Neurogenic Thoracic Outlet Associated With Carpal Tunnel Syndrome: Is This A Clinical Example of Double Crush Hypothesis?

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ABSTRACT

Aim of the work: The aim of this study was to assess the possible contribution of the double crush hypothesis (DCH) for the association of carpal tunnel syndrome (CTS) with neurogenic thoracic outlet syndrome (NTOS) which -if confirmed- can explain some of the not uncommonly persistent CTS cases despite being confirmed and properly treated, particularly that NTOS is potentially treatable.

Subjects and Methods: 137 CTS patients {91 (66.4%) females, 46(33.6%) males}, with mean age 39.4, ranging from 23 to 48 years, were included in the study based solely on electrophysiological criteria of CTS (distal motor latency to abductor pollicis brevis > 4 ms, 3rd digit to wrist orthodromic sensory conduction velocity < 45m/s, or orthodromic median/ulnar latency difference of the 4th digit > 0.4 ms). Patients who proved suffering peripheral neuropathy or entrapment of ulnar nerve were excluded. Twenty apparently healthy individuals, age and sex matched with patients were included as a control group. The patients and control groups were subjected to clinical neurological evaluation. Electrophysiological work up including motor/sensory conduction study of median and ulnar nerves on both sides, and bilateral medial antebrachial cutaneous nerve (MABCN) antidromic sensory, considering side to side MABCN sensory nerve action potentials (MABCN SNAP) amplitude ratio of >2.0 as abnormal. Electrophysiological criteria used for confirming (NTOS), were low median compound motor action potentials (CMAP), low ulnar SNAP, low or normal ulnar CMAP, normal or reduced interference pattern of C8 T1-innervated muscles, and MABCN SNAP interside amplitude ratio >2.0 (the latter was used as a mandatory inclusion criterion). Patients with atypical upper limb pain have undertaken cervical plain X ray. Control group was subjected to complete neurophysiological studies. Student t test was used to compare means of two groups.

Results: Ulnar nerve SNAP amplitudes were found normal. Reduction of median CMAP was reported in 19 patients (13.86%) and it was bilateral in 7 (36.84%). Antidromic MABCN SNAP interside amplitude ratios showed values <2.0 and mean ±SD was 1.273±0.221. Needle examination showed incomplete interference pattern in abductor pollicis brevis muscles in 11 patients (8%). 3 patients (5.26%), 2 males, and 1 female had bilateral bony cervical ribs but non had evidence of NTOS. Conclusions: Neurophysiologically confirmed CTS was not proved to associate NTOS, and the hypothesized relationship between them could not be obtained. This might inspire us to revisit DCH for re-evaluation. Finally, NTOS is still a rare medical condition and scrutinizing suspected cases with thorough clinical assessment, and electrophysiological work up is a must. (Egypt J. Neurol. Psychiat. Neurosurg., 2007, 44(2): 407-419)

INTRODUCTION

The term ‘thoracic outlet syndrome (TOS)’ was described by Schenard in 2005 and referred to a group of disorders affecting the brachial plexus, the subclavian vessels, or both at any point between the base of the neck and the axilla. TOS was further sub classified into vascular, neurogenic, and vascular-neurogenic combined forms. Neurogenic thoracic outlet syndrome (NTOS) is caused by compression of the lower trunk of the brachial plexus by a fibrous band (true NTOS) and this syndrome is exceedingly rare. NTOS consists
of unilateral wasting of the hand associated with a rudimentary cervical rib or elongated C7 transverse process. During surgery a fibrous band is found over which the lower brachial plexus is stretched and angulated.\textsuperscript{4}

Kothari et al. in 1998\textsuperscript{4} reawakened interest in the syndrome and described the neurophysiological findings. The authors showed that nerve conduction studies were quite helpful in localizing the lesion to the lower trunk of the brachial plexus affecting predominantly the C8-T1 roots.\textsuperscript{4,5} Currently the accepted electrophysiological criteria for true NTOS are low amplitude median compound motor action potentials (CMAP), low ulnar sensory nerve action potentials (SNAP), relatively low or normal ulnar CMAP, and normal median SNAP\textsuperscript{4,5,6} and these abnormalities are usually not seen with other lower trunk brachial plexopathies such as those due to injury, tumour, or radiation.\textsuperscript{6}

Medial antebrachial cutaneous nerve (MABCN) SNAP is of value for early diagnosis of NTOS.\textsuperscript{7} This nerve is derived from the C8-T1 roots and is a branch of the medial cord of the brachial plexus and as the nerve goes distally it lies nearby the basilic vein and subsequently branches to supply the median forearm.\textsuperscript{8} Despite that previous electrophysiological criteria for diagnosing NTOS (low median CMAP, low ulnar SNAP, relatively low or normal ulnar CMAP, and normal median SNAP), were not all reported in all NTOS cases, MABCN sensory response was abnormal in all cases in patients studied in two studies.\textsuperscript{7,9} In 2004, Seror\textsuperscript{10} has studied the ability to diagnose mild lower brachial plexus lesion only through abnormal MABCN conduction study and defined abnormality by an interside amplitude ratio of SNAP equal or greater than 2. The author found that MABCN testing was abnormal in all the patients when all other motor and sensory nerve conductions of median and ulnar nerves were normal.

Carpal tunnel syndrome (CTS) is a constellation of symptoms associated with localized compression of the median nerve at the wrist. The pathophysiology of CTS is not fully understood but mechanical aspects of injury within the carpal tunnel are most likely. The issues of ischaemia, mechanical trauma, ectopic impulse generation, demyelination, tendonitis, elevated carpal tunnel pressure, mechanical factors, small and large fiber involvement and the variability of symptoms are presented.\textsuperscript{11} In 2002, a consensus conference was organized and identified a combination of symptoms (numbness, tingling, burning and pain in combination with nocturnal symptoms) plus abnormal median nerve function based upon nerve conduction studies as the best ‘gold standard’ for diagnosis of CTS.\textsuperscript{11}

The double crush hypothesis (DCH) suggests that a peripheral nerve which is compressed proximally is more liable to be compressed distally than a nerve that has not sustained proximal compression.\textsuperscript{12} This hypothesis was supported by studies in the humans\textsuperscript{13-15} and animal models.\textsuperscript{16-18} The comparison of NTOS with CTS is interesting in that CTS as NTOS is a chronic entrapment neuropathy that usually displays a clinical pattern of transient complaints and absence of amyotrophy and hypoesthesia for a long time. Improvements in the methods of CTS electro diagnosis have led to diagnosis at an earlier stage. As a proof of this, Seror\textsuperscript{10} noted that amyotrophy and hypoesthesia were found respectively in 50 and 80\%, of patients. Most of the authors would be interested in discovering early cases of NTOS before occurrence of amyotrophy and hypoesthesia, so that this study was designed aiming at answering the question about the likelihood of NTOS to associate CTS as an example of DCH. NTOS is a potentially treatable co morbidity which may contribute to the not uncommon persistent CTS cases despite being properly diagnosed and treated, so early accurate diagnosis may be of help.

**SUBJECTS AND METHODS**

This study was conducted at Clinical Neurophysiology units of Neurology department and Rheumatology & rehabilitation department, Zagazig university hospitals. A hundred and thirty seven patients (91 (66.4\%) females, 46 (33.6\%) males) were included in this study. They were referred for neurophysiological assessment, a diagnosed CTS based on clinical parameters (permanent hypoesthesia or intermittent symptoms...
of burning, tingling, and paraesthesia in the radial 3 & half digits especially at night or upon awakening, thenar muscle atrophy). Patients were included in the study solely based on electrophysiological proofs of CTS, otherwise they were excluded. Twenty apparently healthy individuals (13 (65%) females and 7 (35%) males), age and sex matched with patients, were included in the study as a healthy control group. Patients who proved clinically and/or electrophysiologically suffering peripheral neuropathy or entrapment of ulnar nerve at wrist or elbow were excluded. Neuropathy was defined by the presence of two or more abnormalities on electrophysiological testing of the nerves examined with regard to distal latency, conduction velocity, amplitude of action potentials, and F wave latency.

All patients were subjected to the followings:

(1) Neurological clinical evaluation: stressing on symptoms/signs of peripheral neuropathy (pain, tingling and numbness, stocking and glove hypoesthesia, distal symmetric weakness, wasting, hypotonia, hyporeflexia, vasomotor changes etc), thoracic outlet syndrome (atypical pain, burning sensation, numbness in digits 4-5 and inner side of the forearm, clumsiness, cramps of the upper limbs, weakness of muscles supplied by C8-T1 not limited to single nerve distribution), as well as signs of entrapment neuropathy of median nerve at wrist, and ulnar nerve at wrist or elbow (paresthesia at the sensory distributions of the median and ulnar nerves, pain etc).

(2) Electrophysiological studies: These studies were carried out using System PLUS Micromed Via Giotto 4, 1-31021 Mogliano Veneto (TU)- Italy as follows: Motor conduction studies of median, and ulnar nerves on both sides: The median nerve was stimulated by surface electrodes at wrist (palmer midwrist 6.5 cm proximal to active recording electrode on abductor digiti minimi, as well as below and above the elbow. Recording was done by monopolar surface electrodes over abductor pollicis brevis for median, and abductor digiti minimi muscle for ulnar nerve. Distal motor latencies in msec were estimated from stimulus artefact to the beginning of CMAP. CMAP amplitudes were measured peak to peak and motor conduction velocities in different segments were calculated. Inter side as well as segmental comparisons regarding conduction velocities were calculated as well. Median and ulnar F-wave latencies were recorded after 10 supra maximal stimulations at wrist and the shortest latencies were obtained. Sensory conduction studies of both median and ulnar nerves on both sides:

Orthodromic sensory studies of median and ulnar nerves were done by ring electrode stimulation of the digit 3 for median and digit 4 for median and ulnar nerves, and recording by surface electrodes at wrist on median and ulnar nerves.

MABCN: MABCN was studied bilaterally with antidromic technique. The antidromic study of the anterior branch of the MABCN was performed with bipolar stimulation 1 to 3 cm above and before the medial epicondyle and bipolar recording 8 to 12 cm distally on the anterior medial aspect of the forearm. Averaging was applied and the amplitudes in µV were measured from peak to peak. The mandatory inclusion criterion was the interside amplitude ratio greater than 2.0 for a unilateral abnormal MABCN. The studies were done bilaterally with the same settings.

Electromyography: was performed with concentric needle examination of the abductor pollicis brevis, abductor digiti minimi, and first dorsal interosseous muscles. The followings were looked for (1) activities at rest (fibrillation potentials), (2)individual motor unit potentials morphology, and (3) interference patterns.

Electrophysiological criteria used in this study for diagnosing CTS were distal motor
latency to abductor pollicis brevis greater than 4 ms, digit 3 to wrist orthodromic sensory conduction velocity lower than 45 m/s, or orthodromic median/ulnar latency difference of the 4th digit greater than 0.4 ms. In cases of bilateral CTS, nerve conduction results from more profoundly affected limbs were processed.

The current study evaluated electrophysiologically the lower brachial plexus lesion, (neurogenic pattern of C8-T1 affection), based on Kothari et al. criteria which are low amplitude of median nerve CMAP, low amplitude of ulnar nerve SNAP, low or normal amplitude of ulnar CMAP as well as normal or slightly reduced interference pattern of some C8-T1 innervated muscles, but MABCN interside amplitude ratio greater than 2.0 by antidromic technique was mandatory for the diagnosis according to Seror postulation. Stimulus duration applied was 0.2 ms, intensity from 5-15 mA, lower/upper filter settings: 20-2000 Hz, time base 20 ms, gain for division 2 µV, and averaging for 50 sweeps.

(3) Plain X ray of cervical spine: That was carried out looking for cervical ribs or elongated C7 processes for 57 patients (31 females, 26 males) having pain numbness and/or tingling, and heaviness vague and generalized over the upper limb, +/- symptom provocation by repetitive or stressful activity of the involved limb, +/- positive Tinel sign.

Healthy controls were subjected to neurological examination exactly the same as patients as well as complete electrophysiological study and their values were used as our norm.

Finally, results were collected and data base processing was done using statistical package of social sciences (SPSS) version 8(24). Student “t” test was used and the results were considered significant if P-value <0.05, while P-value >0.05 indicates non significant and P<0.001 highly significant values.

RESULTS

Total patients included in this study were 137 pts, 91 females (66.4%) and 46 males (33.6%). Age of these patients was ranging from 23 to 48 years and the mean value ± SD was 39.43±7.46 years. Control group included 7 (35%) males and 13 (65%) females, their age was ranging from 19 to 53 years with mean age ±SD (38.60±11.74) which showed a non significant relationship on comparing with that of patients (p=0.713).

Electrophysiological studies:

In electrodiagnosis of CTS, most of the patients subjected to the study showed bilateral entrapment of median nerve at wrist followed in frequency by right unilateral lesion as shown in table (1).

Sensory nerve action potentials of the ulnar nerve studies were done bilaterally for all patients and the mean values ± SD were (14.088±4.832 µV). Bilateral CMAP amplitude measurement of the median nerves in all patients showed reduction of CMAP amplitudes in 19 (13.86%) patients, 7 (36.84%) patients of them were bilateral. The mean CMAP amplitudes for the normal group was 12.137±6.081 mV compared to 2.989±0.580 mV for the abnormal group with statistically highly significant relationship (p<0.001).

Antidromic MABCN SNAP interside amplitude ratio was done and none of the patient had ratio more than 2 and the mean ±SD values were 1.273±0.221 as shown in table (1).

Needle examination showed incomplete interference pattern in abductor pollicis brevis muscles in 11 (8%) cases, 8 (72.7%) females and 3 (27.3%) males and was bilateral in 7 (63.6%) cases 6 (85.7%) females and 1 (14.3%) male.

Fifty seven patients (41.6%), presented with atypical pain in the upper limbs {31 (54.4%) females, 26 (45.6%) males} were subjected to plain cervical x ray and 3 (5.26%) of them (2 males and 1 female) had bilateral bony cervical ribs as shown in figure 3. The electrophysiological testing for them was as shown in table (2).

Electrophysiological values of the control group as shown in tables (3), (4) and (5) were used in the study as a reference values for diagnosing abnormal cases that were excluded (peripheral neuropathy, entrapment of ulnar nerve at wrist or elbow).
Table 1. Neurophysiological parameter results used for detection of CTS and NTOS.

<table>
<thead>
<tr>
<th>Orth.Median SCV/ m/s</th>
<th>Total pts No &amp; %</th>
<th>Abnormal pts No &amp; %</th>
<th>Normal pts No &amp; %</th>
<th>Mean±SD</th>
<th>Bilateral cases No &amp; %</th>
<th>Unilateral cases No &amp; %</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>137pts 100%</td>
<td>*108 78.84%</td>
<td>29 pts 21.16%</td>
<td>35.58±9.2 m/s</td>
<td>62 pts 57.40%</td>
<td>28 pts 25.93% 18 pts 16.67%</td>
</tr>
<tr>
<td>Median/Ulnar Latencies/ msec</td>
<td>112pts 81.75%</td>
<td>*91 81.25%</td>
<td>21 pts 18.75%</td>
<td>1.34±0.89</td>
<td>56 pts 61.54%</td>
<td>21 pts 23.06% 14 pts 15.40%</td>
</tr>
<tr>
<td>Median DML/ msec</td>
<td>94 pts 68.61%</td>
<td>*71 75.53%</td>
<td>23 pts 24.47%</td>
<td>4.52±0.50</td>
<td>39 pts 54.91%</td>
<td>19 pts 26.78% 13 pts 18.31%</td>
</tr>
<tr>
<td>MABCN ratio</td>
<td>137pts 100%</td>
<td>++None</td>
<td>137pts 100%</td>
<td>interside amplitude ratio</td>
<td>1.273±0.221</td>
<td></td>
</tr>
</tbody>
</table>

*Digit 3 to wrist orthodromic sensory conduction velocity lower than 45 m/s (orth = orthodromic, SCV= sensory conduction velocity).
†Orthodromic median/ulnar latency difference of the 4th digit greater than 0.4 ms
‡Median distal motor latency (DML) to abductor pollicis brevis greater than 4 ms.
§Medial antebrachial cutaneous nerve (MABCN) interside amplitude ratio >2.0

Fig. (1): Orthodromic median/ulnar latency difference of the 4th digit in ms.
Fig. (2): MABCN recorded on right and left sides by antidromic technique, interside SNAP amplitude ratio was calculated.
Table 2. Results of Neurophysiological parameters of the three patients presented with cervical ribs in cervical plain X-ray examination.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Median CMAP amplitude/mV</th>
<th>Ulnar SNAP amplitude/µV</th>
<th>MABCN SNAP ratio</th>
<th>Needle Examination</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Right</td>
<td>Left</td>
<td>Right</td>
<td>Left</td>
</tr>
<tr>
<td>1&lt;sup&gt;st&lt;/sup&gt; patient, Male</td>
<td>2.1*</td>
<td>3.5*</td>
<td>17.0</td>
<td>10.4</td>
</tr>
<tr>
<td>2&lt;sup&gt;nd&lt;/sup&gt; patient, Male</td>
<td>21</td>
<td>17</td>
<td>16</td>
<td>13</td>
</tr>
<tr>
<td>3&lt;sup&gt;rd&lt;/sup&gt; patient, Female</td>
<td>13</td>
<td>8.5</td>
<td>11.7</td>
<td>10.1</td>
</tr>
</tbody>
</table>

*Abnormal values

Fig. (3): Plain cervical x ray showing bilateral bony cervical ribs.
Table 3. Mean values of distal latencies (DL), conduction velocities (CV), and CMAP amplitudes in motor nerves in upper and lower limbs of control group.

<table>
<thead>
<tr>
<th>Nerves studied</th>
<th>NC variables</th>
<th>Distal latencies</th>
<th>Motor conduction velocities</th>
<th>CMAP amplitudes</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mean±SD</td>
<td>Mean±SD</td>
<td>Mean±SD</td>
</tr>
<tr>
<td>Median nerve</td>
<td></td>
<td>3.0±0.3 msec</td>
<td>60.3±4.7 m/sec</td>
<td>16.0±6.0 mV</td>
</tr>
<tr>
<td>Ulnar nerve</td>
<td></td>
<td>2.4±0.3 msec</td>
<td>64.1±6.2 m/sec</td>
<td>14.0±4.0 mV</td>
</tr>
<tr>
<td>Radial nerve</td>
<td></td>
<td>2.2±0.4 msec</td>
<td>63.8±5.1 m/sec</td>
<td>15.0±5.3 mV</td>
</tr>
<tr>
<td>Common Peroneal</td>
<td></td>
<td>3.9±0.4 msec</td>
<td>53.7±3.6 m/sec</td>
<td>12.0±3.5 mV</td>
</tr>
<tr>
<td>Posterior Tibial</td>
<td></td>
<td>3.7±0.5 msec</td>
<td>49.3±3.2 m/sec</td>
<td>19.6±7.1 mV</td>
</tr>
</tbody>
</table>

Values beyond ‘two standard deviations’ from normal were considered abnormal.

Table 4. F response in control group.

<table>
<thead>
<tr>
<th>Nerve (distal site)</th>
<th>Mean (msec)</th>
<th>Range (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median (thenar)</td>
<td>26.0</td>
<td>21-30</td>
</tr>
<tr>
<td>Ulnar (hypothenar)</td>
<td>26.3</td>
<td>21-31</td>
</tr>
<tr>
<td>Posterior tibial (AHB)</td>
<td>48.4</td>
<td>39-57</td>
</tr>
<tr>
<td>Common peroneal (EDB)</td>
<td>47.1</td>
<td>36-57</td>
</tr>
</tbody>
</table>

Values beyond ‘two standard deviations’ from normal were considered abnormal.
AHB: Abductor hallucis brevis
EDB: Extensor digitorum brevis

Table 5. Mean values of distal latencies (DL), conduction velocities (CV), and SNAP amplitudes in sensory nerves in upper and lower limbs of control group.

<table>
<thead>
<tr>
<th>Nerves studied</th>
<th>NC variables</th>
<th>Distal latencies</th>
<th>Sensory conduction velocities</th>
<th>SNAP amplitudes</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mean±SD</td>
<td>Mean±SD</td>
<td>Mean±SD</td>
</tr>
<tr>
<td>Median nerve</td>
<td></td>
<td>2.8±0.1 msec</td>
<td>61.0±4.9 m/sec</td>
<td>35.0±12.0 µV</td>
</tr>
<tr>
<td>Ulnar nerve</td>
<td></td>
<td>2.5±0.1 msec</td>
<td>60.0±5.2 m/sec</td>
<td>15.0±4.2 µV</td>
</tr>
<tr>
<td>Radial nerve</td>
<td></td>
<td>2.6±0.2 msec</td>
<td>59.0±4.1 m/sec</td>
<td>28.0±4.0 µV</td>
</tr>
<tr>
<td>Sural nerve</td>
<td></td>
<td>3.1±0.3 msec</td>
<td>48.2±3.5 m/sec</td>
<td>16.3±5.9 µV</td>
</tr>
</tbody>
</table>

Values beyond ‘two standard deviations’ from normal were considered abnormal.

DISCUSSION

Results of this study showed that none of the neurophysiologically proved CTS patients included, had reduction of Ulnar nerve SNAP amplitude. This can be explained in two ways: firstly, patients who showed electrophysiological evidence of ulnar nerve entrapment at wrist or elbow (segmental, and or inter side comparative significant slowing) were excluded from this study, secondly, none of patients showed evidence of lower brachial plexus lesion that could have contributed to a low ulnar nerve SNAP.

Motor study of the median nerve showed that CMAP amplitude was significantly reduced in 19 (13.86%) patients, and that the reduction was bilateral in 7 (36.84%). This finding could be a sequel of severe prolonged entrapment of median nerve at wrist complicated by secondary axonal.
lesion. This explanation might be supported by the fact that nearly 37% of those 19 patients were having bilateral CTS, which usually builds up sequentially, thus taking some time long enough for axonal pathology to ensue. Another possible explanation is coexistence of a lower brachial plexus lesion. As a matter of fact, median nerve CMAP represents mainly a motor function of T1, which is also represented sensorily by MABCN SNAP\(^5,25,26\). So if there is a lesion compromising T1, one can expect affection of median CMAP, and of MABCN SNAP as well, which was not the case in our study. In conclusion, it can be reported that the reduction of median CMAP was mostly a consequence of entrapment of median nerve at wrist rather than of a lower brachial plexus lesion.

Neurophysiological study of MABC nerve in all CTS patients showed within normal SCV, and inter side amplitude ratio. The mean values ± SD of inter side amplitude ratio was 1.273±0.221, which is less than the ratio of 2.0 required for the diagnosis of predominantly T1, and to a lesser extent, C8 involvement in lesion of lower trunk, as well as medial cord of the brachial plexus. In fact, MABCN predominantly carries sensory fibres from the T1 root, which is the first to suffer from angulation on cervical rib, or fibrous bands connecting the first thoracic rib to the scalene tubercle or other spine structures\(^27\). Kothari and colleagues\(^4\), in their study on patients with electrophysiologically proven NTOS have supported the findings of Nishida et al.\(^7\) and recommended that the MABCN sensory study be performed when other standard electrophysiological tests have failed to confirm or satisfactorily localize a lower trunk brachial plexopathy. Subsequently, we can conclude that neurophysiologically evidenced NTOS could not be detected in our CTS patients and we might state that NTOS is still rare in CTS patients, at least less than 1 per 137 patients that meets with Seror\(^22\) findings.

As far as concordance of prominent C7 transverse processes or cervical ribs with NTOS is concerned, bilateral bony cervical ribs were reported in three patients. These patients showed within normal MABCN SNAP inter side amplitude ratio, ulnar SNAP amplitudes, and median CMAP amplitudes except one male patient who showed bilateral reduction of median nerve CMAP, and incomplete interference pattern of adductor pollicis brevis and considered as severe CTS with secondary axonal lesion of the median nerve. This observation could tell us that not all cases of cervical ribs are associated with NTOS, while NTOS cases are frequently (12-100%) associated with cervical rib as compared with the 0.2 or 0.5% expected in the general population\(^5,28,29\).

The above mentioned findings may conclude that NTOS is rare, and its association with CTS may be a mere coincidence, or both have a common aetiology. This calls for re-evaluation of the DCH., particularly as an aetiological factor for the clinical association of CTS and NTOS.

Although the DCH has some experimental and considerable clinical support, present understanding of the anatomy and physiology of peripheral nerves is largely inconsistent with the most common clinical example of this hypothesis which is CTS superimposed on a cervical radiculopathies (CR). CR leading to disruption in axoplasmic flow proximal to the dorsal root ganglion would not be expected to cause distal dysfunction or demyelination of that same axon\(^30\). Wilbourn and Gilliatt\(^30\) suggested that the most common clinical example of the DCH is an increased predisposition to CTS in patients with CR, and reported it as physiologically unsound, where a radicular lesion should have no effect on frequency of CTS and CR should have no effect on distal sensory conduction studies, no effect on distal myelin, and minimal effect on distal motor axon function of the median nerve.

Based on the fact that median sensory component is of C6/C7 origin, and its motor component is primarily of C8 origin, Richardson et al.\(^31\) hypothesized that C6 and/or C7 cases would demonstrate an increased frequency of median sensory mononeuropathy, and C8 cases would demonstrate an increased frequency of median motor mononeuropathy. They also
hypothesized that median sensory and motor response parameters among the same groups would be altered in ways consistent with a proximal influence on distal nerve conduction studies. Although median mononeuropathy was unexpectedly common (22.1%) among cases of CR (which may explain the clinical acceptance of the double crush hypothesis), none of their hypothesis on pattern of median neuropathy determined according to the affected cervical root was supported. This study found no evidence to support a neurophysiological explanation for the double crush hypothesis.

It is unclear why CR and CTS seem to coincide frequently. May be both disorders have common predisposing factors such as upper extremity overuse, or osteoarthritis leading to both cervical foraminal and carpal canal stenosis. Previous work has identified an increased incidence of CTS in patients with cervical arthritis, and small carpal canal size. Upper extremity weakness and pain in patients with CR may cause changes in biomechanics and usage patterns leading to increased upper extremity oedema with resultant increased carpal canal pressures.

Many studies supporting the DCH have often defined CR by clinical or radiological evidence and this may raise the possibility of misdiagnosis. Finally we can summarize that neurophysiologically assessed CTS was not proved to be associated with NTOS that was defined neurophysiologically as ‘low median nerve CMAP amplitude, low ulnar nerve SNAP amplitude, and side to side MABCN SNAP amplitude ratio greater than 2, and the hypothesized relationship between them could not be obtained, and this might call for re-evaluation of the DCH. Also not all cases of cervical rib would be accompanied by NTOS. And we can conclude that NTOS is a rare medical condition, and patients presenting with pain in upper limbs even with x-ray evidenced cervical ribs do not have to be diagnosed as cases of NTOS, unless after thorough clinical examination and specific/sensitive electrophysiological work up.

REFERENCES


الملخص العربي

ارتباط متلازمة مخرج القفص الصدري العصبيه مع متلازمة النفق الرسغي؟

الهدف من البحث:

أن الهدف من هذا البحث هو تمييز أو اختيار افتراضية التحقييم المزدوج ومثال لذلك هو دراسة معدل حدوث متلازمة مخرج القفص الصدري العصبية مع متلازمة النفق الرسغي.

طرق البحث:

انتمت هذه الدراسة على 137 مريضاً من مرضى متلازمة القفص الرسغي وكانوا 91 (66.4%) من المرضى الذكور و46 (33.6%) من المرضى الذكور وقد تراوحت اعمار المرضى ما بين 23 الى 48 عاماً وقد تم تشخيص متلازمة القفص الرسغي معتمداً أساساً على المعايير الكهروفسيولوجية وهي تضمنت الآتي: ضعف قوة الجهد الكهربائي الحركي للعصب.

- فترة الخفاء الحركي القصوى لع鲶ة الاهام المبتدئة القصيرة أكثر من 4 ملي/ثانية.
- سرعة التوصيل الحسية المعتدلة من الأصبع الثالث إلى الرسغ أقل من 45 متر/ثانية.
- الفرق بين الخفاف الحسية المعتدلة للعصب الأوسط و الزلندي للاصبع الرابع أكثر من 0.4 ملي/ثانية.

بالنسبة للمرضى الذين شكل التخصص بالتهاب الأعضاء الطفيفة أو احتقان (اختناق) في العصب الزلندي في الرسغ أو الكوع فقد تم استبعادهم من الدراسة.

أيضاً قد تضمنت هذه الدراسة عشرين شخصاً من الأصحاء وتمجاني من ناحية السن وجنس المرضى كمجموعة ضابطة.

تم إخضاع المرضى والمجموعة الضابطة إلى:

1. فحص أكلينيكي للجهاز العصبي مع التركيز على اعراض وعلامات مرض التهاب الأعصاب واختناق العصب الزلندي.

وذلك لتم استبعادهم أيضاً متلازمة مخرج القفص الصدري ومتلازمة النفق الرسغي.

2. الدراسات الكهروفسيولوجية للمرضى وكانت كالآتي:

- دراسات مزدوجة لسعة التوصيل الحسية/الحركي للعصب الأوسط والزلندي.
- دراسة مزدوجة لتوصيل الحسية غير معدل للعصب المتوسط الجليد قبل العضدي مع حساب نسبة (معدل) الجلد الحسوي بين الطرفين وينبغي أن تكون أكثر من 2.0 وذلك تشخيص اصابة العصب المتوسط الجليد قبل العضدي على أحد الأطراف.
- دراسة رسم العضلات المعززة من الجلد العصبي العنقي الثانى والظهر الأول.

بالنسبة إلى المعايير الكهروفسيولوجية التي استخدمت في هذه الدراسة تشخيص متلازمة مخرج القفص الصدري العصبية كانت كالآتي:

- ضعف قوة الجهد الكهربائي الحركي للعصب الأوسط.
ضعف قوة الجهاد الكهربائي الحسية للعصب النخسي.

ضعف أو ارتفاع قوة الجهاد الكهربائي الحسية للعصب النخسي.

ضعف النموذج الداخلي للعضلات المغذية من الجذور العصبية العقنية الثامن والظهرى الأول.

تم عمل أبحاث أكس عديدة على الفوات العقنية للمرضى بأعراض المطرف العلوي غير تقليدية.

أما بالنسبة للمجموعة الضابطة فقد تم دراستها كهروفسيولوجيًا دراسة كاملة واخيرًا قد تم تجميع البيانات ومقارنة المتوسطات إحصائيًا كما في النتائج.

نتائج الدراسة:

أظهرت الدراسة النتائج التالية:

1. قوة الجهاد الكهربائي الحسية للعصب النخسي كانت في خلال النسبة الطبيعية وكان متوسطها مع مقدار الانحراف
   ±4.8 ±14.08

2. تبين وجود ضعف في الجهاد الحركى للعصب الأسيوط في 19 مريضاً (13.86%) وكان هذا النقص مزدوجا في 7
   %36.84 من المرضى ومتوسط ذلك ± مقدار الانحراف 12.13±0.58 بالمقارنة مع 0.79.

3. معدل الجهاد الحسية للعصب المتوسط الجلدي قبل العضدي للطرفين العليا في جميع المرضى كان أقل من 2.0

4. رسم العضلات أظهر نموذج داخلي غير كامل لعضلة الإبهام السفلي القصيرة في 11 مريضاً (8%) وكان ذلك
   مزدوج في 7 مريض (63.6%).

5. أظهرت النتائج وجود نقص عظمي عقبي في 3 مرضى ولكن لم يظهر فيهم علامات أو مشاكل متزامنة مع
   متلازمة مخرج القفص الصدري العصبية.

ملخص البحث:

أظهر البحث للمؤثرات الكهروفسيولوجية عدم وجود ارتباط بين متلازمة مخرج القفص الصدري العصبيه ومتلازمة
الفق الرسغي وقد تؤدي هذه النتائج إلى إعادة تحليل ودراسة لانفرادية التحليل المزدوجة واحترام يمكن القول بأن متلازمة
مخرج القفص الصدري العصبيه هي حالة طبية نادرة ولا يجب التسرع بتشخيص وبالتالي علاج هذه المتلازمة الا بعد إجراء
فحص اكليتيكي دقيق للجهاز العصبي مصحوب بدراسات كهروفسيولوجية كاملة.