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Investigation of factors affecting shoulder pain in stroke survivors

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ABSTRACT

Background: Shoulder pain is influenced by multiple factors in stroke survivors.
Purpose: This study investigated factors contributing to shoulder pain in stroke survivors and quantified their impact.
Study Design: Sixty-two stroke patients (21 females, 41 males; mean age: 63.97 ± 10.02 years) at Brunnstrom stage 3 or higher were included.
Methods: Shoulder pain was assessed using the Visual Analog Scale, muscle tone with the Modified Ashworth Scale, and myofascial trigger points through palpation. A universal goniometer measured the range of motion, and the Fugl-Meyer Upper Extremity Assessment evaluated upper limb function. Soft tissue conditions were assessed using the Neer Impingement, Apprehension, Acromioclavicular Shear, and Speed tests.
Results: Hemiplegic shoulder pain (HSP) was present in 50% of patients, primarily in the anterior (35.5%) and lateral (32.3%) shoulder. Burning and stinging sensations were common. No significant associations were found with age, gender, or time since stroke. However, HSP correlated with rehabilitation initiation time (p = 0.007, r = −0.34), Brunnstrom stage (p = 0.015, r = 0.31), and Fugl-Meyer score (p = 0.015, r = 0.31). Increased muscle tone in the subscapularis (p = 0.046, r = 0.26) and pectoralis major (p = 0.002, r = 0.38) was linked to HSP. Myofascial trigger points in the levator scapulae, supraspinatus, upper trapezius, teres major, teres minor, and infraspinatus muscles showed significant correlations Soft tissue pathology, indicated by the Neer Impingement (p = 0.000, r = 0.46), speed (p = 0.007, r = 0.34), and apprehension (p = 0.000, r = 0.52) tests, was also associated with HSP.
Conclusions: HSP in stroke survivors is influenced by myofascial trigger points, soft tissue injuries, delayed rehabilitation, increased muscle tone, and lower motor function. Early intervention focusing on shoulder mobility is crucial for prevention and management, promoting functional recovery.

Contribution of the Article

This study provides valuable insights into the multifactorial nature of hemiplegic shoulder pain (HSP) in stroke survivors. By employing a comprehensive approach, it evaluates factors such as muscle tone, myofascial trigger points, and soft tissue pathologies, offering a holistic understanding of HSP. Furthermore, it enriches the existing literature by comparing its findings with prior research, addressing the variability in HSP prevalence reported across studies. These contributions underscore the importance of early and targeted interventions for effective management of HSP, ultimately improving the quality of life for stroke survivors.

Introduction

Stroke is a vascular-origin condition characterized by the rapid loss of brain function. Globally, stroke is the second leading cause of death among adults, following heart disease, and ranks third as a cause of disability.[1,2] Among its many complications, hemiplegic shoulder pain (HSP) is one of the most prevalent, affecting approximately 65%-70% of stroke survivors.[3] Beyond being a symptom, HSP plays a critical role in the functional recovery process. It can

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significantly hinder recovery, increasing the risk of long-term disability. Optimal shoulder function and stability are essential for enabling normal hand mobility, gait, and the performance of daily activities.[4]

Despite its clinical significance, the etiology and treatment of HSP remain poorly understood. Various factors are implicated in its development, including shoulder subluxation, complex regional pain syndrome type 1, biceps tendinitis, rotator cuff abnormalities, adhesive capsulitis, scapulohumeral rhythm loss, prolonged immobilization, and increased muscle tone. These factors can be broadly categorized into impaired motor function and muscle tone, soft tissue pathologies, and sensory problems.[5,6] While motor dysfunction and muscle tone abnormalities are commonly associated with HSP, the pain does not directly correlate with these impairments. Instead, disruptions in scapulohumeral rhythm, uncontrolled movement patterns, altered muscle tone, and imbalances between agonist and antagonist muscles contribute to the condition.[7]

Although numerous studies have investigated the causes of HSP, there is limited understanding of the specific factors that directly contribute to pain and their relative influence. Many studies have focused on a narrow set of muscle groups, leading to incomplete conclusions about the underlying mechanisms of HSP. This study aimed to address this gap by comprehensively investigating the factors influencing HSP and identifying the elements that contribute most significantly to its development.

Method

Study design

This study was conducted between December 2019 and July 2023 among stroke patients treated at the Physical Therapy and Rehabilitation Unit of Pamukkale University Faculty of Medicine Hospital. Patient selection was performed through screening with the Probel system, and those meeting the inclusion criteria and consented to participate were randomly assigned using a random number table.

Data collection was conducted by a designated researcher who performed the measurements. To ensure objectivity, an independent assessor, blinded to the study conditions, evaluated the measurements, while the researcher was excluded from the assessment process.

Ethical approval

Ethical approval was obtained from the Pamukkale University Non-Interventional Clinical Research Ethics Committee (Ethics Number: 6016787-020-/544). All procedures adhered to the ethical standards of the committee for human experimentation and the 1975 Declaration of Helsinki, revised in 1983. Written informed consent was obtained from all participants.

Participants

Stroke patients aged between 20 and 85 were included in the study. The inclusion criteria for this study were as follows: having a diagnosis of hemiplegia following a cerebrovascular accident, experiencing a first-time stroke, having unilateral involvement, and being at stage 3 or above on the Brunnstrom upper limb stages to ensure that the selected tests (eg, Neer impingement test) could be administered. Additionally, participants were required to have no other neurological or orthopedic conditions besides hemiplegia, no history of shoulder problems on the affected side prior to the stroke, and a Mini-Mental State Examination score of 24 or above to exclude individuals with cognitive impairments. There was no time limitation for inclusion participants at all stages of stroke were eligible. Participation in the study was voluntary, and individuals who did not provide consent were not assessed. Participants were excluded if they had a history of shoulder injury on the affected side, neurological or orthopedic conditions unrelated to stroke, a previous stroke, bilateral involvement, noncooperative behavior.

Measurements

We assessed the demographic data of the participants using a demographic data form, shoulder pain severity was evaluated using the Visual Analog Scale during rest, activity, and sleep after recording the pain location and type.

Muscle tone of shoulder flexors, subscapularis, infraspinatus, and pectoralis major muscles were assessed using the Modified Ashworth Scale (MAS).

Myofascial trigger points around the scapula and shoulder muscles were evaluated through manual palpation.

Shoulder flexion, extension, abduction, adduction, and internal and external rotation range of motions (ROMs) were measured using a universal goniometer.

We utilized the Brunnstrom Upper Extremity Scale to classify upper extremity levels, and upper extremity motor impairment was assessed using the Fugl-Meyer Motor Assessment Scale.

We employed Neer, speed, acromioclavicular shear, and apprehension tests for the examination of soft tissue pathologies.

Visual Analog Scale was used to assess the shoulder pain of stroke survivors. The patients were asked to mark the point on a 10 cm line that best represents their current pain, with 0 indicating no pain and 10 indicating unbearable pain. Subsequently, the distance of the point from the 0 point will be measured using a ruler to determine the intensity of the pain. Following this, the patient will be inquired about the localization and type of pain.[8]

The MAS was used to evaluate muscle tone and is commonly applied for spasticity assessment in clinical settings. The original version, known as the Ashworth Scale, categorized the resistance of an extremity to passive movement on a scale of 0 to 4 points. Later, the scale was modified to include a score of 1+, resulting in the 6-point MAS.[9] According to MAS (Table 1).

Fugl-Meyer Motor Assessment Scale was used to evaluate motor recovery after a stroke, it is a disease-specific, reliable, and up-to-

Table 1
Modified Ashworth Scale

Score	Description
0	No increase in muscle tone
1	Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the range of motion
1+	Slight increase in muscle tone, manifested by a catch and release or by minimal resistance throughout less than half of the range of motion
2	A more marked increase in muscle tone through most of the range of motion
3	Considerable increase in tone, passive movement difficult
4	Affected part rigit in range of motion

date scale. It includes sub-sections assessing joint movements, coordination, and reflex activities related to the shoulder, elbow, forearm, wrist, and hand. The maximum score achievable from the upper extremity assessment is 66.[10]

Neer impingement test was used to identify possible subacromial impingement syndrome. The patient's shoulder is passively brought into flexion and internal rotation position. The positive examination finding is the occurrence of shoulder pain during the movement of the arm in this position. The test has a sensitivity of 68% and a specificity of 68.7%.[11]

Speed test was used to identify possible biceps tendon pathologies. When shoulder flexion is performed against resistance with the elbow extended and the forearm supinated, the presence of pain in the bicipital groove indicates a positive test. It is more valuable than the Yergason test in demonstrating biceps tendon pathologies. The test has a sensitivity of 88.9% and a specificity of 38.8%.[12]

Acromioclavicular shear test was used to identify acromioclavicular joint pathology. The examiner cups their hands over the shoulder with the heel of one hand on the clavicle, and the heel of the other on the spine of the scapula, and then squeeze their hands together. A positive result is abnormal movement or pain at the acromioclavicular joint. The test has a sensitivity of 100% and a specificity of 97%.[13]

Apprehension test was used to detect the presence of anterior instability. As the shoulder is passively moved into maximum external rotation in abduction, and forward pressure is applied to the posterior aspect of the humeral head. If the patient expresses concern about dislocation or reports pain in the shoulder, the test is considered positive. The test has a sensitivity of 58% and a specificity of 96%.[14,15]

Blinding

Double-blind study.

Sample size

Based on the results of the power analysis, it was calculated that 80% power could be obtained at a 95% confidence level when at least 62 people were included in the study.[10,16,17]

Statistical analysis

Data were analyzed using SPSS 23 software. Continuous variables were expressed as mean ± standard deviation, and categorical variables were presented as frequencies and percentages. The normality of the data was assessed using the Kolmogorov-Smirnov test. For normally distributed data, t-tests were used for comparisons. Pearson correlation analysis was employed to examine relationships between continuous variables, and Chi-Square tests were used for categorical data comparisons. Statistical significance was set at $p \leq 0.05$.

Results

We included a total of 62 (21 female) stroke patients (25 left hemiparesis) with a mean age of 63.9 ± 10.0 years in this study.

We identified HSP in 31 (50%) patients. Upon examining the distribution of pain based on localization, we identified 22 cases of anterior shoulder pain, 20 cases of lateral shoulder pain, 9 cases of posterior shoulder pain, and 5 cases of upper acromion localization (where patients had the option to select multiple locations). When looking at the type of pain, patients mostly described it as burning and stinging (Figs. 1 and 2).

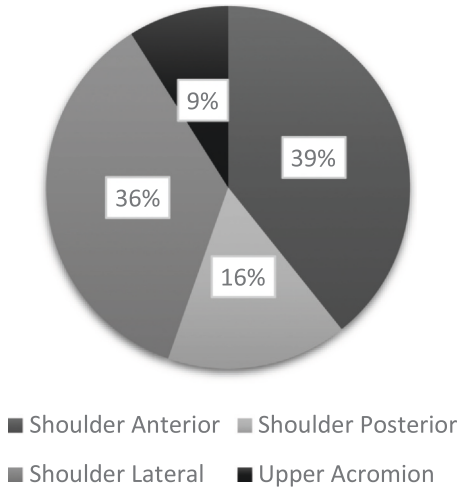

Fig. 1. Pain localizations.

There was no significant relationship found between shoulder pain in stroke survivors and factors such as age ($p = 0.582$, $r = -0.71$), gender ($p = 0.270$, $r = 0.14$), time since stroke ($p = 0.68$, $r = 0.23$), and the affected side ($p = 0.446$, $r = -0.10$) (Table 2).

The rehabilitation initiation periods for individuals with stroke were grouped as acute phase (0-1 month), subacute phase (1-6 months), and chronic phase (6 months and beyond). Of the patients, 42 (67.7%) started rehabilitation in the acute phase, 12 (19.4%) in the subacute phase, and 2 (3.2%) in the chronic phase. Six patients (9.7%) did not receive physical therapy. We found a statistically significant relationship between shoulder pain and the initiation time of the rehabilitation program ($p = 0.007$, $r = -0.34$), Brunnstrom stage ($p = 0.307$, $r = 0.02$), and Fugl-Meyer score ($p = 0.015$, $r = 0.31$) (Table 3).

We found no statistically significant relationship between the muscle tone of shoulder flexor muscles ($p = 0.354$, $r = 0.12$) and infraspinatus muscles ($p = 0.150$, $r = 0.24$) and shoulder pain. We found a statistically significant relationship between shoulder pain and the muscle tone of the subscapularis ($p = 0.046$, $r = 0.26$) and pectoralis major ($p = 0.002$, $r = 0.38$) muscles (Table 4).

When examining the relationship between shoulder pain and myofascial trigger points, we found a statistically significant correlation with trigger points in levator scapulae (p: 0.000, r: 0.47), teres majör (p: 0.013, r: 0.32), teres minör (p: 0.011, r: 0.32), supraspinatus (p: 0.000, r: 0.48), infraspinatus (p: 0.002, r: 0.39), the upper trapezius (p: 0.000, r: 0.50), and middle parts of the trapezius (p: 0.004, r: 0.36) (Table 5).

When examining the relationship between shoulder pain and soft tissue pathologies, we found a statistically significant association between the Neer impingement test ($p = 0.000$, $r = 0.46$), speed test ($p = 0.007$, r: 0.34), and apprehension test ($p = 0.000$, $r = 0.52$) and HSP (Table 6).

Participants with HSP exhibited significant differences in rehabilitation initiation time, muscle tone, ROM, and functional status compared to those without HSP. Rehabilitation was initiated 0.64 months later in the HSP group (mean difference: 0.64, 95% CI [0.18, 1.10], $p = 0.007$). Muscle tone was higher in the subscapularis (mean difference: 0.51, 95% CI [0.10, 1.02], $p = 0.046$) and pectoralis major muscles (mean difference: 0.83, 95% CI [0.31, 1.36], $p = 0.002$) among participants with HSP. Additionally, ROM deficits were observed in the HSP group, with reductions in shoulder flexor (mean difference: −21.45, 95% CI [−35.56, −7.35], $p = 0.004$), abduction (mean difference: −29.96, 95% CI [−49.22, −10.71], $p = 0.003$), external rotation (mean difference: −18.51, 95% CI [−30.55, −6.47], $p = 0.003$), and

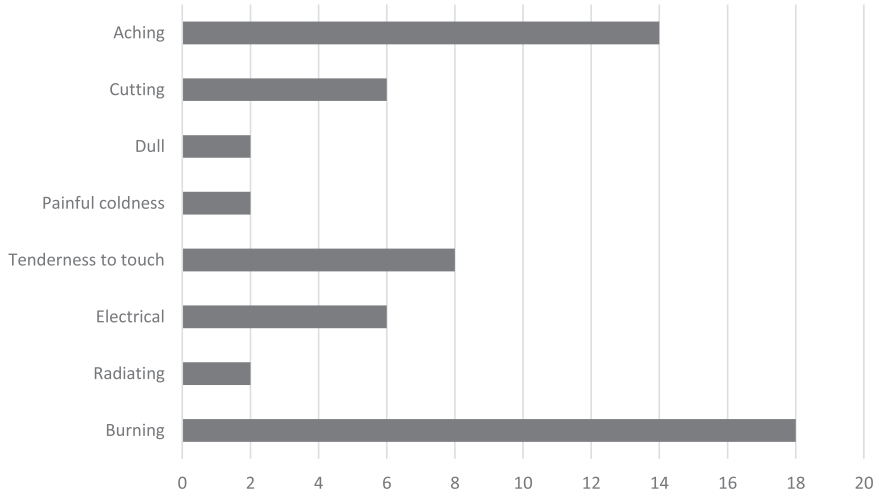

Fig. 2. Types of pain.

Table 2
The correlation between age, gender, stroke duration, affected side, and HSP

		Age	Gender	Stroke duration	Affected side
HSP	*r*	−0.71	0.14	0.23	**−0.01***
	p	0.582	0.270	0.068	0.446

HSP = hemiplegic shoulder pain.
* Pearson correlation coefficient $p < 0.01$.

Table 3
The correlation between initiation time of the rehabilitation program, Brunnstrom stage, Fugl-Meyer score, and HSP

		Initiation time of the rehabilitation program	Brunnstrom stage	Fugl-Meyer Score
HSP	*r*	0.34*	0.31**	0.31**
	p	**0.007**	0.015	**0.015**

HSP = hemiplegic shoulder pain.
* Pearson correlation coefficient $p < 0.01$.
** Pearson correlation coefficient $p < 0.05$.

Table 4
The relationship between HSP and muscle tone

		Shoulder flexors	Subscapularis	İnfraspinatus	Pectoralis major
HSP	*r*	0.12	0.26*	0.15	0.38**
	p	0.354	**0.046**	0.243	**0.002**

HSP = hemiplegic shoulder pain.
* Pearson correlation coefficient: $p < 0.05$.
** Pearson correlation coefficient: $p < 0.01$.

internal rotation (mean difference: −14.35, 95% CI [−25.51, −3.19], $p = 0.013$) ROM. Furthermore, the Fugl-Meyer score was lower by 10.16 points in the HSP group (mean difference: −10.16, 95% CI [−18.25, −2.07], $p = 0.015$), reflecting a significant reduction in motor recovery and functional status (Table 7).

Discussion

The present study provides a comprehensive analysis of the factors associated with HSP in stroke survivors. The findings emphasize the importance of early rehabilitation initiation and considerate assessment of muscle tone, myofascial trigger points, and soft tissue pathologies in managing and preventing HSP, ultimately contributing to improved functional recovery in stroke survivors.

HSP is a commonly encountered problem that can stem from various causes. Knowing the risk factors of this condition, which significantly affects the progression of the rehabilitation program and the patient's functional and psychological state, plays a crucial role in effective rehabilitation.

Reviewing the literature reveals a wide range of prevalence regarding HSP. Koog and colleagues conducted a systematic review that included randomized controlled studies, reporting a range of HSP from 16% to 84%, with another study indicating a variation between 65% and 70%. Our study found a 50% prevalence of HSP, in line with the previously mentioned studies.[18,19]

When examining the relationship between the time elapsed since the stroke and shoulder pain, there is no definitive information in the literature. Shoulder pain can be observed in 75% of patients within the first year.[3] However, Bender and colleagues; review revealed that HSP typically manifests within the first 12 weeks, especially around the 10th week.[18] In our study, there was no statistically significant relationship between the time elapsed since the stroke and HSP. The conclusion reached can be attributed to variations in patients's levels of impairment, differences in their recovery processes, as well as the possibility of the evaluated patients being at different stages in different weeks, which may also affect the outcome.

There are studies in the literature reporting that delayed rehabilitation initiation causes shoulder pain. Wanklyn et al[20] reported that shoulder pain onset occurred within 2 weeks for patients who did not continue rehabilitation and home exercises after hospital

Table 5
The relationship between HSP and myofascial trigger points

		Levator scapulae	Teres majör	Teres minör	Supraspinatus	İnfraspinatus	Upper trapezius	Middle trapezius
HSP	*r*	0.47*	0.32**	0.32**	0.47*	0.39*	0.50*	0.36*
	p	**0.000**	**0.013**	**0.011**	**0.000**	**0.002**	**0.000**	**0.004**

HSP = hemiplegic shoulder pain.
* Pearson correlation coefficient: $p < 0.01$.
** Pearson correlation coefficient: $p < 0.05$.

Table 6
The correlation between HSP and soft tissue

		Neer impingement	Speed	Acromioclavicular shear	Apprehension
HSP	*r*	0.46**	0.34**	0.20	0.52**
	p	**0.000**	**0.007**	0.123	**0.000**

HSP = hemiplegic shoulder pain.
Pearson correlation coefficient $p < 0.05$.
** Pearson correlation coefficient: $p < 0.01$.

discharge. Similarly, in a study conducted by Karaahmet and colleagues, stroke patients who initiated rehabilitation within the first month were compared to those who started after more than a month. A notable difference in the occurrence of shoulder pain was observed between the two groups.[21] Delayed initiation of rehabilitation emerges as a significant risk factor in the development of HSP. Early rehabilitation plays a critical role in preserving shoulder biomechanics through the management of spasticity and the prevention of muscle imbalances. Increased tone in muscles such as the subscapularis and pectoralis major disrupts joint biomechanics, limiting shoulder ROM and exacerbating pain. Interventions aimed at reducing spasticity in these muscles are of great importance for the prevention and management of HSP. In our study, we found a significant relationship between the onset of shoulder pain and the initiation of rehabilitation. Delayed initiation of rehabilitation appears to be a risk factor for HSP.

Researchers observe a 30%-40% increase in muscle tone after a stroke.[22] There are studies in the literature indicating that spasticity disrupts extremity kinematics, leading to HSP. Fernandez et al[23] have stated that an increase in tone in the subscapularis and pectoralis major muscles restricts shoulder external rotation and abduction capabilities, narrows the subacromial space, and predisposes to soft tissue injuries and pain. According to O'Sullivan et al,[24] pain is caused by problems with the scapulohumeral rhythm, which is caused by abnormal muscle tone.[24] This causes tears in the rotator cuff muscles and changes in the structure of the subacromial region. In our study, similar to the literature, we found a significant relationship between the muscle tone of the subscapularis and pectoralis major muscles and HSP. Spasticity, leading to the disruption of normal joint biomechanics, is identified as a risk factor for HSP. The changes caused by spasticity in these muscles create a risk factor for HSP and also reduce the joint's functional capacity.

Gomez et al[25] argued that myofascial trigger points developed on the affected side after a stroke could cause shoulder pain, and therefore, rehabilitation should include attention to myofascial trigger points. Villafane et al[26] reported activated myofascial trigger points in the supraspinatus, infraspinatus, teres minor, and upper trapezius muscles on the affected side with frequencies of 34%, 50%, 12%, and 34%, respectively. They also found a significant relationship between the myofascial trigger point in the supraspinatus muscle and shoulder pain. In our study, we found a significant relationship between myofascial trigger points in the levator scapulae, teres major, teres minor, supraspinatus, infraspinatus, and upper and middle parts of the trapezius muscles, and shoulder pain. In light of the current studies, it may be said that myofascial trigger points are frequently observed in stroke survivors; however, it is not possible to assert that myofascial trigger points directly pose a risk for shoulder pain. The activation of trigger points, particularly in muscles such as the supraspinatus, infraspinatus, teres minor, and trapezius, can increase the spread and intensity of pain. This highlights the need for rehabilitation strategies to include approaches such as manual therapy, myofascial release techniques, and exercise therapy.

In the literature, there is a prevalent view that various changes occur in the affected shoulder joint after a stroke, and these changes contribute to pain. Bendidayi et al[27] stated that prolonged immobilization and spasticity can lead to adhesive capsulitis. They also mentioned that disruptions in scapulohumeral rhythm make the shoulder more susceptible to soft tissue injuries.[27] In a study that examined the shoulder joint arthrographically in 32 stroke patients, researchers detected adhesive capsulitis in 50% of the patients, subluxation in 44%, and rotator cuff injury in 22%.[28] In our study, we detected a 45% positive result in the apprehension test, a 38% positive result in the Neer impingement test, a 24% positive result in the speed test, and a 20% positive result in the shear test. We found a significant relationship between shoulder pain and positive provocation tests, Neer impingement, and apprehension tests. Based on these findings, we believe that stroke-related changes may contribute to HSP.

The results of provocation tests provide important insights into the underlying mechanisms of HSP. In particular, positive apprehension test results suggest shoulder instability or subluxation, indicating that weakened rotator cuff muscles and capsular laxity following a stroke contribute to these pathologies. This condition increases movement between joint surfaces, which, in turn, triggers pain by causing soft tissue injuries. A positive Neer Impingement test indicates subacromial impingement syndrome, highlighting that disruptions in shoulder kinematics narrow the subacromial space and place stress on soft tissues. These findings emphasize the importance of addressing instability and impingement issues in rehabilitation planning.

In light of these findings, addressing HSP with a multifaceted approach will enhance the effectiveness of rehabilitation programs, thereby contributing to the quality of life of individuals with stroke.

Limitations

Some potential limitations may exist in this study. First, the absence of a control group limits the ability to establish causal

Table 7
Comparison of participants with and without HSP

	With HSP (X ± SD)	Without HSP (X ± SD)	Mean difference	%95 confidence interval [lower, upper]	T value	*p*-value
Stroke duration (months)	42.9 ± 53.1	70.4 ± 63.0	−27.5	[−57.1 to 2.1]	−1.85	0.068
Initiation time of rehabilitation (months)	0.8 ± 1.1	0.2 ± 0.6	0.7	[0.2-1.1]	2.81	**0.007***
Muscle tone of shoulder flexor	1.5 ± 1.7	1.1 ± 1.7	0.4	[−0.5 to 1.3]	0.93	0.354
Muscle tone of subscapularis	0.9 ± 1.0	0.4 ± 0.8	0.5	[−0.1 to 1.0]	2.04	**0.046****
Muscle tone of infraspinatus	0.0 ± 0.3	0.2 ± 0.8	0.2	[−0.5 to 0.1]	−1.17	0.245
Muscle tone of pectoralis major	0.9 ± 1.4	0.0 ± 0.3	0.8	[0.3-1.4]	3.18	**0.002***
Shoulder flexor ROM (°)	116.4 ± 23.9	137.8 ± 31.0	−21.5	[−35.6 to −7.4]	−3.04	**0.004***
Shoulder extensor ROM (°)	23.3 ± 10.3	28.0 ± 12.0	−4.7	[−10.4 to 1.0]	−1.65	0.103
Shoulder abduction ROM (°)	88.4 ± 34.8	118.3 ± 40.7	−30.0	[−49.2 to −10.7]	−3.13	**0.003***
Shoulder adduction ROM (°)	27.6 ± 9.8	32.2 ± 11.5	−4.6	[−10.0 to −0.9]	−1.66	0.099
Shoulder external rotation ROM (°)	52.7 ± 25.4	71.2 ± 21.8	−18.5	[−30.6 to −6.5]	−3.07	**0.003***
Shoulder internal rotation ROM (°)	62.2 ± 24.0	76.6 ± 19.6	−14.4	[−25.5 to −3.2]	−2.57	**0.013****
Fugl-Meyer Score	42.8 ± 17.5	53.0 ± 14.0	−10.2	[−18.3 to −2.2]	−2.51	**0.015****

HSP = hemiplegic shoulder pain; X = mean; SD = standard deviation; T = independent simple t test; ROM = range of motion.
* $p < 0.01$.
** $p < 0.05$.

relationships between the variables studied and HSP. Including a control group would allow for comparisons and provide more robust evidence of the intervention's effects. Secondly, the lack of an objective pain assessment method may have introduced variability in pain reporting. While subjective patient-reported outcomes are valuable for understanding the individual's experience, combining them with objective measures (eg, pressure algometry) could provide a more comprehensive evaluation of pain. Lastly, the inclusion of patients at different stages of recovery may have introduced heterogeneity in the results, as patients in acute, subacute, and chronic phases of stroke recovery may experience distinct biomechanical and physiological changes. A more homogeneous sample or subgroup analysis could help identify stage-specific factors associated with HSP and guide tailored rehabilitation approaches.

Conclusion

HSP is a common and multifactorial condition. Myofascial trigger points and soft tissue injuries are contributing factors to HSP. Delayed initiation of rehabilitation, increased muscle tone (especially in supraspinatus and pectoralis major), and low motor level are identified as risk factors for HSP. Disruption of shoulder biomechanics (joint, stability, muscles imbalance, tone) may lay the groundwork for the formation of pain; therefore, early emphasis on shoulder mobility is crucial. Our study may be enhanced by including patients in the same stage of recovery, forming homogeneous groups based on different stages of recovery, or utilizing technological and objective assessment methods.

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CRediT authorship contribution statement

Dengiz Aziz: Writing – review & editing, Writing – original draft, Validation, Supervision, Methodology, Investigation, Formal analysis, Conceptualization. **Baskan Emre:** Writing – review & editing, Writing – original draft, Methodology, Investigation, Formal analysis, Data curation. **Arslan Cemil:** Validation, Supervision, Resources, Methodology, Investigation, Conceptualization.

Declaration of Competing Interest

The authors declare the following financial interests/personal relationships, which may be considered as potential competing interests: Aziz Dengiz reports were provided by the health science institution. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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JHT Read for Credit

Quiz: # C29

Record your answers on the Return Answer Form found on the tear-out coupon at the back of this issue. There is only one best answer for each question.

#1. The following factors were evaluated
- a. trigger points
- b. pain
- c. muscle tone
- d. all of the above

#2. Shoulder pain was found in _____% of patients
- a. 25
- b. 35
- c. 50
- d. 75

#3. Function was assessed using the
- a. Fugl-Meyer Upper Extremity Assessment
- b. Quick DASH
- c. Minnesota Arm Survey
- d. Purdue Pegboard Test

#4. Muscle tone was evaluated using the
- a. VAS
- b. MAS
- c. MAD
- d. VIN

#5. The authors strongly recommend early intervention
- a. not true
- b. true