical management of CN can include debridement of non-viable tissues, removal of bony prominences, Achilles tendon lengthening, arthrodesis, open reduction and internal fixation, bone grafting and external fixation.

Surgical treatments differ based on the severity of the condition and surgeon preference and experience. Surgical reconstruction of CN is associated with a high rate of complications. It has been reported that surgeons embarking on such reconstructions should strive for a 90% limb salvage rate.⁵ Among the most common post-surgical complications is that of surgical site dehiscence with subsequent infection. Surgical site infections can result in prolonged healing, hospitalizations, additional surgical interventions, osteomyelitis, and in the worse cases, amputation.

The authors present a case report of a patient with bilateral CN deformities with instability and impending ulceration. Similar reconstructive procedures were performed on both lower extremities. The initial surgical

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years from a chronic Charcot deformity on his left foot. The patient had a history of poorly controlled DM in past years, but recently intensified medical treatments enabled him to get his HbA1c to 6.7. His past medical history was negative for smoking, but positive for lymphedema, hypertension, and hyperlipidemia. His medications include Levemir, Jardiance, Metoprolol 50mg, Losartan/HCT 100/12.5, Gabapentin 100mg TID, Novolog, 81mg Aspirin. He was currently working as an owner/operator of a metal machining shop, where he was working on his feet all day.

Upon exam, it was noted that both lower extrem-

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ities exhibited a rocker-bottom appearance with complete collapse of the medial longitudinal arch with a pre-ulcerative callus at the area of the cuboid prominence. The right foot deformity was more severe than the left. Pulses were 2/4 DP and PT bilaterally.

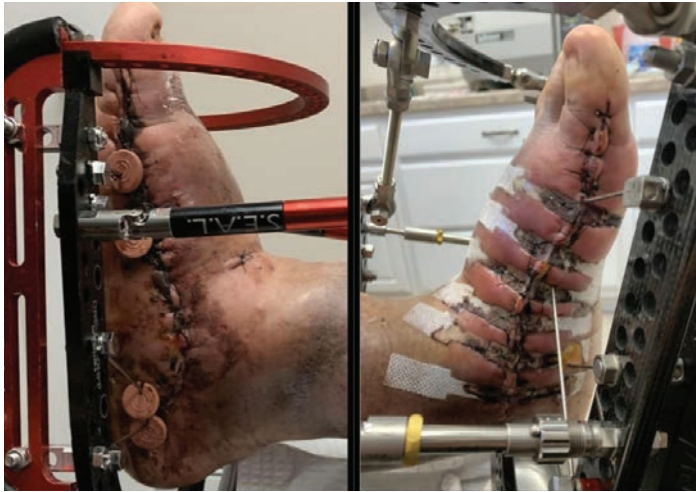


Figure 3: HEMIGARD devices were used to augment skin closure and prevent dehiscence.



Figure 4: HEMIGARD devices were removed 4 weeks post-op.

Non-invasive vascular studies were performed and showed triphasic flow with a 1.3 ABI bilaterally. Review of systems and remaining physical exam were unremarkable. His body mass index (BMI) was 43.5 at the time of initial presentation, but had reduced to 38.2 by the time of surgery.

Plain film X-ray evaluation revealed the following:
Right: subacute collapse of the midfoot at the level of

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the Lisfranc, talonavicular, cuneionavicular, and cuboid 4th and 5th metatarsal joints. Talar declination angle increased to near vertical. Calcaneal inclination angle is now about -15 degrees. Cuboid and 1st cunieform are both plantarly prominent. Rocker bottom deformity. Collapse of the navicular and 1st cunieform. Laterally displaced lesser metatarsals.

Left: Chronic collapse of the talonavicular joint and cuboid 4th and 5th metatarsal joints. Collapse of the inferior portion of the navicular. Talar declination angle has increased to near vertical. Calcaneal inclination angle is now negative by about 25 degrees. Cuboid is prominent plantarly.

At this point, the diagnosis of chronic CN with severe structural changes involving both feet was made. Conservative versus surgical options were discussed with the patient. Ultimately the patient opted to have reconstruct-

tion as opposed to continuing long-term offloading in CROW boots. His current devices had been successful at preventing ulceration; however, the patient felt that they were heavy and difficult to work in. Considering that he now suffers with significant Charcot deformities on both feet, he was at a drastically increased risk for skin breakdown and potential amputation.

Based on the increased severity of the right foot deformity, the patient underwent surgical intervention on this limb first. The patient was taken to the OR and placed on the table in a supine position. He had general anesthesia for the procedure. A thigh tourniquet was applied and inflated. The limb was prepped and draped. A gastrocnemius recession and percutaneous Achilles tendon lengthening was performed prior to any bony

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Figure 5: Long term post-op follow-up, B/L feet

work. A dorsomedial curvilinear incision was made from the talar neck to the first metatarsal phalangeal joint. The tibialis anterior tendon was retracted and bony structures were exposed with careful sub-periosteal dissection.

A midfoot wedge osteotomy was performed and the gap was closed through gentle manipulation. An intramedullary nail was used for medial column fixation. The lateral column was approached using a dorsolateral incision from the midshaft of the 4th metatarsal and extended toward the tip of the lateral malleolus. Soft tissue dissection was performed with care to preserve all vital structures. A large exostosis was resected from the 5th metatarsal base and this resulted in the removal of the peroneal tendon insertion. The peroneal tendon insertion was implanted onto the calcaneus prior to closure.

Resection of the overlapping bone ends was performed to remove excess dorsal and plantar bone and prep the joints for fusion. Internal stabilization of the subtalar joint and lateral column was achieved by using cannulated lag screws. All surgical incisions were closed deeply with 2-0 Vicryl, subcutaneous tissue was closed with 3-0 Vicryl, and skin was closed with 2-0 nylon. Lastly and external fixator was applied to enhance stabilization.

Post-operatively, the patient was non-weight-bearing on the surgical limb. The patient was seen at the clinic weekly for post-op care. Areas of tissue necrosis were clinically noted at the second post-op visit (Figure 1). Nitro patches (0.2mg/hr.) were applied proximal to the wounds for 12 hours a day to aid in improved arterial flow to the incision sites. At three weeks post-op, it was noted that both the medial and lateral surgical sites had dehisced and required wound care (Figure 2). Various wound treatments including serial debridement, antibiotic ointment, collagenase ointment, and platelet-derived growth factor gel were used throughout his course of care.

The lateral surgical incision healed after 4 months of wound care and the medial surgical incision had epithelialized by 7 months post-op. The medial incision, however, developed an infected abscess about a month later that probed to the level of hardware. The patient was taken back to the OR where the intramedullary rod was removed and an antibiotic rod was inserted. After an additional 3 months of wound care, the patient went on to complete healing. He is currently ambulating in a custom shoe with molded insert.

Roughly 7 months later, the patient underwent a similar surgical intervention to correct the CN deformity on the left limb. In order to help to mitigate the post-op complications seen in the previous surgery, several adhesive

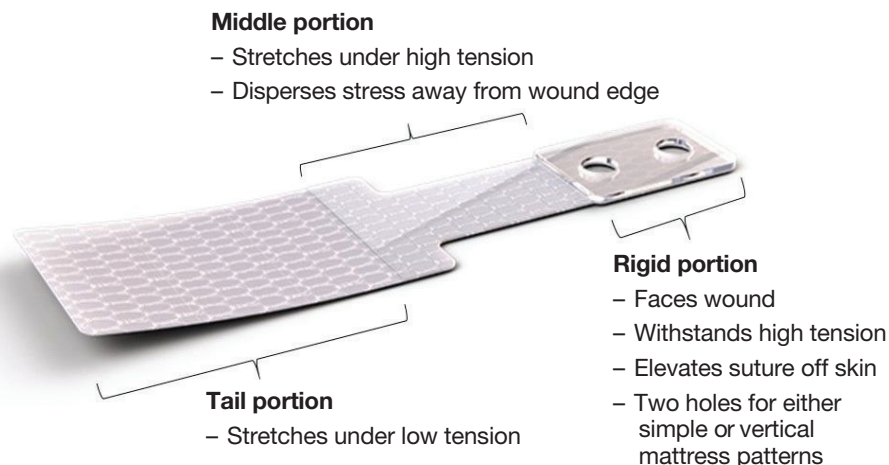


Figure 6: HEMIGARD device

suture retention devices called HEMIGARD were used in addition to deep and subcutaneous sutures to augment skin closure and prevent dehiscence (Figure 3). The patient's post-operative course was uneventful. All surgical incisions were noted to be completely healed by 4 weeks post-op and the HEMIGARD devices were removed (Fig-

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ure 4). The patient went on to complete healing post-operatively without sequelae (Figure 5).

Discussion

Surgical wound dehiscence (SWD) is defined as the separation of the margins of a closed surgical incision that has been made in skin, with or without exposure or protrusion of underlying tissue, organs, or implants.⁶ The re-approximation of skin incisions after invasive surgical interventions can be challenging and SWD is a common complication. SWD can be a result of suture breakage, poor incisional or flap design, and/or closure under tension. Decreases in dermal collagen integrity occur with age and can result in friable skin that can easily separate with sutures alone. This case report demonstrates the significant sequelae that can result when surgical incisions are closed under tension with sutures alone. Very little data is available on SWD in patients who have had foot and ankle surgery. Most available information on morbidity, mortality, and healthcare costs are not specific to lower extremity SWD. However, as illustrated in this case report, SWD can prolong healing and even cause the need for additional costly surgical intervention.

The best course of treatment for SWD is prevention, and the use of the HEMIGARD device appears promising.

SWD can be a result of suture breakage, poor incisional or flap design, and/or closure under tension.

The HEMIGARD device is made up of 3 zones.⁷ (Figure 6) Zone A is the water-resistant rigid, elevated wound end zone with 2 holes to accept and withstand forces of high-tension non-absorbable simple interrupted or vertical mattress suture.⁷ This zone elevates the suture above the skin surface. Zone B is the less rigid, non-elevated, water-resistant middle zone that transmits force of wound closure away from the wound edge.⁷ Zone C is the least rigid zone with a monolayer of stretchy non-woven polyester that allows for reduction of shear forces from high tension wound closures.⁷ In this case report, the addition of the HEMIGARD constructs to the post-surgical skin closure on the left foot allowed for transfer of tension away from the vulnerable incision site during episodes of post-op edema, thus preventing incision dehiscence.

Wound healing by secondary intent can take many months and increases the risk of infection and morbidity. The HEMIGARD has shown favorable results in Mohs surgery, and this case report suggests that there are similar advantages seen when the device is used in complex skin closures following surgical reconstruction in the foot and ankle. The authors believe that the HEMIGARD is a simple, inexpensive device that can easily be added to standard of care skin closure techniques to prevent SWD and improve patient outcomes. **PM**

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