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LITERATURE REVIEW

Underlying Pathology and Associated Factors of Hemiplegic Shoulder Pain

ABSTRACT

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The prevalence of hemiplegic shoulder pain is approximately 22%–23% in the general population of stroke survivors and approximately 54%–55% among stroke patients in rehabilitation settings. Hemiplegic shoulder pain causes a reduced quality-of-life, poor functional recovery, depression, disturbed sleep, and prolonged hospitalization. Herein, we attempted to understand, based on a literature review and experts' opinion, the pathologic processes underlying hemiplegic shoulder pain and the major associated factors contributing to its development. The systematization of underlying pathologies was proposed, which might eventually enable a more constructive clinical approach in evaluating and treating patients with hemiplegic shoulder pain.

Key Words: Stroke, Hemiplegic Shoulder Pain, Risk Factors, Etiology

Hemiplegic shoulder pain (HSP) is a general term used to describe shoulder pain after a stroke. Precise etiology of HSP is often difficult to assess, probably because of the diverse possible underlying pathogenesis involved in its development. The typical clinical picture usually includes severe paralysis of the plegic side, glenohumeral subluxation, shoulder pain (occasionally radiating down to the elbow and hand), and localized tenderness over the biceps brachii and supraspinatus tendons.[1–3] Although pain may be present at rest, the patient usually complains of increased pain during passive motion or dependent position of the arm. The most painful and limited shoulder movement is usually lateral (external) rotation, followed by abduction.[1] Most HSP patients experience moderate to severe pain,[4] which may intensify at night, interfering with sleep. HSP in addition to patient suffering is associated with a reduced quality-of-life,[5] poor functional recovery,[6,7] depression,[8] disturbed sleep,[8] and prolonged hospitalization.[6,7,9]

The aim of the present review was to understand the pathologies underlying HSP and the factors contributing to its development.

SEARCH STRATEGY

PubMed, the Cumulative Index to Nursing and Allied Health, Google Scholar, and the ISI web of Science databases were searched from inception until September 2010 using the key words *stroke*, *hemiplegia* or *hemiplegic*, *shoulder pain*, *shoulder subluxation*, *frozen shoulder*, *shoulder-hand syndrome*, *complex regional pain syndrome*, *central pain*, or a combination of these terms. There were no language restrictions. The results were pooled, and duplicates were deleted, leaving 101 references. The titles, abstracts, and reference lists of all articles were reviewed. A total of 114 articles were included. Studies of any design and methodologic quality were included. We are aware that this traditional approach to narrative reviews has much more potential for bias than do systematic reviews or meta-analyses; however, we have endeavored to be inclusive and open minded. We also consulted physical therapists and rehabilitation medicine experts to construct this narrative review on the etiology and associated factors of HSP.

RESULTS AND DISCUSSION

Prevalence

Reported prevalence of HSP varies drastically from 5% to 84% (Table 1),[1,7,10–19] possibly derived from either a disparity in defining shoulder pain or differences in recruitment criteria, such as duration of hemiplegia and severity of paralysis. Another possible reason is the inconsistency in the quality of care of poststroke patients.[18,20,21] In addition, data on HSP prevalence was gathered mostly from patients enrolled in inpatient rehabilitation programs. These patients inevitably are also the most severely physically disabled.[1,22] However, recently, two large population-based studies (in New Zealand[19] and Sweden[4]) found an almost identical prevalence of HSP: 22%–23%. On the other hand, the largest study reviewing the medical records of 1000 patients in a rehabilitation hospital in Turkey[9] found a 55% prevalence of HSP. This prevalence is very close to the mean prevalence of 54% calculated by Turner-Stokes[23] based on 16 reviewed studies.

Herein, we describe the pathologic processes underlying HSP and subsequently discuss the factors that may be associated with these processes and, consequently, with HSP.

Anatomy Overview

The shoulder girdle is comprised of three bones (clavicle, scapula, and humerus), three synovial joints synchronically working to allow a full range of shoulder motion in all three dimensions (sternoclavicular, acromioclavicular, and glenohumeral) and in two articulations (scapulothoracic articulations between the scapula and thoracic rib cage (ribs 2–7) and acromiohumeral articulation). The

TABLE 1 Prevalence of hemiplegic shoulder pain

Author	Year	Origin	Setting	Number of Participants, *N*	Prevalence of HSP, *n* (%)
Najenson et al.[16]	1971	Israel	Rehabilitation hospital	32	27 (84)
Brocklehurst et al.[22]	1978	England		135	37 (27.4)
Tepperman et al.[38]	1984	United States	Rehabilitation hospital	85	17 (21)
Bohannon et al.[46]	1986	United States	Rehabilitation hospital	50	36 (72)
Parker et al.[113]	1986	Switzerland		187	9 (5)
van Ouwenaller et al.[17]	1986	Switzerland		219	157 (72)
Bohannon and Andrews[114]	1990	United States	Rehabilitation hospital	24	17 (21)
Poulin de Courval et al.[31]	1990	Canada	Rehabilitation hospital	94	45 (47.9)
Ring et al.[21]	1993	Israel	Rehabilitation hospital	80	43 (53)
Braus et al.[80]	1994	United States	Rehabilitation hospital	132	36 (27)
Roy et al.[7]	1994	New Zealand	General hospital	76	55 (72)
Jespersen et al.[15]	1995	Denmark	Rehabilitation hospital	173	38 (22)
Wanklyn et al.[18]	1996	England	Cohort study	108	69 (63.8)
Zorowitz et al.[99]	1996	United States	Rehabilitation hospital	20	9 (45)
Gamble et al.[92]	2002	England	Cohort study	123	49 (40)
Ratnasabapathy et al.[19]	2003	New Zealand	Population-based study	1201	284 (23)
Aras et al.[30]	2004	Turkey	Rehabilitation hospital	85	54 (63.5)
Demirci et al.[9]	2007	Turkey	Rehabilitation hospital	1000	548 (54.8)
Lindgren et al.[4]	2007	Sweden	Population-based study	327	71 (22)
Dromerick et al.[42]	2008	United States	Rehabilitation hospital	46	17 (37)
Suethanapornkul et al.[57]	2008	Thailand	Cohort study	327	62 (19)
Barlak et al.[6]	2009	Turkey	Rehabilitation hospital	187	114 (61)

glenohumeral joint, which is the traditional "ball-and-socket," is the most movable joint in the body, important for arm function but at the expense of stability. Static stabilizers consist of the articular anatomy, glenoid labrum, joint capsule, glenohumeral ligaments, and inherent negative pressure in the joint. The shoulder joint's freedom of movement is also attributable to a vast articular capsule and minor ligamentous guiding. The glenohumeral joint is secured mostly by musculature, dynamic stabilizers including the rotator cuff muscles, the long head of the biceps tendon, and other shoulder girdle muscles (e.g., pectoralis major, latissimus dorsi, and serratus anterior).The rotator cuff muscles and tendons further envelope the glenohumeral joint, contracting and relaxing in a coordinated manner to keep the humeral head centered on the glenoid fossa. These are the main dynamic stabilizers of the joint. Imbalance in the strength or coordination of the rotator cuff muscles and the long head of biceps muscle commonly contributes to the development of glenohumeral instability and capsular laxity. Compression of the subacromial soft tissues between the humeral head and the coracoacromial arch may result in subacromial impingement.

Glenohumeral joint movement requires motion of the other joints of the shoulder complex. The coordinated movement of these joints during arm movement is known as the scapulohumeral rhythm. For every180 degrees of shoulder abduction, 120 degrees occur at the glenohumeral joint, and 60 degrees occur at the scapulothoracic articulation. Any disturbances to the scapulohumeral rhythm caused by periscapular muscle spasticity or by motor control tract lesions with unsynchronized muscular activity might damage the soft tissues of the shoulder.

Underlying Pathologies

Morbidities in the shoulder area, potentially associated with HSP, include rotator cuff disorders,[9,10,16,24,25] adhesive capsulitis,[6,9,24–28] shoulder (glenohumeral) subluxation,[9,16,25,29–32] and spasticity of the shoulder muscles.[17,33,34] HSP may also be caused by shoulder-hand syndrome.[6,12,25,30,35–38] Central pain (e.g., thalamic pain) and central sensitization can also play an important role in HSP development,[39] in addition to remote sources of shoulder pain (e.g., neck problems, visceral referred pain).[1] These underlying pathologies may present separately, or several pathologies may coexist. Each pathology may trigger the development of another; for example, rotator cuff impingement can trigger the development of adhesive capsulitis or shoulder-hand syndrome.

To systematize the pathologies underlying HSP and its major contributing factors, we suggest distinguishing three types of pathologic processes: (1) soft-tissue lesions, (2) impaired motor control (muscle tonus changes), and (3) altered peripheral and central nervous system (CNS) activity.

Soft-Tissue Lesions

This group includes rotator cuff tendinopathies and tears, biceps tendinopathy, bursitis (subacromial and subdeltoid), adhesive capsulitis, and myofascial pain in the shoulder region.

Rotator Cuff and Bicipital Tendon Disorders (Tears and Tendinopathies): Several studies conducted on hemiplegic patients revealed a high incidence of impingement syndrome and rotator cuff tears.[2,6,10,16,24,25,40,41] Barlak et al.[6] found that 61% of HSP patients in a rehabilitation center had impingement syndrome. Rotator cuff tears were found in 33% of the patients. Examining only patients with severe paralysis, Najenson et al.[16] found a 40% prevalence of rotator cuff rupture on the affected side compared with a 16% prevalence on the nonaffected side. An association was also found among pain, rotator cuff rupture, and subluxation.[16]

The study by Dromeric et al.[42] of 46 hemiplegic patients treated in an inpatient rehabilitation center reported 17 (37%) patients with shoulder pain, 7 of whom had experienced pain before stroke. Among all hemiplegic patients, 54% had bicipital tendon tenderness, and 48% had supraspinatus tenderness. Patients with HSP had a significantly higher prevalence of tendon tenderness of the biceps and supraspinatus than those without HSP.

The sonographic study by Lee et al.[43] of 71 consecutive stroke patients with HSP found subacromial-subdeltoid bursal effusion (50.7%), supraspinatus tendinosis (9.9%), partial-thickness tear of the supraspinatus tendon (8.5%), full-thickness tear of the supraspinatus tendon (2.8%), and biceps tendon sheath effusion (54.9%). In a magnetic resonance imaging study of 89 chronic stroke survivors with HSP,[41] 35% of the subjects exhibited a tear of at least one rotator cuff, biceps, or deltoid muscle; 55% exhibited tendinopathy of at least one rotator cuff, biceps, or deltoid muscle.

Najenson et al.[16] suggested that the cause of rotator cuff rupture in hemiplegia may by impingement during passive abduction greater than 90 degrees[2] or forced abduction without lateral rotation, especially with overhead pulleys.[16] Other

authors suggested that rotator cuff rupture may be facilitated by previous degenerative changes, subluxation,[16] trauma during falls,[40] or pulling of the hemiplegic arm by the nursing staff.[18,21,44,45]

Adhesive Capsulitis (Frozen Shoulder): Adhesive capsulitis (frozen shoulder) was found in as many as 43%–77% of stroke survivors.[6,24–27,46,47] Hakuno et al.[24] performed arthrography on affected and unaffected shoulders in 77 randomly selected hemiplegic patients and found that adhesive changes were noted in both paralyzed and nonparalyzed shoulders; 55% of the paralyzed shoulders with adhesions had multiple adhesion sites, compared with 3% of nonparalyzed shoulders.[24] Rizk et al.,[27] using arthrography, found adhesive capsulitis in 77% of painful and stiff hemiplegic shoulders in patients who had been having HSP for more than 3 mos. Lo et al.[25] demonstrated that many patients with adhesive capsulitis have an irregular capsular margin and a longer duration of shoulder pain. In patients with adhesive capsulitis, shoulder range of motion (ROM) was most restricted in external rotation and abduction; however, adding an irregular capsular margin caused limitation in shoulder flexion.

In hemiplegic patients, development of adhesive capsulitis has been related to paralysis,[24,47] unconsciousness,[47] impingement pain,[2] and subluxation.[25] Causal relationships between HSP and adhesive capsulitis can be bidirectional. An HSP may trigger the development of adhesive capsulitis because of immobilization, disuse atrophy, contracture, or varying degrees of disability.[48] Alternatively, adhesive capsulitis especially during its first stage is very painful.[10]

High prevalence of adhesive capsulitis in a hemiplegic shoulder supported our conclusions that maintaining shoulder ROM must be one of the priorities in stroke rehabilitation, potentially preventing ROM limitations and subsequent adhesions.

Myofascial Pain: Latent trigger points in the shoulder girdle muscles were found in 54% of the women and 45% of the men who were completely asymptomatic.[49] Myofascial trigger points were also associated with shoulder pain in a nonstroke population.[50] Muscle imbalance, postural disturbance, immobilization, and emotional distress—common findings in poststroke patients—were found to be predisposing factors for the appearance of myofascial trigger points.[51,52] However, no studies that evaluated myofascial pain as a source of HSP were found. One study using dry needling of myofascial trigger points in the shoulder area to treat HSP[53] found that those who received the dry needling in addition to standard rehabilitation therapy reported a significant reduction in frequency and intensity of pain and better quality of sleep than did patients who received only standard rehabilitation therapy. We believe that myofascial pain can potentially be one of the contributing causes of HSP, and further studies are needed to assess this assumption.

Impaired Motor Control (Muscle Tonus Changes)

CNS lesions lead to motor control disturbances reflected in tonus changes that can be further divided into spasticity (an augmented hypertonicity) and flaccidity. Spasticity has been defined as an increase in muscle tone caused by hyperexcitability of the stretch reflex and is characterized by a velocity-dependent increase in tonic stretch reflexes.[54] Spasticity is the component of the upper motor neuron syndrome.[55] There are differing opinions as to the association between spasticity and HSP. Several cross-sectional studies found no relationship,[6,10,21,30,46,56,57] others found a positive association.[17,31,58] van Ouwenaller et al.[17] in a study of 219 patients with hemiplegia, 1 yr after stroke, found that 85% of patients with spasticity experienced pain, compared with 18% of those with flaccid hemiplegia.

Flaccidity may lead to shoulder subluxation, which in turn, can cause peripheral nerve entrapment and soft-tissue lesions.

Spasticity of the Shoulder Girdle Muscles: Numerous studies have proposed that the spasticity of the shoulder girdle muscles, especially the subscapularis, might be one of the causes of HSP.[34,59,60] Two patients with HSP showed pain reduction and immediate improvement in external rotation, abduction, and flexion after phenol motor point blocks were applied to the subscapularis muscle.[61] Braun et al.[60] suggested that shoulder girdle muscle spasticity, especially in the subscapularis, which pulls the arm into medial rotation, may cause pain by traction on the periosteum at the muscle insertion. They reported pain relief when the muscular contracture was surgically released, with the capsular contracture left intact.[60] Injections of botulinum toxin A into spastic shoulder muscles was also found to be effective in decreasing pain and increasing ROM in HSP patients.[33,34,62–64]

A recent Cochrane systematic review[65] of six randomized controlled trials that included 164 poststroke patients with shoulder pain caused by spastic hemiplegia or arthritis also showed that an injection of botulinum toxin A seemed to reduce pain severity and improve shoulder function and

ROM when compared with placebos in patients with shoulder pain. Nevertheless, it is unclear whether the therapeutic effect was derived from the neurolytic or the analgesic effect of the botulinum toxin injection. Based on the aforementioned studies, we believe that spasticity of the subscapularis is a very promising target for HSP treatment. Additional studies are needed to evaluate the different treatment options. Spasticity of the muscle responsible for scapular movement may lead to development of soft-tissue lesions. O'Sullivan et al.[66] proposed that abnormal muscle tone can result in abnormal scapulohumeral rhythm and, in turn, lead to the impingement of the rotator cuff or other structures in the subacromial space.

Loss of Motor Function: Evidence exists as to the positive association between HSP and loss of motor functions. Patients with minor paresis rarely complain of shoulder pain, whereas patients with severe paralysis frequently develop HSP during rehabilitation.[2,4,7,10,19,30] Najenson et al.[16] reported that 84% of hemiplegic patients with severe paralysis had moderate or severe shoulder pain. In a prospective study, Fugl-Meyer et al.[67] noted that HSP developed in patients who initially had poor motor function. In a recent sonographic study of the hemiplegic shoulder,[68] 34 acute stroke patients were divided into two groups according to their motor impairment; 31% of the individuals with higher motor functioning (Brunnstrom stages 4–6) had soft-tissue injuries upon admission and 2 wks after rehabilitation, whereas 33% of patients with lower motor functioning (Brunnstrom stages 1–3) had soft-tissue injuries upon admission; this number increased to 71% 2 wks after rehabilitation. The authors concluded that acute stroke patients with poor upper limb motor functions are more prone to soft-tissue injury of the shoulder during rehabilitation.

Loss of motor function does not cause HSP. However, severe motor impairment most probably causes an alteration of scapulohumeral rhythm or prolonged immobilization of the shoulder and upper limb. In addition, patients with severe motor impairment need more intensive and prolonged nursing care. All these factors may facilitate the soft-tissue injury of the shoulder structures and, therefore, HSP.

Altered Peripheral and Central Nervous Activity

Altered peripheral and central nervous activity includes peripheral nerve entrapment, shoulder-hand syndrome, central sensitization, and central poststroke pain (CPSP).

Peripheral Nerve Entrapment: Several different locations and mechanisms of possible nerve entrapment have been proposed as sources of HSP. Some authors have suggested that damage to the upper trunk of the brachial plexus is responsible for HSP.[69,70] The study by Chino[69] based on needle electromyography reported that 75% of the supraspinatus and deltoid muscles on the hemiplegic side had neuropathic responses. Ring et al.[71,72] suggested that downward subluxation might produce traction and subsequent demyelinization, even axonopathy of the axillary nerve. However, no association was found between brachial plexus injury or proximal mononeuropathy and HSP in the prospective study of 50 patients by Kingery et al.[73] Other studies found no evidence of brachial plexus injuries in hemiplegic patients.[27,74–76] Lee and Khunadorn[77] performed EMGs of the suprascapular nerve in 30 men with HSP to evaluate the association between possible entrapment of the suprascapular nerve and HSP. Only three patients showed increased latency on the affected side. A suprascapular nerve block did not completely relieve the shoulder pain in these patients; therefore, the authors concluded that a suprascapular nerve lesion was not responsible for the painful contracted shoulder of the hemiplegic patient, although such a lesion may incidentally exist. Accidental injuring of the brachial plexus in hemiplegic patients is probably caused by a lack of support of the paralyzed flaccid shoulder[69–71,78] or pulling of the flaccid arm by personnel moving the patient,[78] thus increasing rehabilitation time because of the slow rate of nerve regeneration. Functional recovery of shoulder movement may subsequently be delayed by 8 to 12 mos.[70] Based on the conflicting evidence presented here, it is hard to draw a definite conclusion regarding the role of peripheral nerves in the development of HSP. Nevertheless, when there is peripheral nerve entrapment, it could probably contribute to the development of the central sensitization discussed later in the text.

Shoulder-Hand Syndrome (Complex Regional Pain Syndrome Type I, Reflex Sympathetic Dystrophy): The incidence of shoulder-hand syndrome among hemiplegic patients is ambiguous. No cases were found in a follow-up study of 24 patients, 1 yr after stroke.[71] In a retrospective study of 540 hemiplegic patients, Davis et al.[79] reported a 12.5% incidence of shoulder-hand syndrome. Braus et al.,[80] in a prospective study of 132 hemiplegic patients, found that 27% developed shoulder-hand syndrome. van Ouwenaller et al[17] found a 28% incidence of shoulder-hand syndrome in 215 hemiplegic patients.

Finch and Harvey,[81] in a prospective study of 20 stroke patients, reported that 50% of the patients developed signs of shoulder-hand syndrome. Two other studies reported a prevalence of 32%[17] or 61.4%.[6] The varying incidence of shoulder-hand syndrome in hemiplegia is most likely caused by the inconsistent diagnostic criteria.

A positive association between the incidence of shoulder-hand syndrome and spasticity,[17,80,82] confusion, and greater sensory loss[81,82] has been observed in several studies. The association between shoulder subluxation and shoulder-hand syndrome is not completely understood. A number of studies found that patients with shoulder-hand syndrome had a higher incidence of subluxation than did other hemiplegic patients.[80,81,83] Other studies did not find an association.[6,82] In addition, motor deficit was found to be a significant contributing factor of shoulder-hand syndrome.[6,84] Finch et al.[81] reported that half of the patients who developed shoulder-hand syndrome had a history of cardiac abnormalities.

Damage of the soft tissues surrounding the hemiplegic shoulder may play a significant role as a source of shoulder-hand syndrome.[85] Peripheral nerve damage is also believed to be a fundamental pathophysiologic factor in the development of shoulder-hand syndrome. It is hypothesized that a localized neurogenic inflammation is the basis for peripheral sensitization.[86] On the other hand, recent studies have demonstrated brain tissue abnormalities in patients who developed shoulder-hand syndrome. Geha et al.[87] reported gray matter atrophy in the right insula, ventromedial prefrontal cortex, and nucleus accumbence related to pain intensity and duration in shoulder-hand syndrome patients. Fukumoto et al.[88] found an alteration in thalamic perfusion, whereas other authors confirmed abnormalities and reorganization of the somatosensory cortex and motor networks[89] by assessing functional imaging studies of shoulder-hand syndrome patients. Whether primary lesions in the previously mentioned areas after stroke or secondary neuroplastic changes in the pathogenesis of an ongoing poststroke promote development of the shoulder-hand syndrome is yet to be confirmed.

A definite conclusion cannot be drawn as to the association or causal relationship between shoulder-hand syndrome and HSP, most probably because of diagnostic criteria inconsistency. Nevertheless, the coexistence of these phenomena in poststroke patients need to be further studied.

Role of the CNS in HSP: Except for the possible role CNS plays in the shoulder-hand syndrome, as earlier described, there are CNS-related phenomena that can potentially play an important role in HSP, such as neglect and sensory impairment, CPSP, and central sensitization.

Neglect and Sensory Impairment: Several authors[9,10,43,90] have found a significant association between neglect and HSP. Joynt[10] suggested that neglect might lead to increased trauma or disturbed perception of the quality of pain, thereby producing pain sensation without the usual pathology. Other studies did not find an association between neglect and HSP.[30]

Sensory impairment was also found to be associated with HSP.[4,31,90–92] Suethanapornkul et al.,[57] in a study of 327 stroke patients, found a significant association between loss of proprioceptive sense and HSP. Additional studies are needed to assess the mechanism of association and the causal relationship between sensory impairment and HSP.

CPSP: CPSP, formally called thalamic pain syndrome, is a neuropathic pain syndrome that can occur after a stroke at any level of the somatosensory pathways of the brain, including the medulla, thalamus, and cerebral cortex.[93] An injury of the spinothalamocortical pathway seems to be a prerequisite for developing central pain.[94] Neural plastic changes and reorganization of the thalamus can occur with pain after stroke.[95] CPSP is characterized by pain in the sensory territory that has been injured. It is hard to distinguish CPSP from other types of pain occurring after stroke. Stroke patients can experience various combinations of one or several types of pain that might overlap with CPSP, including musculoskeletal, neuropathic and spastic pain after stroke.[93]

Central Sensitization: Central sensitization can also play a role in HSP, especially in chronic cases. Unfortunately, no studies have been found that directly addresses this subject. Therefore, we will describe the phenomenon of central sensitization and will speculate about its possible association with HSP.

Central sensitization is defined as an increased responsiveness of nociceptive neurons in the CNS to normal or subthreshold afferent input.[93] Fischer[96] suggested that the development of central sensitization in the corresponding spinal segments (Spinal Segmental Sensitization [SSS]) explains the development of chronic pain originating from musculoskeletal or neuropathic lesions. According to Fischer,[96] the SSS can stem from the sensitization of the spinal nociceptive neurons, regardless of the original provoking events. One such event may be the injury to peripheral structures around the shoulder.

After tissue injury, alteration in the production and secretion of neurotransmitters, neuropeptides, cytokines, and growth factors may increase nociceptor sensitivity, called peripheral sensitization.[97,98] Changes in the expression or activity of the receptors for these secreted ligands and changes in the physical interactions between cells (such as axonal sprouting) in relevant segments of the spinal cord (spinal central sensitization) lead to ongoing noxious signals to the brain, subsequently contributing to the development of supraspinal central sensitization.[97] The sensitization (both peripheral and central) contributes to HSP chronicity. SSS may also produce muscle spasms in the corresponding myotome through the segmental anterior horn motor cells, generating taut muscular bands, muscle spasms, and myofascial trigger points.

We hypothesize that spinal and supraspinal central sensitization plays a role in the development of chronic HSP after soft-tissue and/or peripheral nerve injury in the shoulder region. This mechanism could coexist with CPSP when sensory abnormalities are presented in the hemiplegic arm.

Factors Contributing to the Development of HSP

There is uncertainty as to the factors associated with HSP development. Identifying these factors is necessary to characterize patients with a high risk of developing HSP. These patients must be treated with extra caution, and early prevention procedures must be applied.

Age and Sex

In a retrospective study by Demirci et al.[9] on 1000 hemiplegic patients and in a cross-sectional study of 85 consecutive patients,[30] an incidence of HSP was found to be significantly associated with age. A magnetic resonance imaging study showed an association between age and rotator cuff tears in HSP patients.[41] Higher prevalence of HSP in women (53% *vs.* 47% in men) was reported in only one study.[9] On the other hand, several studies, including a prospective study[92] and a large population-based study,[19] did not find any association between age, sex, and HSP.[6,57,99]

Age may be associated with HSP in several ways. First, older individuals have a higher prevalence of shoulder pain and shoulder pathology independent of stroke.[44,100,101] Second, in general, older patients have more co-morbidities and a lower functional level. After stroke, these patients require more assistance in activities of daily living such as dressing and locomotion, which, in turn can increase risk of hemiplegic shoulder injury.

Preexisting Shoulder Pain and Degeneration

Stroke occurs in all age groups, but the older population has a higher risk for stroke than does the general population. This risk increases with age: 95% of strokes occur in people older than 45 yrs, and two-thirds of strokes occur in those older than 65 yrs.[102] On the other hand, shoulder joint pain and degeneration are also associated with age. For example, painless shoulder ROM decreases with age as a result of postural change and degeneration in the articular surfaces and soft tissues surrounding the shoulder girdle.[103] The prevalence of shoulder pain in the general adult population varies between 7% and 30%,[104] with the highest prevalence occurring during the fifth to the seventh decades.[105] Therefore, there is a considerable probability that stroke survivors had shoulder pain or degeneration before stroke. There are three reasons for this: (1) some cases of shoulder pain are not caused by hemiplegia and should not be diagnosed as HSP; (2) preexisting shoulder pain will most probably be influenced by hemiplegia, but no studies have as yet evaluated these influences; and (3) patients with preexisting shoulder pain or degeneration need to treated with extra caution to prevent aggravating the shoulder pain.

Shoulder (Glenohumeral) Subluxation

Shoulder subluxation usually develops in the first weeks after hemiplegia[106]; however, its relationship with HSP is still controversial. Several studies have found an association between HSP and subluxation, claiming that anteroinferior subluxation of the humeral head can be a triggering factor for HSP.[4,16,30–32,57] Other studies found no association between subluxation and HSP.[6,10,26,29,90,107,108] Suethanapornkul et al.,[57] in a recent and relatively large study, found that shoulder pain was significantly more frequent in subjects with shoulder subluxation. However, approximately 50% of patients with shoulder subluxation did not develop HSP. Another recent study of 187 stroke patients,[6] similar to several previous studies,[107,108] indicated that there was no statistical difference in the amount of subluxation in patients with or without pain. One of the explanations for the lack of association between subluxation and HSP is that a higher grade of subluxation of the glenohumeral joint may not result in more extensive tissue damage. It is not only the passive overstretching of the periarticular tissue that allegedly causes pain but also the resulting injury

to that tissue. The extent of such damage may depend more on how often and how long the arm was left hanging unsupported rather than on the degree of subluxation itself.[29]

Hakuno et al.[24] found a significant correlation between subluxation and arthrographically demonstrated adhesive changes in the bicipital tendon sleeve. Subluxation may cause traction injury to the upper trunk of the brachial plexus.[69] Ring et al. reported electromyographic evidence of axillary nerve dysfunction in patients with glenohumeral joint subluxation and hypothesized that the initial stages of subluxation may cause axillary nerve compression.[71]

Inverse correlation was found between the presence of shoulder subluxation and the age of stroke patients.[109] The older population has a lower risk for shoulder subluxation after stroke, probably because of the loss of elasticity of the periarticular tissues.

Perhaps not only the subluxation but also the actual loss of the mechanical stability after improper muscles activation caused by the damaged motor control pathways amplifies the risk for further soft-tissue lesion and HSP. Improper positioning in bed, lack of support while in an upright position, or pulling on the hemiplegic arm when transferring the patient all contribute to glenohumeral subluxation. The resulting mechanical effect is the overstretching of the glenohumeral capsule, flaccid supraspinatus, and deltoid muscles, which may increase the risk of soft-tissue injury and pain.[57,58]

Time Since Stroke

Several studies[18,42] have found that approximately 16%–20% of stroke patients reported shoulder pain immediately after stroke. However, the majority developed HSP only weeks or months later. Roy et al.[7] evaluated the frequency of HSP during the first 12 wks after an acute stroke in 76 patients, finding that the elapsed time period since the stroke was strongly associated with HSP. Ratnasabapathy et al.,[19] in a population-based study of 1761 patients, found that self-reported HSP increased from 17% at 1 wk to 20% at 1 mo to 23% at 6 mos after stroke. Several other studies confirmed the association between the duration of hemiplegia and HSP.[6,9,22,46,57] It is possible that late HSP can be caused by the abnormal posture of the hemiplegic shoulder that damages the surrounding tissues in time.

Hemiplegic Side

The association between the hemiplegic side and HSP is still not resolved. Several studies found a higher prevalence of HSP in patients with left hemiplegia.[9,10,31] Demirci et al. described a possible involvement of perception in the pathogenesis of the HSP.[9] In contrast, two studies found a predominance of right hemiplegia associated with HSP.[79,107] Many studies have failed to find any association between the hemiplegic side and HSP.[6,30,56]

Co-Morbidities

Lindgren et al.,[4] in a prospective population-based study, found a significant association between self-perceived ill health and HSP. Ratnasabapathy et al.,[19] in a large population-based study, found a positive association between diabetes and the incidence of self-reported HSP. Another large study also found a significant association between diabetes mellitus, heart disease, and HSP.[9] Several studies,[6,57] on the other hand, found no association between HSP and diabetes mellitus, hypertension, and heart disease.

Iatrogenic causes including improper handling by the rehabilitation staff and exercises or tasks using excessive involvement of the plegic hand can cause HSP. Improper passive or assisted active exercise and improper handling, pooling, or lifting performed by health care professionals, family, or the patients themselves may be a major cause of shoulder pain in hemiplegic patients.[2,16,20–22,44,45,66,80]

Wanklyn et al.[18] evaluated 107 hemiplegic patients at discharge and at 8 wks and at 6 mos after discharge; 39 patients with HSP were discharged from the hospital, 59 had HSP at 8 wks after discharge, and 36 had HSP after 6 mos. Patients requiring help with transfers were more likely to have HSP. Nine caregivers reported lifting the patient by pulling on the hemiplegic arm, even though six of them had received advice about correct lifting techniques. The authors concluded that stroke patients and their caregivers need guidance as to the correct handling of the hemiplegic arm. More awareness is essential in ensuring the correct handling of high-risk patients after discharge. Ring et al.[21] reported that some referring hospitals had consistently high rates of HSP, whereas others had a much lower rate. These differences could not be accounted for other factors such as age, long waiting periods, and others, and were therefore attributed to poor handling techniques. It is possible that improper rehabilitation further influences the patients with poor upper limb function.[68]

Braus et al.[80] evaluated the efficacy of an education program for shoulder-hand syndrome prevention. All members of the diagnostic and

therapeutic teams, as well as the patients and their family members, were instructed on how to avoid injuries to the affected limb. The results showed that the awareness of potential injuries to the structure of the shoulder joint reduced the frequency of HSP from 27% to 8%.

Exercises or tasks using excessive involvement of the plegic hand usually occur during therapeutic exercises commonly used in rehabilitation of poststroke patients. Several studies have found that exercises involving the shoulder's full ROM could cause HSP. One study evaluated the effectiveness of a protocol designed to restrict passive movements of the affected arm in preventing shoulder-hand syndrome.[110] A group of patients who received physical treatment with restricted passive movements (n = 81) showed significantly lower incidence of shoulder-hand syndrome (18.5%) than did the group who received regular treatment (n = 71; incidence, 32.4%). The use of overhead pulleys was also found to cause HSP and rotator cuff rupture.[16,108,111] Kumar et al.[108] assigned 28 poststroke patients to one of three groups: (1) ROM exercises by a physical therapist, (2) skateboard, and (3) overhead pulley. A significant difference (P = 0.014) was found in the number of patients who developed HSP between the groups: 8% in the first, 12% in the second, and 62% in the third. On the other hand, a shoulder exercise program according to the Bobath method successfully alleviated pain in HSP patients.[112]

In summary, several factors were found to be associated with a high probability of developing HSP: higher motor impairment, neglect, sensory impairment, older age, and co-morbidities. Improper handling by the rehabilitation staff and exercises or tasks requiring excessive involvement of the plegic hand can cause HSP. Therefore, instruction and awareness by nursing personnel, especially caregivers, must be an essential part of stroke rehabilitation.

CONCLUSIONS

Our goal was to systemize the possible underlying pathologies into an integrated picture. We thus categorized them into three categories (Fig. 1): (1) impaired motor control (muscle tonus changes), (2) soft-tissue lesions, and (3) altered peripheral and central nervous activity. These pathologies may present separately, may coexist simultaneously, or may evolve during the rehabilitation period by triggering each other's development.

Impaired muscle tone results from a direct lesion of the motor control pathways (upper motor neurons). Tonus changes can be further divided into spasticity (hypertonicity) and flaccidity. Spasticity of the shoulder girdle muscles, especially of the subscapularis, can generate local pain by pulling on its periosteal attachments. Spasticity also impairs the scapula-humeral rhythm, leading to

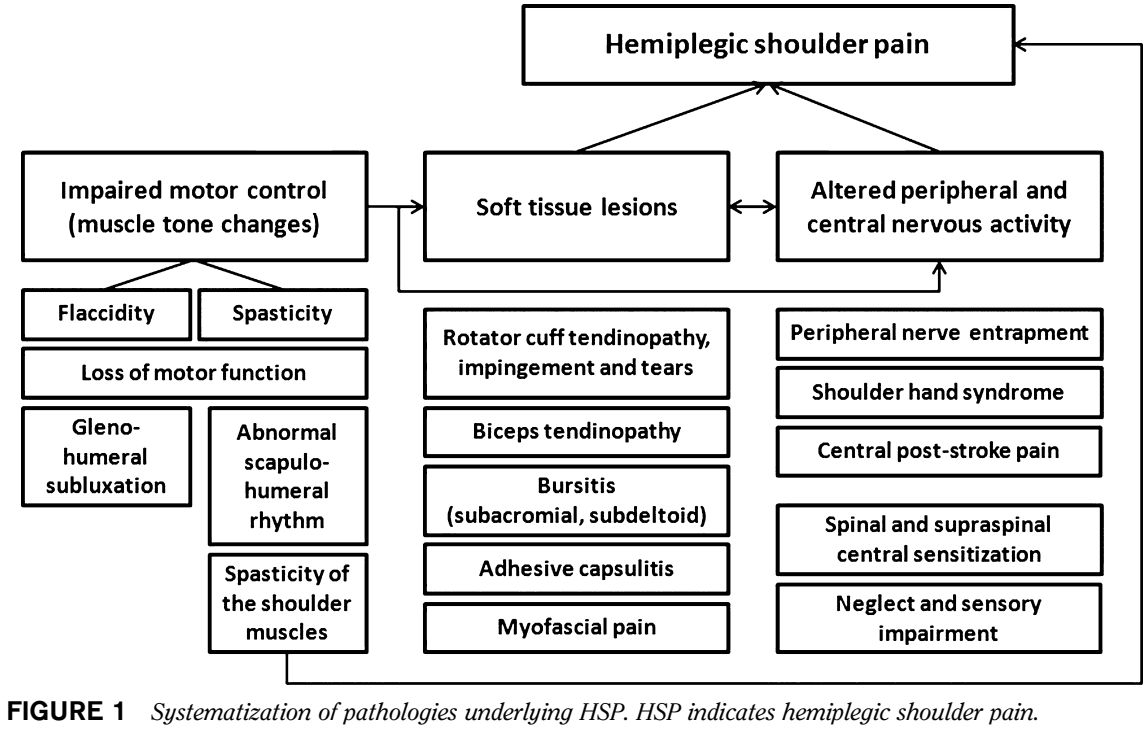

FIGURE 1 *Systematization of pathologies underlying HSP. HSP indicates hemiplegic shoulder pain.*

soft-tissue injuries around the shoulder. Flaccidity, on the other hand, may lead to glenohumeral subluxation, which, in turn, might stretch the peripheral nerves, causing entrapment, thus triggering soft-tissue lesions by overstretching the glenohumeral capsule. Motor deficit, muscle tone changes, and shoulder subluxation may all coexist with shoulder-hand syndrome or even predispose it.

Soft-tissue lesions include rotator cuff tendinopathy, tears, impingement, biceps tendinopathy, bursitis, adhesive capsulitis, and myofascial pain in the shoulder region. Rotator cuff and biceps tendinopathy, as well as bursitis, can trigger the development of adhesive capsulitis. Soft-tissue lesions may also encourage the development of SSS and supraspinal central sensitization followed by neural plastic changes in the CNS and shoulder-hand syndrome.

Altered peripheral and central nervous activity includes peripheral nerve lesions, shoulder-hand syndrome, central sensitization, and CPSP. Peripheral nerve lesions can be one of the reasons causing shoulder-hand syndrome.

The development of central sensitization in the corresponding spinal segments after soft-tissue lesions or peripheral nerve entrapment could contribute to the development of HSP and to ongoing noxious signals sent to the brain contributing to the development of supraspinal central sensitization. The SSS could affect the corresponding myotome, generating muscle spasms and myofascial pain. The primary lesion in the CNS after stroke can lead to CPSP encompassing the shoulder region.

The systematization of the underlying pathologies suggested in this review may lay the foundation for a constructive clinical approach enabling a tailor-made treatment of every painful hemiplegic shoulder.

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