Dynamic Chiropractic

MUSCULOSKELETAL PAIN

The Tarsal Tunnel Syndrome -- A Conservative Approach to Treatment

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The tarsal tunnel syndrome is represented clinically by a constellation of symptoms which affect the foot. Most commonly, it is the result of compression neuropathy of the posterior tibial nerve at the point of its passage through the fibro-osseous tunnel beneath the flexor retinaculum on the medial aspect of the ankle. The neurovascular bundle, in which it is housed, is bound to the tibia by a ligament, which contributes to the formation of the tunnel in conjunction with the fibro-osseous tunnel. Since this neurovascular bundle occupies a relatively fixed position within the tunnel, any additional tissue accumulation in the tunnel tends to compress the nerve. Also, by passively forcing the heel and forefoot into valgus, the flexor retinaculum and point of origin of the abductor hallucis tightens, compressing the nerve. Accordingly, the process of this compression neuropathy is similar to that of carpal tunnel syndrome involving the hand.

Early in this compression process, there may be recurring episodes of transient ischemia which results in burning pain and paresthesias. With prolonged, extreme compression, nerve demyelination with Wallerian degeneration may take place with resulting numbness, muscular weakness, and atrophy. At this point, nerve conduction velocity tests demonstrate a time delay.

A common symptom is nocturnal burning pain causing the patient to awaken and results in the patient hanging the leg over the edge of the bed and shaking it, massaging the foot, or walking to gain relief. The patient may also complain of intermittent burning pain, tingling or numbness, which is located in the toes and distal components of the foot. This latter form of complaint is usually a product of prolonged standing and/or walking and is relieved by rest and shoe removal. The sensory loss finding is characteristically in the region of distribution of the medial and lateral plantar nerves. There may be diminution of two point discrimination and hypoesthesias to pin prick, with a positive (+) Tinel's sign. Not uncommonly, there is tenderness over the nerve at the tarsal tunnel, and/or the medial aspect of the foot. Good recovery should not be expected with the presence of obvious motor weakness and muscle atrophy.

Diagnostic aids to be considered by the physician should include nerve conduction velocity tests, an electromyograph, and some authors propose a tourniquet test which involves inflating a sphygmomanometer cuff proximal to the tarsal tunnel lesion to produce venous congestion and attempt to reproduce the symptoms. Of course, these procedures are requested with a neurological consult. In this author's opinion, the patient should be referred for neurosurgical consult when the neurological report involves nerve demyelinization and/or Wallerian degeneration. Prior to the presence of such findings, conservative care should be the regimen used when symptoms are of a transient character.

At the earliest presentation of clinically confirmed symptoms, the use of cryotherapy may be applied using ice water by immersion for 15 to 20 minutes. In the presence of skin pallor, continue the treatment in the absence of other contraindications, but in the event of skin cyanosis, treatment must be interrupted to avoid damage to subcutaneous tissues. It may be recalled that the reduction of skin temperature by five to 10 degrees C. is capable of reducing the nerve

conduction velocity sufficiently to relieve pain and spasm. Following this, and with future treatments as resolution of symptoms permit, application of 0.5 percent hydrocortisone and 2.5 percent lidocaine (xylocaine) ointment should be transferred by pulsed phonophoresis directly to the region of the tarsal tunnel using 0.50 W/cm2 energy for 10 minutes. Following this, if necessary to reduce the pain level, interferential current therapy may be applied to the lesion with careful attention to correct placement of the electrodes. It is recommended that the beat frequency be about 100Hz to avoid vascular ischemia, which is already a problem.

In the event that this conservative regimen does not prevent progression of this lesion to nerve demyelination and/or Wallerian degeneration, the patient must be referred for neurosurgical consultation. Clinically, this is demonstrated by the presence of numbness, muscular weakness, and atrophy; neurologically by delayed nerve conduction velocity test, and abnormal findings on electromyography.

In this author's experience, tarsal tunnel lesions which have progressed beyond the early stage in which transient symptoms are present, to a point wherein the symptoms have become marked by a history of relatively constant distress, should be referred for neurosurgical consultation.

References

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