

# Differential Diagnosis of Shoulder Pain Followed by Progressive Weakness: A Case Report

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## **ABSTRACT**

Upper extremity weakness can be the result of a myriad of conditions ranging from contractile tissue injury, joint injury, or injury to central or peripheral nervous system components. Accurate diagnosis is important in establishing an optimal treatment regimen and sound prognosis. This report provides an overview of the diagnosis and treatment of Parsonage-Turner Syndrome, a relatively rare cause of upper extremity weakness and dysfunction.

## **OBJECTIVES**

1. Distinguish between Parsonage-Turner Syndrome and other causes of neurological upper extremity weakness.
2. Recognize key subjective findings of Parsonage-Turner Syndrome.
3. Recognize appropriate ancillary tests to aide in the differential diagnosis of Parsonage-Turner Syndrome.

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Accurate diagnosis of acute onset of upper extremity weakness can be a diagnostic enigma. In the SOF community musculoskeletal conditions involving the shoulder girdle are frequently encountered. Neurological conditions may mimic non-neurological conditions and clinicians must be aware of these less frequently encountered but equally impacting conditions. The combination of a thorough subjective and objective examination will enable the medical provider to develop accurately a list of possible diagnoses. Appropriate selection of ancillary tests will often aide in correctly diagnosing the injury. Specific diagnosis of acute upper extremity weakness will help guide appropriate management and avoid unnecessary utilization of medical resources as well as optimize practitioner and patient time.

## **CASE REPORT**

### **SUBJECTIVE HISTORY**

A 32 year-old healthy active duty U.S. Navy servicemember presented to the physical therapist three days following the onset of right medial scapular border and glenohumeral joint pain. The shoulder discomfort awoke him from his sleep. Aside from a six-hour flight three days prior to the onset of pain he reported no antecedent trauma or aggressive workout routine. In the 72-hour period from the onset of pain until reporting to the medical department he noted a gradual reduction in shoulder pain and new onset of, and progressively worsening, weakness in the right upper extremity.

His past medical history included a few episodes of “stingers” while playing rugby in college over 10 years ago. He had occasional neck pain over the past few years without any episodes of radiating pain from the cervical region into the shoulder or distal upper extremity. He denied any family history of similar conditions. There was no history of viral or bacterial illness in the past six months, nor immunizations during this period.

#### **PHYSICAL EXAM AND EARLY INTERVENTION**

Upon exam he was afebrile with normal vital signs. His cervical mobility was normal in all directions with central lower cervical pain produced at the end range of cervical extension. Active mobility assessment of the right upper extremity demonstrated shoulder flexion and abduction limited to 100 degrees with medial scapular border winging. Full active mobility was present for shoulder internal and external rotation as well as all upper extremity joints distal to the shoulder. Deep tendon reflexes of the upper and lower extremities were intact and bilaterally symmetrical. Babinski and Hoffman reflexes were absent. Gross manual muscle testing demonstrated 3-/5 serratus anterior, 4-/5 external rotation, 4/5 shoulder abduction, and 4+/5 pronator teres. He had 5/5 strength in the right triceps brachii, biceps brachii, wrist flexion and extension, and hand intrinsic muscles. Spurling’s test produced neck pain but no radicular symptoms into the shoulder girdle or distal upper extremity. Manual cervical traction produced no change in the patient’s symptoms. Given the patient’s history and physical exam, the differential diagnosis included C5 radiculopathy, long thoracic mononeuropathy, and Parsonage-Turner Syndrome (acute brachial neuritis). Initial management, following consultation with a dive medical officer included prednisone 50mg daily for five days followed by Naprosyn, 500mg twice a day, for 10 days. Additionally, physical therapy was performed consisting of active assisted range of motion exercises and isometric shoulder girdle exercises.

#### **ANCILLARY TESTS**

To further assist in the diagnosis, x-ray and MRI assessment of the cervical spine was conducted in addition to electrophysiological examination. X-rays showed multi-level degenerative disc disease. MRI exam demonstrated multi-level spondylosis, which was worst at C5-6, but no evidence of nerve root compression. Four weeks following the onset of weakness the patient underwent the electroneuromyogram (ENMG).

During the physical examination prior to the ENMG the patient reported intermittent paresthesias in the lateral volar aspect of the right forearm which did not extend distal to the wrist. The patient reported the onset of altered sensation which began about one week after the onset of shoulder pain. Electromyography (EMG) of the right serratus anterior muscle demonstrated positive sharp waves (1+) and a markedly reduced interference pattern. EMG exam of the cervical paraspinals, deltoid, rhomboid, infraspinatus, biceps brachii, triceps brachii, pronator teres, extensor carpi radialis, flexor carpi radialis, first dorsal interosseous, and left serratus anterior muscles was normal. Motor nerve conduction studies of the suprascapular and axillary nerve were normal. Sensory nerve conduction studies of bilateral median (to digits I and II) and superficial radial nerves were normal. Lateral antebrachial cutaneous (LABC) nerve conduction study demonstrated normal distal latencies but a 65% reduction of the sensory nerve action potential (amplitude) on the right upper extremity.

#### **SUBACUTE MANAGEMENT**

Six weeks following the onset of shoulder pain the patient reported full resolution of the shoulder pain, continued difficulty raising the arm overhead, and minimal change in right arm strength. Active flexion and abduction of the right shoulder was limited to 140 degrees and serratus anterior muscle atrophy was visible. He was provided guidance in strength training of the right upper extremity to enhance scapular stability, rotator cuff strength, and avoid stretching of the proximal neural structures. At three months following onset of symptoms the patient reported noticeable improvement in arm strength and mobility, but continued winging of the scapula. Shoulder flexion and abduction had returned to normal. Aside from 4-/5 serratus anterior muscle strength, all other isolated manual muscle testing of the right upper extremity demonstrated normal (5/5) strength. Rehabilitation instruction consisted of exercises focused on the posterior shoulder girdle muscles (e.g., rowing, pull downs, external and internal rotation, prone scapular retraction, and supine push plus). Six months post onset the patient had resumed his usual workout routine but still noted mild winging of the scapula. Exercises were progressed to include push up exercises on a physioball and push plus exercises with any pressing exercise (e.g., military press, incline press, and bench press). One year post onset of shoulder pain the patient reported full return of right arm strength and absence of scapular winging. Physical exam at this time showed 5/5 strength throughout the right upper extremity.

**DISCUSSION**

Acute shoulder girdle pain and weakness can be the result of numerous conditions (Table 1). The distinguishing factors in the clinical diagnosis of Parsonage-Turner Syndrome (PTS) are sudden onset of shoulder pain, that often awakens the patient, which gradually subsides over the course of days to weeks, only to be replaced by painless weakness.<sup>1</sup> The sever-

tient's history of stingers, occasional neck pain, and pain with cervical extension; however, the Spurling's test was unremarkable. While acute, intense upper extremity pain is possible in the initial presentation of CR, the more common presentation is a gradual progression from the onset of neck pain to varying degrees of peripheral manifestations including upper extremity pain, paresthesias, and weakness. Radiological assessment also supported CR as a possibility. MRI evaluation, while demonstrating multi-level spondylosis, did not indicate nerve root compression. Magnetic resonance neurography, not utilized for this patient, has been recently reported as the preferred imaging modality for the diagnosis of PTS.<sup>2,4</sup> The patient's rapid progression of painless upper extremity weakness, absence of paresthesias or numbness, and normal Spurling's test were not consistent with CR.

While isolated long thoracic mononeuropathy would manifest with scapular winging, there are no nerve fibers to the glenohumeral region to produce the shoulder pain reported by this patient. Furthermore, the diffuse weakness noted on initial examination, in muscles innervated by branches from the upper trunk of the brachial plexus, indicates this was not an isolated mononeuropathy. Electroneuromyography results also supported involvement beyond the long thoracic nerve.

Electroneuromyography (ENMG) is commonly used in the evaluation of upper extremity weakness or sensory deficits. The ENMG should be performed no earlier than three weeks following the onset of symptoms in order to provide ample time for axonal degeneration (muscle denervation) to manifest and be recordable during the exam. The abnormal LABC sensory nerve amplitude corresponded to the region of intermittent paresthesias reported by the patient. Isolated muscle denervation of the serratus anterior muscle indicated partial injury to the axons of the long thoracic nerve. Patients with PTS most commonly present with weakness in muscles innervated by the long thoracic or suprascapular nerves.<sup>1,2</sup> Sensory complaints most commonly involve the LABC or median nerve fields.<sup>2</sup> Absence of electromyography abnormalities in other muscles of the right upper extremity, to include the cervical paraspinals, further ruled out CR.

Early treatment with corticosteroids, as utilized for this patient, has been reported to potentially improve the time course for recovery of strength.<sup>2</sup> Additional early management consisted of isometric shoulder and upper extremity exercises to minimize disuse atrophy without imparting utilization of dyskinetic movement patterns by performing active movement of the shoulder

**Table 1.**  
**Differential diagnosis of acute upper extremity weakness**

<b>Neurological</b>	Cervical radiculopathy Peripheral nerve injury Mononeuritis multiplex Tumors of the brachial plexus Traction injury of the brachial plexus Parsonage-Turner syndrome Hereditary neuralgic amyotrophy Monomelic amyotrophy Central nervous system pathology Trauma Stroke Tumor
<b>Musculoskeletal</b>	Rotator cuff injury Acute calcific tendonitis Glenohumeral or acromioclavicular sprain

ity of pain often leads patients to seek emergency medical care and may result in investigation for myocardial infarction.<sup>2</sup> As the pain recedes, weakness and sometimes atrophy develop. Weakness may be limited to muscles innervated by a single peripheral nerve or any combination of peripheral nerves or the brachial plexus.<sup>1,2,3</sup>

Upon initial examination the possibility of musculoskeletal injury as the primary cause of symptoms was ruled out based upon no preceding trauma, weakness of the arm not accompanied by pain, and absence of localized tenderness of the shoulder. Central nervous system involvement was ruled out based upon the patient's localized symptoms as well as the absence of hyper-reflexia, clonus, cranial nerve involvement, diffuse sensory complaints, and normal mobility (gait and distal upper extremity). Cervical radiculopathy (CR), isolated peripheral mononeuropathy, and PTS were therefore the primary considerations at the initial visit.

Cervical radiculopathy, involving the C5 nerve root, was considered a possibility given the pa-

above 90 degrees elevation. Modalities (e.g., therapeutic ultrasound and electrical stimulation) were not utilized as there is no evidence supporting acceleration of axonal regeneration or gross muscle strength with application following PTS. As the patient's mobility improved, strengthening exercises were progressed, focusing on scapular stabilizing muscles, within the available range of uncompensated active motion. The patient was educated on trying to use the scapular muscles to control scapular winging and to avoid exercises which produced scapular winging.

Recovery of strength commonly begins within the first two months following the onset of shoulder pain. Return of full strength is not always obtained. A protracted recovery of more than a year for return of strength is not uncommon.<sup>1</sup> Patients with primary involvement of the long thoracic nerve have been noted to have more complete strength recovery than patients with primary involvement of the suprascapular nerve.<sup>1</sup>

#### SUMMARY

Diagnosis of acute onset of shoulder pain with accompanying muscular weakness can be challenging. Parsonage-Turner Syndrome, although relatively uncommon, should be considered in the differential diag-

nosis of acute onset of shoulder pain and weakness. The presentation of sensory and muscular deficits is variable in both in distribution of involvement and severity of axonal injury. Ancillary testing such as magnetic resonance neurography in the acute phase or ENMG at one month post onset may prove helpful in diagnosing PTS. Return of strength usually begins within the first couple of months following the onset of symptoms, but maximum recovery can take over a year and does not always result in 100% return of strength and functional ability. Incorporating a progressive submaximal strengthening program focused on the uninvolved scapular stabilizing and rotator cuff muscles is recommended to enhance functional recovery.

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