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ORIGINAL ARTICLE

Hourglass-like constriction of the suprascapular nerve: a contraindication for minimally invasive surgery

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Background: Suprascapular nerve (SSN) entrapment is usually ascribed to static or dynamic compression. When no cause of compression is found, SSN entrapment is defined as idiopathic. Focal hourglass-like constriction (H-LC) of the SSN that results in muscle paralysis represents an unusual condition that may be misinterpreted and erroneously diagnosed as SSN entrapment or as neuralgic amyotrophy.
Methods: With the aim of finding clinical and surgical clues that could differentiate the traditional form of idiopathic SSN entrapment from the rare H-LC, a series of 6 cases of SSN palsy caused by H-LC is presented.
Results: All but 1 supraspinatus muscle recovered M5 muscle strength. The Constant shoulder score was excellent in 3 patients, good in 1, fair in 1, and poor in 1.
Discussion: If a diagnosis is not made in time, H-LC may evolve from mild to severe nerve torsion that may require a shift in surgical procedure from epineurotomy and external neurolysis to focal resection and suture. If an incorrect therapy is chosen, the chance of recovery might be definitively compromised with the persistence of muscle palsy. Conversely, when SSN palsy persists despite notch decompression, especially when it is performed with a limited open approach or arthroscopically, concerns about the real etiology and location of nerve compression responsible for the nerve palsy may arise.
Conclusion: When approaching SSN pathology, H-LC should be considered as a potential cause of nerve palsy, as it may represent a contraindication for a limited open approach or arthroscopic decompression.
Level of evidence: Level IV; Case Series; Treatment Study

Keywords: Suprascapular nerve; nerve palsy; hourglass-like constriction; shoulder arthroscopy; suprascapular nerve decompression; arthroscopic nerve decompression; shoulder palsy; minimally invasive surgery

Suprascapular neuropathy has been extensively studied since its first description in the 1950s.[38] Suprascapular neuropathy gained attention in the late 2000s when it began to be treated arthroscopically.[2,14,20] The degree of suprascapular neuropathy ranges from sensory disturbances[26] to nerve palsy with muscle atrophy,[16] and a great variety of provoking factors have been described[3] (Table I); however, until now, no study has

Institutional review board/ethical approval from our institution was not necessary for this study. Nevertheless, we obtained informed consent from all patients included in the study.

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Table I Etiology of suprascapular neuropathy

Repetitive traction (overhead sports)
GH dislocation
Scapular fractures
Penetrating injuries
Iatrogenic lesions
Microvascular alterations
SC or SG notch anatomic variations
Space-occupying masses
Retracted RCT
Parsonage-Turner syndrome
Idiopathic
Hourglass-like constriction

GH, glenohumeral; *SC*, suprascapular; *SG*, spinoglenoid; *RCT*, rotator cuff tear.
Table created from data used in references 14 and 41.

considered hourglass-like constriction (H-LC), a rare condition that may cause suprascapular nerve (SSN) palsy.[41] Although various theories have been proposed to explain the origin and progression of H-LC, the predisposing factors and etiology remain unclear. It is accepted that it primarily affects nerves located around joints that are subject to a high degree of motion (ie, the elbow, with posterior and/or anterior interosseous nerve palsy,[2,4,5,9-11,15,27,28,39,42-44] and as more recently described, the shoulder, with axillary nerve and SSN palsy).[7,25,30,41] According to the literature, H-LC leading to SSN palsy seems to be extremely rare but could be underdiagnosed. In fact, since the first description of H-LC leading to SSN in 2009,[41] only 2 other cases have been reported, one in 2011 and the other in 2014.[7,30] Similarly, after the first description of H-LC of the axillary nerve in 2006,[25] 5 other cases followed.[7,30,41] It is well known that when a new pathologic condition is reported, surgeons' awareness of that condition increases, and gradually, its diagnosis shifts from rare to uncommon and eventually, after years, may become common.

When imaging investigations (magnetic resonance imaging, ultrasonography, computed tomography scans, and so on) fail to identify the cause of SSN palsy (eg, ganglion cysts, direct trauma, bone alterations resulting from fractures, or bone morphology variations), attention is directed to the suprascapular and spinoglenoid notches, where SSN palsy may result from dynamic postural conditions or idiopathic compression (SSN entrapment syndrome). In such cases, the sectioning of the superior scapular transverse ligament and/or spinoglenoid ligament is the accepted treatment to resolve palsy. When SSN palsy persists despite notch decompression, especially when it is performed with a limited open approach or arthroscopically, concerns about the real etiology and location of the nerve compression responsible for the nerve palsy may arise. In those cases, a wider exploration of the nerves proximal to the suprascapular notch may reveal a different cause of nerve palsy.

We present a series of 6 cases of SSN palsy caused by H-LC and describe its peculiar clinical onset and surgical treatment with the aim of finding clinical and surgical clues that could differentiate the traditional form of idiopathic SSN entrapment from the rare H-LC, which may represent a contraindication for a limited open approach or arthroscopic decompression.

Materials and methods

We present a series of 6 SSN palsy cases in 6 patients. All patients reported that the palsy occurred after a short period of severe pain at the shoulder region as a consequence of an acute muscular effort or unfamiliar posture or work; all presented with a point of major tenderness located at the level of the SSN proximal to the suprascapular notch. In all cases, surgical findings demonstrated the presence of H-LC. Four of the patients were men, and the patients' ages ranged from 23 to 46 years (average age, 33.6 years).

Patient 1 presented with a double episode of palsy, first to the axillary nerve and 7 years later to the SSN.[41] The SSN palsy was triggered by an unusual work position: under a car in the lateral position, with the left arm lifted upward and stretched in external rotation with the forearm supinated. Severe pain appeared the next day and lasted for 4 days. When the pain disappeared, left supraspinatus and infraspinatus palsy occurred that became complete in 10 days. Surgery was performed 4 months after the onset of symptoms.

Patient 2 underwent surgery on her right shoulder for acromioplasty and again 1 year later for a supraspinatus tendon tear. After the first operation, she started to report weakness during external rotation and pain in the shoulder. Two electromyography (EMG) studies documented severe SSN damage at that time. Despite the EMG studies, the patient was surgically treated for a supraspinatus tear. During the postoperative period, while her shoulder pain recovered, the shoulder external rotation impairment worsened, and supraspinatus and infraspinatus palsy appeared. Surgery was performed 13 months after the onset of symptoms.

Patient 3 used axillary crutches for 45 days during the postoperative period after an orthopedic operation on both legs. One month after abandoning the crutches, she reported severe pain in the left shoulder; later, she started to experience a rapid and progressive shoulder external rotation deficit in her left shoulder, which ended with supraspinatus and infraspinatus palsy. Surgery was performed 12 months after the onset of symptoms.

Patient 4, after strenuous work, reported pain for approximately 10 days; the pain subsided and was rapidly followed by impairment of the supraspinatus and infraspinatus muscles. Surgery was performed 6 months after the onset of symptoms.

Patient 5 reported severe shoulder pain 2 days after unfamiliar heavy work. When the pain subsided, palsy of the supraspinatus and infraspinatus muscles rapidly appeared. Surgery was performed 9 months after the onset of symptoms.

In patient 6, the triggering factor was related to body building. The symptoms and onset of palsy were similar to those of patients 1, 3, 4, and 5. Surgery was performed 12 months after the onset of symptoms.

The strength of the supraspinatus and infraspinatus muscles was clinically tested and scored according to the British Medical Research Council degree system for muscle strength (M0-M5). Points of major tenderness along the course of the SSN were detected and registered. In all cases, tenderness and pain were located proximally to the spinoglenoid notch. A visual analog scale score was used to grade the pain level. All 6 shoulders were screened with standard radiographs, magnetic resonance imaging, and EMG studies, and extrinsic causes of SSN palsy were excluded (Table II).