Bilateral neuralgic amyotrophy complicating Weil’s disease

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Summary
The case of a patient with leptospirosis (treated with trimethoprim) with late neurological complications manifesting as a bilateral plexus syndrome is described. The probable reasons for the continued weakness, in this patient, of the muscles supplied by the anterior interosseus nerve, despite improvement in the proximal muscles, are briefly discussed.

Introduction
Neurological complications are rarely seen during the course of Weil’s disease (Edwards and Domm, 1960). The authors now report on the occurrence of bilateral asymmetric neuralgic amyotrophy in a patient suffering from leptospirosis.

Case report
A 48-year-old farmer was admitted to hospital with a 2-day history of malaise, headache and fever. A diagnosis of probable viral infection was made and he was treated with trimethoprim (Septrin) tablets 2 twice/day. Three days following admission he became jaundiced (bilirubin 240 μmol/l) and developed renal failure (urea 21.6 mmol/l; creatinine 957 μmol/l). He was treated with haemodialysis which was continued to the twelfth day and during this period his liver function improved (bilirubin 90 μmol/l) simultaneously with the improvement in his renal function. A diagnosis of Leptospira infection with the strain canicola was made on the basis of Leptospira titres (gross titre 1/40 icterohaemorrhagiae 1/30, canicola 1/100).

On the fourteenth day of his illness, he developed pain around the left shoulder which became increasingly severe over a 24 hr period and lasted for 72 hr. Similar pain was experienced on the right, but of lesser degree. As the pain subsided, the patient noticed progressive weakness of his shoulder girdle musculature, more marked on the left. Over the ensuing seven days the weakness in the left arm spread to involve the distal musculature. Neurological examination on the twenty-first day of his illness showed severe weakness of the left supra and infra spinatus muscles with deltoid, biceps, brachioradialis, triceps and distal musculature being MRC Grade 2 (MRC, 1976) at best.

On the right side there was marked weakness (MRC Grade 3) of the supra and infra spinat and deltoid and mild weakness (MRC Grade 4) of the biceps, brachioradialis and triceps. All the deep tendon reflexes in the upper limbs were absent, except the right triceps; the remainder of the neurological examination was normal.

Electrophysiological studies
These are summarized in Table 1 and show bilateral motor involvement.

The patient was re-examined 6 months later after the onset of his neurological illness. At that time his right-sided muscles had completely recovered and the proximal musculature on the left had improved to MRC Grade 4. There was still marked distal weakness with an anterior interosseus nerve palsy.

Discussion
Neurological complications of leptospirosis fall into two main groups:
(a) Early: A polyradiculo-neuropathy which occurs between the third and eighth days. Motor and sensory signs are present equally and the lower limbs are predominantly affected. Recovery occurs within 6 months.
(b) Late: Mono-, or polyneuropathy, occurring 20–30 days after the onset of the disease, the majority of patients presenting with lower facial palsy. Peripheral nerves are involved, with motor and sensory signs being present equally.

This patient falls into the latter category of late complications; however, the occurrence of a bilateral plexus syndrome has not been previously described.

The pathological basis of neuralgic amyotrophy still awaits clarification. A wide range of precipitating factors have been described, including infection,
### Table 1.

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Motor/sensory</th>
<th>Parameter</th>
<th>November 1976</th>
<th>April 1977</th>
<th>Control values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median</td>
<td>Motor</td>
<td>Conduction velocity m/sec</td>
<td>52.0</td>
<td>51.0</td>
<td>58 ± 5.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Terminal latency</td>
<td>4.0</td>
<td>3.8</td>
<td>2.7 ± 0.5</td>
</tr>
<tr>
<td>Ulnar</td>
<td>Motor</td>
<td>Conduction velocity m/sec</td>
<td>53.0</td>
<td>53.0</td>
<td>57 ± 8.4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Terminal latency</td>
<td>2.8</td>
<td>2.9</td>
<td>3.1 ± 1.1</td>
</tr>
<tr>
<td></td>
<td>Sensory</td>
<td>Amplitude µV</td>
<td>12.0</td>
<td>12.0</td>
<td>12.6 ± 2.0</td>
</tr>
</tbody>
</table>

### Latencies in msec from Erb's point:

<table>
<thead>
<tr>
<th>Muscle</th>
<th>November 1976</th>
<th>April 1977</th>
<th>Normal controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deltoid</td>
<td>R 5.0 N.R.</td>
<td>R 4.8 N.R.</td>
<td>3.9 ± 0.2</td>
</tr>
<tr>
<td>Biceps</td>
<td>N.R. N.R.</td>
<td>9.2 N.R.</td>
<td>4.5 ± 0.3</td>
</tr>
<tr>
<td>Triceps</td>
<td>5.9 6.0</td>
<td>N.T. N.T.</td>
<td>–</td>
</tr>
</tbody>
</table>

N.R. Not recordable, N.T. Not tested.

Trauma, surgical operation and immunization. Recurrence may occur and some familial cases are seen (Bradley et al., 1975).

The rate of recovery is roughly proportional to the distance of the affected muscle from the brachial plexus. Thus it is not surprising, in this patient, that the muscles supplied by the anterior interosseous nerve have remained weak, despite improvement in the proximal muscles.

### References


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