



Parsonage Turner Syndrome: A Review of Brachial Neuritis

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Mitesh Patel, et al.**Abstract**

Parsonage-Turner syndrome (PTS), also known as neuralgic amyotrophy or brachial neuritis, is a neurological disorder characterized by acute onset upper extremity pain followed by progressive muscle weakness. It can be triggered by a variety of insults, including infections, immune-mediated responses, mechanical stressors, or genetic predispositions. The underlying pathophysiology involves inflammation or injury of the brachial plexus, resulting in varying presentations depending on the specific nerve roots or branches affected. Despite its variable manifestations, PTS typically follows a recognizable chronological pattern. Patients first experience the sudden onset of intense shoulder or upper arm pain, often unilateral, lasting for days to weeks. This initial painful phase is followed by muscle weakness and atrophy, which can persist for several months. Recovery of muscle function occurs gradually during the final phase, although some patients may experience incomplete recovery.

We present the case of a 21-year-old male who reported five weeks of progressive left shoulder pain. He was initially evaluated at an urgent care center, where he was prescribed anti-inflammatory medication and provided with a sling. However, due to minimal improvement, he sought further evaluation at a sports medicine clinic. On physical examination, he had full passive ROM but had weakness with active ROM, a positive O'Brien's test, and 4/5 strength of the supraspinatus and infraspinatus. An MRI of the left shoulder (without contrast) was ordered to evaluate the weakness and showed a bright T2 signal in the supraspinatus and infraspinatus muscles with slight loss of muscle bulk, but intact tendons. These findings were consistent with denervation changes. Based on the clinical presentation and imaging results, a diagnosis of Parsonage-Turner syndrome was made. The patient experienced gradual symptom improvement through a combination of home exercises and physical therapy.

In conclusion, Parsonage-Turner syndrome remains an important but often under-recognized cause of upper extremity pain and dysfunction. Awareness of its classic symptom progression and imaging findings is essential for timely diagnosis and appropriate management.

Keywords: Parsonage Turner Syndrome; Brachial Neuritis; Neurological Disorder**Introduction**

Parsonage-Turner syndrome (PTS) is a neurological disorder that typically presents with the sudden onset of severe pain in the shoulder or arm. It is most commonly caused by damage to one or

more nerves of the brachial plexus. This syndrome encompasses several related pathologies, including brachial neuritis, brachial plexus neuritis, brachial plexus neuropathy, and neuralgic amyotrophy. The clinical course of PTS typically progresses through

three distinct phases. The first phase is characterized by the acute onset of intense pain, often lasting from several days to weeks. This is followed by a second phase marked by muscle weakness and atrophy, which emerges after the pain begins to subside. In the final phase, patients experience gradual recovery of muscle strength and function [2].

In this presentation, we discuss a case involving a young male patient who developed sudden-onset left shoulder pain, which progressed to weakness over the following weeks. The sports medicine clinician suspected Parsonage-Turner syndrome, and the diagnosis of Parsonage-Turner syndrome was also suggested by the radiologist after reviewing the MRI findings of the affected upper extremity. Treatment for PTS is primarily supportive and may include non-steroidal anti-inflammatory drugs (NSAIDs) for pain relief, as well as neuropathic agents such as gabapentin, pregabalin, or amitriptyline [3]. Physical therapy and structured exercises play a critical role in restoring muscle function and preventing further atrophy. The timeline of PTS can vary widely, with the acute illness phase typically lasting between 6 and 18 months. Some patients may experience residual weakness or persistent muscle atrophy even after the resolution of acute symptoms [4].

Case Presentation

A 21-year-old male with no significant past medical history presented with left shoulder pain that began approximately five weeks prior to his initial office visit. The onset of pain was associated with performing push-ups, during which he experienced a popping sensation in the shoulder. The pain was severe enough to prevent him from sleeping that night. He was evaluated at an urgent care center the following day and was treated with a shoulder sling and a course of naproxen. While his symptoms initially improved, he continued to experience pain with overhead activity and noted intermittent numbness.

He was subsequently evaluated by a non-operative primary care sports medicine physician. On physical examination, the patient had positive crossover and O'Brien's tests, with a negative Speed's test. There was no overlying erythema or signs of infection. Shoulder range of motion was normal passively, but weak actively. He was non-tender over the sternoclavicular joint, clavicle, anterior joint line, bicipital groove, and greater tuberosity, but demonstrated mild tenderness over the acromioclavicular joint. Strength testing revealed 4/5 strength in the supraspinatus and infraspinatus

muscles, with 5/5 strength of the subscapularis. Examination of the right shoulder was unremarkable. Sensory function was intact. Shoulder X-rays showed no fractures or dislocations, and joint spaces were preserved. Given his clinical presentation and the history of recent vaccination, Parsonage-Turner Syndrome (PTS) was considered as a potential diagnosis. He was advised to modify his activity and begin a rotator cuff strengthening exercise program. An MRI of the shoulder was ordered to further evaluate the cause of weakness.

The patient returned approximately two months later for follow-up. He reported significant improvement in shoulder pain, with persistent but improving weakness. He noted that certain activities-such as lifting, overhead movement, and pulling-remained limited due to discomfort. On physical examination, he demonstrated full passive range of motion and 4/5 strength in the supraspinatus and infraspinatus muscles, with 5/5 strength in the subscapularis. The MRI, as interpreted by a neurologist, showed a T2 hyperintense signal and mild muscle atrophy, with intact rotator cuff tendons. At this stage, the primary treatment focus remained physical therapy to preserve muscle mass and gradually restore strength. Over the following months, his symptoms resolved with continued physical therapy and adherence to a home exercise regimen.

Discussion

As outlined in the introduction, this patient was primarily suspected to have Parsonage-Turner Syndrome (PTS) because his initial presentation involved severe unilateral upper extremity pain, which later progressed to weakness. Relevant clinical findings supporting this diagnosis included positive physical exam and subsequent MRI findings.

PTS can also present with sensory deficits in the upper limb; however, this patient did not exhibit sensory abnormalities. The etiology of PTS is multifactorial, including infectious, immune-mediated, mechanical, and genetic causes. Infectious agents can directly involve the brachial plexus or trigger an immune response that damages the nerves. Notably, recent viral infections-such as Cocksackie B virus, HIV, parvovirus, mumps, and smallpox-have been linked to PTS [3]. Additionally, trauma such as vaccination injections in areas near the brachial plexus may precipitate this condition [3]. Given this potential risk, it is prudent to counsel patients, especially when undergoing vaccinations, surgeries, or childbirth. For patients with recurrent neuropathic attacks, premedication

with intravenous methylprednisolone or oral prednisone prior to immunizations may be considered to mitigate risk.

A rare hereditary form of PTS, known as hereditary neuralgic amyotrophy, is associated with mutations in the SEPT9 gene [2]. This mutation disrupts microtubule bundling and neurite outgrowth in cell cultures, likely through transcriptional dysregulation. However, the exact mechanism predisposing patients to inflammatory nerve reactions remains poorly understood.

Early differentiation of PTS from other musculoskeletal conditions is crucial. Differential diagnoses include adhesive capsulitis, calcific tendinitis, cervical radiculopathy, complex regional pain syndrome, osteoarthritis, rotator cuff tears, and motor neuron diseases [1]. Medial scapular winging is a common sign of PTS, often caused by weakness or atrophy of the serratus anterior or trapezius muscles due to involvement of the long thoracic and spinal accessory nerves [4]. Because PTS can affect various nerves, its clinical presentation varies widely between patients. Although PTS most commonly affects middle-aged adults, cases in younger and older patients, such as this 21-year-old, are rare and noteworthy.

Diagnostic evaluation is supported by electromyography (EMG), which can reveal denervation and axonal loss indicative of peripheral nerve involvement. MRI of the affected shoulder can demonstrate muscle edema or enhancement consistent with nerve injury. Laboratory tests may be useful in excluding autoimmune, infectious, or metabolic causes.

Management of PTS typically includes pharmacological therapies such as NSAIDs and oral corticosteroids to reduce inflammation, neuropathic pain medications (e.g., gabapentin, pregabalin, or amitriptyline), and opioids for refractory severe pain. In cases where an autoimmune trigger is suspected, immunotherapy such as intravenous immunoglobulin (IVIG) or plasma exchange may be employed. Physical therapy plays a critical role in preserving muscle function, preventing atrophy, and improving strength once the acute pain subsides [3]. Range of motion and strengthening exercises are vital to prevent complications such as frozen shoulder. Serial EMG studies can be useful for monitoring nerve recovery over time [5].

This patient was diagnosed with PTS within two months of symptom onset, and his recovery spanned approximately three to four months. Typically, recovery from PTS can take between one to

three years due to the prolonged weakness phase [4]. Although the patient was relatively young, a thorough physical exam combined with supportive MRI findings facilitated an accurate diagnosis and appropriate management.

Conclusion

Parsonage-Turner Syndrome is an uncommon but important cause of acute shoulder pain followed by muscle weakness, often presenting a diagnostic challenge due to its varied clinical manifestations and overlap with other musculoskeletal disorders. Early recognition through detailed clinical evaluation, supported by imaging and electrodiagnostic studies, is essential to differentiate PTS from other shoulder pathologies and to guide appropriate management. Although recovery can be prolonged, timely initiation of physical therapy and symptomatic treatment can significantly improve patient outcomes. This case highlights the importance of considering PTS in younger patients presenting with shoulder pain and weakness, emphasizing a comprehensive approach to diagnosis and rehabilitation.

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