WOUNDS: A Compendium of Clinical Research and Practice

WOUNDS

A Compendium of Clinical Research and Practice

Volume 16, Number 8 August 2004

Biology of Wound Healing

Factors Affecting Tissue Repair

Clinical Management of Wounds

Components of Wound Healing

In this issue:

- ORIGINAL RESEARCH
  Staphylococcus aureus is Associated with High Microbial Load in Chronic Wounds
- ORIGINAL RESEARCH
  Skin Graft Donor Site and Use of Polydeoxyribonucleotide as a Treatment for Skin Regeneration: A Randomized, Controlled, Double-Blind, Clinical Trial
- ORIGINAL RESEARCH
  The Use of Negative Pressure Wound Therapy on Diabetic Foot Ulcers: A Preliminary Controlled Trial
- CASE REPORT
  Development of Foot Drop Following Compression Therapy

Also inside:

- EVIDENCE CORNER
  Matrix Metalloproteinases and Wound Healing

www.woundsresearch.com

Official journal of: AAWC Association for the Advancement of Wound Care

HMP Communications

Case Report

Development of Foot Drop Following Compression Therapy

D. R. Miller, MRCS; S. Enoch, MRCS; D. T. Williams, FRCS;
S. Haglestein, RGN; P. E. Price, PhD; K. G. Harding, FRCS

From the Wound Healing Research Unit, University of Wales College of Medicine, Cardiff, Wales United Kingdom

Abstract: Compression therapy is the mainstay of treatment of venous ulceration. We have observed foot drop, due to compression of the common peroneal nerve on the fibular neck, related to compression therapy. Specifically, we believe this complication arose due to a reduction in the usual amount of padding employed on account of the unusally hot weather at the time. We are unaware of a precedent and seek to alert wound care providers to this potential hazard.

WOUNDS 2004;16(8):270–272

Graduated compression bandaging has been shown by meta-analysis of randomized controlled trials to be the most effective treatment of venous ulceration.[1] We have observed foot drop arising from compression of the common peroneal nerve against the fibular neck in relation to compression therapy and are unaware of a precedent.

Case Report

A 78-year-old gentleman with a chronic venous ulcer of 42 months duration was seen in our clinic, which is set in a United Kingdom (UK) teaching hospital. His previous medical history included deep venous thrombosis in the ulcerated limb eight years previously, mild congestive cardiac failure, and osteoarthritis of the ipsilateral knee. He had no history of diabetes mellitus, rheumatoid disease, or peripheral occlusive arterial disease (both foot pulses were palpable and his ankle brachial index was 1.12). Compression therapy was instituted and a short-stretch compression bandage system (Comprilan®, Beiersdorf AG, Hamburg, Germany) was selected as it was felt to be compatible with the patient's active lifestyle. Bandages are normally applied at full stretch over spirally applied (50% overlap) sub-compression wadding bandage (Soffban Natural,® Smith & Nephew, UK) padding. On this occasion, due to the uncharacteristically warm

Address correspondence to:
Professor Patricia E. Price, Director, Wound Healing Research Unit, Cardiff Medicentre, Heath Park
Cardiff, Wales CF14 4UJ United Kingdom
Phone: 44 2920 682179; Fax: 44 2920 754217; E-mail: pricepe@whru.co.uk

UK weather, a reduced amount of padding was used so that only the ankle and the anterior tibial border were covered by a double figure of eight and a single lengthwise strip, respectively. It was hoped this modification would increase patient comfort and compliance.

Three weeks later, the patient noticed progressive weakness of ankle movement of the treatment limb but took no immediate action. At next clinic review one week later, he was unable to dorsiflex his ankle (power 0/5) or hallux (power 0/5). There was no history of injury to the limb and no other neurological deficit detectable. A clinical diagnosis of common peroneal nerve palsy due to compression at the fibular neck was made. Compression therapy was suspended, an ankle-foot orthosis (AFO) constructed for the patient, physiotherapy instituted, and a neurology opinion sought. Nerve conduction studies supported the clinical diagnosis.

Subsequently the patient made a gradual recovery and at six months follow-up had improved ankle (power 2/5) and hallux (power 1/5 dorsiflexion, which progressed to almost full recovery at nine months follow-up.

Discussion

Reduction of venous hypertension and edema in the lower limbs is the mainstay of treatment for venous leg ulcers. The application of graduated compression bandaging, hosiery, or intermittent pneumatic compression aims to achieve this by forcing fluid in the interstitial spaces back into the vascular and lymphatic systems. The choice of a particular compression system depends on individual patient variables, including ankle circumference, patient mobility, nature of ulceration, degree of edema, and patient tolerance and adherence.

Compression bandages can be classified based on the predetermined levels of compression they provide at the ankle[2] into the following: light (14–17mmHg), moderate (15–24mmHg), high (25–25mmHg), and extra high compression (35–50mmHg). Short-stretch bandages contain no elastomeric fibers and depend on crimped threads for their extensibility, which do not recover after stretching. When applied at full extension, they form an inelastic covering to the leg, which tends to resist any change in the geometry of the calf muscle during exercise, thereby increasing surface pressure in a cyclical fashion and enhancing the action of the calf muscle pump. They exert higher pressures (30–40mmHg) when the patient is upright and walking and lower pressures at rest. In previous studies, healing and complication rates associated with short-stretch bandaging have been comparable with four layer bandaging.[3–5]

Estimation of the actual level of pressure acting on the common peroneal nerve in this case or the threshold needed to cause nerve injury is not straightforward. The pressure generated under a bandage, or sub-bandage pressure, is governed by the tension of the fabric, the radius of curvature of the limb, and the number of layers applied.[6] Furthermore, the pressure generated within the interstitial fluid of the limb is a function not only of this sub-bandage pressure but also of skin tensile characteristics, muscle bulk, and activity level (a particular feature of short-stretch bandaging). We know from studies of lower-limb compartment syndrome (increase in pressure within an osseofacial compartment leading to compromise of neural blood supply) in trauma and reconstructive surgery that pressures in excess of 30–35mmHg are sufficient to cause first sensory then motor deficit signs.[7] A formal study to establish a pressure threshold, although beyond the scope of this clinical report, would probably involve the construction of specialist simulation models. The application of potentially injurious levels of pressure to human subjects would be unethical.

We believe this complication arose as a result of compression therapy with inadequate padding and wish to raise this as a point of caution for those who apply it. We believe the mechanism was compression of the common peroneal nerve at the level of the fibular neck. Our reasoning behind this assertion is based on both the close time association (onset and regression) of symptoms with the institution and cessation of bandaging, respectively, and the findings of nerve conduction studies. The suggested mechanism is set out below.

The common peroneal nerve gives motor supply to the dorsiflexor and evertor muscles of the ankle and toes along with sensory supply to the foot dorsum and lateral ankle. Due to its relatively superficial passage below the knee through subcutaneous tissue around the outside of the neck of the fibula it

is vulnerable to injury at this site. The anatomical basis for compression is the presence of an inelastic band (consisting of tendinous attachment of the peroneus longus muscle blended with crural fascia and the fibular ligament) arching over the nerve fibers which acts as a fixed point for entrapment.[8]

Neurological insult at any level between cerebral cortex and neuromuscular junction can cause foot drop, and a range of systemic disorders should also be considered in clinical evaluation. Among neurological causes, cortical and spinal cord lesions are a relatively rare, with L5 nerve root, sciatic nerve, or common peroneal nerve lesions predominating. Discrimination can usually be made clinically (facilitated by history and examination) and confirmed by nerve conduction studies[9] and magnetic resonance imaging (MRI).[10] The determination between a compression at the fibular neck and an L5 nerve root or sciatic nerve lesion can be particularly difficult; hence, nerve conduction studies and MRI are often employed in tandem.

Successful treatment of foot drop must address the underlying cause. Benefit may also be derived from rest, non-steroidal anti-inflammatory medication, physiotherapy, and use of an ankle-foot orthosis (AFO); the aim of the latter being to aid restoration of normal range of motion and constrain potentially hazardous uncontrolled plantar-flexion. In certain circumstances, surgical decompression may be appropriate. Speed and extent of recovery depends upon the severity of nerve injury. In this case, gradual improvement in dorsiflexion was observed within weeks of cessation of compression therapy, strengthening the case for this to be the causative factor. However, the length of time to full recovery (12 months) suggests the nerve injury sustained was second degree (axonotemesis), with nerve regeneration occurring from the injury site to the neuromuscular junction at a rate of one inch per month, rather than a first degree nerve injury (neuropraxia) which is usually associated with full recovery at twelve weeks.[11]

This case also highlights an example of the difficulty of balancing competing priorities when caring for patients with problematic wounds. Any medical intervention, no matter how effective, is limited by its tolerability and resultant level of patient adherence. The modification to the level of padding made in this case was aimed at increasing these factors. However well intentioned, this maneuver almost certainly lead directly to this complication, thankfully a reversible one. Repetition of this scenario is not inconceivable as clinicians attempt to adapt treatment on an individual patient basis. The authors wish to relate this case as a cautionary tale.

References

1. NHS Centre for Reviews and Dissemination. Compression therapy for venous leg ulcers. *Effective Health Care Bulletin* 1997;3(4).
2. Dowsett C, Scanlon L. Compression bandaging and the clinical governance agenda. *Br J Com Nurs* 2001;6:9(Supp):17–21.
3. Scriven JM, Taylor LE, Wood AJ, et al. A prospective randomised trial of four-layer versus short stretch compression bandages for the treatment of venous leg ulcers. *Ann R Coll Surg Engl* 1998;80(3):215–20.
4. Partsch H, Damstra RJ, Tazelaar DJ, et al. Multicentre, randomised controlled trial of four-layer bandaging versus short-stretch bandaging in the treatment of venous leg ulcers. *Vasa* 2001;30(2):108–13.
5. Iglesias C, Nelson EA, Cullum NA, Torgerson DJ. A randomised controlled trial of two types of bandages for treating venous leg ulcers. *Health Technol Assess* 2004;8(29):1–20.
6. Thomas S. Compression bandaging in the treatment of venous leg ulcers (online publication). Accessed at: www.worldwidewounds.com. 1997;September.
7. Kasabian AK, Karp NS. Lower-extremity reconstruction. In: Aston JS, Beasley RW, Throne CM (eds). *Grabb and Smith's Plastic Surgery, Fifth Edition.* Editors. New York, NY: Lippincott-Raven Publications, 1997.
8. Ihunwo AO, Dimitrov ND. Anatomical basis for pressure on the common peroneal nerve. *Cent Afr J Med* 1999;45(3):77–9.
9. Katirji B. Peroneal neuropathy. *Neurol Clin* 1999;17(3):567–91vii.
10. Bendszus M, Wessig C, Reiners K, et al. MR imaging in the differential diagnosis of neurogenic foot drop. *Am J Neuroadiol* 2003;24(7):1283–9.
11. Brandt KE, Mackinnon SE. Microsurgical repair of peripheral nerves and nerve grafts. In: Aston JS, Beasley RW, Throne CM (eds). *Grabb and Smith's Plastic Surgery, Fifth Edition.* Editors. New York, NY: Lippincott-Raven Publications, 1997.