

## THE TARSAI TUNNEL SYNDROME

CASE REPORT: POSTERIOR TIBIAL LEPROUS NEURITIS ASSOCIATED WITH TENDON SHEATH GANGLION<sup>1</sup>

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Various syndromes are related to entrapment phenomena of the extremities. The usual sites of involvement are anatomic structures such as a groove or tunnel, which predispose to compression of essential structures that course within their confines<sup>(7)</sup>. Late paralysis of the ulnar nerve from involvement within the groove behind the medial epicondyle at the elbow, and of the median nerve within the carpal tunnel, is a recognized clinical entity of the upper extremity. Similar phenomena have been reported producing a tarsal tunnel syndrome in the lower extremity<sup>(6,8)</sup>. The structures that course within the tarsal tunnel include the tendons and sheaths of the posterior tibial, flexor hallucis longus, and flexor digitorum longus muscles, the posterior tibial vessels, and the posterior tibial nerve. These adjacent structures may be separately or concomitantly affected.

Clinical manifestations are produced by compression or less frequently by traction of the structures. Both extrinsic and intrinsic factors are responsible for their development. The most common extrinsic factor is the fracture dislocation involving the fibro-osseous tunnel to produce a deformity with diminution of its diameter. Callus, scar tissue, and traumatic arthritis may initiate delayed manifestations that become evident several months later. Intrinsic factors related to the structures produce increased pressure because of the inelastic confines of the normal fibro-osseous tunnel. These factors include specific and nonspecific diseases or tumors of tendon sheaths, vessels, and nerves. The lesions may be localized or originate from systemic disease<sup>(5, 14)</sup>.

The tarsal tunnel syndrome has been recognized only recently, and as yet there is a paucity of literature regarding it. Although existence of the entity is acknowledged, the synovial, vascular, or neural origin of symptoms has not been differentiated. The tarsal tunnel syndrome is a common problem in leprosy, but it has not been sufficiently emphasized. The syndrome refers primarily to neurologic changes of insidious onset and slow progress. The most common symptom is pain, which varies in quality and intensity, but it is not always present. The discomfort may be intense or mild and unannoying. It may be aching, dull, or throbbing in character, or it may be presented as paresthesias. Swelling due to nerve enlargement from leprosy involvement is palpable proximal and posterior to the medial malleolus. Pressure on the nerve elicits localized pain and tenderness extending distally along the

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path of the nerve. Retrograde distribution of pain occurs less frequently. Associated sensory and motor changes support the concept of neural origin of these findings. Pathologic changes include acute edematous and chronic fibrous neuritis, with rare abscess formation. An increase in the intensity of pain, or failure to obtain relief from narcotics, indicates progression of nerve damage, particularly the development of nerve abscess.

Tendon sheath involvement of nonleprous origin is reported. It is usually manifest by intermittent pain related to joint movement. Generalized swelling of the ankle results from chronic nonspecific tenosynovitis with effusion<sup>(9)</sup>. Stenosing tenosynovitis rarely affects the posterior tibial tendon<sup>(3)</sup>; when it does, locking of the toes occurs late<sup>(11)</sup>. Localized swelling due to ganglion is not detectable clinically. A massive concavity of bone adjacent to and attributed to a ganglion has been reported<sup>(4)</sup>.

Vascular deficiencies are not prevalent as manifestations of the tarsal tunnel syndrome. Serious circulatory changes occur immediately in case of extensive injury to the ankle. Should late manifestations develop, they are usually generalized and vague, a fact creating controversy as to their origin, local or systemic. In late stages vascular changes produce neural ischemia.

Involvement of the peripheral nerves is emphasized as one of the predominant findings in leprosy. The pattern of nerve damage is remarkably constant, with involvement of the nerves at sites of predilection. The posterior tibial nerve is most frequently affected at the level of the ankle. Localized nerve swelling is due to infiltration by mycobacteria and to accompanying inflammatory reaction. Various theories are proposed to explain the selective involvement of nerves at specific sites. Brand<sup>(1)</sup> comments on the constancy of a feature that is common to all affected nerves, viz., "the nearness to the surface of the body." Other factors associated with the development of leprous neuritis include intrinsic anatomic features and various extrinsic factors, particularly trauma.

In review of peripheral neuropathies of the lower extremity, attention is directed to the entrapment points and to their etiologic significance<sup>(7)</sup>. Clinical manifestations occur because of dimensional restrictions due to the inelastic confines characteristic of these sites. The posterior tibial nerve is protected within the leg as it lies deep in the soleus and gastrocnemius muscles. It becomes superficial distally, to course through the tarsal tunnel located in the medial aspect of the ankle. The tarsal tunnel is bordered anteriorly by the medial malleolus and limited medially and posteriorly by the flexor retinaculum (Fig. 1). The development of leprous neuritis in relation to anatomic entrapment points has led to surgical decompression as a method of therapy. Relief from swelling and edema is obtained from neurolysis and also by the division of the flexor retinaculum ligament.

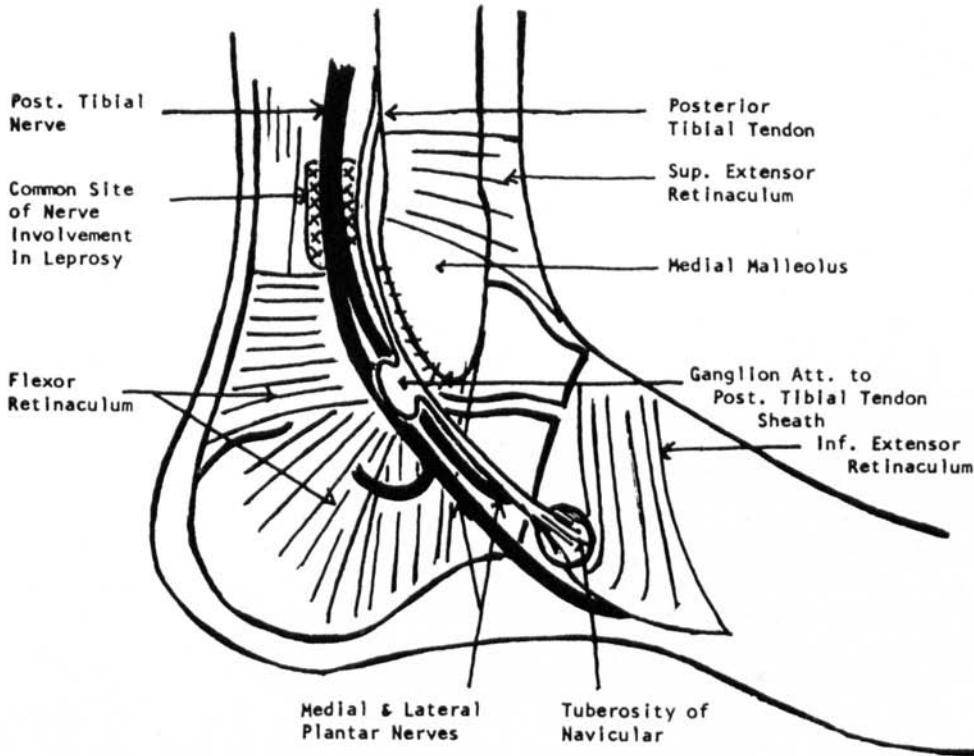


FIG. 1 Posterior tibial nerve within tarsal tunnel.

In our patient a tendon sheath ganglion was encountered, associated with posterior tibial leprosy neuritis within the tarsal tunnel. The existence of a ganglion within the tarsal tunnel may be asymptomatic. However, its presence may contribute to the initiation or aggravation of existing neuritis. In literature reviewed, carpal ganglia are reported associated with neuritic manifestations of the ulnar nerve<sup>(2, 10, 12, 13)</sup>. Cases of nonleprosy posterior tibial neuritis are reported as producing the tarsal tunnel syndrome<sup>(2, 6, 8)</sup>. In one instance there was an associated tarsal ganglion<sup>(15)</sup>. In our case a ganglion of the posterior tibial tendon sheath was encountered within the tarsal tunnel, associated with leprosy neuritis of the posterior tibial nerve.

*Case report.*—A 45 year old white female has had lepromatous leprosy for the past 24 years. The diagnosis was established during 1939, the date of her admission to this hospital. She has remained under continuous care and observation. Her progress, particularly with regard to the feet, had been uneventful until 1954, when she complained of burning and tingling sensations in both feet. Diminished sensory perception affecting both feet was discovered during reexamination in 1950. This was followed by the progressive development of calluses with ulcerations affecting the plantar aspect of both the left great toe and the right fifth metatarsal head. In the former instance healing was

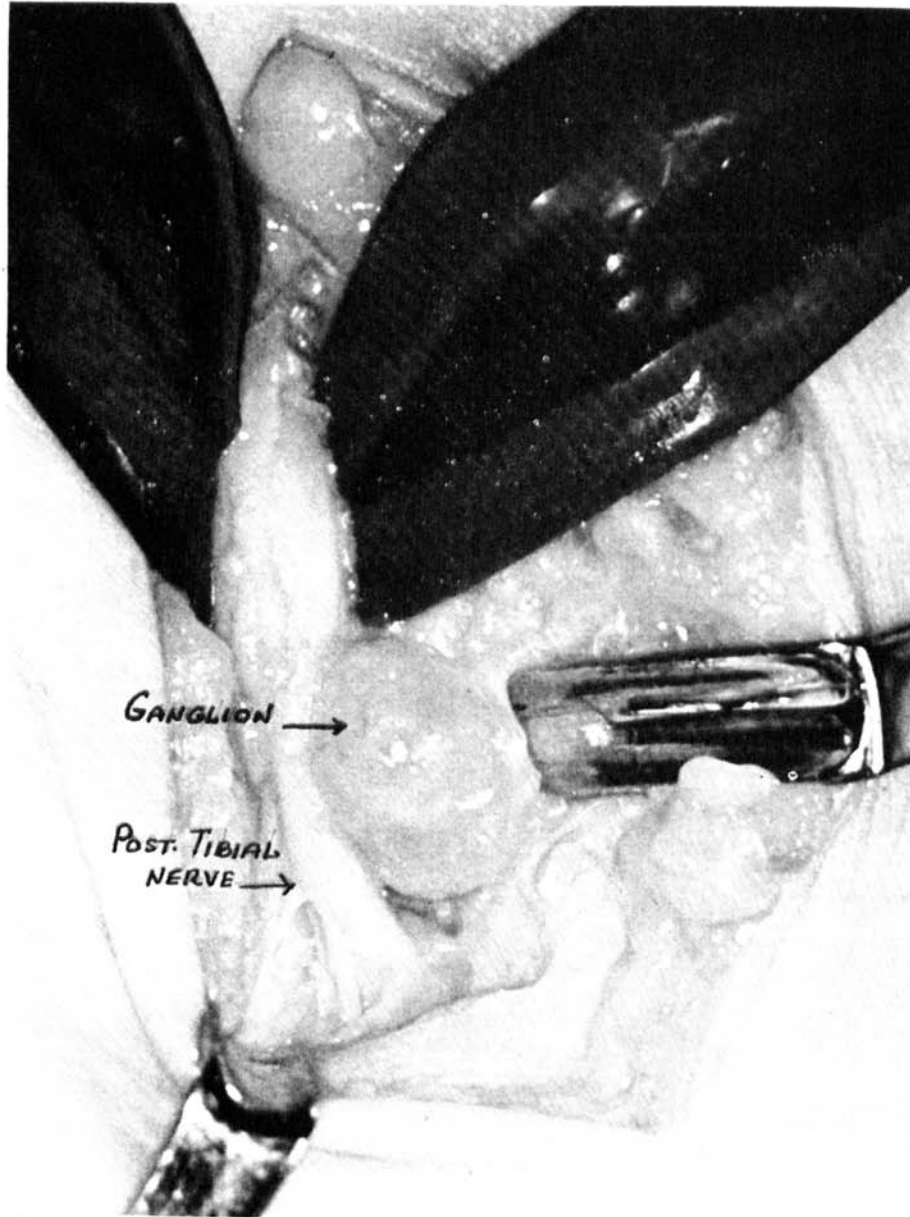


FIG. 2. Ganglion attached to posterior tibial tendon sheath, compressing posterior tibial nerve within the tarsal tunnel.

spontaneous; in the latter instance excision of the fifth metacarpal head was performed to allow healing. Burning and tingling paresthesias, with the recent addition of pain, became progressively more intense, necessitating increasing amounts of narcotics for relief. Examination revealed enlargement with localized tenderness of the posterior tibial nerves bilaterally behind the medial malleolus. The symptoms were

intensified in the peripheral distribution of the nerve by palpation at the site of involvement.

*Surgery.*—On September 24, 1963 a bilateral neurolysis of the posterior tibial nerves was performed at the ankle level. Findings indicative of neuritis were encountered on the right side. On the left side there was neuritis, and, in addition, a ganglion was found attached to the posterior tendon sheath (Figs. 2 and 3). A specimen referred for pathologic examination included the ganglion, a small nerve branch, and perineural fat.

*Pathology report.*—The nerve within perineural fat shows much atrophy, a rare bacillus, and considerable thickening of the perineurium. The fat shows no lesion. The nerve branch shows a dense fibrous mass with some clusters of bacilli within it. A smaller nerve leads away from it, showing a good many bacilli within an axon, a more acute process. The ganglion measures approximately 1.2 x 0.5 cm. At one pole of the sac a tendinous mass is attached to the wall, apparently from the insertion of some ligamentous structure. The sac is lined by a flat

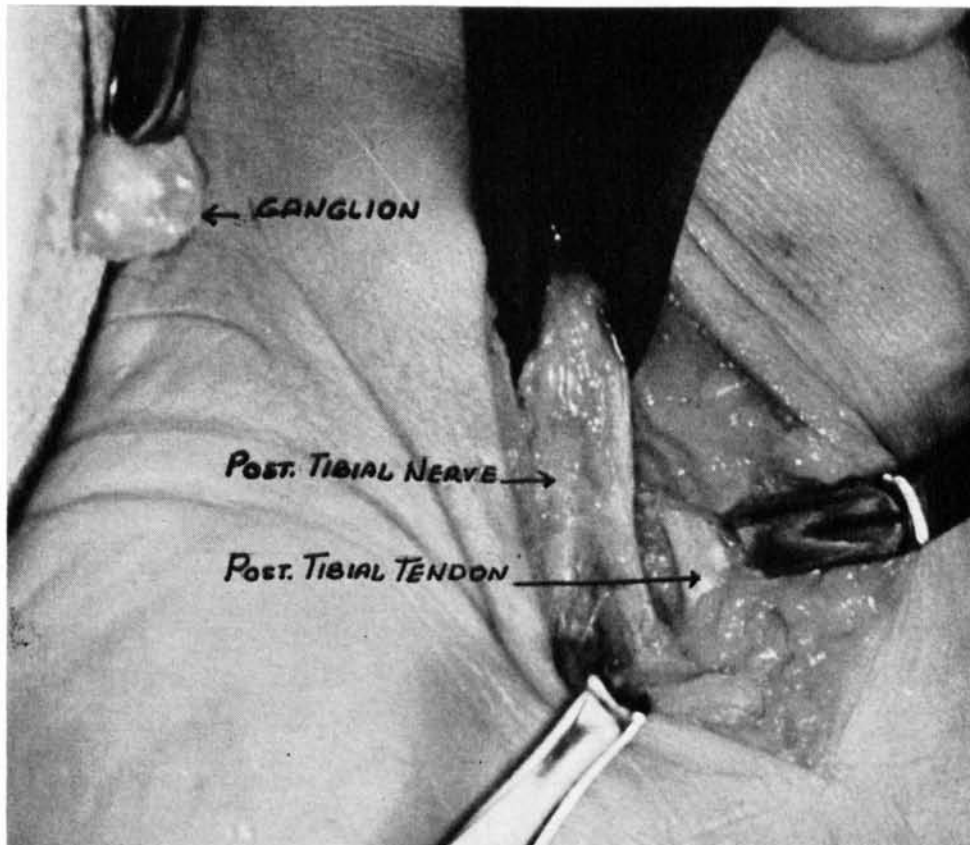


FIG. 3. The intact ganglion has been removed exposing posterior tibial tendon. Compression upon posterior tibial nerve is relieved. The beginning of the division into the medial and lateral plantar nerves is apparent.

mesothelium, and inflammatory changes are insignificant. Diagnosis: a typical ganglion derived from synovial membrane.

*Immediate progress.*—There was immediate relief of acute pain with improvement of the paresthesias, symptoms that were probably due to leprous neuritis. The sensory impairment of both feet remained unchanged.

## SUMMARY

A case report is submitted of tarsal tunnel syndrome due to leprous neuritis of the posterior tibial nerve, associated with a posterior tibial tendon sheath ganglion.

## RESUMEN

Se comunica un caso de síndrome del túnel tarsal, debido a una neuritis del nervio tibial posterior asociada con un ganglión de la vaina del tendón tibial posterior.

## RÉSUMÉ

Un rapport est présenté sur un cas de syndrome du tunnel du tarse dû à une névrite lépreuse du nerf tibial postérieur, associée avec un ganglion de la gaine tendineuse du tibial postérieur.

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