



Carpal Tunnel Syndrome and Shoulder Pain with Particular Attention to Diabetic Patients

Shoulder pain may accompany carpal tunnel syndrome (CTS). We reviewed the clinical characteristics and electrodiagnostic findings of all patients with CTS over a 12-month period, with particular attention to diabetic patients, hoping to clarify the frequency of shoulder pain in CTS of diabetic (NIDDM) and nondiabetic patients. We found that shoulder pain was less common in the diabetic (13%) than the non-diabetic patients (49%). A tentative explanation for this observation is presented. We hypothesize that this may be due to the presence of a small-fiber neuropathy.

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The carpal tunnel syndrome (CTS) is the most common compressive neuropathy.¹ The major complaints of patients with CTS are burning pain, numbness, and tingling, usually in the thumb and palmar surface of the index and middle fingers. These symptoms are especially prominent at night and upon awakening in the morning.^{1,2}

In the pertinent literature of CTS, proximal forearm and shoulder pain is recurrently mentioned as an associated finding of CTS.³ In a review on CTS, Dick and Zadik state, "From our experience and also that of Kremer et al (1953), Garland et al (1957), and Heathfield (1957), it is clear that the original description [of carpal tunnel syndrome] must be widened to include cases which, while having the typical pain and paresthesia, also have wasting of the thenar muscles, impaired sensation in the median area, and pain spreading upward from the hands even as far as the shoulder."⁴

Dr. George Phalen's classic review on CTS states that pain may be referred to the forearm, elbow, or shoulder.² Das and Brown, reviewing complications in carpal tunnel decompression, describe 15 patients who complained of proximal pain; in all of these, the pain disappeared after surgical decompression of the median nerve.⁵

Peripheral neuropathy is a common complication of diabetes. CTS is the most frequent compression neuropathy in diabetes. CTS may occur more frequently in diabetics.⁶ It is postulated that an underlying peripheral neuropathy in diabetics makes compression neuropathy more likely. To our knowledge, shoulder pain in diabetic patients with CTS has not been specifically addressed in prior studies.

The purpose of our study was to investigate the frequency and clinical characteristics of shoulder pain in a group of patients with well-characterized CTS.

Additionally, we hoped to compare shoulder pain in diabetic versus nondiabetic patients. We felt this comparison might assist in our understanding of the mechanisms of shoulder pain in CTS.

Material and Methods

For a 12-month period, all patients who were specifically referred for electrodiagnostic studies with a diagnosis of CTS were evaluated and their electrodiagnostic studies and charts were reviewed. All electrodiagnostic studies were performed by one of the authors (JAC). The laboratory temperature was maintained at 25° centigrade, skin temperature was measured for all patients, and if necessary, the extremity was warmed. CTS was defined electrodiagnostically as an abnormality of one of the following (normal values for standard distances in our laboratory are listed in parentheses).^{1,7}

- 1) Prolonged median distal motor latency (>4.5 msec).
- 2) Prolonged and low-amplitude median mixed nerve action potential (palm to wrist [>2.0 msec, <50 uv]).
- 3) Prolonged and low amplitude median sensory nerve action potential (orthodromic stimulation [>3.5 msec, <6 uv]).
- 4) Fibrillation potentials confined to the abductor pollicis brevis (APB) and no electrophysiological evidence of a more proximal process, such as plexopathy or radiculopathy.
- 5) Normal median nerve conduction velocity and amplitude in the forearm segment.

If a generalized peripheral neuropathy existed by electrodiagnostic criteria, the patient was excluded from the study.

The charts were examined to document: the age and sex of the patient; duration of symptoms of CTS prior to the electrodiagnostic study; which hand was involved; whether shoulder pain existed in temporal association with CTS; symptoms related to CTS; roentgenograms (if performed) of neck, shoulder, wrist, and hands; associated diseases; neurodiagnostic tests; and response to various therapies for CTS. By performing this extensive review, we hoped to exclude all other causes for shoulder pain. The World Health Organization Report of 1985 was used for the confirmation of non-insulin-dependent diabetes mellitus patients.⁸ The patients included in the study were examined by a rheumatologist (D.H.) to exclude other causes for shoulder pain and were also evaluated by a neurologist (J.A.C.). Specifically, the diagnoses of cervical radiculopathy and significant arthritic involvement of the shoulder were excluded. The decision



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to perform x-rays was based on the best clinical judgment of a board-certified neurologist and a board-certified rheumatologist. We felt this represented the “real-world” situation of usual clinical practice in which x-rays are performed only when clinically indicated. Shoulder x-rays performed in eight patients demonstrated no significant joint pathology to account for their shoulder pain. Results of the treatment of CTS and the effect on the shoulder pain were evaluated in person or by telephone interview by one of the authors (J.A.C.).

Results

Eighty-five (85) patients with CTS were identified. Of this group, documentation of clinical characteristics and electrodiagnostic testing were judged to be adequate in 67 patients (94 extremities). The other 18 patients were excluded for the following reasons: Lack of cooperation for complete electrodiagnostic testing, lack of follow-up treatment, or failure of telephone follow-up. The distribution of diabetic (4) versus nondiabetic patients (14) who were excluded was approximately the same as that of the study patients.

Shoulder pain was present in 41% of the patients. Shoulder pain was less frequent in the diabetic (13%) versus nondiabetic patients (49%) (t test $p < .001$). The clinical characteristics of the diabetic (NIDDM) and nondiabetic patients were comparable with respect to sex, age, and side and duration of symptoms (Table 1). The two groups did not differ significantly in their response to therapy such as splints, injection, and surgery (Table 2). The following abnormalities of electrodiagnostic testing were significantly worse in the diabetic patients: median distal motor latency, median distal sensory latency, neuropathic changes of motor unit action potentials in the APB, and fibrillation potentials in the APB (Table 3).

A negative correlation ($r = -0.52$) existed between the presence of denervation potentials in the APB and the presence of shoulder pain.

Discussion

Shoulder pain is relatively common in CTS. In the group of nondiabetic patients it occurred in 49% of the patients. Interestingly, shoulder pain occurred less frequently in the diabetic patients (13%). The clinical characteristics were otherwise similar between the two groups. The electrodiagnostic data demonstrated more severe median nerve involvement in the diabetic patients. In particular, a negative correlation existed between the presence of fibrillation potentials in the APB and the presence of shoulder pain. This negative correlation may support the existence of a small-fiber neuropathy, since the fibrillation potentials suggest axonal injury.

Table 1. Clinical Characteristics

The clinical characteristics of the diabetic and nondiabetic patients were comparable with respect to:

	Diabetic (NIDDM) = 16	Nondiabetic = 51
Sex	15 Female (93%)	38 Female (75%)
Age	56 ± 9 years	49 ± 13 years
Side of CTS	Right - 11 (69%)	Right- 42 (82%)
Duration of Symptoms	19.9 ± 21.4 months	17.3 ± 15.5 months
Presence of SP	13%	49%

Table 2. Response to Therapy

	Diabetic (NIDDM)	Nondiabetic
Splints*	.44 ± .12	.45 ± .07
Injection*	.75 ± .25	.53 ± .16
Surgery*	1.54 ± .25	1.45 ± .18

0 = No response, 1 = partial response, 2 = complete response
* not significant by t test, between the groups

In a study of 60 diabetic patients with shoulder pain,⁹ 37 had hand syndromes such as Dupuytren's contracture or limited joint mobility, but only 6 of those (10% of all patients) had a history of CTS surgery and none had active CTS at the time of the evaluation. This study supports our observation that shoulder pain in diabetic patients is rarely associated with CTS.

We postulate that shoulder pain is less common in diabetic patients with CTS because of the presence of a small-fiber peripheral neuropathy which may interfere with the phenomenon of referred pain. We believe referred pain is the most likely mechanism of shoulder pain in the setting of CTS. We assume that the referred shoulder pain of CTS is dependent on distal nerve fibers.

Absence of shoulder pain in CTS of diabetic patients may thus be analogous to the absence of pain with myocardial ischemia in diabetic patients. This phenomenon of silent or painless myocardial ischemia in diabetic patients is explained by the loss of visceral afferent fibers,^{10,11} which are small, lightly myelinated or unmyelinated fibers. In diabetes, the peripheral neuropathy may affect small-fiber function. We hypothesize that this loss of small-fiber function may result in the loss of shoulder pain otherwise commonly associated with CTS.¹² Excluding patients who demonstrated a generalized peripheral neuropathy on electrophysiologic testing did not exclude the possibility that a small-fiber neuropathy existed in our patients, since nerve conduction studies only assess the largest, fastest myelinated fibers.¹³ Assessment of a small-fiber neuropathy is difficult at best, usually requiring a nerve biopsy for confirmation.¹⁴ The confirmation of a small-fiber neuropathy by autonomic testing is not always reliable.¹³

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Table 3. Electrodiagnostic Testing		
These were significant differences (t test p<0.05) as to the following electrodiagnostic values:		
	Diabetic (NIDDM)	Nondiabetic
Median distal motor latency*	5.9 ± 0.33 msec	4.9 ± 0.17 msec
Median distal sensory latency*	6.4 ± 0.9 msec	4.8 ± 0.18 msec
"Neuropathic"*** changes of MUAPs in the APB	93% ± .02	84% ± .06
Fibrillation potentials in the APB	91% ± .08	76% ± .07
* An absent potential = 7 msec for statistical purposes		
** High amplitude, prolonged duration, increased polyphasia of motor unit potentials		

In addition, the severity of the electrodiagnostic findings in diabetic patients may also be related to compromised small-fiber sensation, less perception of pain, and a later clinical presentation of CTS.

In summation, we believe that our study supports the relation between the presence of a small-fiber neuropathy and the absence of shoulder pain in diabetic patients. Unfortunately, the evidence is indirect because of the difficulties in conclusively documenting the presence of a small-fiber neuropathy. ❖

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" A poorly observed fact is more treacherous than a faulty train of reasoning."

Paul Valery, French philosopher