A unique case of ulceration secondary to heel pad dystrophy

Maddaford L • Foley L

Abstract

This case study presents a unique cause of dystrophy of the plantar heel pad and its associated ulceration. The heel pad is a specialised structure, designed to shock absorb ground reactive forces during normal gait. Reduction or loss of shock absorption results in abnormal and destructive forces being applied to the plantar surface of the calcaneus, namely the medial calcaneal tubercle. Consequently, lack of tissue resilience over bony prominences, coupled with repetitive constant pressure, and underlying neuropathy, inevitably leads to ulceration. Wound management and providing the patient with functional gait whilst minimising pressure to the heel pad is the therapeutic goal.

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Introduction

Persistent ulceration over weight bearing prominences can be secondary to peripheral neuropathy from systemic conditions such as diabetes. However, neuropathy is associated with a wide variety of medical conditions and the effect on a patient is varied. Tabes dorsalis, syringomyelia, Hansen's disease, spina bifida, meningomyelocele and chronic alcoholism all produce some form of peripheral sensory change and create a risk of ulceration.

The following case history will illustrate the effect of iatrogenic unilateral lower limb neuropathy and the resultant chronic ulceration of the heel secondary to the routine insertion of an epidural catheter for intra and postpartum analgesia.

Linda Maddaford
Undergraduate, Podiatry
Curtin University of Technology
Kent Street, Bentley, WA 6102
Tel: (08) 9245 3127

Laurence Foley
Dip Ch. Ms C. F. A. Pod. A
Fremantle Hospital
Alma St, Fremantle, WA 6160
Tel: (08) 9431 2330
Fax: (08) 9431 2918

All correspondence to Linda Maddaford

Heel pad anatomy

The structure and design of the heel pad is specialised for its function of shock absorption and attenuation. The intact heel pad is the initial point of contact between the lower leg and the ground during normal gait.

In a healthy individual the heel pad can attenuate up to 80% of ground reactive forces being transmitted to the joints of the lower extremity [1]. Thus, compromise of this structure can lead to arthritic pathology of joints when subject to the physiological loads of daily living [1].

The heel pad itself lies between the bony plantar surface of the calcaneum and the thick plantar dermis and epidermis [2]. It is the organisation of the lipid cells themselves that is unique and enables the heel pad to attenuate shock waves.

The lipid cells, described as highly specialised tissue called corpus adiposum calcanare, are organised into vertical columns separated by septa of fibrous connective tissue. These septae are then anchored to the plantar surface of the calcaneus, and the adipose tissues (lipid cells) are of a spiral orientation [2, 3]. The heel pad is thus able to compress and dissipate force [2].

Neurological supply is via the rami calcanei medialis, derived from calcaneal, and medial and lateral plantar branches of the tibial nerve, spinal level L3-S3. Vascular supply is via the anterior, posterior tibial and peroneal arteries which send branches into the various fibrous septae [2, 3].

Complications of epidural anaesthesia

The reported incidence of neuropathy due to epidural insertion is not uncommon and has been stated to show

increasing prevalence [4]. The exact incidence is open to conjecture in the literature, and is broadly reported between 1 in 10,000 to 1 in 20,000 [5]. This can be attributed to the increased use of epidurals for intra and post-operative analgesia and obstetrics [4]. A review of anaesthetic literature adds to the uniqueness of this case, as articles reviewed involved patients with transient symptoms that resolved in approximately 2-12 weeks.

The majority of cases resulted in temporary foot drop that was attributed to a combination of factors, predominantly positioning of the patient intra and postoperatively. This resulted in a compression neuropathy which, with physiotherapy, was resolved [4-7].

The podiatric literature revealed many case studies of heel pathology of varied aetiology. Neuropathy post-epidural insertion, resulting in plantar ulceration, has not yet been reported. Gait anomalies and post nerve trauma were widely reported, but aetiology was normally congenital or traumatic.

One such case reviewed resulted in a similarly unilateral dysplastic asymmetrical heel pad, the reason being a condition known as calcaneal gait. A case of *cauda equina* syndrome in a 27 year old female resulted in ulceration and hyperplasia of the plantar heel pad secondary to calcaneal gait [8].

The features of this condition included full dorsiflexion and weak plantarflexion; this is in contrast to the contralateral limb which has full sagittal plane motion. When the patient hangs both limbs over a treatment table with no ground contact, the affected limb will exhibit full unopposed dorsiflexion and the normal limb, slight plantarflexion [9].

Case report

The patient was a 43 year old female that had had chronic ulceration of the right plantar heel pad for 5 years. Persistent ulceration and infection had resulted in a dystrophic and dysplastic heel pad. The adipose tissue of the pad had consequently shifted from the plantar surface of the calcaneus and had become displaced to the medial, posterior and lateral borders of the heel (Figure 1). The ulcer itself was surrounded by extensive hyperkeratotic tissue. The patient was able to dress the wound herself with available foams such as Biatain® and attended regular podiatry and ulcer clinic appointments to monitor the problem.

The aetiology for this persistent ulceration was a unilateral iatrogenic neuropathy of the right lower limb. Five years ago the patient underwent routine insertion of an epidural catheter for intra and postpartum analgesia. Physiotherapy was instigated for an assumed foot drop complication; however, this was ineffective in halting the subsequent deterioration of the function and strength of the right lower limb.

Ulceration began soon after discharge from hospital. Full dorsiflexion and weak plantarflexion were exhibited but the diagnostic sign as described previously was negative to calcaneal gait or foot drop on the right foot.

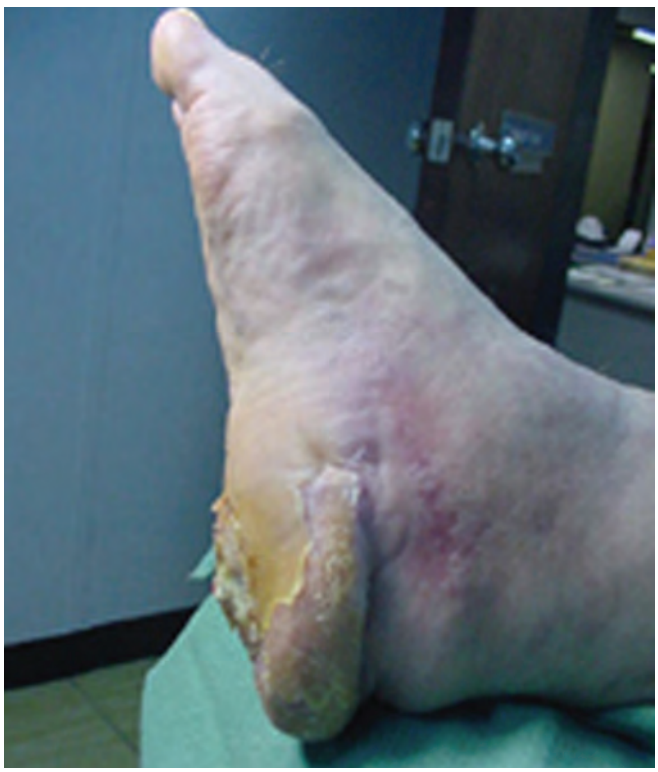
Complications with muscle weakness, reduced reflexes, reduced range of motion and weak dorsiflexion and plantarflexion also became apparent. The foot became hyperhidrotic, pale and cool, with touch evoked allodynia. Dysthesia was present on stimulation and atrophy of the foot musculature was evident.

The patient ambulated with difficulty, using one crutch on the affected side. Gait was antalgic, with wide base of stance, weak forward swing of the right limb and poor propulsion. Gait was therefore inefficient and thus tiring. She was unable to maintain walking activity for greater than 1 hour as the heel pad became hypersensitive and paresthesia and hyperalgesia was experienced.

Podiatric treatment

The podiatric treatment plan evolved with the patient over the 5 years in accordance with the ulcer's cyclic healing and

Figure 1. Medial view of right foot. Note the extent of the heel dysplasia.



re-occurrence, complicated with superimposed infection and occasional need for hospitalisation and intravenous antibiosis. Primary wound care of mechanical debridement of surrounding callosity and basic wound care have been a mainstay.

A variety of foam dressings have been employed to help with absorption and cushioning of the ulcer. More challenging has been the attempt to relieve the area of weight whilst ensuring gait was possible and stable. The patient ambulated in a pair of high quality non-prescription boots with the assistance of forearm crutches (Figure 2). Any 'off the shelf' footwear is a problem due to the asymmetry of the right heel pad.

Therapeutic silicone gel heel cups have also been used in the shoe to cushion the area, with mixed results. At the time of assessment, although a wound was not present over the plantar surface, thick callosity over the medial calcaneal tubercle with dehydrated hypoplastic epidermis was observed (Figure 3). Note also the loss of the heel pad thickness under the heel in Figure 4.

The option of providing the patient with specialised orthotic control known as dynamic orthotics may be a beneficial adjunct. These particular orthotics are designed to induce primitive tonic reflexes and reduce spastic tone common with nerve injury [10]. The patient has had an ankle foot orthosis (AFO). These are commonly prescribed for a variety of conditions to provide specified sagittal plane motion or to off load an area of the foot [11]. The AFO was initially successful in offloading the heel but the materials the device was constructed from fractured from fatigue and became unusable.

Figure 2. Note the bulge of the heel counter to accommodate the dysplastic heel.

Figure 3. Plantar view of right foot. Note ulceration and maceration.

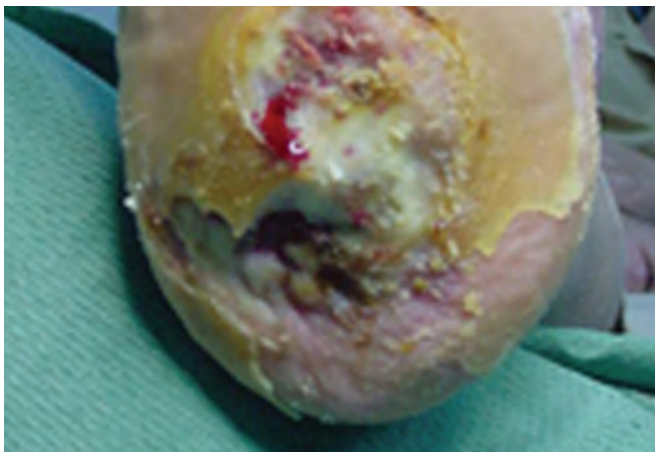
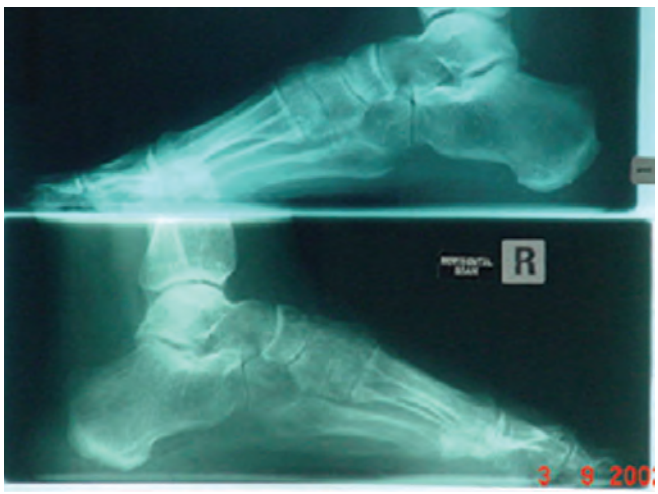


Figure 4. Lateral x-rays of both feet. Note the different thicknesses of the heel pads.



Trials with plaster casts were instigated to attempt to limit dorsiflexion of the ankle past 90^0 as this invariably leads to full heel contact in normal gait. Gait analysis of the patient – with a plaster slab along the anterior of the tibia down over the dorsum of the foot with transverse plaster stirrup in the midfoot – was unsuccessful. This was not due to excessive heel loading but the patient's lack of stability in gait – the knee joint compensated by excessive flexion.

Due to the unique nature of this patient's injury and gait requirements, prescription footwear heavily modified to accommodate the dysplastic and deformed heel pad was trialled (Figure 2). A Spenco product, Dermal pad, has been enclosed in 1.5mm Spenco rubber (Figures 5 & 6) to reduce shear on the area. This appeared to have had a beneficial effect on the area after 1 month (Figure 7).

Figure 5. Spenco dermal pad enclosed in a Spenco cover.

Figure 6. Dermal pad placed in the boot.

Figure 7. Compare the state of the heel wound after 1 month use of the dermal heel pad in new footwear.

Discussion

Future consideration of surgical treatment such as resection of the redundant heel pad, calcaneal osteotomy (either total or sub total) or possible muscle flap has been discussed with the patient. The patient is reluctant at present to explore such options till conservative measures have been exhausted.

Conclusion

The heel pad is a specialised structure that is designed to take initial foot contact during gait. Loss of this shock absorption leads to gait anomalies and local pathology. This case was presented because the aetiology that contributed to the resultant heel pathology was unique and had not been reported in the literature to date. The resultant neuropathy was static but ulceration due to repetitive loading remained problematic.

The challenge is to accommodate the acquired foot and enable a more efficient and stable gait pattern and to reduce wound recurrence.

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