Torsion dystonias have been reported in association with a wide variety of hereditary and degenerative neurological syndromes as well as vascular, traumatic, and toxic brain disorders. The significance of oligoclonal IgG bands in this case is uncertain but if confirmed in other patients would suggest either immune or virally mediated CNS damage as the pathophysiological mechanism in at least some cases of torsion dystonia.

References

Accepted 22 March 1988

Torsal tunnel syndrome secondary to intraneural ganglion

Sir: A 61 year old healthy man complained of burning pains and paraesthesiae, which had been present for 7 months in the toes and along the sole of his left foot. Discomfort was worse at night, and sometimes radiated proximally along the medial aspect of the calf. There was no history of trauma about the ankle. On examination, there was a decrease in two-point discrimination and hypesthesia to pin-prick in the distribution of the medial and lateral plantar nerves. There was no obvious wasting or weakness of the small foot muscles. Tinel's sign was positive in the posterior tibial nerve at the ankle. No mass was palpable along the medial aspect of the ankle. General neurological examination was normal. Radiographs of the left ankle and foot were normal. Electrophysiological tests were performed bilaterally.

EMG studies did not show any spontaneous activity in the small foot muscles. Distal motor latency (ankle to abductor hallucis) was 7.28 ms on the left and 4.21 ms on the right (normal values: 2.7-5.1 ms) with a small compound muscle action potential on the left. The left medial plantar sensory action potential (SAP) was absent; the right SAP was 0.7 μV of amplitude (normal values: 0.1-3 μV), with a sensory conduction velocity of 42 m/s (normal values: 36-50 m/s). The patient underwent surgery. A curved incision was made superior, posterior, and inferior to the medial malleolus. The neurovascular bundle was isolated just proximal to the tarsal tunnel, which was entered by dividing the flexor retinaculum. A calcaneal branch, and the medial and lateral plantar nerves were dissected free. Within the tarsal tunnel, the posterior tibial nerve showed an eccentric, translucent, rubbery, bean-sized swelling. The epineurium was incised longitudinally, and clear gelatinous material evacuated (fig). As the swelling collapsed, a small fusiform cavity, about 2 cm long, with a firm, whitish wall was found within the nerve. Nerve bundles could not be dissected away from the cyst wall. No pedicle was found connecting the cyst to nearby tendon sheaths or joints. The cavity was gently irrigated. A fragment of the cyst wall was taken for biopsy. Microscopically it was composed of lamellar connective tissue with scanty cells. The cyst contained mucoid material.

Symptoms and signs rapidly disappeared following surgery. When seen two years later, the patient did not complain of any symptom, and the neurological examination did not show any abnormality.

Cystic intraneurral tumours filled with gelatinous material and referred to as "ganglia" are rare. Their pathogenesis and patho-

logy have been discussed by Sherman et al.2
Letters

Fig. Artist's drawing showing the intraneural ganglion (white arrowhead) of the posterior tibial nerve just proximal to its division into medial and lateral planter nerves. The flexor retinaculum has been divided, and its edges (black arrowheads) retracted. (1) posterior tibial nerve. (2) posterior tibial artery. (3) medial planter nerve. (4) lateral planter nerve. (5) calcaneal branch piercing the flexor retinaculum.

Descriptions of intraneural ganglia have almost all been of the peroneal nerve, although involvement of other nerves has occasionally been reported. In a review of the English literature concerning intraneural ganglia, we found less than 50 cases; the posterior tibial nerve was involved twice in the popliteal fossa, but never at the ankle. On the other hand, in a review of the literature concerning the causative lesions of the tarsal tunnel syndrome, Matricali in 1980 found only four ganglia plus one personal observation; none was intraneurally located. To the best of our knowledge, the only instance of intraneural ganglion of the posterior tibial nerve at the ankle has been described by Loeffler and Volkmann in 1920. Intraneural ganglia will appear grossly as a dilatation of the nerve trunk. With longitudinal neurotomy, the cyst can be entered and its gelatinous material evacuated by gentle irrigation. Most authors agree that the destruction of nerve tissue to allow excision of the cyst wall is not advisable and even if the cyst wall is left behind, recurrence is rare. Surgical results are generally good, as in the present case.

M Poppi
G Giuliani
E Pozzati
N Acciarri
A Forti

From the Division of Neurosurgery, Ospedale Bellaria and the Department of Neurology, University of Bologna, Bologna, Italy

Address for correspondence: Dr Massimo Poppi, Division of Neurosurgery, Ospedale Bellaria, via Altura 3, 40139, Bologna, Italy.

References


Accepted 3 March 1989

Neopterin: biopterin ratios in Down's syndrome

Sir: Urinary neopterin:biopterin ratios are elevated in senile dementia of the Alzheimer type (SDAT) compared with age matched controls, due to a reduced conversion of dihydronopterin triphosphate to tetrahydrobiopterin. There is a similar elevated neopterin:biopterin ratio in Down's syndrome, a condition producing a similar dementia to SDAT.

Morning urines were collected into ascobic acid (2% final concentration) from a group of 53 Down's sufferers of mixed sex aged between 1 and 70 years without any other disease and, following storage at -20°C, the neopterin and biopterin levels were determined by HPLC after acid iodine oxidation. Compared with a group of 35 healthy controls of mixed sex and aged 23 to 93 years, the neopterin:biopterin ratio was significantly elevated 3.44, 1.73 vs 1.12, 0.55 (mean, SD) p < 0.2% (Table). Against a creatinine baseline biopterin levels were unaffected but the urinary neopterin levels were significantly increased (Table). No age trend was observed suggesting that these metabolic effects precede dementia onset. The elevated neopterin:biopterin ratio could be due to a disease related immune response causing stimulation of neopterin biosynthesis, although all disease states known to produce such were absent and the rise is less pronounced in those conditions. It is known that there are disturbances in the immune system of Down's patients, and it could be suggested that these disturbances are causing the rise in urinary neopterin in a similar manner to viral and malignant disorders. However, it has been found that interferon gamma (IFNg) production is lower in Down's patients than in normal patients with infection. It is IFNg which stimulates the macrophages to produce neopterin in infectious disorders, so any decrease in IFNg levels should result in similar or reduced levels of urinary neopterin and not the increased levels observed. A second possibility is the presence of greater levels of guanosine triphosphate in the purine pool from an increased biosynthesis of purines as a result of the extra chromosome 21, which has the locus for some of the enzymes for purine biosynthesis.

The levels of neopterin and biopterin are measured against a creatinine baseline. In the Down's subjects creatinine clearance is reportedly lower, reflecting a reduced glomerular filtration rate, and this could be causing a distortion in the results. However, creatinine clearance has a positive linear correlation with both neopterin and biopterin so any
Tarsal tunnel syndrome secondary to intraneural ganglion.
M Poppi, G Giuliani, E Pozzati, N Acciarri and A Forti

J Neurol Neurosurg Psychiatry 1989 52: 1014-1015
doi: 10.1136/jnnp.52.8.1014

Updated information and services can be found at:
http://jnnp.bmj.com/content/52/8/1014.citation

These include:

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/