Cervical myeloradiculopathy with entrapment neuropathy: a study based on the double-crush concept

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In an attempt to formulate a standardised approach to the diagnosis and management of patients with the double-crush syndrome, we reviewed 65 surgical cases (39 men and 26 women) with cervical myeloradiculopathy associated with entrapment neuropathy in the arm by examining the clinical features, results of nerve conduction velocity studies and distribution of the vertebral levels of compression. Fifty-three patients (average age, 43 years) presented with a carpal tunnel syndrome at the wrist and cervical lesions (41 with spondylosis and 12 with ossified posterior longitudinal ligament), while 12 had a cubital tunnel syndrome at the elbow with cervical lesions (eight with spondylosis and four with ossification of the posterior longitudinal ligament). In the former group, the lesions were found mostly at C5–6 and C6–7 levels, while in the latter group involvement of C6–7 was frequently observed. Two patients in each group required additional cervical decompression after carpal or cubital tunnel release. A retrospective review of our patients suggested that it is reasonable to pursue an accurate diagnosis followed by treatment based on individual neurological and electrophysiological findings, but taking into consideration the significant level of physical impairment associated with cord compromise, we believe that it may be reasonable to perform a cervical decompression rather than peripheral nerve release in such cases. Our findings suggest that one should be aware of a double-crush during examination of patients complaining of neck and hand problems.

Keywords: cervical myeloradiculopathy; entrapment neuropathy; double-crush syndrome

Introduction

Neurological symptoms and signs in the upper extremity may suggest the coexistence of cervical myeloradiculopathy and entrapment neuropathy of the peripheral nerves.¹ Impingement of the spinal cord, spinal nerves and peripheral nerves of the arm may result in a more complex clinical presentation. Reduced axonal transport in orthograde and/or retrograde pathways may reflect clinically compromised neural conduction and, in turn, mechanical compression of the peripheral nerve may also affect normal axonal flow. Since Upton and McComas's² description, some reports^{3,4} have studied the clinical presentation of this neurological complex (double-crush syndrome), but the diagnosis and treatment are in fact not always easy to deal with in clinical practice. During the last two decades, we have encountered a number of patients who presented with features of the double-crush syndrome, but the available clinical literature on clinical management of such cases seemed to be

limited. We treated these patients rather on a case by case basis, but we have been keen to establish some clinical standards for neurological management of these patients.

The present study was thus designed to retrospectively review our patients who were suspected to have double-crush extending from the cervical cord to peripheral nerves of the upper arm. This short communication also describes our views on the management of such patients.

Patients and methods

Patients

Using our computerised database, we reviewed the records of all patients who underwent cervical cord and nerve root decompression ($n=483$) and upper arm peripheral nerve decompression ($n=285$; carpal tunnel release, 177; and cubital tunnel release, 108) for a proven carpal or cubital tunnel syndrome. These patients represented all those seen in our two University Medical

Centres between 1982 to 1995. A total of 65 surgical cases were confirmed to have a double-crush lesion from the neck to the arm and were enrolled in the present study. The group consisted of 39 men and 26 women. Their average age at operation (cervical decompression or peripheral nerve decompression) was 45.8 years (range, 28 to 72 years). Five patients also had mild diabetes mellitus requiring dietary therapy.

Fifty patients underwent cervical decompression (anterior surgery⁵ in 31 patients; C3–C7 *en bloc* open door laminoplasty^{6,7} in 18; and combined anterior and posterior cervical decompression in one). Of these, two patients underwent carpal tunnel release prior to anterior cervical decompression, and another two patients received cubital tunnel decompression before an anterior fusion in one and laminoplasty in another. Three patients presented with bilateral hand symptoms. Nineteen patients underwent carpal ($n=14$) or cubital ($n=5$) tunnel decompression. None had bilateral cubital tunnel syndrome, and none required additional carpal or cubital tunnel release after cervical decompression.

The patients were conveniently subdivided into groups I and II. The first group consisted of 53 patients who presented with cervical lesions and median nerve palsy (carpal tunnel syndrome), while those of group II ($n=12$) had cervical disease together with ulnar nerve palsy (cubital tunnel syndrome). Representative clinical data, results of median and ulnar nerve conduction velocity measurements, and radiological findings were investigated. The present study was based on the preoperative data of such examinations.

Clinical examination and electrodiagnosis

Radiological examination included cervical plain radiographs (posteroanterior and lateral) and computed tomography myelography in patients seen during the early part of the study, and magnetic resonance imaging. Radiography of the elbow sulcus (tangential view) was taken when needed, but axial viewing of MRI for the carpal tunnel was not performed due to limitation of medical insurance. All patients had compressive lesions of variable degree on radiological examination, such as spondylosis or ossification of the posterior longitudinal ligament (OPLL), at mid- and lower-cervical regions. Seven patients (six men and one woman) showed radiographic abnormalities in the

elbow joint; cubitus valgus deformity (carrying angle $\geq 20^\circ$) in six cases, osteoarthritis of the elbow joint and hypertrophic osseous changes involving the sulcus ulnaris in four.

Epidural spinal cord evoked potential and cortical somatosensory evoked potential recordings were indicated in some patients (spinal cord evoked potential in 17 patients and cortical evoked potential in 13) when needed, to determine the level(s) of myelopathy and assess the severity of cervical cord dysfunction. Our techniques of eliciting these evoked potentials have been detailed previously.^{8–10} However, the results of these tests were unable to be statistically analyzed due to the small population sample.

We also measured the orthodromic sensory and motor nerve conduction velocities (SCV and MCV) of the median and ulnar nerves at the carpal tunnel and the sulcus ulnaris, respectively. The technique to measure the conduction velocity was similar to that described by Oh.¹¹ Denervation discharge, positive sharp wave and fibrillation in the electromyogram, were used as diagnostic criteria of carpal and cubital tunnel syndromes. Large fasciculation potential and high amplitude NMU discharge (≥ 4 mV) were frequently observed, indicating primarily spinal cord involvement.

Statistical analysis

Differences between the groups were examined by one-way layouts analysis of variance (ANOVA) and non-parametric Wilcoxon's signed rank test. A P value less than 0.05 denoted the presence of a statistically significant difference.

Results

Clinical presentation

The average age of the patients in group I was 42.8 years (range, 28–67 years), which included 32 men and 21 women. Patients in group II were slightly older (average, 50.3 years, range, 38–72 years), albeit insignificantly than those of the first group, and consisted of seven men and five women. No difference in gender was noted between the two groups. Tables 1 and 2 summarise the clinical features and treatment modalities in groups I and II, respectively. The clinical features were not different between the two groups.

Table 1 Clinical features and treatment modalities in patients of group I

Treatment	n	Increased spasticity	Sensory disturbance ^a	Radicular signs ^b	Muscle atrophy	Phalen's or Tinel's sign
Cervical decompression	39	19	39	28	10	7
Cervical and carpal decompression	2	0	2	2	1	2
Carpal tunnel release	12	2	12	5	4	11

^a sensory disturbance at palm and index to ring fingers; ^b radicular sign includes positive Spurling's test and Lhermitte sign

Table 2 Clinical features and treatment modalities in patients of group II

Treatment	n	Increased spasticity	Sensory disturbance ^a	Radicular signs ^b	Positive Froment' sign	Claw hand deformity
Cervical decompression	7	5	7	7	4	4
Cervical and cubital decompression	2	1	2	2	1	1
Cubital tunnel release	3	0	3	1	2	2

^asensory disturbance at ulnar side of the forearm and ring and little fingers; ^bradicular sign includes positive Spurling's test and Lhermitte sign

Figure 1 The frequency distribution of the affected, or abnormal, vertebral levels in groups I and II

Radiological findings and conduction velocity

In group I, 41 patients had cervical spondylosis, while the other 12 had cervical OPLL. In group II, cervical spondylosis was detected in eight cases and OPLL in four. There was no significant difference between groups I and II with respect to the cervical spine disease.

Figure 1 shows the spinal levels that were considered to be most responsible for myeloradiculopathy (in patients with cervical decompression), or was considered abnormal (in cases with median or ulnar nerve decompression alone). In group I, C5–6 and then C6–7 levels were frequently involved, while in group II seven patients showed abnormal findings (spondylotic osteophyte or OPLL) at C6–7 level. However, there was no significant difference in the distribution of the affected spinal level between the two groups.

Table 3 Conduction velocity of the median and ulnar nerves

Treatment	unmeasurable	Conduction velocity of the nerve (m/sec)		
		< 20	20 to 40	≥ 40
Group I				
cervical decompression (n=39)				
SCV	0	0	11	28
MCV	0	0	4	35
cervical and carpal decompression (n=2)				
SCV	1	0	1	0
MCV	1	0	0	1
carpal tunnel decompression (n=12)				
SCV	3	5	4	0
MCV	1	3	2	6
Group II				
cervical decompression (n=7)				
SCV	0	1	1	5
MCV	0	0	1	7
cervical and cubital tunnel decompression (n=2)				
SCV	0	1	1	0
MCV	0	0	2	0
cubital tunnel decompression (n=3)				
SCV	2	0	1	0
MCV	1	1	1	0

SCV and MCV, sensory and motor nerve conduction velocities. In group I, SCV and MCV were measured from the median nerve at wrist, and in group II, those were measured from the ulnar nerve at the elbow

Table 3 shows the preoperative measurements of orthodromic SCV and MCV in both groups. Patients were rated according to the motor and sensory conduction velocities. In group I, 72% (28) of cases who ultimately required cervical decompression had SCV ≥ 40 m/sec, while patients who required carpal tunnel decompression had SCV less than 40 m/sec. Similar findings were observed in group II patients. No significant differences were noted in SCV and MCV between the groups.

Discussion

Upton and McComas² postulated that compression of the proximal end of a nerve could reduce its ability to withstand further compression at its distal end. Compressive lesion(s) of the spinal cord and the brachial plexus may thus increase the vulnerability of the median and ulnar nerves at the sites where entrapment neuropathy is frequent.^{1,12,13} Decreased axonal flow at the proximal point of neural impingement is believed to contribute to the increased vulnerability of the nerve and incidence of peripheral nerve entrapment syndrome.^{12,13} Epstein *et al.*⁴ described six patients with cervical spondylosis who had also bilateral carpal tunnel syndrome. These clinical findings signify the role of cervical cord compression in the double-crush syndrome. While double-crush neural injury is infrequently reported and thus the treatment strategy may be difficult to describe, we believe that recognition of this concept is of clinical importance for neurologists and neurosurgeons.

Upton and McComas² also reported that 62 (73%) of 85 patients with carpal tunnel syndrome had cervical lesions, but Dorward¹⁴ showed that only 6.7% of 150 patients with cervical spondylosis had carpal lesions. In another study, Hurst *et al.*¹⁵ reported that the prevalence of carpal tunnel syndrome was 11% (95/888) in patients with cervical compressive lesions. Although a more detailed statistical analysis is necessary, in our present review, 13% of patients who underwent cervical decompression presented with symptoms and signs consistent with carpal tunnel or cubital tunnel syndrome. Conversely, 23% of cases who required carpal or cubital tunnel release had mid- to lower-cervical compressive, or suspected compressive, lesions of the cord and/or nerve roots. A wide variability in the prevalence of the double-crush syndrome is expected partly due to the variability in the clinical criteria used for the diagnosis of both conditions and, therefore, the true incidence remains unclear. However, it is important to determine at an early stage and in an objective manner the extent of neural damage. As Epstein *et al.*⁴ have stressed, such patients require an exhaustive neurodiagnostic assessment.

Interestingly, 2% (12/483) of our patients requiring cervical decompression presented with clinical features of a cubital tunnel syndrome. Of these patients, seven had radiographic features of a cubitus valgus

deformity (carrying angle $\geq 20^\circ$), osteoarthritis of the elbow joint and hypertrophic osseous changes in the sulcus ulnaris, while the counterpart did not show such abnormalities. Most studies^{2-4,14,15} reported the presence of a median nerve palsy in association with cervical lesions, however, we speculate that such cumulative injury may also occur in the ulnar nerve. Unfortunately, we do not have enough data to accurately estimate the prevalence of ulnar nerve palsy. It is possible that the relatively low frequency of compressive lesions at C6–7 and C7–T1 levels (ulnar nerve level), compared with that between C4–5 and C6–7 (median nerve level), may explain the low prevalence of double-crush affecting the ulnar nerve.¹⁶ In the present study, however, the level of vertebral involvement was not different between the two groups.

Proper management of the double-crush syndrome, including surgery, depends primarily on the extent and severity of neural damage and subsequent neurological impairment. In addition, the severity of clinical symptoms should be taken into consideration when selecting the most appropriate surgical intervention. For example, cervical lesions associated with persistent palmar dysesthesia and characteristic night pain may require a surgical decompression of the carpal tunnel. In contrast, a slowly progressive spastic palsy may require cervical decompression even in a case which shows the effect of transient immobilisation combined with medications on proven ulnar nerve palsy. Therefore, a decision as to which lesion should be treated first is an issue of great debate. In our recent experimental studies using wheat germ agglutinin-horseradish peroxidase-labelling method,^{17,18} we described improvement in the retrograde axonal transport that correlated with functioning spinal cord motoneurons (spinal accessory motoneurons). However, extensive mechanical compression of the cord and nerve roots may result in serious neuronal damage even in a case with normal retrograde axonal transport. Considering the significant level of physical impairment associated with cord compromise, we believe that it may be reasonable to perform cervical decompression rather than peripheral nerve release in such cases. One illustrative case is shown in Figure 2 in whom cervical laminoplasty with left-sided foraminotomy⁷ at C6–7 and C7–T1 for extensive OPLL at the level was performed despite some neurological improvement following cubital tunnel decompression. This man with progressive myelopathy had insignificant elbow osteoarthritis involving the elbow sulcus, but presented with a history of considerable pain and dysesthesia in the ulnar side of the left hand which was associated with diminished orthodromic SCV across the elbow sulcus (26 m/sec), while orthodromic MCV at the same level was 41 m/sec. After laminoplasty, the patient improved neurologically with the exception of minimal numbness in the left little finger. At follow-up about 1 year later, SCV at the elbow was measured as normal (53 m/sec). While there was a lack of evidence

Figure 2 (a) T2-weighted (TR,2449 ms; TE, 100 ms) magnetic resonance image showing severe cord compression between C5 and C7, with hyperintense intensity signal of the cord, 6; C6 vertebra. (b) epidural spinal cord evoked potentials showing abnormal wave at C6 and C6-7 levels; (1) C6 level, (2) C6-7 level, (3) C7 level, (4) C7-T1 level. (c) ulnar nerve-stimulated cortical somatosensory evoked potentials showing insignificant abnormalities in the P11, P13, and N18 spikes (upper, left-sided response; lower, right-sided response). (d) a radiograph taken 3 years after surgery showing excellent enlargement of the cervical canal

in the present study for improvement of median or ulnar nerve conduction velocities following cervical decompression, it is our impression that cervical decompression diminished the severity of pain and dysesthesia in several patients.

In conclusion, by presenting our experience in 65 cases, we stressed the significance of double-crush between the cervical cord and peripheral nerves of the upper extremity. The median nerve appeared to be more likely involved in this process than the ulnar nerve. Though comprehensive neurodiagnostic guidelines are still not available, a thorough neurological workup should identify the coexistence of the double-crush lesions. Management may depend on the predominant site involved along the cervical cord and the nerve, but we suggest cervical decompression rather than peripheral nerve release in order to prevent the development of the more serious complication of tetraparesis.

Acknowledgements

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