POST-INCISIONAL NEUROPATHIES OF THE FOOT AND ANKLE

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Post-incisional acquired neuropathies commonly occur in the lower extremity. There are no nerves in the foot or leg that are immune to injury from compression, traction, laceration, or incarceration in scar.

Unfortunately, there are many iatrogenic causes of nerve entrapment including: tourniquet compression;[1, 2] improper positioning of the anesthetized patient during surgery; bandage or cast pressure;[3] and actual tissue-handling misadventures.[1, 4, 5] Postoperative scarring secondary to normal wound healing can also create an acquired neuropathy; even with proper incision planning, layer dissection, hemostasis, nerve manipulation, and wound closure. Additionally, postoperative infection can cause post-inflammatory fibrosis which can increase the likelihood of peripheral entrapment neuropathy.

PATHOPHYSIOLOGY

Surgical trauma may affect the nerve trunk in a variety of ways. In general, tissue dissection induces an inflammatory response that affects the nerve and surrounding tissue, regardless of whether or not the nerve is directly manipulated during the operative procedure. Anatomically speaking, approximately 50% of the peripheral nerve trunk is made up of connective tissue and it is this intraneural connective tissue element that, when appropriately stimulated, can proliferate and disrupt the internal continuity of the impulse conducting nerve fibers. Resultant intraneural fibrosis combined with extra-neural scarring, may eventually cause the nerve to swell. This swelling may propagate proximally from the point of injury or constriction and effect symptomatic neuritis. Although the gross continuity of the nerve trunk is generally maintained during operative intervention, sectioning injuries can occur. These create immediate cessation of impulse conduction and may require nerve repair if a functional deficit is to be avoided.

The key diagnostic criterion associated with localized post-incisional neuropathy of the foot or ankle is pain created by irritation of a specific nerve. The pathology may affect the entire nerve trunk or asymmetrically involve only a portion of its diameter. Sensory abnormalities tend to predominate over motor dysfunction. Pain is usually well localized over the sensory distribution of the involved nerve and typically has the nature of a sharp or burning sensation. Dysesthesia, hypesthesia, and hyperesthesia may also be present. In advanced cases, severe muscle tenderness may result in disuse atrophy and weakness. Pain caused by peripheral nerve entrapment is usually aggravated by limb motion and patient activity. Rest pain is also a frequent finding in chronic cases and may be severely debilitating.

DIAGNOSIS, HISTORY, AND PHYSICAL EXAMINATION

The actual diagnosis of post-incisional peripheral neuropathy is made following a thorough historical and physical examination of the postoperative patient. Objective evaluation centers around the area of previous surgical dissection and focuses on the sensorimotor examination. Decreased

tactile distinction over the sensory distribution of the involved nerve is an early finding. Usually, if the nerve trunk is not too deeply situated, palpation or percussion of the nerve at the suspected point of irritation can elicit pain and paresthesia. Distal radiation of pain and paresthesia along the sensory distribution of the nerve, (Tinel's sign), is usually present from the early postoperative stages. The Valleix sign or proximal radiation of pain and paresthesia along the neuraxis upon percussion at the point of nerve injury may also be present. Moreover, active or passive manipulation of the extremity may exacerbate symptomatology. Manual muscle testing is not usually very helpful unless the neuropathy is seriously advanced and muscle pain and atrophy are present.

ANCILLARY STUDIES

Electro-diagnostic assessment may be useful in certain cases of nerve entrapment following surgery. Diagnostic local steroid injection, combined with a local anesthetic agent, can be used as part of the evaluation and treatment. Immediate and dramatic resolution of symptoms following injection therapy indicates accurate localization of the nerve trunk lesion. Dramatic relief of symptomatology is usually associated with a local inflammatory process as the cause of irritation. Dramatic relief followed by recurrence of symptomatology after a period of time points toward deep diffuse scarring as the cause of nerve dysfunction.

If a significant portion of the anatomical continuity of the involved nerve trunk is preserved, the prognosis following conservative treatment of post-incisional entrapment neuropathy should be relatively good. Prognosis, however, should be guarded, and varies with patient age, extent of nerve defect, as well as the location and duration of the lesion. In general, the younger the patient, the more distal the site of injury, and the shorter the duration of symptoms; the better the prognosis.

TREATMENT

Conservative measures should initially include removal of any direct extrinsic compression or tension aggravating the nerve trunk, and the use of nonsteroidal anti-inflammatory drugs. Abnormal mechanical stress should be alleviated with the use of orthoses, careful casting or splinting, or application of a desensitizing felt or foam shield as indicated. Seven to ten days of immobilization designed to prevent any motion that may be perpetuating local inflammation and nerve irritation is often beneficial. Local infiltration of a steroid combined with a local anesthetic at the site of entrapment is very useful and is a mainstay of conservative therapy.[6]

Surgical intervention may be indicated if the pain of entrapment is disabling and fails to respond to conservative treatment, the clinical picture worsens with advancing sensory loss that threatens position sense or weightbearing sensation, or if motor weakness and atrophy develop. It is important to inform the patient that symptoms may recur or worsen and that disturbing anesthesia may result following surgery. The primary surgical goal is pain relief.

Surgical management of post-incisional entrapment neuropathy evolves primarily around external neurolysis. It is very important to localize normal nerve trunk either proximal or distal to a region of scar entrapment in order to expedite and make adequate external neurolysis possible. External neurolysis involves incising any adherent fibrous tissue and mobilizing the nerve trunk from the surrounding scar. Thorough intraoperative inspection consists of visual and palpatory examination. Typically, some form of indurated neuroma-in-continuity or a terminal bulb neuroma (amputation or stump neuroma) is identified following previous surgery. The formation of a stump neuroma is the natural response to a nerve sectioning injury and this condition is often associated with recurrent intermetatarsal neuroma. In certain instances, the nerve trunk may appear completely normal, however palpation will reveal pathological intraneural fibrosis. This fibrosis may require internal neurolysis under loupe or microscopic magnification. When neurolysis is completed and deemed adequate, it is beneficial to transpose the freed nerve trunk or stump to a nearby protected, well-vascularized, soft tissue bed.[7]

In the case of severe in-continuity nerve trunk damage or painful stump neuroma, (perhaps with a history of recurrent symptomatology following previous neurolysis) resection of the

involved portion of nerve is often the preferred method of treatment. Neurectomy of a lesion in-continuity involves freeing of the lesion from surrounding tissues and sharp sectioning of normal nerve trunk proximal and distal to the lesion. The cleanly sectioned stumps are allowed to retract back into the protective surrounding soft tissues.

When resecting a stump neuroma the lesion is dissected until normal proximal nerve trunk is identified. (Fig. 1, 2, 3) Sharp transection of the proximal nerve trunk is performed and the fresh proximal stump is allowed to retract into protective soft tissues. Using magnification, the epineurium can be sutured over the cleanly sectioned fascicles. This technique can be used to decrease the likelihood of recurrent symptomatology. The experimental use of various tissue glues such as cyanoacrylate or fibrin adhesive have also been useful in the prevention of the development of painful stump neuromas secondary to excess neurite budding.[8, 9, 10] Another useful technique in the prevention of symptomatic amputation neuroma formation is transposition of the free nerve stump into bone. Neurectomy may serve as a last resort in patients severely debilitated by recalcitrant neuritis, even in cases of recurrent tarsal tunnel syndrome.

Following surgical neurolysis, wound closure is performed in anatomical layers, typically leaving the fascia open or only partially closed if it is likely to impinge upon the healing nerve. It is preferable to relocate the involved nerve trunk away from the vicinity of primarily closed deep fascia. Meticulous hemostasis is mandatory. The patient is usually allowed to freely move the extremity to tolerance. Follow-up physical therapy may be used as indicated and repeat conduction velocity measurements may be necessary to monitor regeneration if clinical improvement is less well marked.

REFERENCES

1. Seddon HJ: A classification of nerve injuries. *Br Med J* 2: 237-239, 1942.
2. Denny-Brown D, Brenner C: Paralysis of nerve induced by direct pressure and by tourniquet. *Arch Neurol Psychiatry* 51: 1-26, 1944.
3. Gordon SL, Dunn EJ: Peroneal nerve palsy as a complication of clubfoot surgery. *Clin Orthop* 101: 229-231, 1977.

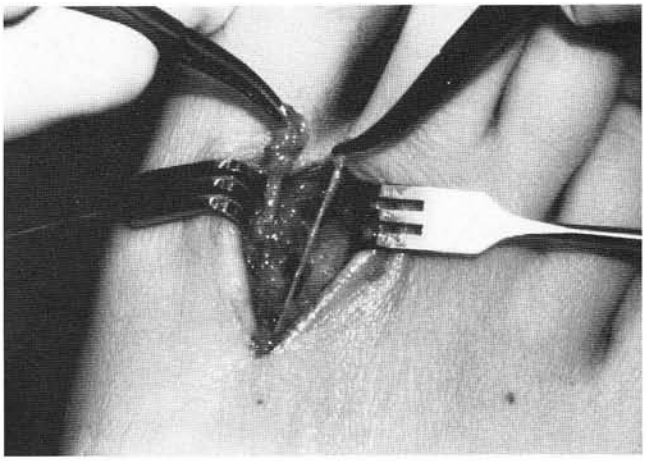

Fig. 1. Post-incisional stump neuroma of the deep peroneal nerve in the first intermetatarsal space.

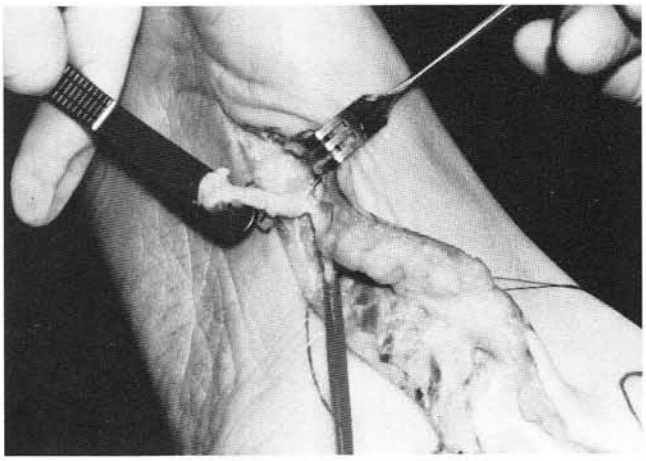

Fig. 2. Post-incisional stump neuroma of the medial plantar nerve in the plantar vault.

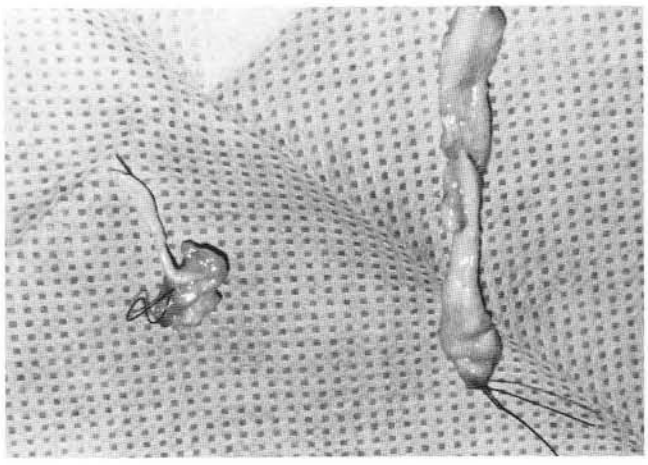

Fig. 3. Gross pathological specimens of post-incisional stump neuroma of medial plantar nerve (left) and deep peroneal nerve (right) following neurectomy.

4. Joplin RJ: The proper digital nerve, Vitallium stem arthroplasty, and some thoughts about foot surgery in general. *Clin Orthop* 76: 199-212, 1971.
5. Kenzora JE: Symptomatic incisional neuromas on the dorsum of the foot. *Foot Ankle* 5: 2-15, 1984.
6. Smith JR, Nerny HG: Local injection therapy of neuromata of the hand with triamcinolone acetonide. *J Bone Joint Surg* 52A: 71-83, 1970.
7. Mackinnon SE, Dellon AL, Hudson AR, Hunter DA: Nerve regeneration through a pseudosynovial sheath in a primate model. *Plast Reconstr Surg* 75: 833-837, 1985.
8. Martini A, Fromm B: A new operation for the prevention and treatment of amputation neuromas. *J Bone Joint Surg* 71B: 379-382, 1989.
9. Moss ALH: Ideas and innovations: the preparation of divided nerve ends. *Br J Plast Surg* 43: 247-249, 1990.
10. Narakas A: The use of fibrin glue in the repair of peripheral nerves. *Orthop Clin N Amer* 19: 187-199, 1988.