

ORIGINAL REPORT

SONOGRAPHY AND PHYSICAL FINDINGS IN STROKE PATIENTS WITH HEMIPLEGIC SHOULDERS: A LONGITUDINAL STUDY

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Objective: To investigate the correlation between hemiplegic shoulder pain factors during the acute and chronic stages of stroke recovery.

Design: A prospective longitudinal study.

Subjects: Seventy-six stroke patients with hemiplegic shoulders.

Methods: Hemiplegic shoulder pain and clinical, physical, and sonography results were recorded at admission and during both acute (before discharge) and chronic stages (6 months after discharge).

Results: During the acute stage, hemiplegic shoulder pain correlated significantly with shoulder motor function level and range of motion limitations. During the chronic stage, hemiplegic shoulder pain correlated significantly with shoulder motor function level, range of motion limitations, spasticity and abnormal sonographic findings. Higher incidence ($p=0.014$) of hemiplegic shoulder pain and pain scores ($p<0.01$) were noted and abnormal sonographic findings of the biceps tendon long head ($p=0.01$) and subscapularis tendon ($p=0.01$) were higher during the chronic stage. Effusion, tenosynovitis or tendinopathy of the biceps tendon long head, and supraspinatus tendinopathy were notable during both stages.

Conclusion: Hemiplegic shoulder pain was correlated with lower motor function level and shoulder range of motion limitation in both stages. Shoulder spasticity and abnormal sonographic findings were correlated with hemiplegic shoulder pain during the chronic stage.

Key words: hemiplegic shoulder pain; stroke; sonography.

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INTRODUCTION

Hemiplegic shoulder pain (HSP) is a major problem in stroke patients. Its incidence varies from 5% to 84% (1–3). HSP results in limited shoulder movement in stroke patients because

they are unable to tolerate passive or active shoulder movements, thus lessening the effectiveness of any motor recovery techniques. Many reports have documented the negative impacts of HSP in stroke patients, including obstruction of the rehabilitation process, delay of motor recovery in the upper extremities, decrease in the functional performance of daily activities, and prolongation of hospital stay (3–5).

Many previous studies have examined the potential factors contributing to HSP, but have not reported consistent results. The most common factors that are correlated with HSP include shoulder subluxation, spasticity, range of motion (ROM) limitation, poor arm motor function, impaired sensation, and soft tissue injuries of the shoulders. However, not all studies report a correlation of all risk factors in all patients. Poulin de Courval et al. (6) reported that stroke patients with HSP had significantly higher levels of spasticity, impaired sensation, and severe shoulder subluxation. Huang et al. (7) found that HSP severity was correlated with poor arm motor function, shoulder ROM limitation, and shoulder subluxation but not with spasticity in acute stroke patients. Joynt (8) concluded that the amount of pain was related to shoulder ROM limitation, but not to subluxation, spasticity, or strength. Bohannon et al. (9) reported that shoulder ROM (external rotation) limitation was correlated significantly with HSP; however, no such correlation was observed with shoulder spasticity and weakness. The lack of consistency between the reported predictors and HSP suggests that these inconsistencies may result from differing patient selection criteria, duration since stroke onset, evaluation methods, and sample sizes.

Some stroke patients may undergo shoulder paralysis during the acute stage of stroke recovery. The ensuing weakness in shoulder girdle muscles may result in failure of the protective mechanisms that prevent shoulder motion impairment in healthy individuals (10). In addition, gravitational forces on flaccid shoulders may lead to traction injuries in soft tissues. However, spasticity eventually develops in most stroke patients. Increased muscle tone may shorten the periarticular soft tissues, resulting in traction forces associated with spasticity in periosteal muscle attachments, thereby causing shoulder pain (11). Thus, there are different manifestations of pain in hemiplegic shoulders at different neurological stages of stroke recovery. Thus, HSP at different motor recovery stages may be associated with different pathological mechanisms and contributing factors.

Many studies have investigated the factors associated with HSP, by using cross-sectional analyses that have included both acute and chronic stroke patients. To best of our knowledge, few researchers have longitudinally followed up on HSP patients to explore the factors associated with the acute and chronic stages of stroke recovery. This is the first longitudinal study to report the correlation of HSP factors in different motor recovery stages. The aim of this study was to investigate the correlation between HSP and the physical findings and to analyse periarticular soft tissue injuries by sonography during both acute and chronic stages of stroke.

METHODS

Study population

Seventy-six patients diagnosed with acute stroke participated in this study. All patients were admitted to the rehabilitation department of the Chang Gung Memorial Hospital, a 2,700-bed teaching hospital in southern Taiwan. Patients who had experienced a stroke for the first time, resulting in unilateral hemiplegia, and who had not experienced shoulder pain in bilateral shoulders in the 6 months before the stroke were enrolled. The exclusion criteria included: severe cognitive impairment; a history of shoulder injuries, rotator cuff disorders, or shoulder surgery; frozen shoulder; and any neuromuscular disorders associated with shoulder weakness.

This study was approved by the medical ethics committee at the hospital; informed, written consent was obtained from each participant before all procedures. During hospitalization, all patients received standard in-patient rehabilitation programmes, which included ROM exercises, strengthening exercises, hand function training, transfer training, balance training, and ambulation training, according to their individual tolerances. The programmes involved 1 h of physical therapy and 1 h of occupational therapy a day, 5 days per week.

Procedures

At admission, the following clinical characteristics were recorded for each patient: age, gender, body weight, height, affected side, type of stroke, and time from stroke onset. The presence of HSP, motor function level (Brunnstrom motor recovery (BMR) stage), shoulder ROM (flexion, extension, abduction, external rotation, and internal rotation), sensation (proprioception, pinprick, and light touch), spastic level of the upper extremity, and glenohumeral subluxation were all recorded by one therapist at admission, before discharge (acute stage), and 6 months after discharge (chronic stage). HSP was considered when a patient reported shoulder pain during passive ROM exercise of the hemiplegic shoulder or while resting.

The severity of HSP was measured using a 10-point visual assessment scale, in which a score of 10 indicated the worst pain possible, and a score of 0 indicated no pain. The functional motor level of the upper proximal extremity was graded according to the BMR stages (12). Patients at Brunnstrom stages I, II, or III were considered to be at a poor BMR stage, and those at stages IV, V, or VI were considered to be at a good BMR stage. Sensation impairment was reported when patients were unable to respond appropriately to sensory stimuli and examinations. The therapist measured the shoulder ROM by goniometry. Spasticity was evaluated using the 5-point Ashworth scale (13). The presence of shoulder spasticity was defined as an Ashworth scale score of ≥ 1 . Glenohumeral subluxation was confirmed when the gap between the acromion and the humeral head was equal to or more than 1-finger breadth.

Sonographic examination

Musculoskeletal sonography of both shoulders was carried out in all patients at admission, before discharge (acute stage) and 6 months after

discharge (chronic stage). A physiatrist with 6 years of experience in musculoskeletal sonography performed the shoulder sonography using a 5–12 MHz linear-array transducer (t3000 Terason; Burlington, VT, USA) and interpreted the images. The physiatrist, certified by the Taiwan Society of Ultrasound in Medicine, was blinded to the clinical information about each patient. Each tendon of the hemiplegic shoulder was scanned in both longitudinal and transverse planes, according to the techniques described by Mack et al. (14) and Middleton (15). The soft tissues in each hemiplegic shoulder, including the long head of the biceps brachii tendon, supraspinatus tendon, infraspinatus tendon, subscapularis tendon, and subacromial-subdeltoid (SA-SD) bursa, were also examined. The sonographic signs of full-thickness cuff tear were as follows: absence of the rotator cuff or naked tuberosity, focal non-visualization of the rotator cuff, anechoic or hypoechoic clefts extending through the cuff, herniation of the deltoid muscle or SA-SD bursa in the cuff, compression of the tendon, and direct joint communication through a tendon gap with SA-SD bursa distension (16). There are two sonographic findings suggestive of a partial-thickness cuff tear: (i) a mixed hypo- and hyper-echoic focus in the critical zone of the tendon; and (ii) either a hypoechoic or an anechoic cleft within the tendon with bursa or articular extension in both the longitudinal and transverse planes (17). An anechoic area (> 2 mm) around the long head of the biceps tendon in the transverse and longitudinal views was interpreted as effusion in the biceps tendon sheath. Bicipital tenosynovitis was confirmed when a thickened hypoechoic area, with increased power Doppler flow, was found around the biceps tendon. Biceps tendinosis was defined as an enlargement of and decreased echogenicity in the biceps tendon. Tendinosis of the rotator cuff was defined as hypoechoic and swelling changes, with a difference in tendon thickness (> 2 mm) in comparison with the healthy side. If fluid accumulation was observed in the SA-SD bursa, with an increased thickness of > 2 mm and hyperaemia as observed by power Doppler imaging, bursitis was confirmed.

Data analyses

Statistical analyses were performed using SPSS software (SPSS v12.0; Chicago, IL, USA). The point-biserial correlation coefficient test was applied to evaluate the correlation between HSP and the BMR stage, shoulder sensation, shoulder spasticity, glenohumeral subluxation, and abnormal sonography findings. The correlation between HSP and shoulder ROM limitation was analysed using the Pearson correlation coefficient test. Correlations between HSP and abnormal sonographic findings in acute and chronic stage patients were calculated using the McNemar test. A paired *t*-test was used to compare the HSP pain score between the acute and chronic stages. Statistical significance was defined as $p < 0.05$.

RESULTS

A total of 104 patients was enrolled during the acute stage of stroke recovery. Of these, 76 patients (30 women and 46 men; age range 30–87 years; mean age 59.7 (SD 13.5) years) completed all clinical and sonographic evaluations before discharge (mean post-stroke time 46.1 (SD 11.6) days) and 6 months after discharge (mean post-stroke time 218.4 (SD 24.3) days).

The correlations between the physical/sonographic findings and HSP during the acute and chronic stage examinations are listed in Table I. During the acute stage, significant correlations were found between HSP and shoulder motor function level ($r = -0.30$; $p = 0.01$) and shoulder ROM limitation, in all 5 motion planes, ($r = -0.25$ to -0.57 ; $p < 0.01$). No significant correlations were observed between HSP and shoulder spasticity, sensation impairment, glenohumeral subluxation, and abnormal findings of shoulder sonography during the acute stage. During the chronic stage, significant correlations between HSP and

Table I. Correlations between hemiplegic shoulder pain (HSP) and the physical/sonographic findings during the acute and chronic stages (n = 76)

	Acute stage		Chronic stage	
	HSP (γ)	p	HSP (γ)	p
BMR stage (poor, good)	-0.30	0.01*	-0.28	0.02*
Sensation				
Light touch	0.14	0.24	0.20	0.08
Pinprick	0.07	0.58	0.21	0.07
Proprioception	0.14	0.24	0.18	0.12
Shoulder ROM limitation				
Flexion	-0.39	<0.01**	-0.52	<0.01**
Extension	-0.33	<0.01**	-0.38	<0.01**
Abduction	-0.57	<0.01**	-0.61	<0.01**
External rotation	-0.36	<0.01**	-0.46	<0.01**
Internal rotation	-0.25	<0.01**	-0.15	0.19
Shoulder spasticity	0.07	0.57	0.28	0.02*
Glenohumeral subluxation	0.20	0.1	0.21	0.08
Abnormal shoulder sonography	0.16	0.19	0.45	<0.01**

*p<0.05; **p<0.01. Point-biserial correlation test was used for Brunnstrom motor recovery (BMR) stage, shoulder sensation, spasticity, glenohumeral subluxation, and abnormal shoulder sonography findings. Pearson correlation coefficient test was used for shoulder range of motion (ROM) limitation.

shoulder motor function level (r=-0.28; p=0.02), shoulder motion limitation (flexion/extension/abduction/external rotation; r=-0.38 to -0.61; p<0.01), shoulder spasticity (r=0.28; p=0.02), and abnormal sonographic findings (r=0.45; p<0.01) were observed. No significant correlations were noted between HSP and glenohumeral subluxation, sensation impairment, and shoulder internal rotation limitation during chronic stage.

Significant correlations between HSP and abnormal sonographic findings (r=0.45; p<0.01) were noted only in chronic stage patients. The correlations between the sonographic findings of periarticular soft tissues and HSP during the chronic stage are presented on Table II. Significant correlations were noted between HSP and bicipital tendinopathy (r=0.24; p=0.03) or supraspinatus tendinopathy (r=0.25; p=0.03) during the chronic stage.

Table III shows the occurrence and severity of HSP and abnormal sonographic findings during both the acute and chronic stages. A significantly higher incidence of HSP was noted during the chronic stage (n=62; 81.6%) than during the acute stage (n=45; 59.2%; p=0.014). The mean HSP pain score (3.99) during

Table II. Correlations between sonographic findings in periarticular soft tissues and hemiplegic shoulder pain (HSP) during the chronic stage

	HSP	
	(γ)	p
Abnormal sonographic findings in hemiplegic shoulders		
Biceps tendon (effusion, tenosynovitis, tendinitis, tear)	0.24	0.03*
Supraspinatus tendon (tendinosis, tear)	0.25	0.03*
Subscapularis tendon (tendinosis, tear)	0.13	0.25
Subacromial-subdeltoid bursa (effusion, bursitis)	0.17	0.14
Total	0.45	<0.01**

*p<0.05; **p<0.01. Point-biserial correlation test was used for abnormal findings of shoulder sonography.

Table III. Comparison of hemiplegic shoulder pain (HSP), pain scores, and abnormal sonographic findings between the acute and chronic stage

	Acute stage	Chronic stage	p
HSP, n (%)	45 (59.2)	62 (81.6)	0.014*
Pain score (0-10), mean (SD)	2.71 (2.86)	3.99 (2.71)	<0.01**
Abnormal sonographic findings, n (%)			
Biceps (effusion, tenosynovitis, tendinitis, tear)	30 (39.5)	44 (57.9)	0.01*
Supraspinatus (tendinosis, tear)	23 (30.3)	31 (40.8)	0.1
Subscapularis (tendinosis, tear)	7 (9.2)	17 (22.4)	0.01*
Subdeltoid (effusion, bursitis)	23 (30.3)	10 (13.2)	0.02*
Total	52 (68.4)	61 (80.3)	0.12

*p<0.05; **p<0.01. The McNemar test was used for hemiplegic shoulder pain and abnormal sonographic findings. Paired t-test was used for pain scores. SD: standard deviation.

the chronic stage was significantly higher than that (2.71) during the acute stage (p<0.001). Abnormal sonographic findings were noted in 52 patients (68.4%) during the acute stage and in 61 patients (80.3%) during the chronic stage; no significant difference was noted in between the two stages in this regard. Effusion, tenosynovitis or tendinopathy of the long head of the biceps tendon (39.5%), supraspinatus tendon tendinopathy (30.3%), and SA-SD bursitis or effusion (30.3%) were the major sonographic findings during the acute stage. During the chronic stage, the most common sonographic findings were effusion, tenosynovitis or tendinopathy at the long head of the biceps tendon (57.9%), and tendinopathy of the supraspinatus (40.8%) or subscapularis tendons (22.4%). In addition, the number of abnormal sonographic findings of the biceps tendon long head (p=0.01) and subscapularis tendon (p=0.01) during the chronic stage was significantly higher than that during the acute stage. Conversely, the incidence of abnormal sonographic findings of subdeltoid bursa was significantly lesser in the chronic stage than in the acute stage.

DISCUSSION

Many studies have investigated the factors associated with HSP by using cross-sectional analyses, but this is the first longitudinal study to report the correlation of HSP factors in different motor recovery stages. During the acute stage, HSP correlated significantly with shoulder motor function level and ROM limitations. During the chronic stage, HSP correlated significantly with shoulder motor function level, ROM limitations, spasticity and abnormal sonographic findings. Effusion, tenosynovitis or tendinopathy of the biceps tendon long head, and supraspinatus tendinopathy were main sonographic findings during both stages.

In normal shoulders, the shoulder girdle muscles pull the humeral head downwards away from the acromion (10), and the scapula elevators and upward rotators (trapezius and serratus anterior muscles) elevate the coracoacromial arch (18). In addition, shoulder flexion combined with internal rotation of the humerus by the subscapularis muscle and shoulder abduction accompanied by infraspinatus muscle control of humerus lateral rotation contribute to the preventing shoulder impinge-

ment (1). Following a stroke, all the protective mechanisms of the normal shoulder girdle muscles are destroyed. The gravitational pull on the unsupported upper limbs may cause traction injuries to periarticular soft tissues and nerves (19, 20). Moreover, repeated inappropriate stretching and passive ROM exercises often result in injury to the rotator cuff (21). Previous research has shown that acute stroke patients with lower BMR stages (I/II/III) in the upper extremities are prone to soft tissue injuries and HSP (7, 22). Roy et al. (5) reported that greater weakness and poorer arm function were noted in patients with HSP during the first 12 weeks following their stroke. HSP was also reported to occur significantly more frequently in patients who demonstrated lower motor function levels in the hemiplegic shoulder (23). In the present longitudinal study, poor shoulder motor function levels were found to be correlated significantly with HSP, not only in acute stage patients, but also in chronic stage patients.

Various researchers have shown strong, positive associations between HSP and reduced shoulder ROM, especially external rotation limitation (5, 6, 8, 9). These ROM limitations in hemiplegic shoulders result from both adhesive capsulitis and spasticity in the affected joint. Results from the present study showed moderate to high correlations between shoulder motion limitations (flexion/extension/abduction/external rotation) and HSP in both acute and chronic stage patients. Shoulder internal rotation was poorly correlated with HSP during the acute stage, and no correlation was observed between shoulder internal rotation and HSP during the chronic stage. In chronic stage patients, the muscle tone of the shoulder internal rotators and adductors increased, resulting in ROM limitation during external rotation and abduction.

Prior investigations have described the local effect of some anti-spasticity medications on HSP relief, indirectly verifying the association between shoulder spasticity and HSP (24–26). However, studies examining the role of spasticity in HSP have quite inconsistent results. Some of these studies have indicated a positive role of spasticity in the development of HSP (6, 27), and others have shown contrary results (5, 23). The contrary results emerging from these studies may be, at least in part, due to the use of different methods for assessing spasticity, different degrees of stroke severity in the patient populations, and different lengths of time since the stroke occurred. Interestingly, some reports failed to show a significant relationship between HSP and spasticity in recent stroke patients (5, 7, 23), while another report demonstrated a strong association between HSP and spasticity in patients who were followed up for a year after stroke (27). Similar to the latter report, the current study found a significant correlation between shoulder spasticity and HSP only in chronic stage patients. This is expected, since chronic stage patients typically progress from having flaccid shoulders to spastic shoulders over time. When patients develop shoulder spasticity, the periosteal muscle attachments, which have sensory receptors, may generate pain sensations due to the traction force caused by the spasticity (11). Spasticity may also cause reflexive, protective muscle spasms, which lead to shoulder pain when the restricted joint is stretched during passive ROM exercises. These mechanisms increase shoulder joint contracture and lead to a vigorous cycle of HSP.

Rotator cuff tendinopathy is attributed to both intrinsic and extrinsic factors (29). In stroke patients, the extrinsic factors may include microtrauma, cumulative traction injuries from gravity or inappropriate exercises, and abnormal humeral and scapular kinematics due to weakness and spasticity of the rotator cuff and scapular muscles. The intrinsic factors, including age-related changes in cell activity, mechanical properties, and poor vascularity, may result in rotator-cuff degradation with tensile or shear overload. Previous research has indicated that rotator-cuff injuries and tendinopathies were highly prevalent in hemiplegic patients and were related to HSP (7, 8, 28, 30). In most studies, arthrography and magnetic resonance imaging (MRI) were chosen to survey the rotator-cuff injury in patients. However, arthrography is relatively invasive, and has some limitations for this purpose (31). Only two studies evaluated shoulder structure abnormalities related to HSP patients by using MRI, and both these studies included chronic stage patients (30, 32). Távora et al. (32) reported that adhesive capsulitis, as determined by MRI, was significantly more frequent in chronic stroke patients with HSP than in those without HSP. Other investigators have concluded that patients with HSP had a higher prevalence of rotator-cuff tears and rotator-cuff and/or deltoid tendinopathy, upon MRI examination (30). Shoulder MRI is usually not practical for stroke patients because of the high expense, limited availability, and some contraindications. Although shoulder sonography has varying sensitivity (56–100%) and specificity (76–94%) for diagnosing rotator-cuff injuries (17, 33, 34), it is useful for surveying stroke patients for soft tissue injuries. Previous studies have shown that structural changes in the supraspinatus tendon and the long head of the biceps tendon are the most common abnormal sonographic findings in patients with HSP (22, 23). Lee et al. (28) reported that the primary sonographic findings in hemiplegic shoulders were bicipital effusion (39%) and supraspinatus/biceps tendinosis (17.3%). These researchers did not conduct any further analysis of the correlation between HSP and abnormal sonographic findings, nor did they clearly limit the length of time since the stroke occurred.

The present study is the first longitudinal study using sonography to follow-up on HSP patients in a cohort of individuals and explore the correlation between abnormal sonographic findings and HSP. In the present study, abnormal sonograms from hemiplegic shoulders correlated with HSP in chronic stage patients, but not in those in the acute stage. Further analysis of the relationships between each shoulder tendon and chronic stage HSP showed a significant correlation linking bicep tendon and/or supraspinatus tendinopathy with HSP in chronic stage individuals. During both the acute and chronic stages, lesions associated with the supraspinatus tendon and the long head of the biceps tendon were the major findings, similar to the findings of previous studies (22, 23, 28). As abnormal sonographic findings of biceps tendon long head ($p=0.01$) and subscapularis tendon ($p=0.01$) increased significantly at chronic stages, therapists and caregivers should take care during therapy to avoid overloading and overstretch exercises of the bicep and subscapularis muscles. This is particularly true since these muscles play important roles in the spasticity of elbow flexion and shoulder internal rotation in stroke patients.

The limitations of this study include the fact that all patients came from a single rehabilitation centre and therefore are not necessarily representative of the overall population of stroke patients. In addition, we did not record the patients with hemineglect and the details of exercise programmes on upper extremity after stroke, which may be correlated with HSP. Sonography was used to assess shoulder muscle abnormalities, instead of MRI, which is often considered the gold standard for such examinations. In the future, a multicentre longitudinal study that follows up patients over a longer period is warranted. In addition, the frequency, types, and duration of the exercise programmes prescribed for the upper extremities should also be recorded, in order to determine if they are related to HSP.

In conclusion, the occurrence of HSP was 59.2% in acute stage stroke patients and 81.6% in those individuals in the chronic stage. Significant correlations were found between HSP and the lower motor function levels and shoulder ROM limitations in both acute and chronic stage patients. Shoulder spasticity and abnormal sonographic findings were highly correlated with HSP in chronic stage patients only. In addition, this study indicated that patients categorized at a lower BMR stage, with respect to their proximal upper limbs, should be especially cautious when performing activities that might lead to shoulder injuries after stroke. For long-term care, all patients should receive continuous therapy to maintain shoulder ROM and reduce spasticity for HSP prevention.

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