Congenital arteriovenous fistula producing carpal tunnel syndrome

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SUMMARY A case of carpal tunnel syndrome resulting from congenital arteriovenous fistula is described.

Carpal tunnel syndrome can result from a wide variety of causes. We wish to report a case of the syndrome resulting from a congenital arteriovenous fistula, believed to be unique.

Case report

A 45 year old man was admitted to hospital because of swelling and pain in the left hand and forearm since the age of 12 years. He used to get pain and paraesthesiae intermittently in the radial three fingers of the left hand. Three weeks before admission he had persistent pain and paraesthesiae in the affected limb with some relief on elevation of the left arm.

The left radial pulse was high volume. The left forearm and hand were swollen. The skin was bluish red, and there was local warmth. The swelling was not tender or pulsatile and there was no bruit. However, it was compressible and on elevation of the limb it decreased considerably and wasting of thenar groups of muscles became obvious. Motor power in the thenar group of muscles was grade 4. There was no objective sensory deficit. No other neurological or other systemic abnormality could be detected.

Routine blood chemistry, haemogram, and cardiac investigations were within normal limits. Skin temperature over the dorsum of the left hand was elevated by 1°C, and oscillometry showed that the fistula was situated at the level of the wrist. An arteriogram performed by percutaneous puncture of the left brachial artery in the anticubital fossa showed a diffuse arteriovenous fistula involving all fingers and wrist (Fig. 1). Nerve conduction studies showed prolonged motor and sensory distal latencies on the left side. There was improvement in the distal latencies and motor nerve conduction velocities on elevation of the arm, and the amplitude of the evoked sensory potential of the left median nerve was increased on elevation of the arm (Table). The distal latency (2.0 ms) and motor nerve conduction velocity in the forearm segment

Fig. 1 Arteriogram of left brachial artery showing diffuse arteriovenous fistula involving all fingers and wrist.

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(67 m/s) of the left ulnar nerve were within normal limits.

The left carpal tunnel was explored surgically. There was extension of the arteriovenous fistula proximally through the carpal tunnel into the forearm. The carpal tunnel was full of engorged and tortuous vessels (Fig. 2). The median nerve was embedded between these vessels. Adequate decompression of the carpal tunnel was performed.

Pain and paraesthesiae decreased considerably immediately after surgery, and subsequently there was complete relief of symptoms. Nerve conduction studies performed one month after surgery showed significant improvement in all the parameters, more so in the sensory conduction (Table).

**Discussion**

The clinical history, examination, and electrophysiological investigations of this patient have shown evidence of compression of the median nerve at the carpal tunnel on the left side.

There seems to be good evidence that intermittent symptoms in carpal tunnel syndrome are the results of ischaemia (Gilliatt and Wilson, 1953; Fullerton, 1963) but the more permanent changes such as increase in terminal latency are not attributable to ischaemia. It has also been shown that a direct mechanical effect on myelin leads to conduction block and conduction delay. Thus, there seems likely to be a dual mechanism for nerve damage (Simpson, 1956; Fullerton, 1963; Anderson et al., 1970).

Intermittent excruciating acroparaesthesias in the initial part of the illness in the present case can be explained by a vascular “steal” phenomenon caused by the fistula. In congenital arteriovenous fistula, arterial blood from a high pressure artery is shunted into a low pressure vein, thus decreasing venous pressure distal to the fistula (Noble, 1974). The increase in severity of symptoms in the present case in the recumbent position can be explained by pooling of blood in the fistula, thereby increasing its volume and resulting in compression of the

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**Table Results of electrophysiological studies**

<table>
<thead>
<tr>
<th></th>
<th>Right median nerve</th>
<th></th>
<th>Left median nerve</th>
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<th>Sensory conduction</th>
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<tr>
<td></td>
<td>Motor conduction</td>
<td>Sensory conduction</td>
<td>Motor conduction</td>
<td>Sensory conduction</td>
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<td></td>
<td>Preoperative</td>
<td>Postoperative</td>
<td>Preoperative</td>
<td>Postoperative</td>
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<td>Arm at rest</td>
<td></td>
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<tr>
<td>Distal latency (ms)</td>
<td>2.8</td>
<td>2.9</td>
<td>3.9</td>
<td>3.4</td>
<td>3.8</td>
</tr>
<tr>
<td>Forearm segment conduction velocity (m/s)</td>
<td>57</td>
<td>55</td>
<td>46</td>
<td>51</td>
<td>51</td>
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<tr>
<td>Sensory action potential (μV)</td>
<td>—</td>
<td>55</td>
<td>—</td>
<td>—</td>
<td>10</td>
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<tr>
<td>Arm elevated for 20 minutes</td>
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<td>Distal latency (ms)</td>
<td>3.5</td>
<td>3.4</td>
<td>3.6</td>
<td>3.4</td>
<td>3.5</td>
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<tr>
<td>Forearm segment conduction velocity (m/s)</td>
<td>51</td>
<td>51</td>
<td>44</td>
<td>51</td>
<td>51</td>
</tr>
<tr>
<td>Sensory action potential (μV)</td>
<td>—</td>
<td>—</td>
<td>30</td>
<td>30</td>
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</tr>
</tbody>
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**Fig. 2** Photograph of carpal tunnel full of engorged and tortuous vessels. The median nerve (arrow) was embedded between these vessels.
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median nerve.

The improvement of sensory nerve conduction after decompression can occur within 30 minutes (Hongell and Mattsson, 1971). The rapidity of this recovery strongly suggests that relief of ischaemia is likely to be responsible. Thus, in the present case the improvement in the motor and sensory conduction parameters after elevation of the limb could be the result of relief of ischaemia after shunting and mechanical compression of the median nerve by engorged veins.

In our patient the attacks of pain were relieved immediately after decompression. The nerve conduction velocities, both motor and sensory, returned to the normal range within one month. Distal venous stasis because of increased venous pressure resulting from the transmission of the arterial pressure to the venous side, together with a leash of blood vessels constituting the arteriovenous fistula increased the volume of the tunnel contents, presumably causing constant compression of the median nerve with reversible and demyelinating conduction block and conduction delay. Thus, in this case there is evidence to suggest a dual mechanism for the nerve damage.

References


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