CASE REPORT

Delayed onset ulnar nerve palsy and carpal tunnel syndrome following blunt injury to the wrist

F. Dakhil-Jerew*, M.S. Ikram, F. Schreuder

Department of Plastic and Reconstructive Surgery, Lister Hospital, Coreys Mill Lane, Stevenage, Hertfordshire SG1 4AB, United Kingdom

Accepted 22 October 2007

Introduction

A peripheral nerve entrapment may be caused by mechanical dynamic compression of a short segment of a single nerve at a specific site, frequently as it passes through a fibro-osseous tunnel, or an opening in fibrous or muscular tissue. At the wrist, the median nerve and the ulnar nerve may be compressed as they pass through the carpal tunnel and Guyon tunnel respectively.

Case study

A twenty-eight-year-old gentleman sustained an industrial injury while operating a roller machine used to compress plastic material into plates. He accidentally placed his left (non-dominant) hand in a narrow gap (approximately 3.5 cm) between the two rolling bullies and it was trapped for 15 min before its extraction.

He was brought into the local accident and emergency department with tender and swollen left hand and wrist. His radial pulse was palpable and there was no sensory or motor deficit. His hand and wrist X-rays did not reveal any fractures.

After one week, pain and tenderness were resolved but his hand was still significantly swollen. On repeated neurological examinations, a slightly reduced light touch sensation in all fingers was found which was attributed to hand swelling. Wrist and fingers’ movements were painless but restricted due to swelling. He was referred for thermoplastic wrist splint and hand physiotherapy to facilitate rehabilitation. After seven days, the patient’s hand appeared to be improving and the swelling had reduced significantly.

The hand therapist then noticed deterioration in patient’s hand function and he was reviewed urgently on the 21st day post-injury. Small muscles of his left hand (dorsal interossi, hypothenar and thenar muscles) appeared wasted in comparison with the right hand (Figs. 1 and 2) and his hand function was significantly restricted. There was clawing of the little and ring fingers. Worsening pins and needles in all four fingers was noted together with positive Wartenberg’s and Froment’s signs. Both Phalen’s and Tinel’s tests were positive. The patient was counselled for surgical decompression of ulnar and median nerves and his wrist was operated upon within three days.

A single volar incision (Fig. 3) was used for release and both Guyon and Carpal tunnels were found to be tight. Median and ulnar nerves were in continuity and intact but both nerves were hyperaemic (Fig. 4).

Close postoperative monitoring showed significant improvement in fingers’ sensations and resolution of little and ring fingers’ clawing (Fig. 5). Phalen’s test became negative (Fig. 6) and the patient achieved a reasonable hand function. He returned back to the same job within eight weeks.
Figure 1  Left hand: preoperative wasting, positive Wartenberg’s signs.

Figure 2  Preoperative clawing of left ring and little fingers.

Figure 3  Single volar incision.

Figure 4  Operative findings both nerves intact within tight compartments.
In this case, the aetiology of delayed onset ulnar and median nerves compression signs and symptoms was related to blunt injury to the wrist. It may be classified as Neurapraxia according to Seddon’s Classification (Table 1) or grade 1 according to Sunderland’s Classification of Nerve Compression (Table 2).

Table 1: Seddon’s classification of nerve compression

<table>
<thead>
<tr>
<th>Type</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurapraxia</td>
<td>Pressure on the nerve with resultant dysesthesias but no loss of continuity.</td>
</tr>
<tr>
<td>Axonotmesis</td>
<td>The neural tube is intact, but the internal axons have been disrupted.</td>
</tr>
<tr>
<td>Neurotmesis</td>
<td>The nerve itself has been completely divided.</td>
</tr>
</tbody>
</table>

Initial clinical assessments did not indicate the presence of acute ulnar or median nerve entrapment; however symptoms evolved over a three week period.

The mechanism of injury suggests a high-energy impact to the wrist and hand. A high index for suspicion was needed to prevent the possibility of overlooking delayed onset signs of damaged structures and might be of value for subsequent medico-legal or industrial compensation claim.

The carpal tunnel is formed by the carpal bones, intercarpal ligaments and flexor retinaculum. The transverse carpal ligament (TCL) forms the roof of the carpal tunnel and the floor of Guyon’s canal. Guyon canal is formed by the pisiform bone, pisohamate ligament and the hook of hamate. The canal contains the ulnar nerve and artery with their venae comitantes and loose fibrofatty tissue. Within the canal, the ulnar nerve divides into superficial sensory and deep motor branches.

Generally speaking, CTS may be caused by repeated compression leading to ischemia, oedema formation and eventually fibrosis, tethering of the nerve due to scar tissue and mechanical pressure from nearby structures.

CTS Clinical assessment includes Phalen’s test (appearance or worsening of paresthesia with maximal passive wrist flexion for 1 min) and Tinel’s sign (paresthesia in the median territory elicited by gentle tapping over the carpal tunnel). In a clinical setting, an assessment of strength, sensory loss, and pain is sufficient to monitor the progress of the syndrome.

Ulnar Tinel’s sign is performed by tapping over pisiform. One of the earliest findings is the ‘Wartenberg’s sign’ where

Table 2: Sunderland’s classification of nerve compression

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Interruption of axial conduction at the site of injury. The axon remains in continuity; some segmental demyelination may be present but not Wallerian degeneration. The condition is reversible.</td>
</tr>
<tr>
<td>2</td>
<td>The axon itself is no longer in continuity. The axon does not survive distal to the level of the injury and for a short distance proximal. The endoneurium is preserved. Full recovery may be expected.</td>
</tr>
<tr>
<td>3</td>
<td>The axon is severed, and Wallerian degeneration develops. The endoneurial tube is lost, and the fascicular anatomy is disturbed. Recovery is incomplete.</td>
</tr>
<tr>
<td>4</td>
<td>Total destruction of the internal architecture of the nerve. The trunk is intact, but a neuroma will form. Spontaneous recovery is rare. Surgical repair is indicated.</td>
</tr>
<tr>
<td>5</td>
<td>Loss of continuity of the nerve trunk. Surgical repair is mandatory.</td>
</tr>
</tbody>
</table>

Discussion

In this case, the aetiology of delayed onset ulnar and median nerves compression signs and symptoms was related to blunt injury to the wrist. It may be classified as Neurapraxia according to Seddon’s Classification (Table 1) or grade 1 according to Sunderland’s Classification of Nerve Compression (Table 2).

Initial clinical assessments did not indicate the presence of acute ulnar or median nerve entrapment; however symptoms evolved over a three week period.

The mechanism of injury suggests a high-energy impact to the wrist and hand. A high index for suspicion was needed to prevent the possibility of overlooking delayed onset signs of damaged structures and might be of value for subsequent medico-legal or industrial compensation claim.

The carpal tunnel is formed by the carpal bones, intercarpal ligaments and flexor retinaculum. The transverse carpal ligament (TCL) forms the roof of the carpal tunnel and the floor of Guyon’s canal. Guyon canal is formed by the pisiform bone, pisohamate ligament and the hook of hamate. The canal contains the ulnar nerve and artery with their venae comitantes and loose fibrofatty tissue. Within the canal, the ulnar nerve divides into superficial sensory and deep motor branches.

Generally speaking, CTS may be caused by repeated compression leading to ischemia, oedema formation and eventually fibrosis, tethering of the nerve due to scar tissue and mechanical pressure from nearby structures.

CTS Clinical assessment includes Phalen’s test (appearance or worsening of paresthesia with maximal passive wrist flexion for 1 min) and Tinel’s sign (paresthesia in the median territory elicited by gentle tapping over the carpal tunnel). In a clinical setting, an assessment of strength, sensory loss, and pain is sufficient to monitor the progress of the syndrome.

Ulnar Tinel’s sign is performed by tapping over pisiform. One of the earliest findings is the ‘Wartenberg’s sign’ where
the little finger is abducted due to weakness of the third palmar interosseous muscle. Froment’s sign is an indicator of motor involvement in ulnar neuropathy. The thumb—forefinger pinch is distorted because of the weakness of the adductor pollicis, the ulnar portion of the flexor pollicis brevis, and the first dorsal interosseous. Montagna in 1994 reported the ‘motor’ Tinel sign in a patient with entrapment of the ulnar nerve at the elbow. The ‘palmaris brevis sign’ helps to differentiate between ulnar nerve compression at the cubital tunnel or at the wrist. In this case ulnar nerve entrapment involved both motor and sensory divisions and it was classified as type I (Table 3).

In our case, diagnosis of nerves compression was adequate from the clinical setting. However in certain conditions, nerve conduction studies are needed to confirm diagnosis. Magnetic resonance imaging and/or ultrasound are indicated in patients with equivocal clinical findings, a suspected mass lesion or persistent symptoms after surgery.

At surgery, both nerves were found to be hyperaemic and tightly compressed within the carpal and Guyon’s tunnel. A possible reason is the swelling and oedema which followed blunt wrist injury.

It is interesting to know that the transverse carpal ligament is common relation to both tunnels and thus changes in the pressure within the Carpal tunnel could affect the ulnar nerve within Guyon’s canal. Seddon treated concomitant CTS and Guyon’s canal compression by release of the carpal tunnel compartment only. Our rationale for early surgical release of both tunnels was to minimize the extent to which neurapraxia progresses to axonotmesis Axonal degeneration may be an early abnormality in distal ulnar neuropathy but segmental demyelination may occur if compression is left untreated.

### Table 3

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Compression is just proximal or within Guyon’s canal and presented with both motor and sensory signs and symptoms.</td>
</tr>
<tr>
<td>II</td>
<td>Compression along deep motor branch, thus presentation is pure motor.</td>
</tr>
<tr>
<td>III</td>
<td>Compression is distal in the end of the Guyon’s canal and it spares the motor branch. Presentation is pure sensory on palmar ulnar distribution.</td>
</tr>
</tbody>
</table>

### Conclusion

Effects of a high-energy blunt injury to the wrist are unpredictable. Early signs may be minimal and surgery may sound as a remote possibility. A high index of suspicion and close observation is required to detect any delayed development of nerve compression. Early surgical decompression is needed to avoid further nerve damage and to restore neuronal function.

### References