Case Report

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Uncovering Parsonage-Turner Syndrome in a Patient Diagnosed with Tendinopathy

Tendinopati Tanılı Bir Hastada Parsonage-Turner Sendromunun Ortaya Çıkarılması

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ABSTRACT

Rotator cuff pathologies are one of the common causes of shoulder pain. However, there are other rare situations that cause pain and limitation in the shoulder. Knowing these pathologies will prevent late or incorrect treatments. Parsonage-Turner syndrome (PTS) (neuralgic amyotrophy, brachial plexitis) is a rare cause of shoulder pain with a frequency of 1.64-3.00 in 100,000. Diagnosis is made by clinical evaluation and electrodiagnostic study. Delayed diagnosis may cause muscle atrophy and functional losses in the patient. The purpose of this case is to increase awareness of PTS, which is a rare condition.

Keywords: Neuralgic amyotrophy, brachial plexus neuritis, shoulder pain, Parsonage-Turner syndrome

ÖZ

Rotator manşet patolojileri omuz ağrısının yaygın nedenlerinden biridir. Ancak nadir de olsa omuzda ağrı ve kısıtlılığa neden olan diğer durumlar da vardır. Bu patolojilerin bilinmesi geç veya yanlış tedavilerin önüne geçecektir. Parsonage-Turner sendromu (PTS) (nevraljik amiyotrofi, brakiyal pleksit) 100.000'de 1,64-3,00 sıklıkta görülen nadir bir omuz ağrısı nedenidir. Tanı klinik değerlendirme ve elektrodiagnostik çalışma ile konur. Tanı gecikmesi, hastada kas atrofisine ve fonksiyonel kayıplara neden olabilir. Bu vakanın amacı nadir görülen bir durum olan PTS hakkında farkındalığı arttırmaktır.

Anahtar Kelimeler: Nevraljik amiyotrofi, brakiyal pleksit, omuz ağrısı, Parsonage-Turner sendromu

Introduction

Parsonage-Turner syndrome (PTS), also referred to as neuralgic amyotrophy or brachial plexitis, is a rare neurological disorder (1). Clinically, it is characterized by acute onset of unilateral shoulder pain, followed by varying degrees of muscle weakness, sensory deficits, and, in more chronic cases, muscular atrophy. The estimated incidence ranges from 1.64 to 3.00 per 100,000 people, although this figure may be unreliable due to challenges in diagnosis, including misdiagnoses, lack of specific biomarkers, and overlap with more common conditions (2).

PTS typically affects individuals between the ages of 30 and 70, with a higher prevalence in males (3). Its pathophysiology remains unclear. Diagnosis is often delayed or incorrect due to its resemblance to more prevalent musculoskeletal issues such as rotator cuff pathologies.

This case report presents an elderly female patient whose symptoms were initially attributed to rotator cuff tendinopathy and who was even considered for surgery before being correctly diagnosed with PTS.

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Case Report

Written informed consent was obtained from the patient for publication of the case report.

A 74-year-old female patient applied to the physical therapy and rehabilitation clinic with complaints of severe pain and weakness in the left shoulder persisting for two weeks. Also, the pain was intense enough to disrupt her sleep. She reported being unable to lift her left arm entirely. There was no history of trauma. She retained mobility in her left elbow, wrist, and fingers. Notably, she had experienced a viral infection approximately one month prior. Her medical history included hypertension but no other significant comorbidities. On physical examination, assessment was limited due to severe pain. Muscle strength in the left shoulder was graded as follows: flexors 4/5, abductors 3/5, adductors 4/5, and extensors 3/5. Passive range of motion was restricted: 70° abduction, 30° adduction, 80° flexion, and 20° each for internal and external rotation. Active movement was preserved in the right shoulder. Sensitivity to touch was present and noted as allodynia. Reflexes were hypoactive. The patient's pain was evaluated as 90 mm with the visual analogue scale (VAS). Neuropathic pain was calculated as 31 with the painDETECT neuropathic pain scale. Routine biochemical and microbiological tests did not detect anything remarkable.

Shoulder magnetic resonance imaging (MRI) performed externally revealed effusion in the subacromial, subdeltoid, and subcoracoid bursae, as well as tendinosis in the supraspinatus and infraspinatus tendons. Based on these findings, the patient was initially recommended for surgery. In another clinic, she was advised complete rest of the left arm for one month. Electroneuromyographic (ENMG) evaluation was recommended for the patient. The ENMG result was reported as brachial plexopathy (brachial plexitis), in which the upper trunk left C5 and C6 roots were affected as shown in Table 1 and Table 2. The electrodiagnostic study showed fibrillation in the left supraspinatus muscle as shown in Table 3. The patient was started on non-steroidal anti-inflammatory drugs (NSAID) and pregabalin, titrated to 150 mg twice daily. A shoulder-focused physical therapy regimen including transcutaneous electrical nerve stimulation (TENS), cold application, and pulsed ultrasound was initiated. The patient was informed that her symptoms were not due to rotator cuff pathology, but rather brachial plexitis. Fifteen days later her VAS score was reduced to 50 mm, allowing the start of joint range of motion exercises. NSAID treatment was discontinued on the 15th day, with pain relief. After one month of exercise, shoulder abduction was 90 degrees, flexion 100 degrees, and internal and external rotations 30 degrees. Pregabalin treatment was continued for 3 months. After three months, the VAS score was evaluated as 30, painDETECT neuropathic pain scale score was 13.

Table 1. Motor nerve conduction study

Nerve	Latency (ms)	Amplitude (mV)	CV (m/s)
Anterior interosseous motor left			
Bl. elbow - Pron. quad / Pron. quad	2.68	2.4	
Ab. elbow - Bl. elbow / Pron. quad	3.29	8	
Anterior interosseous motor right			
Bl. elbow - Pron. quad / Pron. quad	2.57	5.2	
Ab. elbow - Bl.elbow / Pron. quad	2.97	8.1	
Median motor left			
Wrist - APB / APB	3.24	5.4	
Elbow - Wrist / APB	7.94	4.5	44.7
Median motor right			
Wrist - APB / APB	3.84	6.9	
Elbow - Wrist / APB	7.75	6.6	53.7
Ulnaris motor left			
Wrist - ADM / ADM	2.09	6.9	
Ab. elbow - Wrist / ADM	6.98	6.5	45
Bl. elbow - Ab.elbow / ADM	9	6.4	49.5
Ulnaris motor right			
Wrist - ADM / ADM	1.93	10.7	
Ab. elbow - Wrist / ADM	6.48	10.6	50.5
Bl. elbow - Ab. elbow / ADM	8.56	10.3	43.3

Bl: Below, Pron.quad: Pronator quadratus, Ab: Above, APB: Abductor pollicis brevis, ADM: Adductor digiti minimi, CV: Conduction velocity

Table 2. Sensory nerve conduction study

Nerve	Peak latency (ms)	Amplitude (μV)	CV (m/s)
Median sensory left			
Palm - Wrist	4.27	28.5	44.8
Median sensory right			
Palm - Wrist	4.45	41.8	39.5
Ulnaris sensory left			
Dig IV - Wrist	3.79	14	38.9
Median sensory right			
Dig IV - Wrist	3.6	12.7	37.9

CV: Conduction velocity

Table 3. Needle electroneuromyography

Muscle	Spontaneous activity		
	Fib	PSW	CRD
Right deltoideus post	0/10	0/10	
Left inteross dors	0/10	0/10	
Left biceps	6/10	6/10	
Left triceps	0/10	0/10	
Left supraspinatus	4/10	4/10	4+
Left deltoideus post	4/10	4/10	

PSW: Positive sharp waves, CRD: Complex repetitive discharges

Discussion

PTS was first described in the late 19th century and later formalized by Parsonage and Turner (4) in 1948. While relatively uncommon, it remains a frequently missed diagnosis, especially when presenting with symptoms resembling more familiar musculoskeletal issues. It typically begins with sudden and intense shoulder or upper arm pain, followed by progressive weakness in specific muscle groups and sensory disturbances. In some cases, noticeable muscle atrophy develops within weeks.

The pathogenesis of PTS is not fully understood, but a number of accelerating factors have been reported: Infections (particularly viral), vaccination, strenuous activity, surgery, and systemic autoimmune conditions (5). In roughly one-quarter of patients, symptoms follow a recent viral illness. Increasing reports have linked PTS to coronavirus disease 2019 infection and vaccination, supporting its suspected immune mediated nature (6-8). A hereditary form has also been identified, associated with genetic mutations on chromosome 17q25 (9).

In clinical practice, PTS is hard to diagnose due to its overlap with conditions like cervical radiculopathy, rotator cuff pathology, or adhesive capsulitis. These similarities can lead to misdirected interventions, including unnecessary surgeries or prolonged immobilization. Accurate diagnosis hinges on a high level of clinical suspicion and timely use of electrodiagnostic tools. ENMG remains the cornerstone for identifying denervation in affected muscle groups and helps distinguish PTS from compressive or mechanical causes of nerve injury.

Magnetic resonance neurography has shown potential in visualizing inflammatory changes in peripheral nerves, but limited access and high cost restrict its routine use. Importantly, distal sensory and motor nerve conduction studies often appear normal in PTS, as the condition primarily targets proximal nerve segments. This nuance makes localized nerve testing alone insufficient for ruling out PTS. Suprascapular nerve, long thoracic nerve, axillary nerve, and musculocutaneous nerve involvements have been reported in PTS (10,11). In our case, left suprascapular nerve was involved

In our case, the patient was initially misdiagnosed based on her MRI, which revealed mild degenerative changes that are not uncommon in elderly individuals. These coincidental findings led to a treatment plan that included surgery and prolonged immobilization both of which may have exacerbated her symptoms if pursued. Fortunately, the ENMG results shifted the clinical focus toward the brachial plexus and prompted a more appropriate conservative management approach.

Treatment of PTS is supportive and typically unfolds in two stages. The first aims at pain relief using a combination of pharmacologic agents (NSAIDs, gabapentinoids) and physical modalities (TENS, ultrasound, cold packs) (12-14). Acupuncture has also shown benefit in some cases (5).

The second stage focuses on functional recovery and prevention of complications like contractures. Gentle passive exercises are introduced once pain is under control, but strengthening is deferred until there is evidence of muscle reinnervation. This is essential, as prematurely loading denervated muscles may worsen injury or delay recovery (5).

Recovery in PTS is often gradual. Most patients show partial or full improvement within one to three years, though residual weakness or pain is not uncommon. In one study, 80% of patients recovered within two years, and 89% within three (3). Late diagnoses, like in this case, are associated with longer recovery timelines and an increased risk of incomplete resolution. While surgical tendon transfers may be considered in cases with persistent functional deficits, such procedures are generally reserved for selected and chronic cases.

This case reinforces the importance of looking beyond imaging in patients with shoulder pain especially when clinical features don't fully align with common mechanical diagnoses. Rotator cuff pathology may coexist with PTS, but it should not distract from a broader neurological evaluation when signs like weakness or sensory abnormalities are present. In this case, what initially seemed like a straightforward orthopedic issue turned out to be a complex neuropathic condition. A detailed history, careful examination, and timely ENMG helped avoid an unnecessary surgery and set the patient on a more appropriate recovery path.

Conclusion

PTS is a diagnostic challenge that can easily be mistaken for common orthopedic problems. However, the consequences of missing or mislabeling it can be significant leading to delayed recovery, unnecessary procedures, and prolonged disability. Clinicians should maintain a high index of suspicion, especially in patients with unexplained shoulder pain, weakness, and sensory changes. Early electrodiagnostic evaluation and a multidisciplinary approach are key to ensuring timely diagnosis and optimal patient outcomes.

Ethics

Informed Consent: Written informed consent was obtained from the patient for publication of the case report.

Footnotes

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