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Original Article

Sonographic findings of painful hemiplegic shoulder after stroke

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Abstract

Background: Hemiplegic shoulder pain is common in stroke patients and can influence rehabilitation outcome. The underlying pathology can be various: in addition to impaired motor control and altered peripheral and central nervous activity, soft tissue lesions may also play an important role. It remains unclear how these pathologies may interact or correlate with each other.
Methods: This retrospective study collected data from 26 stroke patients who received sonography examination due to shoulder pain. Severity of soft tissue lesion over the shoulder joint was graded on the basis of the sonographic findings. The information regarding cognition, sensory function, spasticity (measured by the Modified Ashworth Scale) and the Brunnstrom stage of motor recovery was collected though medical chart review. This study examined the association between sonographic findings and the clinical findings.
Result: This study showed that sonographic grading of painful hemiplegic shoulder is not statistically associated with impaired cognition and sensory function. (p value = 0.0587 and 0.9776, respectively) In addition, there is no correlation between sonographic grading and motor recovery in patients with hemiplegic shoulder pain. (Spearman's correlation coefficient = −0.0053, p value = 0.9796) Neither is there any statistically significant correlation between sonographic grading and the degree of spasticity. (Spearman's correlation coefficient = −0.0311, p value = 0.8801).
Conclusion: The results of this study suggests that the mechanism through which soft tissue lesions causes hemiplegic shoulder pain may be independent of the mechanisms through which changes of muscle tone and nervous activity causes shoulder pain.

Keywords: Hemiplegia; Shoulder pain; Sonography; Stroke

1. Introduction

Shoulder pain is a common clinical consequence of stroke. Previous studies have reported an incidence of shoulder pain after stroke ranging from 9% to 73%.[1,2] Shoulder pain can occur as early as 2 weeks after stroke, but typically occurs 2–3 months after stroke.[3] The median pain score is most severe at 4 months following stroke.[4] The degree of pain can be moderate to severe, and may intensify at night. It has been reported that shoulder pain is associated with depression, disturbed sleep, and a reduced quality of life.[5,6] Hemiplegic shoulder pain is also associated with prolonged length of hospital stay and poor recovery of arm function during the first 12 weeks of illness.[7]

Due to the various possible underlying pathogeneses involved in the development of hemiplegic shoulder pain, the precise etiology is difficult to assess. Kalichman et al.[5] performed a systematic review regarding associated factors and underlying pathology of hemiplegic shoulder pain. They proposed a systemization for pathologies underlying hemiplegic shoulder pain, which includes impaired motor control (change of muscle tone), altered peripheral and central nervous activity, and soft tissue lesions. These underlying pathologies may

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occur separately or simultaneously. In addition, each pathology may initiate the development of another. It remains unclear how these pathologies may interact or correlate with each other.

Therefore, in order to clarify the associations between the factors that might cause hemiplegic shoulder pain the evaluation of soft tissue lesions is key. The standard imaging modalities for evaluating soft tissue lesions of shoulder region are arthrography and magnetic resonance imaging.[8,9] However, both types of examinations take a long time and cost a large amount of money. In addition, neither of these two imaging modalities is indicated for stroke patients due to limited and intolerable positioning. Sonography on the other hand is a noninvasive method for evaluating soft tissue lesions that is a quick procedure which provides real-time and dynamic evaluation of multi-planar structures and movements.

Therefore, the goal of this study was to examine the sonographic findings of painful hemiplegic shoulder in stroke patients. This study also investigated the association between sonographic findings and clinical findings including cognition, sensory function, spasticity, and motor recovery.

2. Methods

2.1. Study population

This study retrospectively collected data from stroke patients who received sonographic evaluation of their shoulder joint due to shoulder pain between January 2015 and December 2015. Patients with a diagnosis of a new-onset stroke within the previous 6 months were included. Patients were excluded if radiographic examination revealed any humeral fracture or shoulder dislocation.

2.2. Sonographic examination

Shoulder sonographic examinations were performed using a 4.4−13.0 MHz linear transducer (Acuson X300 Ultrasound System, premium edition, Siemens) by three physicians certified by the Taiwan Society of Ultrasound in Medicine. The sonography examination followed a sequence of steps described by previous studies to ensure a complete and thorough evaluation.[10,11] The assessed soft tissues included the long head of the biceps brachii tendon, supraspinatus tendon, subscapularis tendon, infraspinatus tendon, and subacromial-subdeltoid (SA-SD) bursa. Patients underwent the sonography examinations while sitting in their wheelchairs.

The severity of soft tissue lesions over the shoulder joint can be classified by a grading system proposed in previous studies on the basis of the sonographic findings. The grading system is described as follows: grade 1, normal or effusion of the biceps tendon sheath; grade 2, tendinosis of the supraspinatus; grade 3, SA-SD bursitis; grade 4, a partial-thickness tear of the rotator cuff; and grade 5, a full-thickness tear of the rotator cuff. The cases with two or more abnormal sonographic findings were graded on the basis of the most severe finding.[12,13]

2.3. Physical examination

This study collected information regarding clinical findings including cognition and sensation, as well as the spasticity and motor recovery of the affected upper limb. The data were collected through medical chart review. The study variables were measured. Cognitive function was evaluated by assessing judgment, orientation, memory, abstract thinking, and calculating ability. Any impairment of such dimensions of cognition was defined as impaired cognitive function. Sensory function included pinprick sensation, light touch sensation, and proprioception over the hemiplegic side. Any impairment of such dimensions was defined as impaired sensory function. Spasticity was evaluated using the Modified Ashworth Scale, an ordinal scale ranging from 0 to 4, in which 0 refers to no increase in muscle tone; 1 refers to slight increase in muscle tone; 2 and 3 refer to more and considerable increase in muscle tone, and 4 refers to rigid in flexion or extension.[2] Brunnstrom stage was used to evaluate stroke recovery. It consists of seven stages: stage 1, flaccidity with no movement of the affected muscles; stage 2, passive movement and spasticity occur; stage 3, active movement and spasticity occur; stage 4, spasticity decreases and significant motor control emerges; stage 5, spasticity wanes and complex movement develops; stage 6, coordination reappears and spasticity is no longer present; and stage 7, normal function returns and full control of muscle movement is restored.

2.4. Data analysis

This study investigated the correlation between sonographic grading and clinical findings including cognitive function, sensory function, motor recovery (measured by Brunnstrom stage), and spasticity (measured by the Modified Ashworth Scale). The correlations between sonographic grading and cognitive function as well as sensory function were analyzed using the Wilcoxon (Mann−Whitney) rank-sum test. The correlations between sonographic grading and Brunnstrom stage as well as the Modified Ashworth Scale were analyzed using the Spearman's rank correlation test. Statistical significance was defined as $P < 0.05$. Statistical analyses were performed using STATA software (STATA IC 14.2, College Station, TX, USA).

3. Results

A total of 26 patients were included; their mean age was 66.52 years (range: 38.5−90.9 years). Among the patients, 18 were male and 8 were female. Twenty patients had cerebral infarction and 6 had cerebral hemorrhage. Ten patients had right hemiplegia and 16 had left hemiplegia. Shoulder sonography was performed at a mean of 78.15 days after stroke. The characteristics of the study population are shown in Table 1.

All of the 26 patients included in this study presented with at least one abnormal sonographic finding. In addition, more than two-thirds of the patients (N = 18) had more than one

Table 1
Characteristics of the study population.

	Number	Percentage
Age		
Mean ± SD	66.52 ± 14.67	
Range	38.5−90.96	
Gender		
Female	8	30.77%
Male	18	69.23%
Type of stroke		
Ischemic	20	76.92%
Hemorrhagic	6	23.08%
Hemiplegic side		
Right	10	38.46%
Left	16	61.54%
Duration of stroke (day)		
Mean ± SD	78.15 ± 48.21	
Range	3−179	

abnormal sonographic finding. The sonography examinations showed that SA-SD bursitis is the most common finding in hemiplegic shoulder pain; it was seen in 73.08% of the patients (N = 19). Biceps tendinitis was also commonly seen; it occurred in 69.23% of the patients (N = 18). Table 2 summarizes the sonographic findings.

Among the 26 patients, 12 had impaired cognitive function (46.15%); 13 had impaired sensory function (50%); and 18 had spasticity over the affected upper limb with a Modified Ashworth Scale score of 1 in 12 patients (46.15%) and 2 in 6 patients (23.08%). The Brunnstrom stage of the study population ranged from stage 2 to stage 5. The details of the clinical findings are shown in Table 2.

The correlations between sonographic grading and clinical findings are summarized in Table 3. Although patients with impaired cognition had a higher grade of sonographic severity of hemiplegic shoulder pain, there was no statistically significant difference ($P = 0.0587$). Neither was there any statistically significant difference of the sonographic grading between the patients with and without intact sensory function ($P = 0.9776$). This study also examined the relationship between sonographic grading and the degree of spasticity as well as the relationship between sonographic grading and motor recovery. The results showed that there was no significant correlation between sonographic grading and the degree of spasticity as measured by the Modified Ashworth Scale ($P = 0.8801$; Spearman's correlation coefficient = −0.0311). Neither was there any significant correlation between sonographic grading and motor recovery, as measured by Brunnstrom stage ($p = 0.9796$; Spearman's correlation coefficient = −0.0053).

Table 2
Sonographic and clinical findings of the study population.

Variables	Number	Percentage
Sonographic finding		
Biceps tendon lesion	18	69.23%
Supraspinatus tendon lesion	7	26.92%
SA-SD Bursitis	19	73.08%
Partial-thickness rotator cuff tear	6	23.08%
Full-thickness rotator cuff tear	3	11.54%
Cognition		
Intact	14	53.85%
Impaired	12	46.15%
Sensory function		
Intact	13	50%
Impaired	13	50%
Modified Ashworth Scale		
0	8	30.77%
1	12	46.15%
2	6	23.08%
Brunnstrom stage		
2	6	23.08%
3	9	34.62%
4	7	26.92%
5	4	15.38%

Table 3
The correlation between sonographic grading and clinical findings.

Sonographic grading	1	2	3	4	5	*P*
Cognition						0.0587
Intact	0	2	10	1	1	
Impaired	1	0	4	5	2	
Sensory function						0.9776
Intact	0	2	6	4	1	
Impaired	1	0	8	2	2	
Modified Ashworth Scale						0.8801
0	0	1	4	2	1	
1	1	0	7	3	1	
2	0	1	3	1	1	
Brunnstrom stage						0.9796
2	1	0	3	2	0	
3	0	1	4	2	2	
4	0	1	4	1	1	
5	0	0	3	1	0	
Total	1	2	14	6	3	

4. Discussion

The pathogenesis underlying hemiplegic shoulder pain after stroke remains unclear, but clinicians need to identify the precise etiology of hemiplegic shoulder pain in order to achieve a satisfactory treatment outcome for patients. This study reviewed medical chart records from 26 patients diagnosed to have stroke within the previous 6 months. These patients received a sonographic examination due to shoulder pain. This study examined the relationship between sonographic findings and clinical findings for such variables as cognitive function, sensory function, spasticity, and motor recovery. The results showed that there was no statistically significant correlation between sonographic findings and clinical findings.

Impaired motor control and soft tissue lesions are two major causes of hemiplegic shoulder pain. Whether these two causes are associated with each other and, if so, how they are associated remains unclear. Previous studies have provided mixed results. Lee et al. recruited a total of 71 stroke patients and found that there was no correlation in patients with hemiplegic shoulder pain between Brunnstrom stage of motor recovery and the grades of sonographic findings.[12] A longitudinal study by Pong et al. that included 104 patients reported significant correlation between hemiplegic shoulder pain and abnormalities only during the chronic stage of stroke (defined as 6 months after discharge), but not during the acute stage of stroke.[14] Another study by Mohamed et al. reported that the sonographic grading of shoulder lesions was not correlated with Brunnstrom stage of motor recovery.[15] The present study supported the finding of previous studies that no significant correlation exists between sonographic grading of severity and Brunnstrom stage of motor recovery. This may suggest that motor recovery is not associated with soft tissue lesions; it may also imply that patients with different stages of motor recovery may present with different levels of severity or different types of soft tissues lesions. For example, a study in which sonography examinations were performed on 57 hemiplegic patients found that patients with poor motor function (defined by Brunnstrom stage of 1−3) have a significantly higher proportion of abnormal sonographic findings only in the supraspinatus tendon, not in the biceps tendon, subscapularis tendon, or SA-SD bursa.[16] Further studies on the association between motor recovery and each specific type of soft tissue lesion are needed.

Spasticity refers to a velocity-dependent increase in muscle tone that is associated with a hyperactive stretch reflex. The literature shows that hemiplegic shoulder pain is more likely to occur in patients with spasticity than in patients with flaccidity.[17] Spasticity over the shoulder girdle muscles, especially the subscapularis, leads to muscle imbalance and limits external rotation, and may be associated with hemiplegic shoulder pain.[18,19] Very few studies have investigated the association between spasticity and sonographic grading of severity of shoulder lesions. The study by Mohamed et al., which included 80 patients with hemiplegic shoulder pain, showed that the sonographic grading of the shoulder lesion is not correlated with the degree of spasticity.[15] This study showed a consistent finding that the Spearman's correlation coefficient between sonographic grading and the degree of spasticity (measured by the Modified Ashworth Scale) was −0.0311 ($P = 0.8801$). Therefore, this study showed no statistically significant correlation between sonographic grading and the degree of spasticity. This result may suggest that the mechanism through which spasticity causes hemiplegic shoulder pain is independent of the mechanism through which soft tissue lesions cause hemiplegic shoulder pain.

Kalichman et al. considered altered peripheral and central nervous activity as one of the major causes of hemiplegic shoulder pain. This category of causes includes peripheral entrapment, shoulder-hand syndrome, central post-stroke pain, and spinal and supra-spinal central sensitization, as well as neglect and sensory impairment.[5] This study examined the association between sensory function and sonographic grading of severity of hemiplegic shoulder pain, which has not been reported in the literature. The sensory functions assessed in this study included light touch sensation, pinprick sensation, and proprioception. Any impairment was defined as impaired sensory function. This study did not find any statistically significant association between sensory function and sonographic grading of a soft tissue lesion in painful hemiplegic shoulder. This result may be due to the heterogeneous dimensions of sensory function. For example, previous studies showed that hemiplegic shoulder pain is associated with loss of proprioception, but is not associated with light touch and pinprick sensation.[20] As the present study is a retrospective study via medical chart review, the information regarding proprioception in some patients was missing and, therefore, the present study did not examine the association between each specific dimension of sensory function and sonographic grading.

This study showed that patients with impaired cognition function are more likely to have a more severe soft tissue lesion in the shoulder joint, but a statistically significant difference was not reached ($P = 0.0587$). This might be due to insufficient patient number as only 26 patients were included. The reasons why patients with impaired cognition function are more likely to present with soft tissue lesions in the shoulder joint may be due to iatrogenic causes such as improper care given by the health care personnel or excessive exercises regarding the affected limbs. Health care personnel or family may, through improper passive or assisted active exercise and improper handling or lifting, cause shoulder pain for patients.[5] Patients with impaired cognitive function may have problems in timely communication of their pain to health care personnel or family, which results in soft tissue lesions. Therefore, instruction and awareness by health care personnel, especially caregivers, is very important for stroke patients with impaired cognition.

One major limitation of this study is that the patients included might have had a soft tissue lesion over the shoulder joint before the stroke occurred. Patients may have had degenerative soft tissue lesions over the shoulder joint, especially if they were elderly. Shoulder pain may also have developed during a fall or a near fall accident or from traction injury when stroke occurred. Another limitation is that this study did not measure the individual muscle function or detailed sensory impairment of hemiplegic limbs. Therefore, it is impossible to exclude neuropathic pain, cervical radiculopathy, or shoulder-hand syndrome in that limb. In this regard, no definitive statement can be made about isolated influence of the aforementioned issues associated with hemiplegic shoulder pain.

In conclusion, this study showed that hemiplegic shoulder pain is not statistically significantly associated with impaired cognition or sensory function. In addition, there is no correlation between sonographic grading and motor recovery in patients with hemiplegic shoulder pain. Also, neither is there any statistically significant correlation between sonographic grading and the degree of spasticity. This might suggest that the mechanism through which soft tissue lesions cause hemiplegic shoulder pain is independent of the mechanisms through which changes of muscle tone and nervous activity cause shoulder pain.

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