Proximal neuropathies in patients with poststroke shoulder pain

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Background
Poststroke shoulder pain (PSSP) could be due to proximal neuropathy or upper trunk brachial plexus lesion.
Aim
The aim was to detect any electrophysiological abnormality in the proximal nerves supplying shoulder structures that could contribute to PSSP.
Settings and design
Cross-sectional study at institution: a university hospital, tertiary level of clinical care.
Materials and methods
Nerve conduction studies of the axillary, musculocutaneous, suprascapular, and lateral antebrachial nerves were done on both sides. In addition, electromyography of the deltoid, biceps brachii and infraspinatus on the hemiplegic side was performed on 30 stroke survivors with PSSP.
Statistical analysis used
Statistical Package for the Social Sciences (SPSS ver.20). Description and analysis of the obtained data were done using appropriate tests.
Results
Axillary and musculocutaneous motor nerve latencies on the hemiplegic side were significantly prolonged compared with the normal side (P=0.012, 0.029, respectively). Moreover, axillary and suprascapular nerve amplitudes on the hemiplegic side were significantly lower than those on the normal side (P=0.008, 0.002, respectively). Twelve (40%) patients had electrophysiological abnormalities. Upper trunk brachial plexopathy was the most common abnormality which occurred in six (20%) patients. In addition, isolated axillary or suprascapular nerve lesion occurred at a similar frequency (10%).
Conclusion
Proximal nerve lesions are not uncommon in PSSP patients.

Keywords:
brachial plexus, shoulder pain, stroke, survivors

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Introduction

Most patients with poststroke shoulder pain (PSSP) have nociceptive cause for their PSSP. However, a neuropathic cause may also contribute to PSSP. Damage to the upper trunk of the brachial plexus has been suggested to be responsible for PSSP [1,2]. Pulling the flaccid arm during moving the patient or lack of support of the paralyzed flaccid shoulder and/or the weight of the unsupported arm may cause traction damage or injury to the brachial plexus, axillary nerve, and/or the suprascapular nerve [2–4].

The current study was designed to assess proximal nerves supplying the shoulder muscles in patients with PSSP.

Materials and methods

This cross-sectional study was done on 30 stroke survivors (according to WHO) [5] with PSSP. Stroke duration was more than 1 month. Exclusion criteria: previous shoulder trauma, surgery, chronic inflammatory arthritis, and patients known to have peripheral neuropathy and/or other muscular disorders associated with shoulder weakness (e.g. polymyositis, dermatomyositis, myopathies). All participants had a nociceptive cause (according to clinical, laboratory, and imaging techniques) that could explain PSSP, for example, bicipital tendonitis, bursitis, and/or impingement syndrome.

Patients were recruited over 18 months from those attending the Outpatient Clinic of Physical Medicine in a university hospital in the area where the research was done. All participants were informed about the nature of the study and an informed consent was taken and approved by the local ethics committee.

Table 1 Upper limits for side-to-side difference in the studied nerves [12]

	Axillary	Musculocutaneous	Suprascapular	Lateral antebrachial sensory response
The upper limit for side-to-side latency difference (ms)	0.5	0.4	0.4	0.2 onset latency 0.3 peak latency
The upper limit for side-to-side amplitude difference (%)	54%	33%	48%	69% onset to peak amplitude 86% peak to peak amplitude

The following data were collected from all participants: (a) demographic data, (b) stroke history, (c) shoulder muscle power (using Medical Research Council scoring [6]), (d) shoulder subluxation (using the sulcus sign [7,8] and radiography), (e) shoulder muscle spasticity according to the modified-Ashworth scale [9] (spasticity was identified if the Ashworth score was ≥1).

The presence of atrophy and absence of spasticity of the supraspinatus, infraspinatus, deltoid and biceps muscles in the impaired upper extremity, in the presence of increased muscle tone or movement in the distal muscles, was suggestive of associated brachial plexus lesion [10].

Fugl-Meyer for upper extremity motor performance (FMUE) was used to assess the impairment level of the arm motor function. It is composed of 33 items, each item is rated on a three-point ordinal scale (0–2) [11].

Electrophysiological studies included: standard axillary, musculocutaneous, and suprascapular nerve motor conduction studies as well as lateral antebrachial sensory nerve conduction on both sides [12,13].

Needle electromyography (EMG) of the middle deltoid, biceps brachii, and infraspinatus muscles was performed on the hemiplegic side [13]. The following EMG parameters were recorded:

(1) Any spontaneous activity (sensitivity was set at 50 μV/division, analysis time was set at 100 ms).
(2) Recording of motor unit action potential (MUAPs) on minimal volition (sensitivity was set at 200 μV/division, analysis time was set at 100 ms).
    Normal MUAPs were considered if: MUAP amplitude is greater than 100 μV and less than 2 mV, duration=5–15 ms with 2–4 phases [13].
(3) Recording of muscle activity on maximal volition (sensitivity was set at 200 μV/division, analysis time was set at 1 s).

Electrophysiological procedures were done using Neuropack2 Electromyograph (Neuropack 2, Nihon Kohden, Tokyo, Japan).

Electrophysiological studies in hemiplegic patients have a special pattern, as it is complicated by the upper motor neuron lesion (UMNL) manifestations [14]. Accordingly, the criteria used in electrophysiological laboratories under normal circumstances are difficult to be applied in this situation. Therefore, the diagnosis of brachial plexopathy or proximal nerve lesion has been based on side-to-side comparison and designated cutoff points (as shown in Table 1) [12,13] as well as the standard pathological interpretation of needle EMG.

(1) Upper trunk brachial plexus lesion (axonopathic) was considered if the following criteria were met [10]:
    (a) Clinical supportive data: shoulder subluxation with flaccidity and atrophy of supraspinatus, infraspinatus, deltoid, and biceps muscles on the hemiplegic side besides increased muscle tone or the presence of ipsilateral hand movement.
    (b) EMG finding of abnormal rest potentials in the tested muscles.
    (c) Low lateral antebrachial SNAP amplitude.
(2) Upper trunk brachial plexus lesion (demyelinating) was considered if there were prolonged axillary, musculocutaneous, and suprascapular nerve latencies [15].
(3) Isolated axillary demyelinating neuropathy was diagnosed if there was prolonged axillary nerve latency with normal musculocutaneous and suprascapular nerve latencies. The presence of abnormal rest potentials only in the deltoid muscle was considered axillary nerve axonopathy.
(4) Suprascapular neuropathy [4] was considered if there was prolonged suprascapular nerve latency, with normal axillary and musculocutaneous latencies (demyelinating).The presence of abnormal rest potentials only in infraspinatus muscle was considered suprascapular nerve axonopathy.
(5) Isolated musculocutaneous demyelinating neuropathy was diagnosed if there was abnormal musculocutaneous nerve conduction in the presence of normal axillary and suprascapular nerve conduction studies.

Nerve conduction study, functional evaluation, and clinical assessment or radiological examination were done by one and the same doctor.

Statistics

Data were analyzed using the Statistical Package for the Social Sciences (SPSS ver.20; SPSS Inc., Chicago, Illinois, USA). The distributions of quantitative variables were tested for normality using the Kolmogorov–Smirnov test, which revealed that the data were not normally distributed, so nonparametric tests were used. Quantitative data were described using median and range. Qualitative data were described using number and percent. Mann–Whitney test was used to compare the median of quantitative variables between the paretic and the normal side to detect statistical significance at a level of less than or equal to 0.05.

Results

Demographic and clinical characteristics of the study participants are presented in Table 2.

Table 2 Demographic and clinical characteristics of the study participants

	n (%)
Age (years)	
Median (minimum–maximum)	55.50 (29–81)
Sex	
Male	21 (70)
Female	9 (30)
Occupation	
Manual worker	19 (63.3)
Clerk	4 (13.3)
Housewife	7 (23.3)
Duration of stroke (months)	
Median (minimum–maximum)	4 (1.5–60)
Duration of stroke	
<1 year	24 (80)
1 to <5 years	6 (20)
Duration of poststroke shoulder pain (months)	
Range (minimum–maximum)	1–33
Median	3
Muscle power shoulder abductors[a]	
Severe weakness	11 (68.7)
Moderate weakness	8 (53.3)
Mild weakness	10 (38.4)
Normal	1 (4.3)
Fugl-Meyer motor impairment total A-D	
Median (minimum–maximum)	32.5 (4–64)
Spasticity shoulder	
Yes	10 (52.6)
No	20 (32.8)
Shoulder subluxation[b]	
Yes	6 (20)
No	24 (80)

[a]Severe weakness=grade 0–2; moderate weakness=3; mild weakness=4; normal=5. [b]Three patients had grade II and three patients had grade III Sulcus sign.

Nerve conduction results of the axillary, musculocutaneous, suprascapular, and lateral antebrachial nerves of the hemiplegic and normal sides are presented in Tables 3 and 4. Axillary and musculocutaneous motor nerve latencies on the hemiplegic side were significantly prolonged compared with the normal side (P=0.012, 0.029, respectively). Moreover, axillary and suprascapular nerve amplitudes on the hemiplegic side were significantly lower than those on the normal side (P=0.008, 0.002, respectively).

EMG findings of the studied muscles are displayed in Table 5. Different pathophysiological patterns of electrophysiological findings are displayed in Table 6. Upper trunk brachial plexopathy was the most common abnormality which occurred in six (20%) patients. In addition, isolated axillary or suprascapular nerve lesion occurred at a similar frequency (10%).

Discussion

Proximal mononeuropathy and brachial plexus injury have been reported as potential complications in the hemiplegic shoulder [2,16]. The current study showed different electrophysiological abnormalities of the proximal nerves around shoulder in PSSP patients. These abnormalities included prolonged motor latency of the studied proximal nerves, in comparison to the normal side, which was significant for both axillary and musculocutaneous nerves. Also, there was reduction in CMAP amplitude of the studied proximal nerves that was significant for axillary and suprascapular nerves.

In agreement with the current study, Chokroverty and Medina [15] previously reported delayed motor latency of axillary and musculocutaneous nerves in 12 hemiplegic patients within 3 days to 6 weeks of onset of hemiplegia. Chino [17] found prolonged mean proximal latencies in the hemiplegic shoulder compared with latencies found in normal controls.

It has been reported that hemiplegic patients are susceptible to traction of the brachial plexus during the initial acute stage of paralysis [2,4]. In addition, downward subluxation is able to produce traction on the axillary nerve as it winds around the surgical neck of the humeral shaft [18]. Thus, traction on nerves can produce nerve injury.

Table 3 Nerve conduction parameters of the studied motor nerves of the hemiplegic and nonhemiplegic sides among poststroke shoulder pain patients

Nerve	Hemiplegic side (*n*=30)	Nonhemiplegic side (*n*=30)	Test of significance	*P* value
Axillary nerve latency (ms)				
Range (minimum–maximum)	3.20–5.50	2.90–5.30	*U=279.000*	0.012*
Mean±SD	4.39±0.56	4.03±0.51		
Median	4.40	4.1		
Axillary nerve CMAP amplitude (mV)				
Range (minimum–maximum)	0.32–19.5	4.70–40.70	*U=–272.000*	0.008*
Mean±SD	8.14±4.19	12.61±7.26		
Median	8.06	10.66		
Musculocutaneous nerve latency (ms)				
Range (minimum–maximum)	3.80–6.80	3.56–6.70	*U=302.500*	*0.*029*
Mean±SD	5.18±0.63	4.86±0.59		
Median	5.1	4.9		
Musculocutaneous CMAP amplitude (mV)				
Range (minimum–maximum)	1.60–28.30	1.93–23.30	*U*=364.500	*0.206*
Mean±SD	10.23±6.34	11.88±5.75		
Median	9.10	10.90		
Suprascapular nerve latency (ms)				
Range (minimum–maximum)	2.50–6.60	2.30–4.70	*U=372.500*	0.251
Mean±SD	3.70±0.94	3.40±0.70		
Median	3.45	3.22		
Suprascapular nerve CMAP amplitude (mV)				
Range (minimum–maximum)	1.53–22	3.70–25.50	*U=242.500*	0.002*
Mean±SD	9.13±4.47	12.69±5.15		
Median	9.08	11.50		

CMAP, compound muscle action potential; *U*, Mann–Whitney test. *$P \leq 0.05$, statistically significant.

Table 4 Nerve conduction parameters of lateral antebrachial nerve on the hemiplegic and nonhemiplegic sides among poststroke shoulder pain patients

Nerve	Hemiplegic side (*n*=30)	Nonhemiplegic side (*n*=30)	Test of significance	*P* value
Lateral antebrachial onset latency (ms)				
Range (minimum–maximum)	1.56–2.98	1.26–2.50	*U=390.000*	0.375
Mean±SD	2.08±0.34	1.98±0.31		
Median	2.00	1.98		
Lateral antebrachial peak latency (ms)				
Range (minimum–maximum)	2.00–3.96	1.92–3.20	*U=393.000*	0.399
Mean±SD	2.68±0.43	2.58±0.29		
Median	2.65	2.56		
Lateral antebrachial SNAP amplitude (μV)				
Range (minimum–maximum)	1.26–38.50	4.31–40.00	*U=334.000*	0.086
Mean±SD	14.92±8.47	16.62±7.21		
Median	13.33	16.05		
Lateral antebrachial conduction velocity (m/s)				
Range (minimum–maximum)	44.00–89.6	45.00–95.20	*U=423.000*	0.695
Mean±SD	58.54±10.41	59.05±11.09		
Median	60.00	56.25		

SNAP, sensory nerve action potential; *U*, Mann–Whitney test. *$P \leq 0.05$, statistically significant.

Some authors suggested that prolonged motor latencies in the hemiplegic limbs could be related to decreased skin temperature in the affected limbs which may result from inactivity of the limbs and reduced circulation [19]. However, this was true mainly for the distal nerves when studying the median, ulnar, and peroneal nerves rather than the proximal nerves [15]. Since the current study did not show a significant side-to-side (hemiplegic vs. normal) difference in the conduction parameters of the lateral antebrachial sensory nerve, the delayed motor latencies could not be attributed to changes in skin temperatures.

Several studies have noted decreased CMAP amplitudes in the hemiplegic side versus the contralateral side [20–22]. The current study confirmed this finding with the observation that there was diminished CMAP amplitude of the

Table 5 Electromyographic findings in the studied muscles of the hemiplegic side among poststroke shoulder pain patients (*n*=30)

	Deltoid [*n* (%)]	Biceps brachii [*n* (%)]	Infraspinatus [*n* (%)]
Abnormal rest potentials			
Yes	4 (13)	1 (3)	0 (0)
No	26 (87)	29 (97)	30 (100)
On minimal volition			
No significant abnormality	11 (37)	7 (23)	7 (23)
Few normal MUAPs[a]	6 (20)	13 (43)	9 (30)
Few polyphasic MUAPs[b]	5 (17)	4 (13)	3 (10)
Polyphasic MUAPs	5 (17)	3 (10)	3 (10)
Silent	3 (10)	3 (10)	8 (27)
MUAPs duration (ms)			
Range (minimum–maximum)	6–12	6–12	8–10
MUAPs amplitude (μV–mV)			
Range (minimum–maximum)	400 μV–3 mV	400 μV–3.5 mV	400 μV–3 mV
Interference pattern			
Complete	0 (0)	0 (0)	0 (0)
Incomplete	20 (67)	20 (67)	18 (60)
Discrete	7 (23)	7 (23)	4 (13)
Silent	3 (10)	3 (10)	8 (27)
Total	30 (100)	30 (100)	30 (100)

MUAP, motor unit action potential. [a]Few: 1–2 MUAPs of normal parameters. [b]Few: 1–2 polyphasic MUAPs.

Table 6 Patterns of electrodiagnostic abnormalities among the studied patients

	n (%)
Isolated axillary nerve lesion	
Demyelination	2 (6.66)
Axonopathy	1 (3.33)
Upper trunk brachial plexopathy[a]	
Demyelination	3 (10)
Axonopathy	3 (10)
Suprascapular neuropathy	
Demyelination	3 (10)
Axonopathy	0 (0)
No evidence of associated lower motor neuron pathology	18 (60)
Total	30 (100)

[a]C5, C6 lesions. Only 40% of the study patients had evidence of proximal nerve lesion.

studied motor nerves on the hemiparetic side compared with the normal side that was significant for the axillary and suprascapular nerves.

Cortical lesions are believed to cause diffuse peripheral motor axonal degeneration by some authors [14,22,23]. Kingery *et al.* [14] and Zalis *et al.* [23] were able to correlate the diminished amplitude with the degree of the spontaneous activity on needle EMG which was used to consolidate the hypothesis that CMAP reduction in patients with cortical lesions reflects loss of motor axons rather than disuse. However, these changes are mainly seen in distal muscles rather than the proximal ones [24]. Thus, it is unlikely that fibrillation potentials and positive sharp waves at the proximal muscle of the shoulder are caused by UMNL.

The present study showed denervation potentials that are consistent with peripheral nerve lesion (Table 5). This is because we sampled proximal muscles and not distal muscles. Also, Benecke *et al.* [24] reported that denervation potentials in stroke survivors were 'transient' occurring mainly in the first 3 weeks and patients enrolled in this study had stroke duration of 1 month or more. These factors could explain the inability to confirm the existence of central denervation activity in this study.

This study showed different patterns of MUAPs recorded on minimal volition including normal MUAPs, polyphasic motor units, and large motor units (reaching up to 3.5 mV). In some patients, it was observed that there is decreased number of recorded MUAPs on minimal volition (1–2 MUAP

only). These different patterns have been previously reported in stroke patients and they were attributed to loss of the trophic effect from UMNL that could lead to loss of functioning motor units or alter the functional state of anterior horn cells on the affected side without cell loss [25–27]. Similar to the current study, Lukács [27] reported that normal and enlarged motor units could be found. After the acute phase of stroke, there is restoration of neuronal lesions occurring via collateral reinnervation that could lead to an increased number of active muscle fibers and serve to explain the recovery of the M wave in the chronic stage. In severe cases in which motor neurons undergo transsynaptic degeneration, collateral sprouting would start to enlarge the remaining motor units [27].

In this study, electrodiagnosis showed pathological findings in 12 (40%) patients. It was observed that upper trunk brachial plexopathy was the most common abnormality which occurred in six (20%) patients. In addition, isolated axillary nerve lesion occurred at a similar frequency as suprascapular nerve (10%).

This study revealed that among the 12 patients with neuropathic affection; only five patients had shoulder subluxation. Furthermore, the other seven patients had no history of shoulder traction prior to the development of PSSP. This suggests that shoulder subluxation was not the only cause for proximal nerve involvement. Patients with electrophysiological features of upper trunk brachial plexopathies, in whom evident subluxation was not present, may suggest the presence of subclinical proximal neuropathy.

Chokrovery and Medina [15], more than 40 years ago, has raised the issue of associated mild traction plexopathy in some hemiplegic patients, which might not show clinical manifestations and can only be diagnosed by latency determination, while the more severe traction plexopathy might be accompanied by localized muscle wasting, persistent flaccidity of the limb with neurogenic EMG changes. The findings of this study was in agreement with Chekorvey and Medina [15] as it showed associated mild brachial plexopathy in some patients with PSSP. This pattern of traction plexopathy (based on latency determination) has received little attention in the literature.

Axillary nerve lesion was the most common proximal mononeuropathy in stroke survivors that received medical and research attention. Tsur and Ring [3] studied 44 shoulders of 22 patients with flaccid hemiplegia (43±12 days after stroke onset). They found that axillary nerve latency in the paralyzed shoulder was significantly increased versus the sound side ($P<0.001$). Taksandea *et al.* [28] studied axillary nerve motor conduction in flaccid stroke patients and concluded that axillary nerve in the paretic limb is liable for injury as there was significant reduction of axillary CMAP amplitude and increase of motor latency compared with the sound side.

Suprascapular entrapment neuropathy was found in the current study in three (10%) of the studied patients. Similarly, Lee and Khunadorn [4] studied suprascapular nerve conduction in 30 patients with PSSP. They reported prolonged latency in three patients according to side-to-side comparison. However, they denied a direct relationship between the suprascapular nerve lesion and PSSP as the suprascapular nerve block did not relieve pain and that suprascapular nerve lesion was considered an incidental finding in an already painful shoulder.

It should be noted that EMG in the paralytic upper limb was not an easy procedure because of spasticity, uncomfortable position, and the presence of pain. However, careful electrophysiological evaluation should be done with patience because electrophysiology is considered the diagnostic gold standard for brachial plexus injuries. If electrophysiology was impossible because of severe spasticity or pain, neuromuscular ultrasound could be helpful.

In this study, it is unlikely that bias had played a role. Each evaluation step (nerve conduction study, functional evaluation, clinical assessment, or radiological examination) was done by one and the same doctor. Results of the current study could be generalized to all stroke patients who do not have chronic inflammatory arthritis, cognitive impairment and previous shoulder pain or trauma because the appropriate electrodiagnosis was done carefully and with patience. However, further research enrolling larger number of stoke survivors is needed.

Conclusion

Proximal nerve lesions are not uncommon in PSSP patients and may occur subclinically.

Study limitation

The relatively small size of the studied patients is a limitation of the current study. Moreover, neuromuscular ultrasound examination was not done for limitations of the resources and unavailability of high frequency machines.

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Conflicts of interest
There are no conflicts of interest.

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