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Parsonage Turner Syndrome: A Case of Acute Shoulder Pain and Weakness in a Previously Healthy Adult

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Introduction

Parsonage-Turner Syndrome (PTS), also known as idiopathic brachial neuritis or neuralgic amyotrophy, is a rare neurological condition characterized by sudden onset of severe shoulder pain followed by muscle weakness and atrophy. It typically affects the upper brachial plexus and may be triggered by infections, vaccinations, or trauma. The syndrome is often misdiagnosed due to its overlap with cervical radiculopathy, rotator cuff injury and adhesive capsulitis. Diagnosis is largely clinical, supported by Electromyography (EMG) and imaging when necessary. The condition is self-limiting in most cases, but recovery can be prolonged and incomplete. This case highlights the importance of considering PTS in patients with acute shoulder pain and weakness, even in the absence of predisposing factors Parsonage-Turner Syndrome (PTS), also known as idiopathic brachial neuritis or neuralgic amyotrophy, remains an underrecognized cause of acute brachial plexus neuropathy. Its hallmark is the dissociation between intense pain and relatively preserved sensory function, which can mislead clinicians toward alternative diagnoses such as rotator cuff injury, cervical radiculopathy, or adhesive capsulitis. In this case, the absence of sensory loss and normal cervical MRI were critical in redirecting the diagnostic workup toward a peripheral neuropathy [1].

Description

Parsonage-Turner Syndrome (PTS) is believed to be an immune-mediated brachial plexopathy that typically presents with sudden-onset, severe shoulder pain followed by focal muscle weakness and atrophy. The condition often follows a viral illness, immunization, or other immune stimulus, although it can also occur idiopathically. The suprascapular, axillary and long thoracic nerves are among the most commonly affected, leading to characteristic patterns of motor deficits. Pain usually precedes weakness by days to weeks, distinguishing it from structural neuropathies or radiculopathies. Diagnosis is primarily clinical and supported by Electromyography (EMG), which typically shows acute denervation changes in the affected nerve distributions. EMG sensitivity increases when performed two to four weeks after symptom onset. Imaging of the cervical spine and shoulder is often performed to exclude compressive or structural causes, while high-resolution MRI or ultrasound may show muscle edema or atrophy in affected areas. There are no specific serologic markers for PTS and its diagnosis remains one of exclusion. From a therapeutic standpoint, no standardized treatment protocol currently exists. Corticosteroids are frequently used in the acute phase to reduce pain and inflammation, though controlled studies are lacking [2].

Early initiation of physical therapy is critical to preserving joint mobility and minimizing long-term functional impairment. Pain management with neuropathic agents may be necessary in cases of severe neuralgia. Recovery varies widely, with some patients regaining full function, while others experience persistent weakness or fatigue. As understanding of PTS evolves, future research should prioritize the identification of specific immunological or genetic markers that could aid in early diagnosis and risk stratification. Biomarker discovery through proteomic and genomic profiling could pave the way for targeted therapies and better prediction of disease course. Additionally, prospective studies are needed to evaluate the efficacy of corticosteroids and emerging immunomodulatory treatments in altering the trajectory of nerve recovery. Advances in neuroimaging and high-resolution nerve ultrasound may also enhance diagnostic accuracy, particularly in early or atypical cases where EMG findings are inconclusive. Quantitative MRI techniques capable of detecting microstructural nerve changes may become instrumental in monitoring disease activity and guiding rehabilitation efforts. Integration of artificial intelligence into imaging interpretation could support earlier recognition and more precise mapping of nerve involvement [3].

Moreover, establishing international registries and collaborative research networks would facilitate long-term outcome tracking and therapeutic comparisons. Patient-centered care models emphasizing early education, emotional support and multidisciplinary coordination are essential for managing the prolonged recovery phase associated with PTS. Tele-rehabilitation platforms and wearable technologies could play a growing role in monitoring motor recovery and improving access to specialized physical therapy services in underserved populations. Another important area of future development is the refinement of rehabilitation strategies. Current physical therapy approaches primarily focus on maintaining range of motion and preventing disuse atrophy, but individualized regimens based on functional assessments and muscle group involvement may yield better outcomes. Incorporating electromyographic biofeedback and neuromuscular stimulation into rehabilitation could enhance motor re-education and facilitate earlier muscle reactivation. Additionally, longitudinal studies assessing the efficacy of these advanced modalities in promoting nerve regeneration and functional restoration are warranted [4].

The role of autoimmunity in PTS pathogenesis suggests a potential benefit from more targeted immunotherapies. Future clinical trials evaluating treatments such as intravenous immunoglobulin (IVIG), plasma exchange, or newer biologics may clarify their utility in acute or refractory cases. Better understanding of the underlying immune mechanisms could also help differentiate PTS subtypes, such as hereditary neuralgic amyotrophy, from the more common idiopathic form, allowing for more personalized therapeutic strategies. In the meantime, clinical guidelines could be strengthened by including algorithms for early steroid use, timing of rehabilitation and when to consider immunomodulatory therapies. Finally, improving awareness and education about PTS among frontline clinicians including general practitioners, emergency physicians and orthopedic specialists is critical for early recognition. Because PTS is often mistaken for rotator cuff injury, cervical radiculopathy, or adhesive capsulitis, delays in diagnosis remain common. Development of decision-support tools, clinical checklists and continuing medical education programs can improve early identification and referral to neurologists or physiatrists. Digital health tools such as mobile apps or symptom trackers could also empower patients to report progression and receive timely interventions, ultimately improving long-term functional outcomes [5].

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Conclusion

Parsonage-Turner Syndrome is a rare but important differential in cases of sudden shoulder pain followed by weakness, particularly in otherwise healthy individuals. Clinicians should maintain a high index of suspicion for PTS when imaging is unremarkable and neurological findings localize to the brachial plexus. Early diagnosis and a multidisciplinary approach involving pain control and rehabilitation can enhance recovery and minimize disability.

Acknowledgment

None.

Conflict of Interest

None.

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