Migrant sensory neuritis of Wartenberg

Sir: We enjoyed the timely report by Matthews and Esiri in this journal. We agree that this entity is more common than appreciated. Diagnosis of this benign condition is easy when a patient presents with symptoms of several years' duration. Difficulties arise when one encounters a patient early in the course of the disease.

A 42-year-old woman initially experienced constant tingling and pain in both hands which were worse at night and frequently awoke her from sleep. Three weeks after onset of the hand symptoms, she developed painful numbness of both cheeks and the right forehead. Several weeks later tingling and pain began in her feet and the lower posterior aspect of her right thoracic paravertebral area. The abnormal sensation on the trunk cleared within two months. Four months after the onset of her symptoms neurologic examination revealed the following abnormalities: sensation was decreased to pin and touch in the distribution of both sural nerves distal to the ankle and there were patchy areas of decreased sensation in the trigeminal nerve distribution bilaterally. Sensation in the fingertips and hands was normal. Tinel's sign could be easily elicited by light percussion of the median nerves at the wrist level, the sural nerves behind the lateral ankle, and the right supraorbital nerve. Dorsiflexion of the wrist and supination movements of the ankle resulted in an increase in paresthesias and lancinating pain. The remainder of the neurologic examination was normal; in particular there was no muscle atrophy or weakness and the tendon reflexes were brisk and symmetrical with downgoing plantar responses. Vibration and position sense were normal. The following laboratory studies were normal: CBC, chemistry profiles, ESR (on three occasions), serum protein electrophoresis, serum immunoglobulins, urinalysis, RA factor, thyroid profile, cryoglobulins, CSF examination, visual evoked responses. A nerve biopsy was not performed.

Abnormalities on nerve conduction studies consisted of moderate severe prolongation of distal motor latencies for one ulnar, both median and both peroneal nerves. Motor nerve conduction velocities for the proximal segments of these nerves including F-wave latencies were all normal. The radial nerve sensory latencies were normal. Median, ulnar and sural nerve action potentials were variably decreased in amplitude but all of normal latency. Needle electrode examination of proximal and distal limb muscles and the lumbar sacral paraspinal muscles was normal.

During three months of follow-up painful sensory symptoms have decreased considerably in the hands and feet, but her facial "pain" has increased and did not respond to carbamazepine 600 mg per day.

Our patient has signs and symptoms referable to sensory nerves or sensory nerve branches on trunk, hands, feet and face. Symptoms were migratory and fluctuating, but severe enough to interfere with work and social function. The presence of Tinel's signs suggested multiple compression neuropathies at common areas. Generalised increase in motor latencies, however, is consistent with acquired generalised demyelinating polyneuropathy. Moreover, the combination of prolonged distal motor latencies and the presence of normal latencies for the sensory nerve action potentials of the median nerve are against an "ordinary" compression neuropathy.

We are clinically similar to those reported by Matthews and Esiri. Nerve conduction abnormalities, however, suggest that our patient may have a different disease with an uncertain prognosis. We strongly recommend serial nerve conduction studies in patients with migratory sensory symptoms during prolonged follow-up. This may not aid in differential diagnosis and demonstration of objective abnormalities but may lead to better classification of these patients into different disease groups with different prognosis and need for diagnostic work-up.

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References

Matthews and Esiri reply:

We were interested to read the case report and physiological findings described by Streib and Sun. We think that their report emphasises that there are probably a number of different forms of wholly or predominantly sensory mononeuritis, as the clinical features in their case are, we believe, distinct from those we reported. Wartenberg1 certainly described patients with areas of sensory loss on the trunk and Schulz2 reported hyperalgesia of the face. In these cases and that of Streib and Sun the characteristic onset of symptoms with stretch was not present. This is easily distinguishable from painful paraesthesiae induced by stretch in areas already involved by neuropathy. In our case 1 normal motor and sensory conduction has been preserved for more than 30 years since the onset of symptoms and the benign nature of the condition can scarcely be in doubt.

Table Results of nerve conduction studies

<table>
<thead>
<tr>
<th>Motor nerve conduction</th>
<th>Sensory nerve action potentials</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nerve</td>
<td>Amplitude</td>
</tr>
<tr>
<td>R ulnar</td>
<td>5-6 (6-16)</td>
</tr>
<tr>
<td>L ulnar</td>
<td>5-2 (6-16)</td>
</tr>
<tr>
<td>R median</td>
<td>3-6 (5-18)</td>
</tr>
<tr>
<td>L median</td>
<td>4-0 (4-18)</td>
</tr>
<tr>
<td>R peroneal</td>
<td>3-6 (2-12)</td>
</tr>
<tr>
<td>L peroneal</td>
<td>2-2 (2-12)</td>
</tr>
<tr>
<td>R posterior tibial</td>
<td>3-8 (3)</td>
</tr>
<tr>
<td>R sural</td>
<td>—</td>
</tr>
<tr>
<td>L sural</td>
<td>—</td>
</tr>
<tr>
<td>R radial</td>
<td>—</td>
</tr>
<tr>
<td>L radial</td>
<td>—</td>
</tr>
</tbody>
</table>

CV = conduction velocity; DL = Distal motor latency; Latency = latency to peak of distal NAP.
Skin temperature of palm 34°C and dorsum of foot 32°C.
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E Streib and S F Sun

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