SHORT REPORT

The tarsal tunnel syndrome after a proximal lesion

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Abstract

Three patients in whom the first symptoms of the tarsal tunnel syndrome (TTS) emerged after an acute event proximal to but not affecting the ankle are described. These patients suggest that a pre-existing asymptomatic TTS may become manifest after a mechanism akin to that described in the "double crush" syndrome.

The tarsal tunnel syndrome (TTS), consisting of entrapment of the posterior tibial nerve beneath the flexor retinaculum and causing pain and paraesthesia in the foot, was first described by Lam and Keck in 1962.[1 2] The most common causes are local post-traumatic deformities, constrictive adhesions, local tenosynovitis, chronic stasis of posterior tibial veins, and other more rare causes.[3] We know of no report of TTS emerging after more proximally situated tibial nerve involvement. We describe three patients with this condition.

Case reports

Patient 1

A 63 year old man presented at the emergency department 36 hours after acute embolic occlusion of the right common femoral artery. Clinical examination showed a livid and cold leg with absent arterial pulsations. Embolectomy resulted in restoration of the circulation but he then complained of painful paraesthesia, most pronounced in the calf and the sole of the right foot. He was known to have stage II non-Hodgkin lymphoma, which had been treated with a combination of cyclophosphamide, adriamycin, vincristine, and prednisone. The lymphoproliferative disease was in a stable condition. Neurological examination showed a diminished reflex in the right ankle and diffuse hypaesthesia in both feet. Radiography of the lumbar spine showed a grade 1/4 L5–S1 spondylolisthesis, and a radiculogram with metrizamide showed minimal compression of the L5 and S1 roots on both sides.

Conduction velocities of the nerves were measured under control of skin temperature, which was kept between 33°–36° C by warming up the leg in hot water (also done for the two other patients). Low normal or slightly diminished motor nerve conduction velocities were found for the right (33 m/s) and the left (41 m/s) peroneal nerves (normal 41·65 m/s[4]) and for right and left tibial nerves (40 and 39 m/s, lowest normal value 40·63 m/s[4]). The distal motor latencies (DML) were also prolonged in the peroneal nerves (right side 5·9 ms; left side 5·1 ms; normal 4·8 ms[4]). The Hoffman reflexes elicited from the right soleus muscles were slightly slowed on both sides. Electromyography with concentric needles showed active denervation potentials in the abductor hallucis muscle and the extensor digitorum brevis muscle of the right foot. The time-related association of the vascular event and the neurological symptoms suggested aggravation of a S1-radiculopathy, pre-existing polyneuropathy, or both. Symptomatic treatment was prescribed. He was not seen again until four years later for increase of the same painful paraesthesia in the right foot. The TTS was now considered and seemed probable in view of a positive sign of Tinel at the medial ankle of the right foot and atrophy of the right abductor hallucis muscle. Neurophysiological abnormalities were similar to those found four years previously, but additional investigations were consistent with the TTS,[4] including a slowed sensory nerve conduction velocity of the right medial plantar nerve (23 m/s, normal: 27·96 m/s); this contrasted with a normal conduction velocity of the left medial plantar nerve (34 m/s). The DML in the tibial nerves were asymmetrical (right 5·1 ms, left 4·0 ms, normal 5·11 ms[4]). Division of a clearly thickened flexor retinaculum resulted in disappearance of the symptoms, with no recurrence on follow up.

Patient 2

A 57 year old man presented with symptomatic subacute obstruction of the left superficial femoral artery. A popliteal bypass with a venous graft was performed 24 hours later but resulted in pain, paraesthesia, and numbness in the sole of the left foot, which were considered as residual symptoms of limb ischaemia. Persistence of his complaints led to neurological examination, which showed a positive Tinel's sign at the medial malleolus and hypalgesia at the ventro-medial side of the sole of the left foot. Results of nerve conduction studies and electromyography were consistent with the clinical diagnosis of TTS: the DML derived from the abductor hallucis muscle (6·8 ms; normal 5·11 ms) was prolonged, which contrasted with a normal DML derived from the gastrocnemius muscle (5·0 ms; normal 5·3 ms). There was also denervation activity in the left abductor hallucis and a complete sensory conduction block of the left medial plantar nerve

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over the flexor retinaculum. Surgical decompression of the tibial nerve at the ankle resulted in complete and persistent improvement.

Patient 3

A 38 year old football player was admitted with a right sided fracture at the distal third of the tibia and fibula. No evidence was detected of a second lesion near the medial malleolus. The fracture was treated with repositioning, traction, and immobilisation. Two weeks later the patient experienced increasing pain and numbness on the medial side of the right foot. Neurological signs consisted of impaired plantar flexion of the third, fourth, and fifth toes, hypalgesia in the sole of the foot, and diffuse hypaesthesia in the tibial and peroneal nerves distal to the fracture. Percussion of the calf at the site of the fracture elicited paraesthesia in the sole of the foot. Motor nerve conduction studies showed a normal conduction of the right tibial nerve to the gastrocnemius muscle (DML 3·6 ms; normal 5·3 ms) and a distal conduction block to the abductor hallucis muscle. A total conduction block of the medial plantar nerve was present on the right side. Electromyography showed active denervation potentials in the abductor hallucis muscle. A tibial nerve lesion or entrapment at the site of the fracture was suspected. Surgical exploration and neurolysis of a slightly entrapped tibial nerve were carried out but did not result in neurological improvement. Six months later TTS was considered; this was confirmed by clinical re-evaluation and neurophysiological data, which were the same as six months previously. At surgical intervention distinct fibrosis of the flexor retinaculum and a severely compressed and atrophic tibial nerve were found. Neurolysis led to decreased pain and numbness in the sole of the foot.

Discussion

Our three patients were characterised by the onset or aggravation of neurological symptoms in one foot after an event in the leg, proximal to the tarsal tunnel. Although restoration of nerve function was not documented by postoperative neurophysiological reassessment, TTS seemed most probable in view of the persistent improvement after tarsal tunnel decompression in all patients.

In patient 1 aggravation of the radiculopathy or the polyneuropathy after the vascular operation was suspected. In patient 3 damage or compression of the tibial nerve at the site of the trauma seemed the most probable cause. Consequently the diagnosis of TTS was considerably delayed in these two patients. We cannot totally exclude alternative mechanisms explaining the rapid onset of TTS; there may have been prolonged compression of the medial ankle during the operation but this was technically improbable. A concomitant traumatic lesion to the ankle in patient 3 was conceivable, but this was neither clinically nor radiologically apparent. The most plausible aetiology is that an ischaemic (patients 1 and 2) or traumatic (patient 3) proximal nerve damage may have resulted in "decompensation" of a pre-existing asymptomatic TTS, via a mechanism similar to that described by Fullerton in CTS[5]: she compared normal subjects and patients with CTS and found a greater susceptibility to ischaemia in patients with CTS, with early failure of motor nerve conduction. Later, Upton and McComas described a series of patients in whom the median or ulnar nerves were compressed at two different sites and proposed their so-called "double crush theory" to explain that the occurrence of symptoms was facilitated by a concomitant cervical radiculopathy.[6] The proximal compression would decrease the resistance of the nerve to a second, more distal entrapment. This would facilitate the occurrence of the carpal tunnel syndrome or an ulnar nerve compression neuropathy.

Experimental studies have added further arguments to the double crush theory. Shimpo *et al* tied silk ligatures round the sciatic nerve of guinea pigs to produce permanent nerve constriction.[7] They found that local plantar nerve lesions developed in the sole of the foot of the affected side, possibly due to the pressure from the floor of the cage. They suggested an increased sensitivity to local pressure occurred in atrophic nerve fibres distal to the constriction. Nemoto *et al* found that the loss of nerve function after a double lesion in sciatic nerves of dogs was greater than the sum of the deficits after each separate lesion.[8] They concluded that proximal compression of a nerve could lessen its ability to withstand further compression more distally.

In patients who have suffered severe and prolonged limb ischaemia one of the mechanisms leading to a double crush phenomenon might be that axonal anoxia and subsequent deficient oxidative phosphorylation result in disturbed axoplasm transport, which is closely dependent on oxidative metabolism.[9] This might lead to an increased susceptibility of the distal axon to further ischaemic or compressive damage.[7 9] In two of our patients prolonged ischaemia of the leg probably resulted in ischaemic nerve damage,[10] as has been previously described by Welti *et al*[11] and Ferguson and Liversedge.[12] These authors described selective peroneal palsies after acute limb ischaemia, attributing this ischaemic mononeuropathy to an increased susceptibility of the nerve after a relatively poor blood supply in the region of the fibular head. We could trace no more reports on TTS emerging after a second more proximal lesion so further studies are needed to confirm the theory that some cases of TTS may be due to a "double lesion neuropathy", a term that seems more appropriate than "double crush syndrome",[11] as in most cases nerve damage is not due to crushing.

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1 Lam SJS. A tarsal tunnel syndrome. *Lancet* 1962;ii:1354–5.
2 Keck C. The tarsal tunnel syndrome. *J Bone Joint Surg* 1962;44A:180–2.
3 Edwards WG, Lincoln CR, Bassett FH, Goldner JL. The tarsal tunnel syndrome: diagnosis and treatment. *JAMA* 1969;207:716–20.

4 Oh SJ. Tarsal tunnel syndrome. In: *Clinical electromyography: nerve conduction studies*. Baltimore: University Park Press, 1984:408–10.
5 Fullerton MP. Effect of ischaemia on nerve conduction in carpal tunnel syndrome. *J Neurol Neurosurg Psychiatry* 1963;26:385–97.
6 Upton ARM, McComas AJ. The double crush in nerve-entrapment syndromes. *Lancet* 1973;ii:359–61.
7 Shimpo T, Gilliatt RW, Kennett RP, Allen PJ. Susceptibility to pressure neuropathy distal to a constricting ligature in the guinea-pig. *J Neurol Neurosurg Psychiatry* 1987;50:1625–32.
8 Nemoto K, Matsumoto N, Tazaki K, Horiuchi Y, Uchinishi K, Mori Y. An experimental study on the "double crush" hypothesis. *J Hand Surg* 1987;12A:552–9.
9 Ochs S. Basic properties of axoplasmic transport. In: *Peripheral neuropathy*. Dyck PJ, Thomas PK, Lambert EH, Bunge R, eds. Philadelphia: Saunders, 1984:453–76.
10 Daube JR, Dyck PJ. Neuropathy due to peripheral vascular diseases. In: *Peripheral neuropathy*. Dyck PJ, Thomas PK, Lambert EH, Bunge R, eds. Philadelphia: Saunders, 1984;1459–78.
11 Welti JJ, Melekian B, Réveillaud M. Paralysies périphériques ischémiques. *Pr Med* 1961;69:333–4.
12 Ferguson FR, Liversedge LA. Ischaemic lateral popliteal nerve palsy. *Br Med J* 1954;2:333–5.