Prevalence of Raynaud’s phenomenon in patients with idiopathic carpal tunnel syndrome
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Both idiopathic carpal tunnel syndrome (CTS) and Raynaud’s phenomenon (RP) are common, and may have similar clinical symptoms. The degree of their coexistence is uncertain.

We have examined 30 patients, who were diagnosed clinically and electromyographically as having idiopathic CTS, for the presence of RP using a cold provocation test with photoplethysmography. The patients’ hands were exposed in water at 10°C for five minutes.

A total of 18 patients (60%) was found to have RP; this is much greater than would be expected from the prevalence in the general population. Raynaud’s phenomenon should be considered when treating patients with CTS because of the possibility of coexistence and the similar symptoms of these two disorders.

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Carpal tunnel syndrome (CTS) may be associated with systemic disease such as obesity, diabetes mellitus, thyroid dysfunction, and Raynaud’s phenomenon (RP). CTS and RP may present with similar symptoms such as tingling and numbness.

The association between CTS and RP was first recorded by Garland, Bradshaw and Clark. Since then, the possible relationship between the two conditions has been suggested many times. Linscheid, Peterson and Jeurgens reported CTS associated with vasospasm, and Waller and Dathan described cases of RP in which digital ischaemia appeared to have been exacerbated by CTS. Pal et al reported the association in a more systematic study using groups of controls. The studies published so far are equivocal since the diagnosis of RP was not made on objective criteria. To do so, we have tested patients’ sensitivity to cold by a provocation test using photoplethysmography and investigated its prevalence in patients with idiopathic CTS.

Patients and Methods

We included 30 patients with idiopathic CTS in the study. We excluded those who had an obvious underlying cause such as rheumatoid arthritis, malunited Colles’ fracture, a history of manual work with exposure to vibratory tools, and definite evidence of peripheral neuropathy such as diabetes mellitus. Patients were included only if they had: 1) symptoms of CTS such as paraesthesia, numbness and pain at night; 2) more than one clinical sign such as positive Tinel and Phalen’s tests and atrophy of the thenar muscles; 3) abnormalities in electrophysiologic studies, such as delayed nerve conduction distal to the wrist; and 4) surgery to release the volar carpal ligament with a follow-up of more than three months. The conduction time was considered to be prolonged if the terminal latency of the motor impulse exceeded 4.5 ms or if the sensory antidromic latency exceeded 3.5 ms.

There were two men and 28 women with a mean age of 54 years (36 to 67). At the time of surgery, the mean duration of symptoms was 4.5 years (5 months to 20 years). Fifteen patients had unilateral and 15 bilateral involvement. No cases were recurrent and no patients had a history of taking medication such as ergot, beta-blocking agents and cytotoxic drugs, which are known to induce RP. A cold provocation test was carried out.

Photoplethysmography was used to measure the systolic blood pressure of the thumb or index finger before and after cooling the hand by immersion in water at 10°C for five minutes. It was regarded as a positive response if there was a decrease of more than two-thirds in the amplitude of the arterial pulse within one minute of exposure to cold water (Fig. 1). We used the Medasonic vascular model PPG 13 (Medasonics, California) for photoplethysmography. The general vascular status of the patients was evaluated to exclude the possible association of an obstructive disorder, such as Buerger’s disease. RP was diagnosed when the patient showed both a positive cold provocation test, had experienced one or more typical vasospastic episodes, such
as colour changes in the skin, and had a history of severe pain in the fingers after exposure to cold.

We used the chi-squared and Student’s t-tests for statistical analysis.

Results

Of the 30 patients with CTS, 20 had a history of unusual sensitivity to cold. Of these, 19 had a positive response to the cold provocation test, only one of whom reported no vasospastic episodes. Thus, 18 patients (60%) had RP.

Of the 18 patients with both CTS and RP, 11 had bilateral CTS and seven were affected on only one side, but all had RP in both hands. No significant differences were observed in the distribution of age and gender. No statistical significance (p = 0.13) was seen in the duration of the symptoms in the patients with both conditions, although there was a tendency for a longer duration in these patients (mean, 5.7 years) compared with those with CTS only (mean, 2.8 years) (Table I).

Studies of motor-nerve conduction showed no significant differences. There was statistical significance (p = 0.04) in sensory nerve conduction, with a mean distal sensory latency, which represents conduction velocity, of 7.3 ms in patients with CTS and RP compared with 5.1 ms in the patients with CTS only.

A comparison of the duration of symptoms and conduction velocities suggests that the patients with both conditions had a longer period of nerve compression.

Discussion

In our study, the prevalence of RP in patients with CTS was 60%, which is considerably more than in the general population. Walker et al. reported a prevalence of 9.5% in men not exposed to vibration; Leppert et al. described 15.6% in women. In Orientals, the prevalence was 2.5% and 4.6% for Japanese men and women, respectively, and 1.6% in Chinese working men. Thus, the prevalence of RP in the general population is about 10% to 15% in Caucasians and 2% to 5% in Orientals.

Garland et al. reported five patients (9%) with RP in 53 with CTS, while Linscheid et al. and Pal et al. described 28 in 2800 (1%) and in 34 in 93 (37%), respectively. These differences in prevalence were probably due to lack of a precise definition for RP. The most important factors which affect prevalence are an exact definition and objective diagnostic criteria; it is questionable whether these reports would satisfy the current, generally recognised definition of RP. In our study, we used the cold provocation test, since it is reproducible, and the sensitivity and the specificity of plethysmography after cold provocation are generally accepted as reasonable.

Episodic digital ischaemia can result from two distinct pathophysiological mechanisms, arterial obstruction and vasospasm. In contrast to the relatively straightforward relationship between arterial occlusive disease and RP, the pathophysiology of spastic RP is unclear. Originally, Raynaud ascribed colour changes and digital ischaemia to an enormous exaggeration of vasomotor innervation. Lewis suggested a 'local fault' in the blood vessel, such as a hypersensitivity of the vascular walls. Significantly elevated levels of alpha-2 adrenergic receptors in circulating platelets and an increase in the levels of plasma norepinephrine and cyclic nucleotides have been found in patients with RP, indicating that the central mechanism of RP involves the sympathetic nervous system. It is generally accepted that, regardless of vasospastic relationships to central or local mechanisms, the sympathetic nervous system probably plays a significant role in the pathogenesis.

Table I. A comparison of the 30 CTS patients with and without RP

<table>
<thead>
<tr>
<th></th>
<th>RP-positive</th>
<th>RP-negative</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>CTS patients</td>
<td>18</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>Male:female</td>
<td>2:16</td>
<td>0:12</td>
<td></td>
</tr>
<tr>
<td>Mean age in years ± sd (range)</td>
<td>54 ± 5.0 (40 to 63)</td>
<td>53 ± 8.7 (36 to 67)</td>
<td>0.75</td>
</tr>
<tr>
<td>Mean duration in years ± sd (range)</td>
<td>5.7 ± 7.3 (0.2 to 20)</td>
<td>2.8 ± 2.1 (0.2 to 20)</td>
<td>0.13</td>
</tr>
<tr>
<td>Bilateral</td>
<td>11</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Unilateral</td>
<td>7</td>
<td>8</td>
<td>0.14</td>
</tr>
<tr>
<td>Mean motor latency ± sd (ms)</td>
<td>5.9 ± 2.0</td>
<td>5.8 ± 2.1</td>
<td>0.81</td>
</tr>
<tr>
<td>Mean sensory latency ± sd (ms)</td>
<td>7.3 ± 2.9</td>
<td>5.1 ± 2.4</td>
<td>0.04</td>
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</table>
Kramer and Todd\(^{14}\) reported a greater vasomotor supply to the distal than to the proximal segments of extremities. Woollard and Phillips\(^{15}\) noted that vasomotor fibres followed the distribution of sensory fibres in the median and ulnar nerves. Recently, Campero, Verdugo and Ochoa\(^{16}\) stated that the median nerve provides supplementary vasomotor innervation to the hand in addition to the area which it normally supplies. As paraesthesiae reflect compression of the sensory fibres of the median nerve vasospasm may be an expression of irritation of the autonomic fibres. Sympathetic nerves are also compressed in the carpal tunnel.

Raynaud’s phenomenon cannot simply result from local compression of sympathetic fibres, since all the patients with both conditions showed RP in both hands, regardless of whether they had unilateral or bilateral symptoms of CTS. Ramieri et al\(^{17}\) reported that the sympathetic axons survived unaltered even in patients with a long clinical history of profound sensory impairment and Lang et al\(^{18}\) found that the function of their sympathetic fibres did not help in the diagnosis of CTS. The exact mechanism of RP in CTS is still unclear, although local or systemic irritation of the autonomic nervous system is thought to play a part.

If the vasospasm occurs because of entrapment of the median nerve at the wrist, RP should disappear after decompression of the carpal tunnel. Porter et al\(^{19}\) found that release of the carpal tunnel did not affect the symptoms and signs of RP. Our study has not included postoperative follow-up of such symptoms, although the patients are still closely observed in the outpatient clinic. Persistent symptoms after surgical release are a source of great frustration for both surgeon and patient, and sometimes lead to medical and legal problems. Many surgeons emphasise the importance of confirming a double-crush phenomenon in CTS and for a suboptimal clinical outcome of treatment. We think that it is essential to understand the possibility of the coexistence of RP in the treatment of CTS.

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References


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