

Short report

The tarsal tunnel syndrome in hypothyroidism

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SUMMARY Nine patients with myxoedema and carpal tunnel syndrome have been studied clinically and electrophysiologically to determine the presence or absence of the tarsal tunnel syndrome. Four patients had electrophysiological evidence of the tarsal tunnel syndrome, three of whom were mildly symptomatic. This would suggest that the tarsal tunnel syndrome is frequently encountered in myxoedema in association with the carpal tunnel syndrome.

The neurological complications of hypothyroidism include the carpal tunnel syndrome, a myopathy, neuropathy, cerebellar syndrome, dementia, psychosis and coma.¹ The clinical diagnosis of a carpal tunnel syndrome which is often bilateral may be confirmed by electrodiagnostic studies. Since the median nerve is often vulnerable to compression within the carpal tunnel by the flexor retinaculum in hypothyroidism, we were interested to determine whether a similar compression occurred of the posterior tibial nerve under the flexor retinaculum on the medial aspect of the ankle in hypothyroidism, producing the clinical and electrophysiological features of the tarsal tunnel syndrome.²

Patients and methods

We have studied nine consecutive patients with primary hypothyroidism who were referred for electrodiagnostic studies with a clinical diagnosis of possible carpal tunnel syndrome (table 1). Six of these patients were untreated and three had recently begun treatment with l-thyroxine. All patients had classical symptoms and signs of unilateral or bilateral carpal tunnel syndrome. Three of the patients had unilateral symptoms of the tarsal tunnel syndrome, characterised by tingling on the medial half of the sole and the heel of the foot. Their foot symptoms were aggravated by walking but were not worse at night in bed. On examination pain sensation was decreased in the distribution of the medial plantar nerve. One patient had local tenderness below the medial malleolus. All the patients had motor and sensory conduction studies performed in the upper and lower limbs. In the median nerve the distal motor latency from the wrist to abductor pollicis brevis and F responses were determined bilaterally using surface electrodes. Simi-

lar studies were carried out bilaterally in the posterior tibial nerve, stimulating at the ankle and recording with surface electrodes from the abductor hallucis. The normal distal motor latency for the median nerve was taken as less than 4.3 milliseconds, and for the posterior tibial nerve as less than 6.5 milliseconds. Sensory conduction studies were performed in both median nerves, but in most patients similar studies could not be carried out in the medial plantar nerve because of ankle and foot oedema. Eight of the patients had thyroid function tests performed at the time of the electrodiagnostic study and one a month earlier.

Results

All the patients had thyroid function tests performed at the time of the electrodiagnostic study and one a month earlier. The clinical details, biochemical parameters, and electrophysiological results on the nine patients studied are shown in tables 1 and 2. All the patients had symptoms and signs of either a unilateral or bilateral carpal tunnel syndrome. Three patients had unilateral symptoms and signs of the tarsal tunnel syndrome (patients 2, 5 and 9). All these symptoms were mild and less distressing to the patient than those in the hand.

All the patients had electrophysiological evidence of carpal tunnel syndrome, five of which were bilateral. Patient 8 had an abnormal median sensory potential in one hand with normal distal motor latencies bilaterally. Four patients had electrophysiological evidence of a bilateral tarsal tunnel syndrome (patients 2, 5, 7, and 9). All the patients with clinical symptoms of the tarsal tunnel syndrome had electrophysiological evidence of that condition. All the patients had normal F responses in the upper and lower limbs.

The two patients with the most severe electrophysiological evidence of the tarsal tunnel syndrome also had the most prominent carpal tunnel syndrome (patients 5 & 7). The most prominent

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Table 1 Clinical and biochemical data on the nine patients with primary hypothyroidism.

| Patient No | Age (yr) | Sex | Length of history (months) | T4 (nmol/l) (NR 58-140) | TSH (mu/l) (NR <7) | ESR (mm/hr) | Antibodies to: | |
|------------|----------|-----|----------------------------|-------------------------|--------------------|-------------|--------------------|---------------|
| | | | | | | | Thyroid microsomes | Thyroglobulin |
| 1 | 65 | F | 3 | <15 | 99.4 | 50 | +ve | -ve |
| 2 | 63 | F | 3 | <15 | 53 | 47 | - | - |
| 3 | 50 | F | 24 | <15 | 106 | - | +ve | +ve |
| 4 | 62 | F | 36 | 45* | 6.9 | - | - | - |
| 5 | 70 | F | 1 | <15 | - | - | +ve | +ve |
| 6 | 72 | F | 24 | <15 | > 60 | - | +ve | -ve |
| 7 | 73 | F | 6 | <15 | 25 | 88 | +ve | +ve |
| 8 | 50 | F | 18 | <15† | 95† | 33 | -ve | +ve |
| 9 | 59 | F | 2 | 52‡ | -‡ | 39 | +ve | -ve |

*NR 50-140 nmol/l.

†Measurement made one month before electrophysiological study (prior to commencement of L-Thyroxine therapy).

‡Two months before study (prior to commencement of L-Thyroxine therapy): T4 < 10 nmol/l; TSH = 45.2 mu/l.

Table 2 Electrophysiological data on the nine patients with primary hypothyroidism

| Patient No | Distal latencies (ms) | | | |
|------------|---------------------------|------|-------------------------------------|-------|
| | Median nerve (N < 4.3 ms) | | Medial popliteal nerve (N < 6.5 ms) | |
| | Right | Left | Right | Left |
| 1 | 5.3 | 6.1 | 3.9 | 3.9 |
| 2 | 5.8 | 6.0 | 7.2 | 7.8* |
| 3 | 5.2 | 4.2 | 5.8 | 5.6 |
| 4 | 5.6 | 5.5 | 4.2 | 4.6 |
| 5 | 6.2 | 8.4 | 7.2 | 10.5* |
| 6 | 3.9 | 4.7 | 5.5 | 5.0 |
| 7 | 15.1 | 9.4 | 8.0 | 7.9 |
| 8 | 3.6 | 3.4 | 5.1 | 5.0 |
| 9 | 4.0 | 4.7 | 6.7 | 10.7* |

*Symptomatic

electrophysiological abnormalities of the carpal and tarsal tunnel syndrome were found in those patients with a short clinical history (less than 6 months.)

Discussion

The tarsal tunnel syndrome is characterised by intermittent burning pain, tingling, and numbness of the feet, usually aggravated by prolonged standing and walking long distances.^{3,4} Some patients also have symptoms while lying in bed. The sensory symptoms can affect either the medial or lateral part of the sole of the foot, but occasionally only the heel is involved. On examination there may be a positive Tinel's sign on percussion just below the medial malleolus with diminished pain and light touch sensitivity on the sole of the foot on its medial and lateral aspects. There may be some atrophy of the abductor hallucis with no apparent weakness. The electrical parameters for the diagnosis include the distal motor latency from the ankle to abductor hallucis,^{2,5} the abductor digiti minimi, and measurement of sensory potentials of the medial and lateral plantar branch of the posterior tibial nerve.^{6,7} Denervation of the abductor hallucis muscle can also be assessed. Using

all three parameters, the diagnostic yield can be increased.⁸ The diagnosis has to be distinguished from an S1 root lesion, which can be determined electrophysiologically by a delayed F response.

The known causes of the tarsal tunnel syndrome include post-traumatic fibrosis in the region of the tarsal tunnel, hypertrophy of the abductor hallucis muscle, tenosynovitis, a ganglion, ankylosing spondylitis, rheumatoid arthritis,¹⁰ a neurolemmoma, diabetes mellitus, and leprosy.¹¹ There have been occasional reports of patients having both the carpal tunnel syndrome and tarsal tunnel syndrome.^{4,12-14} The carpal tunnel syndrome is often bilateral and a common finding in myxoedema, but the tarsal tunnel syndrome has only rarely been recognised in this condition.

Murray and Simpson¹⁵ described 26 of 35 patients with paraesthesae of the fingers and myxoedema, of which only two had tingling in the toes, but these patients were not investigated for possible tarsal tunnel syndrome. Linscheid *et al*,⁴ described 34 patients with tarsal tunnel syndrome one of whom had myxoedema and a bilateral carpal tunnel syndrome. Torres and Moxley¹⁶ produced evidence in a single patient with severe hypothyroidism of carpal

and tarsal tunnel syndrome which slowly improved over 3 years with replacement therapy.

In the electrophysiological evaluation of the tarsal tunnel syndrome the distal motor latency of the tibial nerve from medial malleolus to abductor hallucis may be increased. Kaeser² found an increased distal latency in 40% of his cases. Measurement of sensory potentials in the medial plantar nerve increases the diagnostic yield.⁷ Because many of our patients had marked ankle swelling measurement of the sensory potentials with surface electrodes was not possible. As a result the number of patients with physiological evidence of the tarsal tunnel syndrome may have been underestimated. This study has demonstrated that the tarsal tunnel syndrome is a common finding in myxoedema, and is occasionally symptomatic, but is less frequently encountered than the carpal tunnel syndrome.

References

- ¹ McKeran RO, Slavin G, Ward P *et al.* Hypothyroid myopathy. A clinical and pathological study. *J Pathol* 1980;**132**:35–54.
- ² Kaeser HE. Nerve conduction velocity measurements. In: Vinken PJ, Bruyn GW, *et al.* *Handbook of Clinical Neurology*. Amsterdam: North Holland 1970:Vol. 7, 164.
- ³ Edwards WG, Lincoln CR, Bassett FH. III, Goldner JL. The tarsal tunnel syndrome. *JAMA* 1969;**207**:716–9.
- ⁴ Linscheid RL, Burton RC, Frederichs EJ. Tarsal tunnel syndrome. *South Med J* 1970;**63**:1313–23.
- ⁵ Goodgold J, Rapell HP, Spielhal MI. The tarsal tunnel syndrome. *N Engl J Med* 1965;**273**:742–5.
- ⁶ Oh SJ, Sarala PK, Kuba T, Elmore RS. Sensory nerve conduction velocity of the plantar nerves: a superior objective diagnostic test for tarsal tunnel syndrome. *Trans Am Neurol Assoc* 1978;**103**:256–8.
- ⁷ Oh SJ, Sarala PK, Kuba T, Elmore RS. Tarsal tunnel syndrome: electrophysiological study. *Ann Neurol* 1979;**5**:327–330.
- ⁸ Ludin HP. *Electromyography In Practice*. Stuttgart: Thieme Verlag 1980;88–89.
- ⁹ Di Stefano V, Sack JT, Whittaker R, Nixon JE. Tarsal tunnel syndrome. *Clin Orth Rel Res* 1972;**88**:76–9.
- ¹⁰ Baylan SP, Paik SW, Barnett AL *et al.* Prevalence of the tarsal tunnel syndrome in rheumatoid arthritis. *Rheumatol Rehabil* 1981;**20**:148–50.
- ¹¹ Bourel P, Reg A, Blanc JF *et al.* Syndrome du canal tarsien. *Rev Rheumatol* 1976;**43**:723–8.
- ¹² McGill DA. Tarsal tunnel syndrome. *Proc R Soc Med* 1964;**57**:1125–6.
- ¹³ Paterson DC. Bilateral carpal and tarsal tunnel syndrome. *Med J Austr* 1966;**421**:1–2.
- ¹⁴ Lam SJS. Tarsal tunnel syndrome. *J Bone Joint Surg* 1967;**49B**:87–93.
- ¹⁵ Murray IPC, Simpson JA. Acroparaesthesia in myxoedema a clinical and electromyographical study. *Lancet* 1958;**1**:1360–63.
- ¹⁶ Torres CF, Moxley RT. Clinical and electrophysiological changes in nerve hypothyroidism. Follow up with thyroid replacement alone. Fifth International Congress on Muscle disease Marseille, 1982. (published only at the meeting)