Section: Neurology

Original Research Article

HEMIPLEGIC SHOULDER PAIN AND SUBLUXATION AFTER STROKE: CLINICAL AND MRI CORRELATES

Fazal Ghafoor P A[1], Mallikarjuna Reddy[2], Mohamed Rafeequ P[3]

[1]*Professor & HOD, Department of Neurology, MES Medical College, Perinthalmanna, Malappuram, Kerala, India*
[2]*Senior Resident, Department of Neurology, MES Medical College, Perinthalmanna, Malappuram, Kerala, India*
[3]*Associate Professor, Department of Neurology, MES Medical College, Perinthalmanna, Malappuram, Kerala, India*

Received *: 26/09/2026*
Received in revised form *: 20/10/2026*
Accepted *: 03/11/2026*

Corresponding Author:
Dr. Fazal Ghafoor P A,
Professor & HOD, Department of Neurology, MES Medical College, Perinthalmanna, Malappuram, Kerala, India
Email: drfazalmes@yahoo.in

DOI: 10.70034/ijmedph.2026.4.275

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (4); 1811-1817

ABSTRACT

Background: Hemiplegic shoulder pain (HSP) and glenohumeral subluxation impede post-stroke rehabilitation, but magnetic resonance imaging (MRI)-based data from India are scarce. The objective is to determine the prevalence and the clinical and MRI correlates of HSP and subluxation in post-stroke hemiplegia.

Materials and Methods: This cross-sectional study (December 2022–December 2023) enrolled 70 consecutive patients with first-ever stroke within three months of onset, affected upper-limb power of Medical Research Council grade ≤3 and Mini-Mental State Examination score ≥24. Subluxation was assessed on upright bilateral shoulder radiographs and soft-tissue lesions on 1.5-T MRI; pain was rated on a visual analogue scale (VAS). Chi-square, Fisher's exact and t-tests and unadjusted odds ratios (OR) with 95% confidence intervals (CI) were used.

Results: Mean age was 61.7 years; 58.6% were men. HSP was present in 54 patients (77.1%) and subluxation in 32 (45.7%). HSP was associated with joint effusion (OR 10.29; 95% CI 2.58–41.10), supraspinatus tendinitis (OR 6.94), subscapularis tendinitis (OR 6.81) and infraspinatus tendinitis (OR 5.60) (all $P \leq 0.020$), but not significantly with subluxation ($P = 0.058$). Subluxation was associated with severe pain (VAS 7–10; 78.1% vs 23.7%; OR 11.51; $P < 0.001$), joint effusion ($P = 0.002$), infraspinatus tendinitis ($P = 0.041$) and higher glycated haemoglobin ($P = 0.045$). Vascular risk factors were associated with neither outcome.

Conclusion: HSP affected over three-quarters of patients with moderate-to-severe post-stroke arm weakness and was linked to rotator cuff tendinitis and joint effusion; subluxation was associated with pain severity. Systematic shoulder assessment, including targeted imaging, may guide rehabilitation.

Keywords: Stroke; Hemiplegia; Shoulder Pain; Shoulder Dislocation; Magnetic Resonance Imaging.

INTRODUCTION

Stroke remains the second-leading cause of death and the third-leading cause of combined death and disability worldwide.[1] In India, it is now the fourth-leading cause of death and the fifth-leading cause of disability, with a crude annual incidence of 108–172 per 100 000 population in prospective studies.[2] Weakness of the upper limb is its most frequent acute impairment, affecting more than three-quarters of patients with a first-ever stroke,[3] and only a minority of patients who present with a flaccid arm regain some dexterity by six months.[4] Shoulder complications add substantially to this disability. Hemiplegic shoulder pain (HSP) has been linked to poor functional recovery, reduced quality of life, depression, disturbed sleep and prolonged hospitalisation, and it hinders rehabilitation of upper-limb motor function.[5,6]

HSP refers to pain in the shoulder on the hemiplegic side after stroke. Its reported frequency varies widely with geographical region, care setting and timing of assessment. A meta-analysis of 21 studies estimated a pooled incidence of 29% and a pooled prevalence of 33%, with significant differences between regions,[7] and a systematic review of 18 studies

reported an incidence of 10–22% and a prevalence of 22–47%, identifying age, female sex, increased muscle tone, sensory impairment, left-sided hemiparesis, haemorrhagic stroke, hemispatial neglect and poor National Institutes of Health Stroke Scale (NIHSS) scores as predictors.[8] Frequencies are considerably higher in rehabilitation settings, where approximately half of patients are affected.[5]

Glenohumeral subluxation, the second major shoulder complication of hemiplegia, is generally attributed to loss of muscular support of the joint during the flaccid stage, which leaves the humeral head unable to resist the downward pull of the unsupported arm; its pathomechanics, however, are not fully understood.[9] Subluxation has been reported in up to 81% of patients after stroke, is frequently associated with poor upper-limb function.[10] and is regarded as an important risk factor for shoulder pain,[9] although its relationship with HSP has not been consistently demonstrated.[11]

A spectrum of structural lesions has been described in the painful hemiplegic shoulder, including rotator cuff tendinopathy and tears, glenohumeral joint effusion, subacromial–subdeltoid bursitis, long-head biceps tendon pathology, adhesive capsulitis and labral injury.[11–14] Ultrasonography is a practical bedside tool for detecting many of these abnormalities,[13] whereas MRI is regarded as the reference standard for evaluating the rotator cuff and the glenohumeral joint.[14] Most imaging series, however, have examined only painful shoulders,[11,12,14] and few studies have compared painful with pain-free hemiplegic shoulders or related imaging findings to radiographically measured subluxation.

Prospective Indian data that combine clinical grading of shoulder pain, upright radiographic assessment of subluxation and MRI characterisation of the hemiplegic shoulder remain scarce. We therefore aimed to determine the prevalence of HSP and glenohumeral subluxation among patients with moderate-to-severe upper-limb weakness within three months of a first stroke, to identify the clinical and MRI correlates of each complication and to examine the relationship between subluxation, HSP and pain severity. Such data could help clinicians to identify, early in rehabilitation, the patients who need targeted shoulder protection and treatment.

MATERIALS AND METHODS

Study Design and Setting: This hospital-based, cross-sectional analytical study with prospective enrolment was conducted in the Department of Neurology, MES Medical College Hospital, Perinthalmanna, Kerala, India, between December 2022 and December 2023. The study is reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement.[15]

Ethical Considerations: The study protocol was approved by the Institutional Ethics Committee of MES Medical College Hospital (approval number IEC/MES/74/2022). The study was conducted in accordance with the ethical principles of the Declaration of Helsinki, and written informed consent was obtained from every participant before enrolment. Participant confidentiality was maintained throughout the study.

Participants: Consecutive patients attending the Department of Neurology for follow-up and rehabilitation after stroke were screened for eligibility. Patients were included if they were older than 18 years; had a first-ever ischaemic or haemorrhagic stroke confirmed by computed tomography (CT) or MRI of the brain; were within three months of symptom onset; had muscle power of Medical Research Council (MRC) grade 3 or less in the affected upper limb; and had a Mini-Mental State Examination (MMSE) score of 24 or more, which ensured that they could reliably report pain and cooperate with examination.

Patients were excluded if they had recurrent stroke; pre-existing pathology, trauma or surgery of the affected shoulder, including known rotator cuff disease; shoulder pain before the stroke; expressive or receptive aphasia that precluded reliable pain rating; hemispatial neglect; trunk control inadequate for upright radiography; contraindications to MRI, such as metallic implants, cardiac pacemakers or severe claustrophobia; or if they declined to give consent.

Sample Size: The minimum sample size was calculated with the formula for estimating a single proportion, $n = Z21{-}\alpha/2 \times p \times q / d2$. Taking the prevalence of HSP as 63.8% ($p = 0.638$; $q = 0.362$), as reported by Wanklyn et al.,[16] a 95% confidence level ($Z1{-}\alpha/2 = 1.96$) and a relative precision of 20% ($d = 0.20 \times 0.638 = 0.1276$), the minimum required sample size was 55. Seventy patients were enrolled to allow for incomplete imaging and to improve the precision of the estimates.

Clinical Assessment: A structured proforma was used to record age, sex, vascular risk factors (diabetes mellitus, systemic hypertension, dyslipidaemia, current or past smoking and alcohol use), glycated haemoglobin (HbA1c), side of hemiparesis and MRC grade of the affected upper limb. The shoulder was examined at rest and during passive range of motion, and pain intensity was rated on a 10-cm visual analogue scale (VAS; range 0–10). HSP was considered present when the VAS score was 1 or more. Pain severity was categorised as none (VAS 0), mild-to-moderate (VAS 1–6) or severe (VAS 7–10).

Radiographic Assessment of Subluxation: Anteroposterior radiographs of both shoulders were obtained with the patient seated upright and both arms hanging unsupported. Radiographic measurement is considered the best method of quantifying glenohumeral subluxation.[9] The vertical acromiohumeral distance, measured from the inferior border of the acromion to the superior aspect of the humeral head, was recorded on both sides, and inferior subluxation was diagnosed by comparing the

distance on the hemiplegic side with that on the unaffected side.

MRI Protocol and Interpretation: MRI of the affected shoulder was performed on a 1.5-T superconducting scanner (Magnetom Avanto; Siemens, Erlangen, Germany) with the patient supine and the arm in a neutral position alongside the body. T1-weighted, T2-weighted and proton-density fat-suppressed sequences were acquired in the axial, oblique coronal and oblique sagittal planes. All radiographs and MR images were interpreted on a dedicated workstation by an experienced musculoskeletal radiologist who was blinded to HSP status and VAS scores. The radiologist recorded supraspinatus tendinitis (increased intratendinous signal on T2-weighted or proton-density fat-suppressed images with tendon thickening), infraspinatus tendinitis, subscapularis tendinitis and glenohumeral joint effusion (abnormal fluid distension of the glenohumeral joint recesses).

Statistical Analysis: Data were entered in Microsoft Excel and analysed with IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables are presented as mean ± standard deviation (SD) and were compared between groups with the independent-samples t-test. Categorical variables are presented as frequencies and percentages and were compared with Pearson's chi-square ($\chi 2$) test, or with Fisher's exact test when any expected cell count was less than 5. Unadjusted odds ratios (OR) with 95% confidence intervals (CI), calculated by the logit (Woolf) method, quantified the strength of each association. The distribution of pain-severity categories was compared between patients with and without subluxation with the $\chi 2$ test for a 3 × 2 table. A two-tailed P value of less than 0.05 was considered statistically significant. Because the sample was not powered for multivariable modelling, no adjusted analyses were performed, and P values were not corrected for multiple comparisons; the analyses of association are therefore exploratory.

RESULTS

Participant Characteristics: All 70 enrolled patients completed both radiographic and MRI evaluation and were included in the analysis. Their mean age was 61.7 ± 9.3 years, 41 (58.6%) were men and 41 (58.6%) had left-sided hemiparesis. Diabetes mellitus (58.6%) and hypertension (50.0%) were the most common vascular risk factors [Table 1].

HSP was present in 54 patients (77.1%); pain was mild-to-moderate in 20 (28.6%) and severe in 34 (48.6%). Radiographic subluxation was identified in 32 patients (45.7%), and 28 (40.0%) had both HSP and subluxation. On MRI, supraspinatus tendinitis was the most frequent abnormality (65.7%), followed by glenohumeral joint effusion (58.6%), subscapularis tendinitis (51.4%) and infraspinatus tendinitis (37.1%).

Factors Associated with Hemiplegic Shoulder Pain: Age, sex, vascular risk factors, HbA1c and side of hemiparesis did not differ significantly between patients with and without HSP [Table 2]. All four MRI abnormalities were significantly more frequent in painful shoulders [Figure 1]. Joint effusion showed the strongest association (70.4% vs 18.8%; OR 10.29, 95% CI 2.58–41.10; P < 0.001), followed by supraspinatus tendinitis (75.9% vs 31.3%; OR 6.94, 95% CI 2.03–23.68; P < 0.001), subscapularis tendinitis (OR 6.81, 95% CI 1.73–26.78; P = 0.003) and infraspinatus tendinitis (OR 5.60, 95% CI 1.16–27.07; P = 0.020). Subluxation was about twice as frequent in patients with HSP as in those without (51.9% vs 25.0%), but the difference was not statistically significant (OR 3.23, 95% CI 0.92–11.29; P = 0.058).

Factors Associated with Glenohumeral Subluxation: Age, sex, vascular risk factors and side of hemiparesis were not associated with subluxation [Table 3]. Diabetes mellitus was numerically more frequent in patients with subluxation (65.6% vs 52.6%; P = 0.272), and their mean HbA1c was significantly higher (8.56 ± 2.65% vs 7.37 ± 2.22%; P = 0.045). Joint effusion (78.1% vs 42.1%; OR 4.91, 95% CI 1.71–14.13; P = 0.002) and infraspinatus tendinitis (50.0% vs 26.3%; OR 2.80, 95% CI 1.03–7.62; P = 0.041) were more frequent in subluxed shoulders, whereas supraspinatus and subscapularis tendinitis were not significantly associated with subluxation [Figure 1].

The distribution of pain severity differed markedly according to subluxation status ($\chi 2$ = 20.97, degrees of freedom = 2; P < 0.001). Severe pain was reported by 25 of 32 patients (78.1%) with subluxation but by only 9 of 38 (23.7%) without it (OR 11.51, 95% CI 3.74–35.39; P < 0.001), whereas mild-to-moderate pain or no pain predominated among patients without subluxation [Figure 2].

Table 1: Baseline demographic, clinical and imaging characteristics of the study participants (N = 70)

Variable	Category	n (%) or mean ± SD
Age (years)	Mean ± SD	61.7 ± 9.3
Sex	Male	41 (58.6)
	Female	29 (41.4)
Side of hemiparesis	Left	41 (58.6)
	Right	29 (41.4)
Vascular risk factors	Diabetes mellitus	41 (58.6)
	Hypertension	35 (50.0)
	Smoking (current or past)	22 (31.4)
	Dyslipidaemia	15 (21.4)
	Alcohol use	6 (8.6)

HbA1c (%)	Mean ± SD	7.9 ± 2.5
Hemiplegic shoulder pain	Present	54 (77.1)
	Absent	16 (22.9)
Pain severity (VAS)	None (0)	16 (22.9)
	Mild-to-moderate (1–6)	20 (28.6)
	Severe (7–10)	34 (48.6)
Glenohumeral subluxation	Present	32 (45.7)
	Absent	38 (54.3)
HSP with subluxation	Both present	28 (40.0)
MRI findings (affected shoulder)	Supraspinatus tendinitis	46 (65.7)
	Glenohumeral joint effusion	41 (58.6)
	Subscapularis tendinitis	36 (51.4)
	Infraspinatus tendinitis	26 (37.1)

HbA1c, glycated haemoglobin; HSP, hemiplegic shoulder pain; MRI, magnetic resonance imaging; SD, standard deviation; VAS, visual analogue scale.

Table 2: Clinical, radiographic and MRI factors associated with hemiplegic shoulder pain (N = 70)

Variable	Category	HSP present (n = 54)	HSP absent (n = 16)	OR (95% CI)	P value
Age (years)	Mean ± SD	61.41 ± 9.31	62.69 ± 9.58	—	0.633‡
Sex	Female	23 (42.6)	6 (37.5)	1.24 (0.39–3.89)	0.716
	Male	31 (57.4)	10 (62.5)	Reference	
Diabetes mellitus	Yes	33 (61.1)	8 (50.0)	1.57 (0.51–4.83)	0.428
	No	21 (38.9)	8 (50.0)	Reference	
HbA1c (%)	Mean ± SD	8.03 ± 2.50	7.51 ± 2.46	—	0.466‡
Hypertension	Yes	28 (51.9)	7 (43.8)	1.38 (0.45–4.26)	0.569
	No	26 (48.1)	9 (56.3)	Reference	
Dyslipidaemia	Yes	11 (20.4)	4 (25.0)	0.77 (0.21–2.85)	0.734†
	No	43 (79.6)	12 (75.0)	Reference	
Smoking	Yes	16 (29.6)	6 (37.5)	0.70 (0.22–2.26)	0.551
	No	38 (70.4)	10 (62.5)	Reference	
Alcohol use	Yes	5 (9.3)	1 (6.3)	1.53 (0.17–14.14)	1.000†
	No	49 (90.7)	15 (93.8)	Reference	
Side of hemiparesis	Left	34 (63.0)	7 (43.8)	2.19 (0.70–6.78)	0.171
	Right	20 (37.0)	9 (56.3)	Reference	
Glenohumeral subluxation	Present	28 (51.9)	4 (25.0)	3.23 (0.92–11.29)	0.058
	Absent	26 (48.1)	12 (75.0)	Reference	
Supraspinatus tendinitis	Present	41 (75.9)	5 (31.3)	6.94 (2.03–23.68)	<0.001*
	Absent	13 (24.1)	11 (68.8)	Reference	
Infraspinatus tendinitis	Present	24 (44.4)	2 (12.5)	5.60 (1.16–27.07)	0.020*
	Absent	30 (55.6)	14 (87.5)	Reference	
Subscapularis tendinitis	Present	33 (61.1)	3 (18.8)	6.81 (1.73–26.78)	0.003*
	Absent	21 (38.9)	13 (81.3)	Reference	
Joint effusion	Present	38 (70.4)	3 (18.8)	10.29 (2.58–41.10)	<0.001*
	Absent	16 (29.6)	13 (81.3)	Reference	

Values are n (%) unless stated otherwise. Odds ratios compare the first category listed with the reference category. P values are from Pearson's $\chi 2$ test unless marked: †Fisher's exact test (expected cell count <5); ‡independent-samples t-test. *Statistically significant ($P < 0.05$).

CI, confidence interval; HbA1c, glycated haemoglobin; HSP, hemiplegic shoulder pain; MRI, magnetic resonance imaging; OR, odds ratio; SD, standard deviation.

Table 3: Clinical and MRI factors associated with glenohumeral subluxation (N = 70)

Variable	Category	Subluxation present (n = 32)	Subluxation absent (n = 38)	OR (95% CI)	P value
Age (years)	Mean ± SD	62.69 ± 9.49	60.87 ± 9.22	—	0.420‡
Sex	Female	13 (40.6)	16 (42.1)	0.94 (0.36–2.45)	0.900
	Male	19 (59.4)	22 (57.9)	Reference	
Diabetes mellitus	Yes	21 (65.6)	20 (52.6)	1.72 (0.65–4.52)	0.272
	No	11 (34.4)	18 (47.4)	Reference	
HbA1c (%)	Mean ± SD	8.56 ± 2.65	7.37 ± 2.22	—	0.045‡*
Hypertension	Yes	15 (46.9)	20 (52.6)	0.79 (0.31–2.04)	0.631
	No	17 (53.1)	18 (47.4)	Reference	
Dyslipidaemia	Yes	7 (21.9)	8 (21.1)	1.05 (0.33–3.30)	0.933
	No	25 (78.1)	30 (78.9)	Reference	
Smoking	Yes	11 (34.4)	11 (28.9)	1.29 (0.47–3.54)	0.626
	No	21 (65.6)	27 (71.1)	Reference	
Alcohol use	Yes	4 (12.5)	2 (5.3)	2.57 (0.44–15.06)	0.402†
	No	28 (87.5)	36 (94.7)	Reference	
Side of hemiparesis	Left	20 (62.5)	21 (55.3)	1.35 (0.52–3.52)	0.540
	Right	12 (37.5)	17 (44.7)	Reference	
Supraspinatus tendinitis	Present	23 (71.9)	23 (60.5)	1.67 (0.61–4.57)	0.319

	Absent	9 (28.1)	15 (39.5)	Reference	
Infraspinatus tendinitis	Present	16 (50.0)	10 (26.3)	2.80 (1.03–7.62)	0.041*
	Absent	16 (50.0)	28 (73.7)	Reference	
Subscapularis tendinitis	Present	19 (59.4)	17 (44.7)	1.81 (0.70–4.68)	0.222
	Absent	13 (40.6)	21 (55.3)	Reference	
Joint effusion	Present	25 (78.1)	16 (42.1)	4.91 (1.71–14.13)	0.002*
	Absent	7 (21.9)	22 (57.9)	Reference	

Values are n (%) unless stated otherwise. Odds ratios compare the first category listed with the reference category. P values are from Pearson's χ2 test unless marked: †Fisher's exact test (expected cell count <5); ‡independent-samples t-test. *Statistically significant (P < 0.05).

CI, confidence interval; HbA1c, glycated haemoglobin; MRI, magnetic resonance imaging; OR, odds ratio; SD, standard deviation.

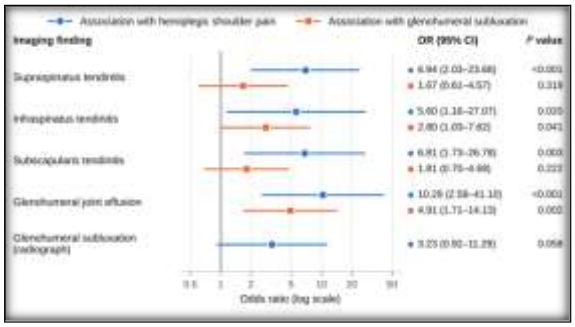


Figure 1: Unadjusted odds ratios (95% confidence intervals) for the association of magnetic resonance imaging (MRI) findings and radiographic subluxation with hemiplegic shoulder pain (circles), and of MRI findings with glenohumeral subluxation (squares). Logarithmic scale; vertical line, odds ratio = 1.

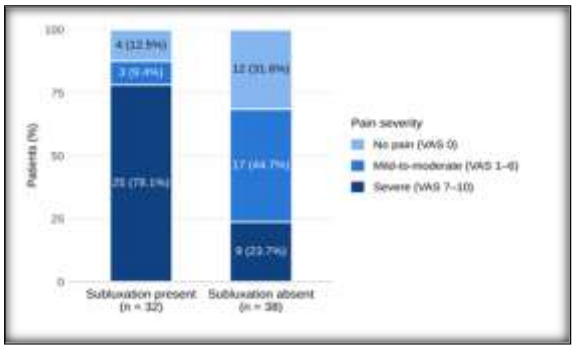


Figure 2: Distribution of shoulder pain severity on the visual analogue scale (VAS) according to glenohumeral subluxation status (N = 70). Values are n (%). χ2 = 20.97, P < 0.001; severe pain: odds ratio 11.51 (95% CI 3.74–35.39).

DISCUSSION

In this cohort of patients with moderate-to-severe upper-limb weakness within three months of a first stroke, more than three-quarters had HSP and nearly half had radiographic glenohumeral subluxation. HSP was strongly associated with MRI evidence of rotator cuff tendinitis and glenohumeral joint effusion, whereas its association with subluxation did not reach statistical significance. Subluxation, in turn, was closely associated with severe pain, joint effusion, infraspinatus tendinitis and higher HbA1c, and conventional vascular risk factors were associated with neither complication. To our knowledge, this is one of the few studies from India to combine upright bilateral radiography and MRI of the hemiplegic shoulder with a direct comparison of painful and pain-free shoulders.

The prevalence of HSP in our study (77.1%) exceeds the pooled prevalence of 33% reported in a global meta-analysis,[7] the range of 22–47% reported in a systematic review,[8] and the 55–59% observed over four months in a Chinese rehabilitation-unit cohort.[6] Several features of our design probably explain this difference. First, we enrolled only patients with MRC grade 3 or weaker power in the affected arm, and impaired arm motor function and greater stroke severity are among the most consistent predictors of HSP.[8,17,18] Second, pain was assessed during passive range of motion as well as at rest, and passive movement testing elicits shoulder pain more often than self-report does.[19] Third, HSP is more frequent in rehabilitation settings, where about half of patients are affected.[5] Our estimate therefore applies to patients with moderate-to-severe arm weakness undergoing rehabilitation rather than to unselected stroke survivors. The burden was nonetheless substantial: pain was severe in 34 of the 54 patients with HSP, and in a population-based cohort most patients with post-stroke shoulder pain rated it as moderate-to-severe, with frequent restriction of dressing and walking.[17]

The 45.7% prevalence of subluxation lies within the wide range reported after stroke.[9,10] Subluxation was twice as frequent among patients with HSP as among those without, but the difference was not statistically significant and the confidence interval was wide. This finding is consistent with the uncertain relationship between subluxation and HSP described in earlier work: Xie et al. found rotator cuff injury and bursal effusion to be common in painful hemiplegic shoulders irrespective of subluxation,[11] whereas Li et al. identified subluxation, together with increased biceps or triceps tone and limited passive range of motion, as factors associated with HSP.[6] In our cohort, subluxation was related more closely to the intensity of pain than to its presence, with more than 11-fold higher odds of severe pain among patients with subluxation. Inferior displacement of the humeral head is thought to stretch the joint capsule and rotator cuff and to expose these structures to repeated strain during transfers and passive mobilisation,[5,9] which could amplify pain arising from coexisting soft-tissue lesions. Because functional electrical stimulation and strapping can prevent subluxation in the acute stage,[9,10] and

several interventions have shown benefit for established HSP despite limited evidence,[20] early identification of patients with subluxation may help to target preventive measures.

Rotator cuff tendinitis was common, and each of the three tendons examined was significantly associated with HSP. Supraspinatus tendinitis was present in three-quarters of painful shoulders. This agrees with ultrasound studies in which supraspinatus tendon thickening was the most common lesion in patients with HSP.[6] and supraspinatus abnormalities were significantly more frequent on the hemiplegic than on the unaffected side.[13] In contrast, Shah et al. found tendinopathy of the rotator cuff, biceps or deltoid in 53% of chronic stroke survivors with shoulder pain but no relationship between tendinopathy and pain severity.[12] Unlike that study, which included only painful shoulders, our comparison of painful and pain-free hemiplegic shoulders within three months of stroke suggests that rotator cuff tendinitis is closely linked to the presence of pain in the early post-stroke period. Infraspinatus tendinitis was associated with both HSP and subluxation, whereas subscapularis tendinitis was associated only with HSP. The infraspinatus contributes to dynamic stabilisation of the humeral head and may be strained when the head displaces inferiorly, whereas the subscapularis, the principal internal rotator, is exposed to spasticity and shortening of the internal rotators; these explanations remain hypotheses that require confirmation in studies measuring spasticity.

Glenohumeral joint effusion showed the strongest association with HSP and was also associated with subluxation. Effusion was the most frequent imaging abnormality (47.3%) in painful hemiplegic shoulders in an Egyptian series evaluated with both ultrasound and MRI,[14] and bursal effusion was common in painful shoulders with and without subluxation in the study by Xie et al,[11] Distension of the joint capsule may directly activate capsular nociceptors, and effusion may also reflect synovial inflammation secondary to rotator cuff injury and abnormal glenohumeral mechanics. Because imaging and pain were assessed at the same time, however, we cannot determine whether effusion precedes pain or results from it; serial imaging studies could clarify this temporal relationship.

Conventional vascular risk factors, including a diagnosis of diabetes mellitus, were associated with neither HSP nor subluxation. Findings on diabetes have been inconsistent: diabetes was an independent risk factor for HSP in a large retrospective series.[18] and was associated with HSP in the Egyptian study,[14] but not in our cohort. We did, however, observe a higher mean HbA1c among patients with subluxation. Chronic hyperglycaemia promotes the accumulation of advanced glycation end-products, which impair the biological and mechanical properties of tendons and ligaments,[21,22] and poorly controlled diabetes might therefore increase the vulnerability of periarticular tissues to the gravitational load of a weak arm. Given the borderline significance of this finding and the number of comparisons performed, it should be regarded as hypothesis-generating.

These findings have practical implications for stroke rehabilitation services. Because more than three-quarters of patients with marked arm weakness had shoulder pain and most painful shoulders showed at least one of the soft-tissue lesions examined, the hemiplegic shoulder should be examined routinely during rehabilitation, including assessment of pain on passive movement. Inferior subluxation is best quantified on radiographs,[9] which should be obtained with the patient upright because the gravitational traction that produces subluxation is absent in the supine position used for MRI. Where MRI is not readily available, ultrasonography, which has shown good to very good agreement with MRI for joint effusion, rotator cuff disorders and subacromial–subdeltoid bursitis,[14] offers an accessible alternative, and management can then be tailored to the individual presentation, as recommended for this heterogeneous condition.[20] Careful handling of the weak arm also deserves emphasis, because carers have been observed lifting patients by pulling on the hemiplegic arm despite having received advice on correct technique.[16]

This study has limitations. First, it was conducted at a single centre and included only patients with moderate-to-severe arm weakness who could reliably report pain; patients with aphasia, hemispatial neglect or cognitive impairment, groups in whom HSP may be more frequent,[8] were excluded, which limits generalisability. Second, the cross-sectional design precludes causal inference, and because the onset and severity of HSP may peak around four months after stroke,[19] a single assessment within three months may not capture its full course. Third, MRI was performed only on the hemiplegic shoulder, so pre-existing degenerative rotator cuff changes, which become more common with age,[12] cannot be excluded; moreover, images were read by a single radiologist, and inter-observer agreement was not assessed. Fourth, the modest sample produced wide confidence intervals, the associations were unadjusted and multiple comparisons were made without correction. Finally, spasticity, sensory impairment, stroke type and severity, and the interval from stroke onset to assessment were not included in the analysis; lesions such as long-head biceps tendon pathology, subacromial–subdeltoid bursitis and adhesive capsulitis were not analysed; and functional outcomes were not assessed. The strengths of the study include prospective consecutive enrolment, standardised upright bilateral radiography, MRI interpretation blinded to pain status and direct comparison of painful with pain-free hemiplegic shoulders.

CONCLUSION

Among patients with moderate-to-severe arm weakness within three months of a first stroke, HSP

affected more than three-quarters and glenohumeral subluxation nearly half. HSP was strongly associated with MRI-detected rotator cuff tendinitis and glenohumeral joint effusion, whereas subluxation was associated with severe pain, joint effusion, infraspinatus tendinitis and higher HbA1c. Systematic assessment of the hemiplegic shoulder, including upright radiography and targeted imaging when pain is present, may help clinicians to identify treatable soft-tissue lesions and to plan early protective measures during rehabilitation. Longitudinal multicentre studies with adjusted analyses are needed to confirm these associations.

REFERENCES

1. GBD 2019 Stroke Collaborators. Global, regional, and national burden of stroke and its risk factors, 1990-2019: a systematic analysis for the Global Burden of Disease Study 2019. Lancet Neurol. 2021;20(10):795-820. DOI: 10.1016/S1474-4422(21)00252-0
2. Jones SP, Baqai K, Clegg A, et al. Stroke in India: a systematic review of the incidence, prevalence, and case fatality. Int J Stroke. 2022;17(2):132-140. DOI: 10.1177/17474930211027834
3. Lawrence ES, Coshall C, Dundas R, et al. Estimates of the prevalence of acute stroke impairments and disability in a multiethnic population. Stroke. 2001;32(6):1279-1284. DOI: 10.1161/01.str.32.6.1279
4. Kwakkel G, Kollen BJ, van der Grond J, Prevo AJ. Probability of regaining dexterity in the flaccid upper limb: impact of severity of paresis and time since onset in acute stroke. Stroke. 2003;34(9):2181-2186. DOI: 10.1161/01.STR.0000087172.16305.CD
5. Kalichman L, Ratmansky M. Underlying pathology and associated factors of hemiplegic shoulder pain. Am J Phys Med Rehabil. 2011;90(9):768-780. DOI: 10.1097/PHM.0b013e318214e976
6. Li Y, Yang S, Cui L, et al. Prevalence, risk factor and outcome in middle-aged and elderly population affected by hemiplegic shoulder pain: an observational study. Front Neurol. 2023;13:1041263. DOI: 10.3389/fneur.2022.1041263
7. Zhang Q, Chen D, Shen Y, Bian M, Wang P, Li J. Incidence and prevalence of poststroke shoulder pain among different regions of the world: a systematic review and meta-analysis. Front Neurol. 2021;12:724281. DOI: 10.3389/fneur.2021.724281
8. Anwer S, Alghadir A. Incidence, prevalence, and risk factors of hemiplegic shoulder pain: a systematic review. Int J Environ Res Public Health. 2020;17(14):4962. DOI: 10.3390/ijerph17144962
9. Paci M, Nannetti L, Rinaldi LA. Glenohumeral subluxation in hemiplegia: an overview. J Rehabil Res Dev. 2005;42(4):557-568. DOI: 10.1682/jrrd.2004.08.0112
10. Ada L, Foongchomcheay A. Efficacy of electrical stimulation in preventing or reducing subluxation of the shoulder after stroke: a meta-analysis. Aust J Physiother. 2002;48(4):257-267. DOI: 10.1016/s0004-9514(14)60165-3
11. Xie HM, Zhang XT, Xu L, et al. Magnetic resonance imaging findings in painful hemiplegic shoulder patients with or without subluxation: a retrospective cohort study. Front Neurol. 2022;13:1032676. DOI: 10.3389/fneur.2022.1032676
12. Shah RR, Haghpanah S, Elovic EP, et al. MRI findings in the painful poststroke shoulder. Stroke. 2008;39(6):1808-1813. DOI: 10.1161/STROKEAHA.107.502187
13. Lin TY, Shen PC, Chang KV, Wu WT, Özçakar L. Shoulder ultrasound imaging in the post-stroke population: a systematic review and meta-analysis. J Rehabil Med. 2023;55: jrm13432. DOI: 10.2340/jrm. v55.13432
14. El-Sonbaty HAE, Abou Elmaaty AA, Zarad CA, El-Bahnasawy AS. Clinical and radiological assessment of hemiplegic shoulder pain in stroke patients. Egypt J Neurol Psychiatry Neurosurg. 2022; 58:41. DOI: 10.1186/s41983-022-00474-w
15. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP; STROBE Initiative. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. Lancet. 2007;370(9596):1453-1457. DOI: 10.1016/S0140-6736(07)61602-X
16. Wanklyn P, Forster A, Young J. Hemiplegic shoulder pain (HSP): natural history and investigation of associated features. DisabilRehabil. 1996;18(10):497-501. DOI: 10.3109/09638289609166035
17. Lindgren I, Jönsson AC, Norrving B, Lindgren A. Shoulder pain after stroke: a prospective population-based study. Stroke. 2007;38(2):343-348. DOI: 10.1161/01.STR.0000254598.16739.4e
18. Hao N, Zhang M, Li Y, Guo Y. Risk factors for shoulder pain after stroke: a clinical study. Pak J Med Sci. 2022;38(1):145-149. DOI: 10.12669/pjms.38.1.4594
19. Adey-Wakeling Z, Arima H, Crotty M, et al. Incidence and associations of hemiplegic shoulder pain poststroke: prospective population-based study. Arch Phys Med Rehabil. 2015;96(2):241-247.e1. DOI: 10.1016/j.apmr.2014.09.007
20. Dyer S, Mordaunt DA, Adey-Wakeling Z. Interventions for post-stroke shoulder pain: an overview of systematic reviews. Int J Gen Med. 2020; 13:1411-1426. DOI: 10.2147/IJGM.S200929
21. Abate M, Schiavone C, Salini V, Andia I. Occurrence of tendon pathologies in metabolic disorders. Rheumatology (Oxford). 2013;52(4):599-608. DOI: 10.1093/rheumatology/kes395
22. Lui PPY. Tendinopathy in diabetes mellitus patients—epidemiology, pathogenesis, and management. Scand J Med Sci Sports. 2017;27(8):776-787. DOI: 10.1111/sms.12824