

THE ELECTRODIAGNOSTIC SENSITIVITY OF PROXIMAL LOWER EXTREMITY MUSCLES IN THE DIAGNOSIS OF L5 RADICULOPATHY

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ABSTRACT: The aim of this study was to assess the electrodiagnostic (EDx) sensitivity of proximal lower extremity muscles, including tensor fascia lata (TFL) and gluteus medius (GMED), in the diagnosis of L5 radiculopathy. Eleven EDx recordings with L5 radiculopathy were collected. The motor unit action potentials were assessed for morphology, stability, and firing characteristics. A descriptive analysis was performed. In proximal L5-supplied muscles, 4 of 11 recordings were abnormal in TFL only, with normal GMED; 4 of 11 recordings had similar findings in both muscles; 2 of 11 had abnormal findings in both muscles, but TFL had more noticeable findings; and 1 had abnormal findings in both muscles, but GMED findings were more noticeable. No patient had abnormalities limited to GMED. TFL was more sensitive than GMED in detecting L5 radiculopathy. Knowing which muscles are more likely to show abnormalities can improve the efficiency of EMG and reduce patient discomfort.

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Comparisons of the electromyographic (EMG) findings of proximal lower extremity muscles for single root lesions have not been performed in detail.^{1,2} Sampling a proximal L5 muscle is a key part of EMG to verify diagnosis of an L5 radiculopathy and to eliminate possible superimposed sciatic or fibular mononeuropathies or polyneuropathies.^{3,4} Thus, we investigated the electrodiagnostic (EDx) sensitivity of proximal lower extremity muscles, including tensor fascia lata (TFL) and gluteus medius (GMED), in the diagnosis of L5 radiculopathy.

METHODS

We collected prospectively 11 EDx recordings from 9 subjects with L5 radiculopathy. Those with EDx evidence of polyneuropathy or focal mononeuropathy were excluded from the study. In the clinical findings, 7 of 9 subjects had low back pain (LBP), foot drop (weakness of foot dorsiflexion and foot

eversion), and L5 dermatome numbness; 1 of 9 subjects had LBP and L5 dermatome numbness; and 1 of 9 had LBP and radiating leg pain. Radiological imaging of the patients showed herniated disks at the L4 and L5 levels. Of these patients, 4 previously underwent L5 discectomy surgery.

A routine EDx study was performed, including nerve conduction studies (NCS), using standard laboratory techniques. Routine needle EMG studies included examination of the tibialis anterior (TA), tibialis posterior (TP), medial gastrocnemius (MGast), vastus lateralis (VL), GMED, TFL, and paraspinal muscles. EMG testing was conducted by two board-certified electromyographers. Interpretation of the EMG was performed according to accepted guidelines⁵ in order to minimize inter-rater reliability.

The demographic features of patients were recorded, including gender, age, clinical symptoms, duration of symptoms, and time between surgery and EDx. A descriptive analysis (with number and percentages) was performed to assess abnormal findings.

RESULTS

Eleven EDx recordings were obtained from 9 patients. The average duration of symptoms was 31.2 ± 25.8 months (minimum 3 months, maximum 65 months). Four subjects (subjects 1, 2, 4, and 6) had laminectomy and/or fusion surgery due to L5 radiculopathy prior to electrodiagnostic testing. The duration between the surgery and EMG testing was 9 months for subject 1, 36 months for subject 2, 72 months for subject 4, and 51 months for subject 6. In subject 4, the symptoms initially started 6 years previously, prompting L5 laminectomy. Symptoms resolved after surgery, but returned 4 months prior to EDx.

In 5 subjects, NCS showed decreased amplitude of the fibular compound motor action potential (CMAP). Two studies showed reduced amplitude of tibial CMAP, possibly related to an unusual L5 contribution or coexisting S1 radiculopathy. NCS were normal in the other 4 subjects.

In the EMG assessments of *distal* L5-innervated muscles, 11 of 11 recordings from TA muscles and 3 of 3 recordings from TP muscles showed active

Abbreviations: AH, adductor hallucis; CMAP, compound motor action potential; EDB, extensor digitorum brevis; EDx, electrodiagnostic; EHL, extensor hallucis longus; EMG, electromyography; FL, fibularis longus; GMED, gluteus medius; MGast, medial gastrocnemius; NCS, nerve conduction study; TA, tibialis anterior; TFL, tensor fascia latae; TP, tibialis posterior; VL, vastus lateralis

Key words: electrodiagnostic sensitivity, gluteus medius, L5 radiculopathy, lumbosacral radiculopathies, tensor fascia latae

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Table 1. Comparison of denervation findings of TFL and GMED muscles.

Subject number	DOS (months)	TFL						GMED					
		Spontaneous		Volitional				Spontaneous		Volitional			
		Ins	Fib/ PSW	Rec	Amp	Dur	Poly	Ins	Fib/ PSW	Rec	Amp	Dur	Poly
1	15	Inc	0	SR	2+	2+	2+	Nml	0	SR	1+	1+	1+
2	65	Nml	0	SR	1+	1+	1+	Nml	0	Nml	0	0	0
3	24	Inc	0	MO	2+	2+	2+	Inc	2+	MO	2+	2+	2+
4	4	Inc	1+	SR	0	0	0	Nml	1+	SR	0	0	0
5	6	Nml	2+	SR	1+	1+	1+	Nml	2+	SR	0	0	0
6a*	52	Nml	0	SR	1+	1+	1+	Nml	0	SR	1+	1+	0
6b*		Nml	0	MK	2+	2+	0	Nml	0	Nml	0	0	0
7	60	Nml	0	Nml	1+	1+	1+	Nml	0	Nml	0	0	0
8	?	Nml	0	MO	1+	1+	1+	Nml	0	Nml	0	0	0
9a*	3	Inc	0	SR	1+	1+	1+	Inc	1+	SR	1+	1+	1+
9b*		Inc	0	Nml	0	0	1+	Inc	0	MO	1+	1+	0

Color scale of denervation: white = 0; light gray = +1; dark gray = +2; black = +3. Amp, amplitude; Dur, duration; DOS, duration of symptoms; Fib/PSW, fibrillation potentials/positive sharp waves; GMED, gluteus medius muscle; Inc, increased; Ins, insertion; MK, marked; MO, moderate; Poly, polyphasia; Rec, recruitment; SR, slightly reduced; TFL, tensor fascia lata.

*Both lower limbs were examined.

and/or chronic denervation. Subjects 1, 2, and 4 had active denervation findings with fibrillation potentials and positive sharp waves in distal L5-innervated muscles, including the TA and TP muscles. In addition, subjects 3, 4, and 5 had active denervation of paraspinal muscles based on EMG findings. No active distal denervation was detected in 3 recordings.

In assessing the EMG findings of proximal L5-innervated muscles, 4 of 11 recordings showed acute and/or chronic denervation (increased amplitude, increased duration, polyphasia, and decreased recruitment) in TFL only with normal findings in GMED; 4 of 11 recordings had similar findings in both TFL and GMED; 2 of 11 recordings had abnormal findings in both muscles, although TFL had more noticeable findings than GMED; and 1 recording had abnormal findings in both muscles, with GMED findings more noticeable than TFL (Table 1). No patient had abnormalities limited to GMED.

DISCUSSION

A logical muscle selection is essential for accurate diagnosis of lumbosacral radiculopathy.⁵⁻⁷ Different muscles innervated by the same myotome may not demonstrate similar EMG findings and may have different sensitivity.^{1,3,5} Based on two published myotome charts,^{5,8} we compared TFL and GMED for L5 radiculopathy.

Distal L5 muscles, such as TA, fibularis longus (FL), TP, and extensor hallucis longus (EHL), are sensitive in the EDx of L5 radiculopathy.^{4-6,9,10} In our study, the distal L5 muscles were more sensitive than the proximal L5 muscles. However, to verify the diagnosis of an L5 radiculopathy and to eliminate superimposed sciatic or fibular mono-

neuropathies or polyneuropathy, proximal L5-innervated muscles should be sampled.^{3-5,7} There is no consensus on which proximal muscles are more sensitive for EDx of L5 radiculopathy.^{1,3,5,6} The active and chronic denervation findings can be highly variable.^{3,5} In the Tsao et al. study,¹ the muscles affected by L5 radiculopathy included FL (16 of 16), TFL (4 of 4), GMED (13 of 26), TP (23 of 25), extensor digitorum brevis (20 of 24), TA (20 of 26), EHL (8 of 11), and paraspinal muscles (12 of 25). Although the number of TFL muscles sampled was smaller than for GMED muscles, the TFL was more sensitive for detection of abnormality. In another study,⁹ the most affected muscles were EHL (20 of 23; 87%), PL (16 of 19; 84%), TP (21 of 26; 81%), TA (32 of 41; 78%), TFL (14 of 57; 52%), and GMED (2 of 5; 40%) in L5 radiculopathy. In our study, the affected muscles included TA (11 of 11), TP (3 of 3), TFL (10 of 11), and GMED (7 of 11) in L5 radiculopathy. Overall, the distal L5 muscles were superior to the proximal L5 muscles for detection of active and/or chronic denervation.^{1,11} The TFL muscle was more sensitive than the GMED muscle in the diagnosis of L5 radiculopathy. However, if one accepts active denervation with fibrillation potentials as a more important indicator of muscle involvement, then the TFL is at least as sensitive as the GMED. It may also be more easily accessible in obese patients.

Active and chronic denervation findings were detected simultaneously (8 of 11 recordings; 72%) in most cases. The presence of myotomal fibrillation potentials and chronic neurogenic motor unit action potential changes is usually consistent with either chronic, ongoing, active radiculopathy or a recent active root lesion superimposed upon a

long-standing pre-existing one.⁵ The increased frequency of active denervation findings in distal muscles in our patients may relate to the nature of the reinnervation process, as proximal muscles are reinnervated earlier than distal muscles.^{3–5,12}

In conclusion, the TFL is at least as sensitive as the GMED for detection of L5 radiculopathy. Knowing which muscles are more likely to show abnormalities can improve the efficiency of EMG and reduce patient discomfort. Further studies that include larger numbers of patients are warranted to confirm our results.

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PROGNOSIS OF ACUTE COMPRESSIVE RADIAL NEUROPATHY

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ABSTRACT: *Introduction:* Small published case series suggest that compressive radial neuropathy is often a self-limited phenomenon with a favorable prognosis. Due to paucity of data, we sought to clearly define prognosis. *Methods:* To define clinical and electrodiagnostic features in this condition, we retrospectively reviewed consecutive cases of compressive radial neuropathy confirmed using electrodiagnostic studies at a large tertiary center over a 10-year period. *Results:* A total of 51 patients (26 men, 25 women, mean age 46 years \pm 15; range, 19–83 years) with compressive radial neuropathy were identified and reviewed. All patients in whom clinical follow-up was available (23 [45%] of the 51 patients identified) experienced complete recovery. Mean duration from onset to resolution of symptoms was 3.4 months. *Conclusions:* Our results support a good prognosis in essentially all patients with acute compressive radial neuropathies. This report provides valuable information to assist in counseling patients who may present with profound clinical deficits.

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Radial neuropathy is the third most common focal neuropathy of the upper limb, and the spiral groove is the most common site of injury.¹ Although reports have examined natural history and prognostic data in radial neuropathies associated with traumatic injuries, few address these topics in nontraumatic compression-related radial

neuropathy at the spiral groove (“Saturday night palsy”). In 1945, Sunderland reported seven patients with radial neuropathy related to compression, and complete recovery was noted in all within an average of approximately 4 months after injury.² Additional small case series in adult and pediatric populations suggested a good prognosis in 67–100%.^{3–5} We sought to define prognosis and describe the natural history of compression-related radial neuropathy at the spiral groove in a large cohort.

METHODS

Potential patients were identified from billing records over the last ten years at a large tertiary medical center using the diagnostic code of radial neuropathy (ICD-9 354.3). We performed a retrospective chart review of electrodiagnostic and clinical records. Patients were required to have at least one electrodiagnostic study with findings consistent with radial neuropathy at the spiral groove. Electrodiagnostic studies included, at minimum, the following on the ipsilateral limb: (1) Sensory conduction studies of the median, ulnar, and radial nerves; (2) Motor conduction studies of the median and ulnar nerves; and (3) Needle electrode examination of radial-innervated muscles as well as other muscles not innervated by the radial nerve. Ipsilateral radial motor conduction studies were performed in some but not all patients. Patients were excluded if electrodiagnostic studies demonstrated an alternative or concomitant

Abbreviations: CMAP, compound muscle action potential; SNAP, sensory nerve action potential

Key words: compression, electrodiagnosis, prognosis, radial neuropathy, Saturday night palsy

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diagnosis of C7 radiculopathy, posterior interosseous neuropathy, or brachial plexopathy. Patients were also excluded if there was a history of known trauma to the limb beyond simple compression, or if an alternate etiology of radial neuropathy such as vasculitis (mononeuritis multiplex), neuralgic amyotrophy, or multifocal motor neuropathy with conduction block was identified.

RESULTS

A total of 91 individuals with radial neuropathy who had at least 1 electrodiagnostic evaluation were identified. After patients with alternate diagnoses or a clear history of trauma beyond compression were excluded, the cohort included 51 patients (26 men, 25 women; mean age, 46 years \pm 15; range, 19–83 years) with compressive radial neuropathy. Examination demonstrated complete paralysis in a radial nerve distribution distal to the triceps muscle in 17 (33%). Sensory symptoms of numbness or paresthesia were noted in 26 (51%). In 13 (25%), mild burning or aching pain was noted, typically in the distribution of the superficial radial sensory nerve. Symptoms were first noted upon awakening in 22 (43%), and alcohol or opiate use was noted in 6 (12%) and 4 (8%), respectively. An increased incidence of coexistent systemic metabolic disorders was noted. Hypothyroidism and diabetes were documented in 12 (24%) and 7 (14%), respectively. In 26 (51%), no potential contributing factors were identified. In 23 (45%), clinical follow-up was available, and, in all, complete clinical recovery without residual weakness or sensory deficit was documented. In 14, the duration of symptoms was explicitly recorded in the chart; their mean time to complete recovery was 3.4 months (range, 0.5–6 months). Similar to the entire cohort, 11 (48%) of the 23 patients with clinical follow-up had no identifiable risk factor.

The radial sensory nerve action potential (SNAP) amplitude was frequently preserved and was abnormal in only 5 (17%) of the 29 studies performed at least 10 days after clinical onset. Radial SNAP conduction velocity across the forearm segment was normal in all cases. Radial motor conduction studies were performed 7 days or later in 21 of the patients. The distal radial compound muscle action potential (CMAP) amplitude was reduced in 2 (10%). Conduction block or reduced conduction velocity in the spiral groove were each noted in 12 (57%). Despite prominent conduction study features of demyelination or conduction block, abnormal spontaneous activity was present universally in all patients who underwent electrodiagnostic study at least 3 weeks after symptom onset.

DISCUSSION

This study represents the largest series of patients with acute compressive radial neuropathy. Other previous reports which described outcome and prognosis included no more than 7 subjects with compression radial neuropathy.^{2,3,5} The details how the cases were included were not described in these reports. Mondelli et al. reported the clinical and electrophysiological features of 91 consecutive cases of radial neuropathy of mixed etiologies. Of 27 cases of nontraumatic radial neuropathy, 10 cases were localized to the spiral groove. In this report no data was reported regarding outcome.¹ The limited data available prompted this study. The data from our cohort are consistent with the classical clinical presentation of compressive radial neuropathy with weakness in the distribution of the radial nerve, triceps sparing, and inconsistent sensory loss in the distribution of the superficial radial sensory nerve.^{2,6} Surprisingly, all patients in our series who returned for clinical follow-up had no residual clinical deficits. While follow up was available in only 45%, it is expected that patients who experience rapid spontaneous recovery are less likely to return for follow-up.

Electrodiagnostic data in our patients showed a high frequency of SNAP and distal CMAP amplitude preservation, suggesting that axon loss is less prominent as compared with focal demyelination, which agrees with the favorable clinical outcome. However, the universally noted abnormal spontaneous activity on the needle electrode examination indicates that mild motor axon loss does occur. Thus abnormal spontaneous activity in these patients does not necessarily portend a poor prognosis. In all patients, a radial SNAP could be recorded, and in the majority, the amplitude was normal using our laboratory's reference values. The frequency of radial SNAP amplitude preservation is particularly important to recognize as a potential cause for incorrectly diagnosing a preganglionic process (i.e., C7 radiculopathy) or motor predominant disease such as multifocal motor neuropathy.

Our results indicate that there is a good prognosis in essentially all patients with acute compressive radial neuropathies. This report provides valuable information to assist in counseling patients who may present with profound clinical deficits sometimes causing significant psychological distress.

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DOES THE PRESENCE OF THE PALMARIS LONGUS TENDON INFLUENCE MEDIAN NERVE FUNCTION?

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ABSTRACT: *Introduction:* The objective of this study was to determine if the presence or absence of a palmaris longus tendon influenced the function of the median nerve across the wrist. The primary hypothesis was that the presence of a palmaris longus tendon would be associated with more median nerve entrapment at the wrist. *Methods:* This was a cross-sectional study. Subjects were recruited at a screening of dental professionals. The median and ulnar sensory nerve latencies across the wrist (relative prolongation of the median compared with the ulnar) and the presence or absence of the palmaris longus tendon were the primary outcome measures. *Results:* A total of 462 subjects were recruited into the study of which 16.2% lacked a palmaris longus tendon. There was no difference in the median nerve function or the percentage with a 0.5 ms prolongation of the median sensory latency when comparing subjects with and without a palmaris longus tendon. *Conclusions:* The presence of a palmaris longus tendon does not influence the median nerve function across the wrist.

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The palmaris longus tendon is present in approximately 80% of people and attaches to the flexor retinaculum at the wrist near the carpal tunnel. The lack of a palmaris longus muscle is considered a normal variant. It is been suggested that presence of the palmaris longus tendon increases the pressure on the median nerve within the carpal tunnel and may increase the prevalence of carpal tunnel syndrome (CTS).¹ Several case reports have supported this supposition by demonstrating hypertrophied palmaris longus tendons as the possible cause of CTS.^{2–5} Recently, two studies have demonstrated conflicting results, one supporting the relationship between the presence of a palmaris longus tendon and a higher prevalence of CTS and one demonstrating the opposite.^{3,6}

Our goal was to study the relationship between the presence or absence of the palmaris longus tendon and its influence on median nerve function. We hypothesized that presence of the tendon would lead to more compression within the carpal tunnel and thus cause slowing of the median sen-

sory nerve conduction across the wrist.

METHODS

The study was done as part of a larger screening study for CTS in dental professionals at their annual meeting in 2011. The IRB of the American Dental Association approved the screening study. Participants chose to have the nerve conduction screening for CTS as one of many options at the health fair that is offered to registered dental professionals at their annual meeting. All subjects had nerve conduction studies of the median and ulnar sensory nerves at the wrist of their dominant hand. All studies were done using antidromic stimulation at 14 cm.⁷ The hands were warmed if the surface temperature was under 32°C. Peak latency, amplitude and palm temperature were recorded for each nerve and the median and ulnar peak latencies were compared. A 0.5 s difference was considered to indicate median mononeuropathy at the wrist. Presence of the palmaris longus tendon was confirmed by having the patient press the thumb and the fifth digit together while flexing the wrist. The examiner used visual inspection and palpation to carefully assess whether the palmaris longus tendon was present medial to the flexor carpi radialis tendon.

RESULTS

Of the 462 subjects tested, 75 (16.2%) did not have a palmaris longus. The median nerve conduction measures are shown in Table 1. The *t*-test for comparison of the mean peak latency and amplitude were not significant, $P = 0.34$ and $P = 0.58$ respectively. The chi-squared for comparing the percentage with a median mononeuropathy was not significant, $P = 0.99$. We did not find any influence of gender or hand dominance in this cohort, but only the dominant hand was evaluated.

DISCUSSION

Although there is some literature which suggests that the presence of a palmaris longus tendon may contribute to the prevalence of CTS, this study

Abbreviations: CTS, carpal tunnel syndrome; IRB, institutional review board

Key words: carpal tunnel syndrome, compression neuropathy, median nerve, palmaris longus, tendon

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Table 1. Median peak latency, amplitude, and % with a median mononeuropathy (median latency prolonged >0.5 ms compared to the ulnar peak latency) in subjects with and without a palmaris longus tendon.

	Median peak latency (ms)	Median amplitude (μ V)	% with median mononeuropathy
+ Palmaris longus, N=387	3.64 (0.03)	38.2 (8.7)	22.7
- Palmaris longus, N=75	3.56 (0.08)	27.3 (1.7)	22.7

Mean (standard deviation).

does not support that position. This is the largest study of this relationship and uses objective criteria for median nerve function. We chose to use objective outcome measures of median nerve latency and amplitude instead of subjective symptoms of numbness and tingling in the hand, which has a variable distribution in subjects with CTS.⁷ Using these measures of median sensory nerve function as an indicator of the health of the nerve, we have demonstrated that the presence or absence of the palmaris longus tendon has no influence and thus cannot be considered a risk factor for CTS. We recognize that the palmaris longus tendon does not pass through the carpal tunnel but terminates at its proximal end. Therefore, it theoretically could increase the pressure on the carpal canal, but this

study demonstrates that its presence does not have any effect on median nerve function. We believe that the use of objective measures of median nerve function is a better measure of median nerve health compared with the subjective symptoms associated with CTS. There is some variation of the anatomy of the palmaris longus muscle and tendon and as such, it may have a differential influence on the median nerve. This could be evaluated with high resolution ultrasound and should be considered in future studies.

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