Expanding the Differential of Shoulder Pain: Parsonage-Turner Syndrome

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A 44-year-old man was in his car when it was rear-ended in a minor motor vehicle collision, during which his right forearm contacted the steering wheel. Shortly thereafter, pain in his right shoulder developed, but initial work-up was unremarkable. His pain progressed to shoulder girdle weakness over several months and did not improve after 2.5 years. At the time of consultation, he complained of right-sided neck pain radiating to the right deltoid muscle and axilla as well as right shoulder blade pain with shoulder girdle weakness. Repeated electrodiagnostic studies revealed denervation limited to the serratus anterior and right deltoid muscles without evidence of cervical radiculopathy. He was diagnosed with Parsonage-Turner syndrome, which is a neurologic condition characterized by acute onset of shoulder and arm pain followed by weakness and sensory disturbance. The authors review patient presentation, physical examination, and work-up needed for diagnosis of this syndrome to help physicians avoid administering unnecessary tests and treatment.

J Am Osteopath Assoc. 2009;109:415-422

In 1943, Spillane\(^1\) reported approximately 100 cases of “localized shoulder girdle neuritis,” a condition that typically included shoulder pain, weakness, asymmetric muscle wasting, and sensory symptoms. Five years later, Parsonage and Turner\(^2\) described a similar syndrome they termed *neuralgic amyotrophy* but stressed that the pain was of sudden onset and was often severe without any “constitutional disturbance” present from the neck to the hand. Although reports have used the terms *shoulder syndrome of Parsonage and Turner*,\(^2\) *paralytic brachial neuritis*,\(^3\) *brachial plexus neuropathy*,\(^4\) and *acute brachial neuritis* to convey this syndrome,\(^5\) we have elected to use *Parsonage-Turner (PT) syndrome* because, in our experience, it is the term used most often by clinicians.

In the present report, we first review the literature on PT syndrome, describing its etiologic process as well as tools for diagnosis and prognosis. We then present a report of a patient who initially was thought to have radiculopathy. Based on clinical presentation and evidence of weakness in two muscles that are not primarily innervated by the same nerve root, it was clear the patient had PT syndrome. Finally, we include an appendix that outlines the proper physical examination of muscles affected by PT syndrome. As the present report suggests, a thorough medical history, physical examination, and electrodiagnosis are necessary for proper diagnosis of this condition.

**Parsonage-Turner Syndrome**

Parsonage-Turner syndrome is well described in the literature. Although its etiologic process is unknown, various stressors have been correlated to disease onset. As described earlier, acute onset of pain followed by muscle weakness in a separate peripheral nerve distribution (ie, axillary and thoracic nerves) rather than a root distribution (ie, two muscles primarily innervated by nerve root C6) is a key element of diagnosis. Specifically, two weakened muscles sharing primary root innervation would suggest a diagnosis of radiculopathy in the proper clinical context. However, in PT syndrome, muscles that share root innervation with weakened muscles will be uninvolved. Electrodiagnostic studies should be used to confirm clinical diagnoses.

**Etiologic Process**

The etiologic process of PT syndrome has not been elucidated. In 1948, Parsonage and Turner\(^2\) correlated infection, minor surgery, minor gunshot wounds, and minor trauma with the precipitation of the syndrome. Several other stressors, including unaccustomed strenuous exercise, parturition, and surgery, have also been reported to precede the onset of PT syndrome.\(^3,4,6\) Autoimmune diseases, hereditary factors, and routine vaccinations have also been implicated.\(^4,7\) Although there have been parallels between various stressors and the onset of PT syndrome, an antecedent stressor is not always found.

**Patient Presentation**

Although the symptoms of PT syndrome vary, patients generally describe a triad of pain, weakness, and sensory symptoms. The onset is sudden and most often occurs at night.\(^4\)
CASE REPORT

Discomfort may last for prolonged periods. One study reported that 10% of patients had initial pain that lasted longer than 60 days. More than 75% of patients had two additional phases of neuropathic pain that were elicited by movement or prolonged posturing (eg, lying on of the affected limb). These pain phases lasted up to several months. Approximately 65% of patients reported persistent musculoskeletal-type pain at the origin or muscle insertion of the paretic or compensating muscles. Patients also had glenohumeral joint pathology, including subluxation and frozen shoulder. In addition, 29% of patients had chronic pain resistant to therapy.

Generally, weakness manifests days to weeks after the onset of pain and is commonly found in the shoulder girdle and serratus anterior muscle, though other individual muscles supplied by the brachial plexus may be affected. However, some reports have described weakness or paralysis occurring concurrently with the onset of pain. Sensory loss can occur in up to two-thirds of individuals and is commonly noted in the lateral distribution of the arm and posterior forearm, with paresthesias in one-third of patients.

Diagnostics

Electrodiagnostic evaluation of PT syndrome is most helpful in confirming diagnosis and localizing the lesion. This evaluation consists of two parts: nerve conduction studies (NCS) and needle electromyography (EMG). This two-part test is sometimes referred to as simply EMG, implying both NCS and EMG.

Motor and sensory nerves are measured by NCS, while motor response is measured by several parameters. The most important parameter for evaluating PT syndrome is compound muscle action potential, which represents the summation of all underlying individual muscle fiber action potentials. The second part of the evaluation is EMG, which involves inserting a needle into individual muscles to detect nerve injury. The most important aspect of evaluating PT syndrome is finding nerve injury in individual muscles. These muscles should not share root innervation, which may imply radiculopathy.

Cwik et al reported that median and ulnar motor and sensory nerve conduction studies are abnormal in only 15% of patients with a typical history of PT syndrome. However, Dimitru reported that 50% of patients had abnormalities that were found in motor studies of the serratus anterior, biceps brachii, and deltoid muscles as well as sensory studies of the median and lateral brachial cutaneous nerves. Although F waves may be abnormal, they have minimal value in localization. With abnormal motor studies of the musculoskeletal and axillary nerves innervating the biceps and deltoid muscles, respectively, the compound muscle action potential can determine the amount of axonal loss and, therefore, prognosis.

Unfortunately, the commonly affected long thoracic and suprascapular nerves are not amenable to reliable surface recording. Lo and Mills reported conduction block by stimulating at the cervical nerve roots, but this is contrary to the conventional belief that PT syndrome results from an axonal process. Suarez reported that 96.3% of patients with PT syndrome have abnormal EMG results that are consistent with the condition.

Needle EMG is useful because it localizes the axonal lesion. The most common patterns are single and multiple mononeuropathies. Overlapping myotomic abnormalities, along with concomitant paraspinal muscle denervation, would implicate a more proximal root lesion. With the exception of the anterior interosseus nerve to muscles such as the flexor pollicis longus, flexor digitorum profundus, and pronator quadratus, PT syndrome usually affects proximal musculature, most of which are the serratus anterior, biceps brachii, rhomboid, supraspinatus, and infraspinatus muscles. However, any part of the plexus—and, clinically, any muscle—could be involved.

There are several reports of phrenic nerve involvement with PT syndrome. Technically, a needle study can reveal Wallerian degeneration from axonal loss (ie, decreased recruitment, fibrillation and positive sharp waves, and polyphasic motor unit potentials), which can be very specific with patchy involvement to specific fascicles of a single nerve. With the suspicion of PT syndrome, evaluations should include screening nerve roots and paraspinal muscles as well as the serratus anterior, rhomboid, supraspinatus, and infraspinatus muscles. Depending on patient presentation, muscles innervated by the anterior interosseus nerve should be screened. With comprehensive screening, PT syndrome with single or multiple mononeuropathies can be differentiated from radiculopathy with myotonic involvement.

In a study by van Alfen and van Engelen, MRI scans of the cervical spine were not found to correlate anatomic pathology with clinical findings. Furthermore, results of MRI scans of the brachial plexus were abnormal in less than 10% of patients. These findings support the suggestion that an MRI scan of the plexus and shoulder girdle or upper arm is seldom required to establish a diagnosis. However, T2-weighted MRI scans of clinically weak muscles may reveal high signal intensity of the affected muscles. A 2007 report revealed that findings from magnetic resonance neurography may have clinically important benefits compared to those from MRI scans and may benefit patients by providing earlier diagnoses of acute or chronic PT syndrome.

In summary, patients with PT syndrome present with a sequence of sudden pain, weakness, and sensory loss, which can be confirmed by EMG and NCS.
Prognosis

The prognosis of PT syndrome is generally favorable. The duration of pain and severity of muscle weakness and atrophy prognosticates recovery. Severe pain typically resolves, but the persistence of pain negatively impacts the time for motor recovery.4,8

Motor restoration is unpredictable—two-thirds of patients may begin recovery in as early as 1 month, with proximal involvement improving better than distal involvement.4,8 Unilateral disease has demonstrated quicker recovery after 1 year and was similar to bilateral disease after 2 years. In one study,3 approximately 60% of upper trunk mononeuropathies recovered to normal function in 1 year, in comparison to lower trunk mononeuropathies, which resolved in 1.5 to 3 years. Although patients with weakened muscles recovered within 4 weeks and demonstrated “complete recovery within 6 months,” general motor recovery can continue for up to 3 years.4

A less favorable prognosis was found in patients with early, severe, and rapid weakness and wasting and prolonged or recurrent pain patterns with no motor recovery signs before 3 months.5,6 This finding is inconsistent with Tsairis et al,4 who reported that the rate of motor recovery was 36% within the first year, approximately 75% the second year, and 89% by the third year.

Report of Case

A 44-year-old man was in his car when another vehicle hit the rear end of his car 2.5 years ago. His right forearm made contact with the steering wheel. He was seen in an emergency department and was discharged the same day.

Shortly after the collision, right shoulder pain developed. The patient followed up with an orthopedist soon after the motor vehicle crash. Electromyography, NCS, and magnetic resonance imaging (MRI) were performed 2 months after onset of symptoms, but results revealed that the cervical spine and right shoulder were normal. During the following several months, his pain did not improve but progressed to shoulder girdle weakness.

The patient returned to the orthopedist 2.5 years after the motor vehicle collision. At his initial presentation, the patient complained of right-sided “achy, stabbing, and burning” neck pain that radiated into the deltoid muscle, axilla, and right shoulder. He quantified the severity of pain as seven out of 10 on a visual analog scale. He also complained of shoulder girdle weakness that not only limited his ability to lift his arm but also limited his ability to perform activities of daily living, including sleep. His medical history was not clinically significant for neurologic or musculoskeletal disease.

On initial presentation, the patient was in discomfort. He was afebrile with stable vital signs. The patient’s trapezius, serratus anterior, deltoid, supraspinatus, infraspinatus, and teres minor muscles were thoroughly examined using the techniques described in the Appendix. Physical examination revealed 70 degrees of active shoulder abduction with right medial scapular winging in forward flexion. Upper limb deep tendon reflexes, sensation, and motor power—including shoulder internal and external rotators—were normal with the exception of 2/5 strength in the right shoulder abductors and serratus anterior muscle. Provocative maneuvers were performed to further assess the patient, but Hawkins’, Neer’s, O’Brien’s, Scarf’s, Speed’s, and Spurling’s maneuvers17 were negative.

Results from a repeated MRI scan of the cervical spine and right shoulder remained unremarkable. Results from nerve conduction studies of the bilateral motor median, ulnar nerves with F waves, bilateral sensory median, ulnar, and radial nerves were normal. Needle EMG revealed small diffuse fibrillations and large polyphasic voluntary motor unit potentials with marked decreased recruitment consistent with denervation of the serratus anterior and right deltoid muscles. Results from needle EMG of the left supraspinatus, infraspinatus, rhomboid, cervical, biceps, triceps, pronator teres, and first dorsal interosseous muscles were normal.

Because of the severe sharp acute onset of shoulder pain followed by profound weakness and evidence of denervation in the serratus anterior and deltoid muscles, in the absence of any abnormalities in the cervical spine and shoulder on MRI, the patient was diagnosed as having PT syndrome. The diagnosis was reported to the referring physician with recommendation for conservative treatment with pharmaeologic pain management and aggressive outpatient physical therapy.

Discussion

Shoulder pain, muscle weakness, and sensory abnormalities are common complaints in patients with PT syndrome. Various more common pathologies, such as cervical radiculopathy, shoulder pathology, osteoarthritis of the neck or shoulder, somatic dysfunction, and myofascial pain, must be differentiated by medical history, physical examination, and selective use of electromyographic and imaging studies. Concomitant disorders may complicate the diagnosis of PT syndrome vs other conditions. A full neurologic and musculoskeletal examination of the upper limbs—focusing on the trapezius, serratus anterior, deltoid, and rotator cuff muscles—will implicate specific peripheral nerve pathology.

Although electrodiagnostic tests are needed, such tools have certain pitfalls (eg, patient intolerance, technical error). Another pitfall may occur with EMG. On needle EMG, up to 25% polyphasic motor unit potentials in the deltoid muscle may be normal.18 On a root screen to evaluate the patient for radiculopathy, when polyphasic motor unit potentials are encountered in the deltoid, findings may be considered normal unless other C5 and C6 innervated muscles have abnormal motor unit potentials or abnormal spontaneous activity. The contralateral deltoid muscle may be studied for proportional polyphasicity. In PT syndrome, if isolated polyphasic motor unit potentials are disregarded, then axillary nerve involvement may be overlooked.

A detailed review of physical examination techniques is
provided in the Appendix. If the trapezius or serratus anterior muscle is weak, scapular stabilization will be affected, and shoulder muscles will demonstrate pseudo-weakness if not positioned in the proper plane. The scapula is stabilized by the trapezius muscles in the frontal plane—also commonly referred to as the coronal plane—and the serratus anterior muscle in the sagittal plane. The instability typically affects the rotator cuff muscles of external rotation and the deltoid muscles.

If a patient’s serratus anterior muscle is weak, the physician should test the patient’s external rotators with the patient’s arms abducted 90 degrees. If the external rotators are tested in positions of scapular instability, pseudo-weakness can implicate rotator cuff pathology or suprascapular neuropathy. Middle deltoid strength will seem compromised if the trapezius muscle is weak. The axillary innervated anterior deltoid muscle can be tested in the sagittal plane, where the scapula is stabilized by the serratus anterior muscle.

Current treatment options for patients with PT syndrome include physical therapy, osteopathic manipulative treatment, and medication. Physical therapy should focus on active and passive range-of-motion exercises to prevent disuse atrophy and shoulder contracture. Mobilization of the region should include the treatment of the glenohumeral and scapulothoracic joints as well as the cervical spine. Use of osteopathic manipulative treatment to mobilize the shoulder, neck, and surrounding thoracic and scapulothoracic joints with myriad techniques, including the “seven stages of Spencer,” may help relieve restriction and pain. Ultrasound, electrical stimulation, and heat may be beneficial for the patient. Adequate analgesia is necessary for patient comfort, but there is no literature supporting a specific class of medication. Likewise, there is limited literature supporting the use of oral steroids for patients with PT syndrome.

In the present report, we discuss physical examination in detail because findings of weakness are easily missed or misinterpreted. Examination of less commonly examined muscles must be considered when shoulder range of motion is limited with scapular winging. Proper positioning during physical examination prevents detection of pseudo-weakness in the rotator cuff, deltoid muscle, or both, which can implicate incorrect peripheral nerve injury and possible misdiagnosis.

Generally, imaging provides no correlative data toward PT syndrome diagnosis, and a routine needle EMG root screen may miss the muscles affected. Therefore, examination of the commonly affected serratus anterior, rhomboid, supraspinatus, and infraspinatus muscles are necessary. Medical history, physical examination, and results from EMG will prevent misdiagnosis as well as unnecessary tests and procedures.

Conclusion
Parsonage-Turner syndrome must be included in the differential diagnosis of patients with shoulder pain, weakness, and sensory abnormalities. With a high index of suspicion, medical history, muscle weakness, and electrodiagnostic abnormalities will elucidate the diagnosis.

Acknowledgments
We thank Jeremy Simon, MD, and Theera Vachramukunkiet, MD, for allowing us to use their photographs in the present report.

References
A thorough physical examination is essential for physicians to determine the presence and extent of muscle weakness in patients suspected of having Parsonage-Turner syndrome. The following paragraphs describe muscle-testing techniques for examining various muscles of the shoulder. More information and videos on muscle-testing techniques are available through the Thomas Jefferson University JEFFLINE Web site.1

**Trapezius**

The trapezius muscle is divided into the upper, middle, and lower regions and is innervated by the spinal accessory nerve from the C3 and C4 nerve roots. The upper fibers of the trapezius muscle originate at the occipital protuberance and the upper ligamentum nuchae and insert on the lateral third of the clavicle and acromion. These fibers elevate and retract the scapula. The middle fibers of the trapezius muscle originate at the C7 through T5 spinous processes and insert on the acromion and lateral spine of the scapula to retract the scapula. The lower fibers of the trapezius muscle originate from the T6 through T12 spinous processes and insert on the medial spine (medial to the axis of rotation) of the scapula. These fibers depress and retract the scapula—performing the opposite function of the upper fibers. All three regions of the trapezius muscle rotate the glenoid fossa of the scapula toward the frontal plane (Supplemental Figure 1).

To assess the upper fibers, have the patient in a seated position and ask him or her to shrug and retract his or her shoulders. Observe for any side-to-side differences in elevation. Then, forcefully depress the shoulders, noting any muscle weakness (Supplemental Figure 2).

To examine the middle trapezius, have the patient flex forward 90 degrees at the waist and horizontally abduct the arms to 120 degrees. Monitor for any lateral winging and inability to fully abduct the arms. Then, depress the patient’s arms at the elbow (ie, toward the ground), observing for decreased horizontal abduction and lateral sliding of the scapula to retract the scapula.

Supplemental Figure 1. Action of the trapezius muscle. The upper fibers cause scapular Elevation and Retraction (A), the middle fibers cause scapular Retraction (B), and the lower fibers cause scapular Depression and Retraction (C). Each of the three regions of the trapezius muscle contribute to the Upward Rotation of the glenoid fossa of scapula in the frontal plane. Abbreviations: A, anterior; L, lateral border of scapula; M, medial border of scapula; P, posterior; S, superior border of scapula.
scapula (Supplemental Figure 3).

To examine the lower fibers, the patient remains at 90 degrees of flexion at the waist and places their shoulders in the diving position of 180 degrees flexion. Look for the arms to be at the same level. The examiner pushes down at the elbow and looks for decreased flexion and sliding of the scapula cephalad over the upper ribs (Supplemental Figure 4). Alternatively, the patient may be tested in the prone position.

Another method of detecting trapezius weakness is to have the patient place their back to the wall and abduct their shoulder in the frontal plane. If the trapezius is weak, the patient will compensate by trying to bring his or her shoulder into the sagittal plane, using the serratus anterior muscle as the primary abductor (Supplemental Figure 5).

Serratus Anterior

The serratus anterior muscle is innervated by the long thoracic nerve arising from the C5, C6, and C7 nerve roots. The muscle originates on the upper eight ribs and inserts on the costal aspect of the medial border of the scapula. It protracts the scapula and rotates the glenoid up in the sagittal plane (Supplemental Figure 6).

To examine the strength of the serratus anterior muscle, place the patient’s arm to 90 degrees of flexion with the elbow fully flexed, and position the elbow in slight horizontal adduction across the body. From behind the patient, place one hand on the opposite scapula to stabilize the patient while the other hand is on the patient’s elbow, pulling the shoulder posterior and inferior, noting any weakness or scapular winging (Supplemental Figure 7).

Deltoid

The deltoid muscle is innervated by the axillary nerve from the C5 and C6 nerve roots. The deltoid originates at the acromion and inserts on the deltoid tubercle. It primarily abducts the humerus.

To examine the deltoid muscle, have the patient abduct the arm to 90 degrees with the elbow flexed, and push the arm just proximal to the elbow toward the floor. This technique primarily isolates the middle deltoid. If there is suspicion of trapezius weakness,
have the patient place his or her elbow in flexion, and then flex and internally rotate the shoulder to 90 degrees to test the anterior deltoid.

**Supraspinatus, Infraspinatus, and Teres Minor**

The rotator cuff is made up of four muscles, three of which—the supraspinatus, infraspinatus, and teres minor muscles—contribute to external rotation of the humerus. They originate at the supraspinatus fossa, infraspinatus fossa, and lateral border of the scapula, respectively, and insert on to the greater tubercle of the humerus. The supraspinatus and infraspinatus are innervated by the suprascapular nerve and the teres minor by the axillary nerve, both from the C5 and C6 nerve roots.
Examination of the external rotators may be done with the patient’s arms at his or her side with the elbows at 90 degrees flexion if there is suspicion of weakness in the serratus anterior muscle. Force the arm into internal rotation by holding the elbow at the side with one hand (A) and forces the patient’s arm into internal rotation by pushing with the other hand, which is positioned proximal to the patient’s wrist (B).

Supplemental Figure 8. Examination of the external rotators of the humerus with serratus weakness. The examiner holds the patient’s elbow at the side with one hand (A) and forces the patient’s arm into internal rotation by pushing with the other hand, which is positioned proximal to the patient’s wrist (B).

Supplemental Figure 9. Examination of the external rotators of the humerus with trapezius weakness. Patient’s arm is abducted. The examiner holds the patient’s elbow with one hand (A) and forces the patient’s arm down with the other hand, which is positioned proximal to the patient’s wrist (B).

References