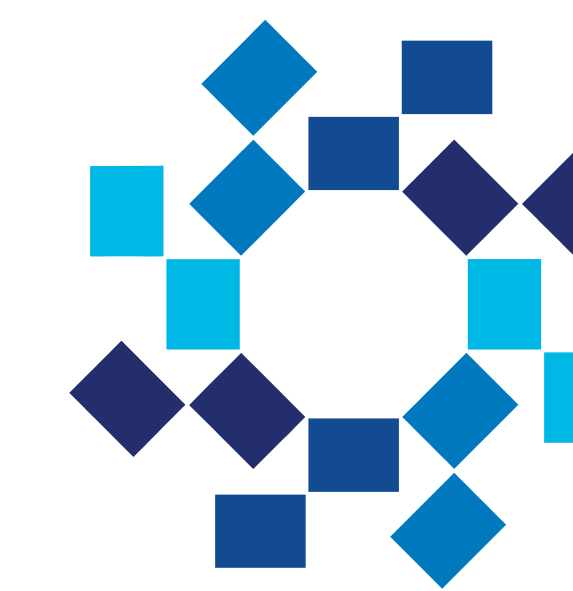


Staged Reconstruction of an Acquired Equinovarus Deformity Secondary to Nerve Injury and Lumbosacral Plexopathy



Hackensack
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PURPOSE

Purpose: Neurologic damage from pelvic ring and sacral fractures is a well documented injury. This case study presents the sequelae of a pelvic ring and sacral fracture affecting the function of the lower extremity causing an equinovarus foot deformity and its staged surgical correction.

REVIEW OF LITERATURE

Neurologic injuries from pelvic ring and sacral fractures is a well-documented sequelae. Most of the literature regarding pelvic injuries have focused on the nerves affected, the motor/sensory findings, and the recovery prognosis. The most frequently associated nerve injuries involve the L5 or S1 nerve roots [1]. Injury to the deep branch of the peroneal nerve or to the L5 nerve root results in primary peroneal muscle weakness and diminished dorsiflexion and eversion strength [2]. This enables the actions of the tibialis posterior and long toe flexor muscles to be unopposed, resulting in a varus hindfoot and forefoot contracture [2]. With damage to the L5 and S1 nerve roots, other muscles in the distal leg can be affected and significantly weakened by the proximal nerve injury. Reilly, *et al.* reviewed 90 patients with unstable fractures of the pelvic ring and found neurologic injuries in 21% of patients. Of those patients, 37% had sensory deficits and the remaining 63% had motor and sensory deficits. In the patients with motor dysfunction, the majority involved L5 or S1. L5 function was least likely to progress to full recovery [1]. Kabak, *et al.* retrospectively reviewed the outcomes of 40 patients with unstable pelvic fractures and 7 sustained neurologic injury [3]. Two out of these patients required a posterior tibial tendon transfer for a residual foot deformity [3].



Figure 1: Clinical appearance of the foot and radiographs of the equinovarus contracture.

CASE PRESENTATION

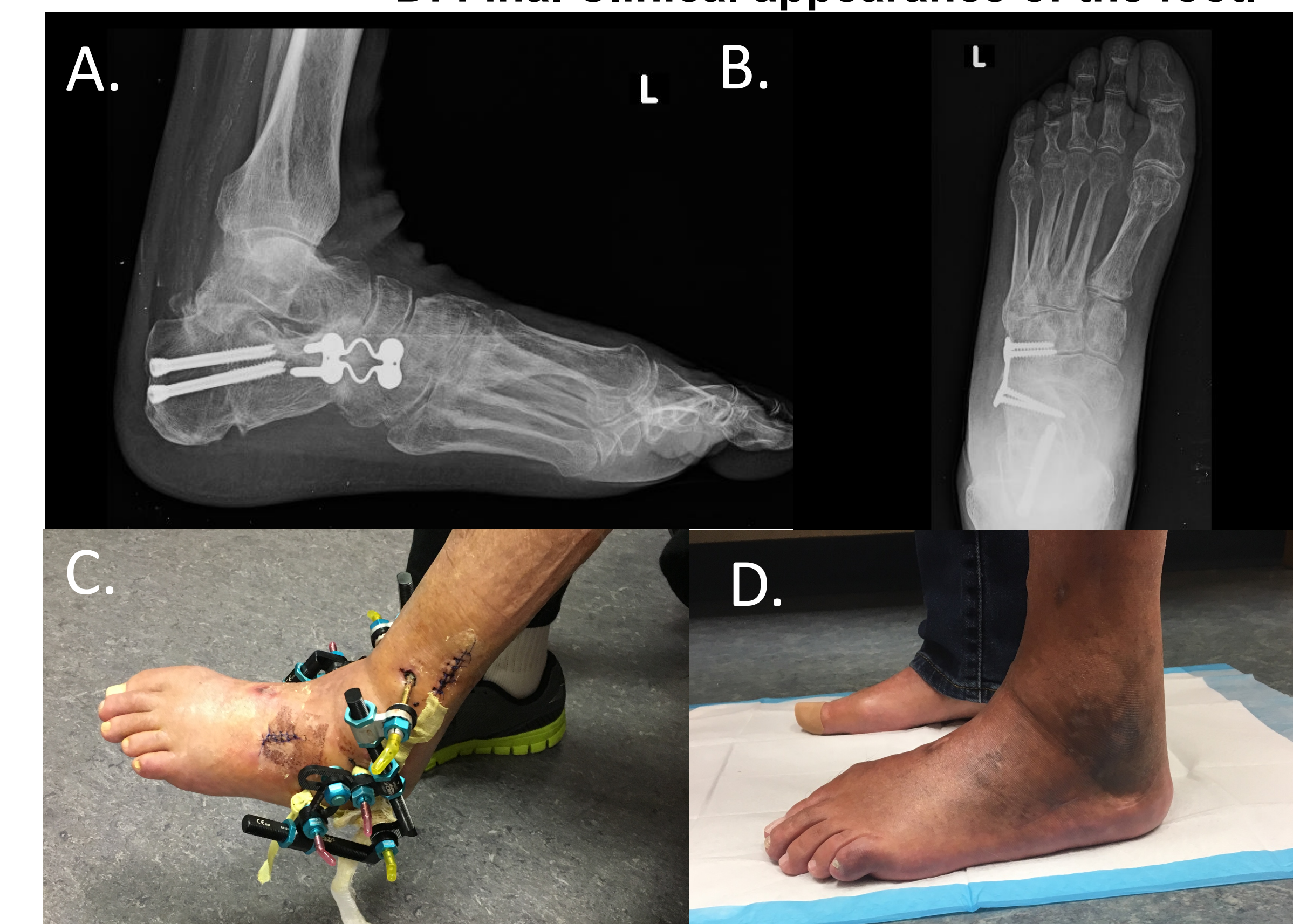
70-year-old male fell approximately 20 feet sustaining a severe vertical shear pelvic fracture, severely comminuted zone 3 sacral fracture, left 5th MPJ dislocation, and L5-S1 nerve injury. Upon presentation, he was noted to have neurologic compromise with documented L5-S1 nerve injury. After fixation of the multiple orthopedic issues, the patient subsequently developed a left drop foot and a left foot equinovarus contracture (Figure 1). The patient had significant difficulty with ambulation secondary to his weakness and foot position. Manual muscle testing revealed grades of 0/5 eversion or peroneal strength, 0/5 strength to the extensor hallucis longus muscle, and 1/5 strength to the tibialis anterior muscle. EMG testing revealed abnormal results, most consistent with axonal motor lumbosacral plexopathy at the L5-S1 nerve roots. Denervation was seen throughout the L5-S1 myotomes in the left limb. Magnetic resonance imaging of the foot and ankle showed a chronically torn ATFL and CFL. The peroneal and extensor tendons, however were intact. Approximately 4 months after the patient's initial injury, reconstructive surgery for correction of his equinovarus foot deformity was performed. The posterior tibial tendon (PTT) was transferred through the interosseous membrane to the lateral cuneiform with a tenodesis screw. The FDL tendon was then transferred into the navicular tuberosity with a bone anchor. A percutaneous triple-hemisection of the Achilles tendon was also performed. A repair of the lateral ankle ligament complex was performed with a modified Brostrom-Gould procedure. Additional reduction was necessary and further manipulation of the foot and ankle was performed gradually over time via medial and lateral external fixator device. The external fixation devices allowed the foot to be corrected acutely and then more gradually into a more rectus alignment (Figure 2C). The patient's complete loss of peroneal muscle power persisted. An AFO device with lateral suspender was trialed for loss of eversion power. Slight rearfoot varus and adduction in the midfoot/metatarsus adductus persisted. In order to further reduce this residual deformity, a secondary procedure was performed at 12 months after the initial injury. A lateralizing calcaneal slide osteotomy was performed in combination with a calcaneocuboid arthrodesis (Figure 2A and 2B).

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Figure 2:

A and B: Final radiographic appearance of foot.
C: Medial and lateral external fixator.
D: Final Clinical appearance of the foot.



RESULTS

At a final follow-up of 26 months, after the initial injury, the patient is ambulating pain free with continued use of an AFO brace with a lateral suspender. He has a rectus foot type with a mild residual metatarsus adductus deformity (Figure 2D). He continues to have loss of peroneal and EHL muscle power but is able to actively dorsiflex his ankle to 5 degrees as a result of the posterior tibial tendon transfer.

DISCUSSION

Our experience with correction of the equinovarus foot deformity is similar to the existing literature. A posterior tibial tendon transfer has been used successfully to correct equinovarus deformity in spastic disorders, leprosy, clubfeet, and polio. This transfer removes the deforming force and additionally assists dorsiflexion during the swing phase of gait [4]. The correction of rigid varus deformities requires combining multiple osseous procedures involving midfoot and hindfoot osteotomies or arthrodesis [5]. The lateralizing calcaneal osteotomy can effectively reduce the varus moment arm of the Achilles tendon at the ankle during stance phase and reduce the contribution of the Achilles tendon toward the tibialis posterior in favor of the peroneus brevis during toe off [2]. An osteotomy is indicated for a mild to moderate fixed deformity that persists after appropriate tendon releases in a patient without arthritic changes in the surrounding joints [2]. The calcaneal-cuboid arthrodesis assisted heavily in reducing the persistent midfoot adduction deformity and was performed to provide a permanent correction.