Orijinal Araştırma / Original Investigation
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Axillary Nerve Involvement in Hemiplegia

Hemiplejide Aksiller Sinir Tutulumu

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ÖZET

AMAÇ: Hemiplejik omuz subluksasyonuna bağlı brakiyal pleksus ve periferik sinir hasarı görülebilir. Çalışmamızda omuz subluksasyonu ile aksiler sinir hasarı arasındaki ilişkiyi araştırmayı amaçladık

GEREÇ VE YÖNTEM: Çalışmaya, inmeli 60 hasta alındı. Brunnstrom Skalası ve Ashworth Skalası sırasıyla motor ve kas tonusunu değerlendirmek için kullanıldı. Fonksiyonel değerlendirme Fugl-Meyer Ölçeği ve İnme Bozukluğu Değerlendirme Seti ile yapıldı. Ön-arka omuz grafisinde omuz subluksasyonunu derecelendirmek için Van Langenberghe yöntemi kullanıldı. Normal ve hemiplejik tarafların aksiller sinir hasarı elektromiyografi kullanılarak araştırıldı.

BULGULAR: Hemiplejik tarafın aksiller sinir latansı (4.2 ± 0.75 msn), normal taraf latansı (3.7± 0,7 msn)' na kıyasla uzamış tespit edildi. Bulgular istatistiksel olarak anlamlıydı (p<0.001)..

SONUÇ: Omuz subluksasyonu olan hastalarda periferik sinirler ve brakiyal pleksus gibi aksiller sinir de hasar görebilir. Omuz subluksasyonu ve ek aksiller sinir hasarının tespiti, uygun bir rehabilitasyon programının planlanmasına yardımcı olabilir ve komplikasyonları önleyerek fonksiyonel iyileşmeye katkıda bulunabilir.

Anahtar Kelimeler: inme, aksiller sinir, rehabilitasyon, omuz subluksasyonu

ABSTRACT

OBJECTIVE: Brachial plexus and peripheric nerves of the upper extremities can be damaged in hemiplegic shoulder subluxation. This study aimed to determine the relationship between shoulder subluxation and axillary nerve injury.

MATERIALS AND METHODS: Sixty patients with stroke were included in the study. The Brunnstrom Scale and the Ashworth Scale were used to evaluate the motor and muscle tone, respectively. Functional evaluation was performed with the Fugl–Meyer Scale and the Stroke Impairment Assessment Set. The Van Langenberghe method was used to grade shoulder subluxation on anterior-posterior shoulder X-ray. Latencies and amplitudes of the axillary nerves of the unaffected and hemiplegic sides were compared using electromyography.

RESULTS: The latency of the axillary nerves of the hemiplegic sides (4,2±0,75 msn) was prolonged when compared to that of the healthy sides (3,7±0,7 msn), and this difference was statistically significant (P<0.,001). The amplitudes of the compound muscle action potential of the hemiplegic sides (4,1±4,22 mv) lower than that of the healthy sides (6,1±6,57 mv) (P<0,001). .

CONCLUSION: Shoulder subluxation may cause injury not only to the axillary nerve but also to other peripheric nerves and the brachial plexus. Detecting shoulder subluxation and additional axillary nerve injury could in help planning an appropriate rehabilitation program and contribute to functional recovery by preventing complications.

Key Words: stroke, axillary nerve, rehabilitation, shoulder subluxation

Introduction

Stroke is a severe health problem, with negative effects on a person's quality of life. The socio-economic importance of stroke, that leads to longtime disability, is increasing (1). While the chances of surviving stroke patients to walk independently are 82%, the chances of functional use of their upper limbs are 50% (2).

After Stroke loss of muscle control, abnormal motion patterns, spasticity, and soft tissue changes that block movement are the main causes of impaired shoulder biomechanics. Disruption of the shoulder biomechanics has been shown to be the cause of the majority of complications seen in the hemiplegic upper extremity (3). Shoulder lesions and glenohumeral subluxation are

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frequently observed in stroke (2-4). Shoulder subluxation results in deterioration of biomechanics (3). Complications of shoulder subluxation (SS) include complex regional pain syndrome, lesions of the rotator cuff, adhesive capsulitis, rupture of tendons, tendinitis, brachial plexus and peripheral nerve lesions and shoulder pain (2-4).

While several investigations have reported on the dysfunction of brachial plexus and other peripheral nerves in patients with SS (5), other studies have found no such evidence (6). Depression of the paralytic shoulder due to gravity, can cause peripheric nerve lesions or rotator cuff rupture. Atzmon et al. reported that SS causes axillary nerve injury due to the traction effect (7). Chino also suggested that SS can cause traction of the brachial plexus (5).

In stroke rehabilitation, it is aimed to bring the patient to the maximal functional capacity as soon as possible and to bring it as independent and productive as possible (2). The functional independence level after stroke is associated with the motor impairment, and the predictor of the functional prognosis of the upper limb is related with the severity of the initial motor involvement (8). Shoulder subluxation and axillary nerve injury are two complications that can cause prolongation in the rehabilitation program. Thus, careful physical examination of the shoulder and/or radiographic examination are routinely performed for hemiplegic individuals with stroke who are undergoing rehabilitation. However, it is difficult to recognize the sensory or motor symptoms of peripheric nerve involvement in stroke patients during their first motor neuron clinic follow up. Additional cognitive and communication problems must also be considered. Although several studies have discussed this issue, there is still a gap regarding the involvement of the axillary nerve at the hemiplegic side individuals with stroke. The aim of the present study was to investigate the presence of underdiagnosed axillary nerve involvement with electromyography (EMG) in individuals with stroke, as well as determine the relationship between factors like SS, spasticity, motor functions and nerve involvement..

Materials and Methods

Between October 2011 and July 2012, 60 consecutive stroke patients in Ankara Physical Medicine and Rehabilitation Education and Research Hospital were recruited for this study. The study was approved by the local ethics committee, and consent was obtained from the patients or their relatives at the beginning of the study.

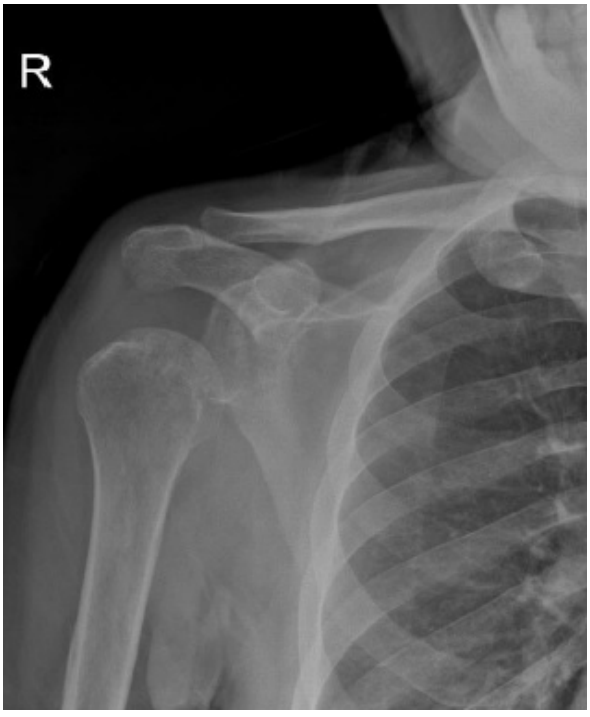
Inclusion criteria; 1) history of stroke 2) history of both flaccid and spastic paralysis 3) sufficient cooperation

Exclusion criteria: Those who 1) were unconscious, 2) had bilateral stroke, 3) had cardiac pacemaker, 4) had known axillary nerve or brachial plexus lesions 5) had known previous shoulder trauma.

As muscle tone of patients in the early stage is more prone to be flaccid (6, 9, 10) and SS is associated with flaccid periods (11); no restriction was applied to the duration of hemiplegia of the patients included in this study. This is because spasticity itself may also provoke SS (11,12).

All consecutive subjects underwent a physical examination conducted by N.Y. The age, sex, poststroke duration, stroke etiology (ischemic or hemorrhagic), side of stroke, family history presence of comorbid diseases, and smoking status were recorded. Following physical examination, radiographs of the shoulder were taken. The degree of SS (DSS) in anteroposterior radiography was assessed according to the classification developed by Van Langenberghe et all (13) (Figure 1).

Figure 1. *Shoulder x-ray of a patient with stage 3 shoulder subluxation according to Van Langenberghe method*



Electrophysiological assessment was performed by a researcher who did not know the radiologic DSS of a patient. The Medtronic Keypoint 4C EMG equipment was used. Motor conduction study of the axillary nerves was performed with the patient in a supine position in a quiet and well-lit room with a constant temperature of 22–24°C. Patients' skin temperature was not allowed to drop below 31°C. The axillary nerves in the hemiplegic and normal extremity of each patient were stimulated at Erb's point. A needle electrode was used to record EMG signals from the posterior deltoid muscle. The electrophysiological study was repeated three times for each axillary nerve. The average of the latencies and the compound muscle action potentials (CMAPs) were recorded (Fig. 2-3).

Figure 2. *Right axillary nerve compound muscle action potential of a patient with left hemiplegia*

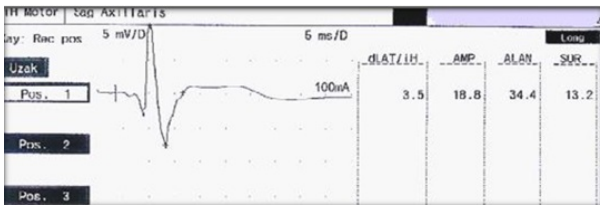
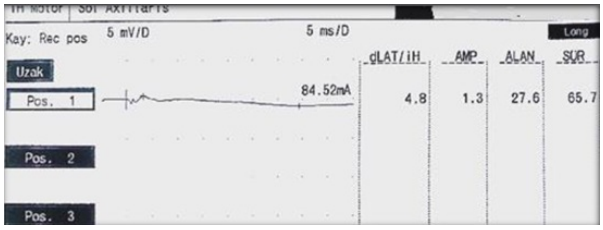


Figure 3. *Left axillary nerve compound muscle action potential of the same patient*



The Brunnstrom neurophysiological assessment, Fugl–Meyer method, and Stroke Impairment Assessment Set (SIAS) were used to evaluate the patients' hemiplegic upper extremities and hand motor functions (2,14-16). The Fugl–Meyer method evaluates motor function recovery. The SIAS estimates motor function, tonus, sense, range of motion, pain, trunk function, visuospatial function, speech, and the healthy side. We used only the upper extremity part of the test for hemiplegic upper extremity. The Ashworth Scale (AS) was used to assess muscle tone (17).

Statistical analysis

Analysis of data was performed using the IBM SPSS Statistics 17.0 package program (IBM Corporation, Armonk, NY, USA). The Shapiro–Wilk test was used to determine whether the distribution of continuous variable was near normal. Descriptive statistics for continuous variables are shown as the mean ± standard deviation, and the median or interquartile range is shown as the minimum-maximum. Categorical variables are shown as the number of cases and percent value.

The Wilcoxon-signed rank test was used to determine whether there were significant differences in the level of latency and the amplitude between the hemiplegic and normal sides. The significance of the difference in the mean values between the groups was investigated with a Mann–Whitney U Test. Categorical variables were evaluated using Pearson's chi-square or Fisher's exact test. The presence of significant differences among the amplitudes, latencies, and DSS and differences between these and other clinical variables were evaluated using Spearman's correlation test. Axillary neuropathy was investigated based on reference values. Reference cut of value for latency is 5,5 ms and reference cut of value for amplitude is 10,8 mVolt. During the evaluation of axillary nerve EMG findings and SS, the values over 5.5 ms for latencies were considered as pathologic and the values lower than 10.8 mVolt for amplitudes were considered as abnormal. A P value <0.05 was accepted as statistically significant.

Results

Sixty patients (27 males, 33 females) were included in the present study. The mean age of the patients was 61.5 years. The mean duration of stroke was 1.2 years. Thirty-two patients had comorbid disease; 20 had diabetes mellitus, 41 had hypertension, 18 had hyperlipidemia, and 6 had chronic heart disease. Twenty-three (38%) of the patients had a history of smoking.

The mean of the axillary nerve latency was 4.2±0.75 ms, and the mean of the amplitude was 4.1±4.22 mVolt. The axillary nerve latencies (4.2±0.75 ms) of the hemiplegic sides were longer (in 38 case) when compared with those of the normal sides (3.7±0.7 ms) (P<0.001). The CMAP of the hemiplegic sides (4.1±4.22 mVolt) was decreased (in 45 case) when compared with the CMAP of the normal sides (6.1±6.57 mVolt). These findings were statistically significant (P<0.001). Needle EMG of the deltoid muscle of the normal and hemiplegic sides revealed no spontaneous activity (Table1).

Table 1. *Latency and amplitude measurements of axillary nerves at the hemiplegic and normal sides*

	Hemiplegic side	Normal side	P*
Latency (msn)	4,2 (0,75)	3,7 (0,70)	**<0,001**
Amplitude (mV)	4,1 (4,22)	6,1 (6,57)	**<0,001**

**p<0,05 Wilcoxon sign test*

The axillary nerve conduction velocity is not calculated because the present study included women and men, whose body sizes are different. Thus, standardization of axillary nerve conduction velocity would have been impossible.

According to radiographic evaluation, 19 patients had stage 1 DSS, 12 had stage 2, and 6 had stage 3. None of the patients had stage 4 DSS. Twenty-eight (38%) patients had normal DSS. In the present study, the frequency of SS was 71.7%. Of the patients with SS, 31.7% cases had grade 1, 20% had grade 2, and 10% had grade 3 (Table 2).

Table 2. *Distribution of cases with shoulder subluxation according to muscle tone*

DSS	Flaccid	Normotonic	Spastic	p*
Normal	1 (11,1%)	10 (52,6%)	12 (37,5%)	0,107
Abnormal	8 (88,9%)	9 (47,4%)	20 (62,5%)	

DSS: Degree of shoulder subluxation

Since normal values fort the axillary nerve latency and amplitude were not generated in our EMG lab, axillary nerve electrophysiological changes and DSS were evaluated based on reference values. These findings were not statistically significant however there was a moderate correlation in the negative direction between amplitude and SS (P=0,041) (Table3).

Table 3. *Correlation Coefficients and Significance Levels between Demographic and Clinical Features with Latency and Amplitude Measurements in Hemiplegic and Counterparts*

	Hemiplegic Side				Normal Side			
	Latency		Amplitude		Latency		Amplitude	
	r-value	*p-value*	*r-value*	*p-value*	*r-value*	*p-value*	*r-value*	*p-value*
Age	0,006	0,961	-0,021	0,875	-0,029	0,828	-0,128	0,328
Stroke Duration	0,056	0,668	0,018	0,890	-0,163	0,213	0,134	0,309
DSS	-0,066	0,617	-0,264	**0,041**	0,024	0,855	-0,141	0,281
Upper Brunnstrom	0,122	0,354	0,075	0,568	-0,072	0,584	0,113	0,388
Hand Brunnstrom	0,070	0,597	0,189	0,147	-0,123	0,350	0,135	0,304
AS	0,249	0,055	-0,009	0,945	0,055	0,675	0,061	0,641
FUGL	0,104	0,427	0,095	0,468	-0,069	0,598	0,080	0,543
SIAS	0,084	0,526	0,118	0,369	-0,083	0,526	0,072	0,587

*Spearman's Correlation test. AS: Ashworth Scale, DSS: Degree of shoulder subluxation. * p<0,05 p<0,05 Pearson's Chi-square test*

Statistical analysis showed that 66.7% of patients had simultaneous prolonged axillary nerve latency and SS. In addition, 60.7% of the patients showed simultaneous SS and decreased axillary nerve amplitudes.

We evaluated whether comorbid disease had any effect on the amplitudes and the latencies of the axillary nerve. There was no statistically significant relation between the amplitudes (P=0,619) and the latencies (P=0,336) of the axillary nerve (P=1) between the patients with and without comorbid diseases (Table 4).

Table 4. *Latency and Amplitude Measurements According to Hemiplegic and Normal Side in Comorbid Disease and Distribution of DSS*

Variables	Comorbid Disease (-) (n:11)	Comorbid Disease (+) (n:49)	p-value
Hemiplegic side			
Latency	4,2 (0,30)	4,2 (0,70)	0,336
Amplitude	3,6 (4,10)	4,2 (4,10)	0,619
Normal side			
Latency	3,7 (0,50)	3,8 (0,80)	0,788
Amplitude	7,9 (13,00)	6,1 (6,65)	0,260
DSS			1,000
Normal	4 (%36,4)	19 (%38,8)	
Abnormal	7 (%63,6)	30 (%61,2)	

Data; as shown Median (width between quarters), + Man Whitney U test DSS: Degree of shoulder subluxation

Examination of the muscle tone of the patients revealed 9 with flaccid muscle tone, 19 with normotonic muscle tone, and 32 with spasticity. When we evaluated the spasticity, the findings were as follows: 5 patients were at stage 1, 19 at stage 2, and 8 at stage 3. None of the patients were at stage 4 spasticity.

Comparison of the DSS and muscle tone values of patients revealed that among flaccid patients DSS was normal in only 1 of 9 cases. Eight patients had SS. The muscle tone of 19 patients was normal, and 20 patients with spasticity had SS. When muscle tone and axillary nerve values were evaluated, the findings were not statistically significant (p=0,055 andp=0,675).

The results of the Brunnstrom evaluation of the upper extremities of the patients were as follows: There were 27 (45%) patients at stage 1, 12 (20%) patients at stage 2, five (8.3%) patients at stage 3, five (8.3%) patients at stage 4, 10 patients at stage 5 (16.7%), and one patient at stage 1 (1.7%). The results of the Brunnstrom evaluation of the hand were as follows: There were 32 (53.3%) patients at stage 1, eight (13.3%) patients at stage 2, five (8.3%) patients at stage 3, four (6.7%) patients at stage 4, 10 (16.7%) patients at stage 5, and one (1.7%) patient at stage 6.

The mean score for the Fugl–Meyer test was 6.5 out of 36, and the mean score for the SIAS was 2 out of 10.

Analysis of the potential relationship between age, post stroke duration, Brunnstrom value, and spasticity revealed

a statistically significant relationship between The Brunnstrom Scale and the DSS, the Fugl–Meyer scale and the DSS, and the SIAS and the DSS (r: 0.35, P<0.006; r: -0.40, P<0.001; and r: -0.33, P<0.009, respectively) (Table 3).

Discussion

Under tension, peripheral nerve undergoes strain and glides within its interfacing tissue (19). Persistent traction of the axillary nerve may cause demyelination. We can visualize this as a prolonged latency on EMG. Axonopathy may also occur as demonstrated by low amplitudes on EMG.

The humoral head usually displaces inferiorly during the flaccid stage (11) this may cause nerve traction (7). İn addition the weight of the paralyzed arm may cause traction damage to the axillary nerve. Ring et al recommended that downward subluxation is able to produce traction on the axillary nerve as it wings around the surgical neck of the humoral shaft (20).

In a study of 21 hemiplegic cases with SS, Chino showed that when compared with previously notified normal values, there are considerably delay in the latency of the suprascapular, axillary, musculocutaneous, and radial nerves with denervation findings on needle EMG. Chino further suggested that SS can cause traction of the brachial plexus (5). In their case series of 8 patients, Kallio et al. emphasized on the importance of EMG in the diagnosis of patients with teres minor damage which could not diagnosed clinically (21).

In the present study the axillary nerve latencies (4.2±0.75 ms) of the hemiplegic sides were longer when compared with those of the normal sides (3.7±0.7 ms) (P<0.001). The CMAP of the hemiplegic sides (4.1±4.22 mVolt) was decreased when compared with the CMAP of the normal sides (6.1±6.57 mVolt) (P<0.001). These results were in agreement with those reported in the literatüre (5,7). Atzmon et al. indicated that SS caused axillary nerve injury because of the traction effect in 22 patients with 25–87 days poststroke duration (7). These patients had flaccid or atrophic shoulder muscle; as well as SS. When axillary nerve motor conduction studies of the hemiplegic sides of stroke patients were compared with studies of the normal sides, the latency of the axillary motor nerves of the hemiplegic sides were increased significantly. The same studies detected a statistically significant decrease in the amplitude of the axillary motor nerves of the hemiplegic sides as compared with those of the normal sides (7).

Kingery et al (22) and Enam et al (23) also studied axillary nerve electrophysiology similar with Tsur et all in early stage poststroke patients who have SS. However, in all three studies hemiplegic patients without shoulder subluxation were not included in the study. When axillary nerve motor conduction studies of the hemiplegic sides of stroke patients were compared with studies of the normal sides, the latency of the axillary motor nerves of the hemiplegic sides were increased significantly. The same studies detected a statistically significant decrease in the amplitude of the axillary motor nerves of the hemiplegic sides as compared with those of the normal sides.

We believe that the inclusion of patients without shoulder subluxation with stroke should be more meaningful in order to evaluate the effect of SS on axillary nerve neuropathy more effectively. By this reason when the patients included the study the present study did not assess the presence of shoulder subluxation. EMG and physical examination were done by different researchers. Results assessed in the statistics phase.

When it was evaluated the relationship between axillary nerve and shoulder subluxation, it was found that there were no statistically significant results. But there was a moderate correlation in the negative direction between amplitude and SS. When the research was carried out, it was decided based on the reference values in the book whether axillary nerve damage was present. Because the normal values for the axillary nerve were not established in the present EMG lab. It may have affected these results. When compared to the healthy side, more patients had axillary nerve damage than using the reference values.

Although the room temperature was well controlled by a standard thermometer, temperatures of patients' hemiplegic side may be lower than normal side (due to reduced circulation) and EMG results might be affected.

In the present study among sixty patients, 37patient (88.9%) had SS. There were 32 cases with spasticity and of these, 74% had SS. Nine patients had flaccid muscle tone and of these 8 (%88.9) patient had SS. The detection of SS in both flaccid and spastic cases supports the two opinions in

literature (11,12). According to the literature, both spastic and flaccid periods contribute to SS in hemiplegic patients. Several studies have suggested that in the spastic period, an increase in scapular rotation causes SS because the pectoralis major, pectoralis minor, rhomboideus, levator scapula, and latissimus dorsi muscles are hypertrophic. In contrast, during the flaccid period, inferior displacement of the humeral head with the loss of normal muscle activity of the supraspinatus and deltoid muscles lead to SS (11,12).

Unlike the literature the present study investigated the parameters like age, sex, stroke duration, stroke etiology, motor function level, tone of muscles and comorbid diseases which may affect the axillary nerve electrophysiology in hemiplegic patients. There was no statistically significant relationship between these parameters and axillary nerve EMG results. (Table 3)

Chang et al. and Daviet et al. suggested that an increased Brunnstrom stage and recovery of motor function reduced SS. Zorowitz detected reduced SS in stroke patients, with marked motor recovery (24). In this research, the relationships among SS, axillary nerve and functional status were also investigated. Similar to the literature it was found that the SS decreased as the functional status improved. However, there was no significant relationship between axillary neuropathy and functional recovery.

In conclusion, axillary nerve electrophysiology in hemiplegic patients investigated in the present study. In hemiplegic patients, shoulder problems are common in daily practice. When the patients were seen for the first time without notice, either early or late, spastic tonus or flask tonus, presence of shoulder subluxation and axillary nerve damage should be assessed with a good examination. If necessary, routine EMG should be performed for axillary nerve and other peripheral nerves if circumstances permit. Thus, good and comprehensive rehabilitation practice is provided. However, the axillary nerve has a rather complicated course through several soft tissue compartments of the shoulder and axilla. Therefore, imaging of the nerve with sonography is not troublesome for experienced sonographer (25). Thus, sonographic assessment of axillary nerve nicely suggested for pre-surgery exploration (25). In our study ultrasound imagining of the peripheral nerves is absent. This lack can be seen as the limitation of the study.

In summary, SS in hemiplegic patients may damage the axillary nerves, which may delay the healing process. Detecting SS and additional axillary nerve injury could help in planning an appropriate rehabilitation program and contribute to functional recovery by preventing complications.

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