The Painful Neuroma and the Use of Conduits

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KEYWORDS

• Neuroma • Conduit • Graft • Transposition

After nerve injury, a reparative response inevitably takes place, where neurotrophic factors aid axons to regenerate from the proximal stump, and a wallerian degeneration occurs in the distal axon. Degeneration occurs also for a variable distance on the proximal axon. After the period of degeneration, myelinated and unmyelinated fibers grow from the proximal stump, trying to reinnervate the distal stump.[1] If a minimal amount of structures is left of the distal axon, namely, its endoneurial basement membrane, the regenerating fibers reach the end organ. When the complete connective tissue framework is damaged, as in a Sunderland grade IV injury, there is an exaggerated inflammatory response, with extensive deposition of collagen tissue, together with inflammatory cells, and the regenerating fibers do not reach their destination, proliferating in a chaotic manner. The resulting bulb-shaped tissue is known as a neuroma, where an unorganized network of connective tissue is intermingled with nerve fibers, Schwann cells, macrophages, fibroblasts, and myofibroblasts, the latter thought to contribute to pain causing the collagen matrix to contract around nerve fibers.[2] There is a paucity of studies dealing with neuromas in the foot and ankle, and most knowledge comes from level 4 studies, with small numbers of patients involved, and most of them come from hand surgery.

According to the anatomy of peripheral nerves, each nerve is encircled by an external epineurium. Nerve fascicles are group of nerve fibers embedded in endoneurium, correspondingly encircled by perineurium. This structure is the smallest one capable of accepting sutures.[1] Many fascicles are grouped together, surrounded by epineurium, thus forming a classic peripheral nerve. Nerve fibers occupy 25% to

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75% of the cross-sectional area of a nerve, compared with the cross-sectional area of a neuroma, which is occupied by 80% of connective tissue.[3] The most common mechanism of nerve injuries are stretch and contusive forces. Common causes include gunshot wounds, fractures, iatrogenic causes, lacerations, and so forth. Classically, nerve lesions have been classified according to Sunderland, where mild lesions with no apparent structural damage are grade I lesions, and grade IV lesions affect all component of the nerve architecture.[1] Every nerve injury, as contusion, avulsion, or direct injury, may produce a neuroma. Neuromas are classified in 3 types: neuromas in continuity, neuromas in completely severed nerves, and amputation neuromas.[2] This article focuses on neuromas in continuity and after completely severed nerves.

PATHOPHYSIOLOGY

A neuroma alters normal signal conduction through the nerves, and it affects neighboring nerves too. Normal nociceptive responses are augmented, and small stimuli generate abnormal responses, creating, for example, hyperesthesia. This hyperexcitability may be explained by an abnormal accumulation of potassium ion channels and sodium ion channels on the axons in the neuroma.[3] This hyperexcited state explains what has been called ectopic neuralgia, where a patient suffers spontaneous pain discharges without any external stimulus.[4] Locally, the disorganized connective and neural tissue, where the nerve fibers innervate scar tissue and skin, yield neuromas sensitive to mechanical stimulation.[5] This has been called nociceptive neuralgia.[4]

PATIENT EVALUATION

A complete understanding of the neural anatomy in the foot and ankle is needed to understand and interpret symptoms and clinical signs. A review of the pertinent anatomy is beyond the scope of this article, but it is recommended for understanding how to proceed with neuroma treatment. The mechanism of injury is important, because it is different when dealing with a gunshot wound or an open fracture, where nerve scarring is expected, a gradual decrease in nerve function may be observed, and a longer waiting time before intervention is warranted, compared with a clean, sharp injury where immediately afterwards a deficit in nerve function appears and a sooner operative intervention is recommended.

Pain in relation to a surgical scar which also radiates along the course of a nerve is a typical clinical sign of a neuroma. There may be a Tinel sign on top of the scar and altered sensation in the territory corresponding to that specific nerve. The authors agree with Mackinnon in that patients who are willing to palpate and massage the area of pain are not good surgical candidates. The area of discomfort should be painful to light touch, and pain proximal to the suspected area of neuroma is a frequent finding (sometimes called “Mackinnon sign”) and should be looked for.[3] The complete area and its surroundings should be inspected and palpated to find additional territories innervated by nerves which may be involved in the neuropathic pain. These additional nerves have to be addressed when performing a surgical intervention.

Imaging studies generally are not necessary, except when dealing with previous open multiple fractures or gunshot wounds, where the exact location of the injury is not completely clear. Ultrasound imaging may assist in being certain where the lesion is. An MRI can also be obtained, although its utility is best when dealing with bone or soft tissue tumors, if no history of injury is clear. A history of chronic symptoms may indicate the need for an electrodiagnostic study. This study is also useful when following the recovery of an injured nerve. Diabetes and hypothyroidism have to be