Cauda Equina Syndrome Resulting In Late Sequela of Calcaneal Gait And Neuropathic Heel Ulcer

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The case of a 27-year-old female, 5 years after a motor vehicle accident that resulted in spinal cord damage, is reviewed. She progressed well after her accident for 4 years, at which time she developed a neuropathic heel ulceration, aggravated by her calcaneal gait and pregnancy weight gain. The rarity of such a case involving neuropathic ulceration and calcaneal gait after partial deficit of cauda equina function prompted this article.

Trauma to the spinal column can result in complete or incomplete injury to the spinal cord, cauda equina, or peripheral nerve roots.[1] Complete lesions are characterized by a lack of motor and sensory function below the level of injury, whereas incomplete lesions retain a mixture of motor and sensory function. Although the majority of incomplete lesions have no definite pattern of recovery, some partial spinal cord injuries do result in a particular clinical picture or syndrome, such as cauda equina syndrome.[2]

The cauda equina roots are responsible for the sensory and motor innervation of most of the lower extremities, the pelvic floor, and the sphincters. They also carry the autonomic supply to and from these structures, including bowel and bladder control.[3] Cauda equina syndrome usually presents with low back pain, bilateral sciatica, saddle anesthesia, and weakness in the lower extremities associated with paralysis of the bowels and bladder.[4] Complete lesions of the cauda equina (consisting of the anterior and posterior nerve roots) result in a lower motor neuron clinical picture with flaccid paralysis and loss of reflex activity and sensation. Because of the mobility of the nerve roots of the cauda equina within the neural canal, lesions of the cauda equina are most often incomplete.[1]

Weakness, diminished deep tendon reflexes, and pain and sensory disturbances associated with cauda equina syndrome may be seen in the territory of any of the roots forming the cauda equina (L2, 3, 4, 5, and S1).[5] The conus medullaris is often spared, and thus bladder and bowel sphincter function and motor and sensory functions of S2 through S4 (sacral sparing).[5] The signs and symptoms of cauda equina syndrome are difficult to differentiate from conus medullaris damage; indeed, often the most reliable test is that of the bulbocavernosus reflex, which, if intact, signifies an intact conus medullaris (personal communication, David Polly, MD).

The most common causes of disease of the cauda equina are injuries, followed by compressive etiologies (spinal neoplasms and herniated lumbosacral discs). Differential diagnosis includes lesions of the conus medullaris, lumbosacral plexus neuropathy, and Guillain-Barré syndrome.

Case Report

A 27-year-old 6-month pregnant female presented with a right heel ulcer that had been present for the past year. Five years ago, she sustained a fracture to the third lumbar vertebrae in a motor vehicle accident that left her with complete paraplegia and lower

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extremity sensory loss. Automobile accidents typically cause hyperflexion of the spine, which leads to disruption of the posterior ligaments of the spine with the vertebral body often being split transversely into two parts or displaced anteriorly.[1] Back surgery was performed for the fractured vertebra at the time of the accident. Within 5 months after the injury, she began walking again. Rehabilitation consisting of exercises, heat, and electrical stimulation improved her motor recovery and she progressed from a walker to crutches and was discharged 1 year later.

She continues to suffer bladder dysfunction since the accident, and currently takes nitrofurantoin macrocrystals, 100 mg daily, for repeated urinary tract infections. The patient has experienced back pain and weakness of the hips and legs since the accident. The cavus attitude of the right foot was noticed approximately 2 years after the initial injury. Twelve months prior to presentation, a mild abrasion first appeared on the right heel that deteriorated to the presenting ulcer.

Physical examination revealed a healthy 27-year-old pregnant female with her right foot in exaggerated dorsiflexion when in the sitting position (Fig. 1). The right foot was increased ⅛-inch in heel width and reduced ¼-inch in forefoot width as compared with the left foot (Fig. 2). There was a total loss of sensation in the abdomen and on the right side there was a loss of sensation in the buttock, posterior thigh, posterior leg, and the dorsal and plantar aspects of the foot. On the left, sensation was diminished on the plantar aspect of the foot only. Deep tendon reflexes at the knee and ankle were diminished bilaterally. Passive ankle range of motion for the right foot was 45° of dorsiflexion (Fig. 3) and plantarflexion to the neutral position (Fig. 4). The left foot had 45° of passive dorsiflexion and 30° of plantar flexion. The gastrocnemius and soleus muscle group was weak (3/5) and the patient experienced difficulty with active plantar flexion with the right side worse than the left (Fig. 5). However, full strength was maintained for the anterior muscle group (5/5) and the patient could actively dorsiflex both feet (Fig. 6). A calcaneus gait was observed on ambulation, with increased time spent on the rearfoot with diminished push-off. The F-scan diagram depicts increased load on the opposite left heel, as the patient compensates for the right heel ulcer with an antalgic gait (Fig. 7). She was able to walk on her heels (Fig. 8), but unable to walk on her toes. There was no atrophy present in the calf. The right calf was ⅛-inch wider than the left calf (Fig. 9).

The heel ulcer measured 2 × 2 cm and was classified as Wagner grade two with a red granular bed, serosanguinous drainage, slight surrounding hyperkeratosis, and mild periwound erythema (Fig. 10). Radiographs of the right foot showed increased soft tissue density about the heel with a soft tissue defect, and an increased calcaneal inclination angle (Fig. 11). There was no evidence of osteomyelitis.

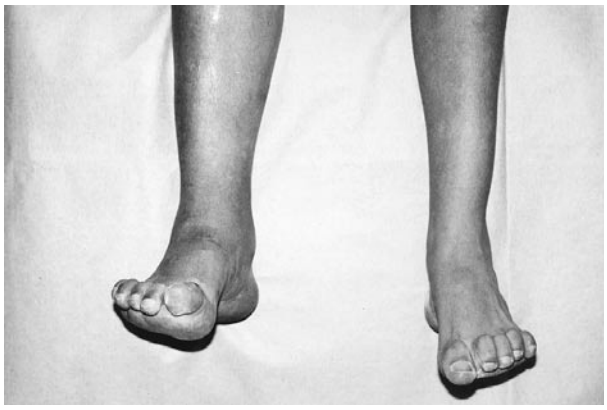


Figure 1. The right foot is pointed upward with the patient seated.



Figure 2. Comparison of the plantar aspects of the right and left feet shows the right foot is increased in the width of the heel and decreased in forefoot width.

The ulcer was treated with mild debridements weekly, dilute Betadine®[1] cleansing and dressings daily, and limited ambulation in a surgical shoe with the aid of a cane. Pressure dispersion was accomplished with a ¼-inch Plastazote®[2] inlay with cutout for the ulceration. After 5 months of only modest healing, a rotation skin flap combined with a calcaneal osteotomy was planned for after the delivery of the baby. A cesarean section was performed because of her inability to deliver vaginally because of the neurologic deficit. The ulcer showed full healing within 8 weeks of delivery (7 months after initial

®[1]Purdue Frederick, Norwalk, CT.
®[2]BXL Plastics Limited, ERP Division, Croydon, England.



Figure 3. Right foot shows passive dorsiflexion to 45°.



Figure 4. Right foot shows passive plantarflexion to the neutral position.



Figure 5. The patient is actively trying to plantarflex the right foot. Active plantarflexion is possible only to the neutral position.



Figure 6. Active dorsiflexion to 45°.

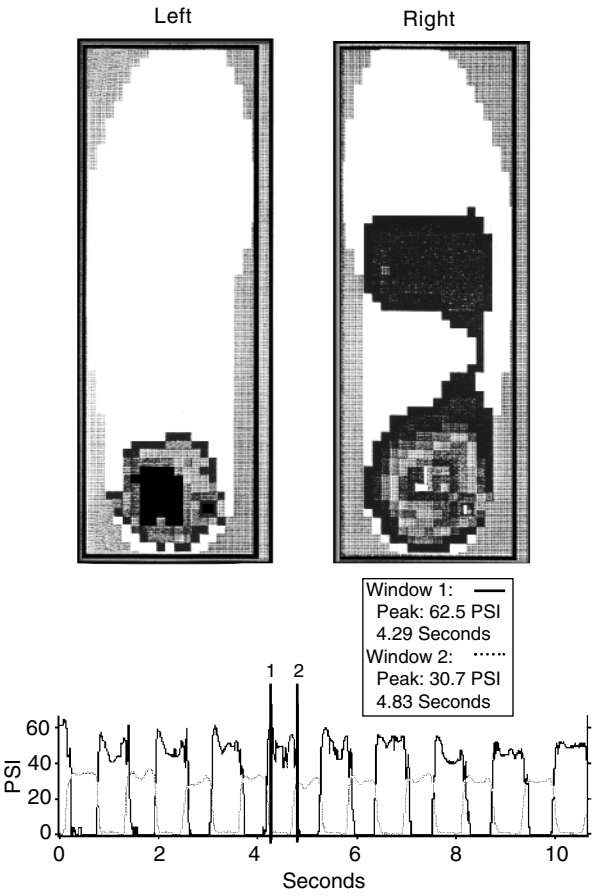


Figure 7. F-scan printout shows peak pressure on the left heel double that on the right heel with absence of weightbearing on the left forefoot. Increased pressure on the unaffected left heel represents antalgic compensation for right heel ulcer.



Figure 8. The patient is able to walk on her heels showing good muscle strength of the anterior muscle group.



Figure 9. Posterior view of patient standing shows wide heel on the right.



Figure 10. Heel ulcer as it appeared at the time of initial presentation.



Figure 11. Radiograph of the right foot revealing soft tissue defect, plantar calcaneus.

presentation), thus obviating the need for surgery (Figs. 12 and 13). Simple cushioning with Plastazote inserts with a cutout heel was sufficient mechanical therapy for this patient. An ankle-foot orthosis with a dorsiflexion stop or locked ankle may be used if a patient exhibits dorsiflexion weakness. In cases where a heel lesion is too painful to tolerate any weightbearing, a patellar tendon-bearing orthosis would provide unweighting of the heel. In this case, meticulous wound care, combined with pressure-reducing inserts and a reduction in body weight, lead to resolution of the heel ulcer.

Discussion

The patient in this report showed many of the typical manifestations of lesions of the cauda equina that included lower back pain, lower extremity weakness,[6-15] voiding problems,[9, 11, 12] sensory deficits to the lower extremities,[4, 7, 10] diminished deep tendon reflexes,[10, 16] and pes cavus foot morphology.[16] Full recovery of muscle function in patients with cauda equina syndrome may occur as long as 10 years after initial paralysis.[6, 10] The patient presented here had initially suffered total lower extremity paralysis for 5 months, which resolved.

The distinguishing characteristic of the authors' patient was that her injuries resulted in a calcaneus gait rather than drop foot, which is by far the more common pattern of a brain or spinal cord injury and cauda equina lesions.[4, 6, 9, 16-18] Weak plantarflexors with calcaneal gait may be the only neuromuscular deformity present in lower lumbar injuries.[19] However, only one report specifically describes persistent weakness of the plantarflexors of both feet in a patient with cauda equina syndrome operated on for lumbar disc herniation.[9] There is a similar case in



Figure 12. The healed ulcer 7 months after the patient began treatment and 1 month after giving birth.



Figure 13. Two months later, the ulcer is gone.

which a woman suffered a burst fracture of L1 with resultant calcaneal gait (personal communication, David Polly, MD). Although the calcaneus gait with weak plantarflexors and adequate dorsiflexors is less dysfunctional than drop foot gait and a relatively minor residual problem compared with the initial paralyzing injury, the neuropathy in conjunction with the stress of pregnancy resulted in a large heel ulcer that could have required amputation.

Concerning treatment, the patient was a young, healthy female who was able to heal with aggressive wound care and weight reduction after giving birth. The ulcer was totally resolved 2 months after the delivery. However, this was not anticipated and a surgical date was planned. Management of patients with a neuropathic ulceration should generally involve various types of pressure reduction. It has been shown that an alteration in gait pattern reduces excessive peak plantar pressures and can be an adjunct to wound healing.[20] Weakness of ankle plantarflexors or dorsiflexors in patients with spinal cord injuries can be treated with an ankle-foot orthosis.[21, 22] Finally, it is necessary to allow adequate time prior to surgical intervention.

Conclusion

The patient reported here suffered complete paralysis of the lower extremities that lasted 5 months as a result of a fracture to the third lumbar vertebrae. She was left with residual weakness of the plantarflexors resulting in calcaneal gait. Calcaneal gait, although a relatively mild deformity as compared with some of the more dramatic neuromuscular sequella resulting from spinal cord injury, in conjunction with the sensory impairment and increased weight and stress of pregnancy, was sufficient to produce a Wagner grade two neurotrophic heel ulcer. Meticulous wound care with reduction of excessive peak plantar pressure and weight reduction after giving birth resulted in resolution of the ulcer.

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