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The enigmatic tale of parsonage-turner syndrome: An intricate case study unveiling diagnostic puzzles and therapeutic insights

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Abstract---Neuralgic Amyotrophy, commonly referred to as Parsonage-Turner Syndrome (PTS), stands as a clinically diagnosed yet frequently overlooked inflammatory ailment that primarily targets the brachial plexus. [1] Regrettably, accurate identification of the condition is often delayed, subjecting patients to significant morbidity characterized by excruciating pain, muscle weakness, and sensory abnormalities in the upper extremity. Although the underlying pathophysiological mechanisms remain partially elusive, PTS typically emerges following a discernible triggering event, while the road to complete recovery can span from months to even years. [2] Within this context, we present a compelling case of PTS, aiming to shed light on the intricacies of the disease and foster heightened awareness to mitigate the risks associated with its under-diagnosis.

Keywords---enigmatic tale, parsonage-turner syndrome, unveiling diagnostic puzzles, therapeutic insights.

Introduction

Parsonage-Turner Syndrome, originally documented by M.J. Parsonage and J.W. Turner in 1984, and referred to as neuralgic amyotrophy/shoulder girdle syndrome [3][4][5], manifests with an acute onset of shoulder and arm pain, accompanied by a spectrum of sensory and motor deficits along the distribution of the brachial plexus. Unfortunately, this syndrome is frequently disregarded by physicians, who misinterpret it as a mere muscular spasm. The diagnosis of PTS relies primarily on a comprehensive evaluation of the patient's clinical history, presenting symptoms, and findings from physical examination [6]. Additional diagnostic investigations, including imaging and electromyography, can contribute to the confirmation of PTS by effectively ruling out other potential causes [6]. However, such patients should be approached with caution, considering the associated risk of increased morbidity, which could potentially result in significant disability.

Despite advancements in medical knowledge, the etiology and pathophysiology of PTS remain elusive. The syndrome has been reported in various contexts, including post-operative, post-infectious, and post-vaccination scenarios [7]. Furthermore, there is a possibility of an underlying genetic component, as certain TSP variants demonstrate hereditary patterns [8]. Although PTS typically follows a self-limited course with complete recovery, a subset of patients continues to experience chronic pain and persistent muscle weakness, leading to ongoing challenges in their daily lives.

Case Presentation

A 50-year-old male patient, diagnosed with type 2 diabetes mellitus, presented with an abrupt onset of severe pain in his right shoulder, ranking it as a 10 out of 10 on the pain scale, accompanied by a disruptive burning sensation that disrupted his sleep. The pain gradually intensified, extending to his neck, right arm, and medial scapula. The patient denied any history of trauma, strenuous activity, fever, chills, infections, or prior rheumatological conditions.

Initial attempts at self-management with over-the-counter analgesics and muscle relaxants proved ineffective, prompting multiple outpatient visits for the same complaints. Empirical treatment with pregabalin, targeting nerve pain, yielded minimal symptomatic relief. Over the course of a month, the patient experienced additional symptoms including paresis, numbness, tingling, and burning sensations along the lateral aspect of his right upper arm. Throughout this period, the patient remained stable from a hemodynamic perspective. Physical examination revealed restricted shoulder abduction, internal rotation, and limited flexion at the elbow joint, while no signs of redness, warmth, or swelling were observed. Active movements were impeded due to tenderness, yet passive movements remained intact. Notably, the patient retained normal range of motion in the neck.

Baseline investigations, encompassing parameters such as Erythrocyte Sedimentation Rate, C-reactive protein, and Anti-nuclear antibody levels, returned within normal ranges. Shoulder joint X-ray imaging failed to reveal any discernible abnormalities. However, electromyography conducted after four weeks exhibited signs of right brachial plexopathy, characterizing moderate involvement of the upper trunk with evidence of denervation and re-innervation, thus favoring a diagnosis of right brachial plexitis. Subsequent MRI evaluation of the right shoulder joint unveiled degenerative changes in the acromioclavicular joint, subacromial bursa fluid, impingement of the supraspinatus tendon by the acromion leading to partial tear/tendinosis, and concomitant adhesive capsulitis. Edema within the supraspinatus tendon was also noted. Contemplating the possibility of Parsonage-Turner Syndrome, a comprehensive treatment regimen was initiated, encompassing analgesics, glucocorticoids, and a structured rehabilitation program. As a result, the patient experienced remarkable improvement in pain symptoms and achieved nearly complete restoration of shoulder and elbow movements within a span of two months.

Discussion

Parsonage-Turner syndrome (PTS), an infrequent yet debilitating condition, presents with sudden onset, severe, nocturnal unilateral shoulder girdle pain, followed by progressive muscle weakness and varying degrees of paresthesia and numbness [3-5]. The syndrome typically exhibits pain, paresthesia, and sensory deficit that do not affect the same nerve distribution, while preserving passive range of motion [6]. Despite its distinct clinical features, PTS often encounters initial misdiagnosis as cervical radiculopathy or glenohumeral bursitis, highlighting the importance of prompt and accurate recognition for optimal management outcomes. PTS is thought to have an immune-mediated etiology, with approximately 50% of cases associated with preceding infections. Additionally, exercise, vaccination, surgery, and pregnancy have been identified as potential triggers, potentially eliciting an autoimmune response and subsequent brachial plexus inflammation [5].

Early identification of PTS assumes critical significance due to its potential to inflict significant pain and disability [2-5]. Treatment approaches commonly involve the administration of steroids to curb inflammation and alleviate pain. Complementary to pharmacotherapy, physical therapy plays an indispensable role in the comprehensive management of PTS, aiding in the preservation of range of motion and prevention of muscle atrophy. Notably, even with timely steroid administration, complete recovery may require months to years [5].

Conclusion

The present case serves as a compelling reminder of the pivotal role played by early diagnosis and timely initiation of steroid therapy in mitigating the morbidity and mortality associated with this condition. It is disconcerting that a considerable number of cases are initially misdiagnosed, leading to unfortunate delays in implementing appropriate treatment measures, consequently resulting in pronounced pain and neurological deficits. Therefore, meticulous attention to the patient's clinical history, presentation, comprehensive examination, and

electromyographic studies assumes paramount importance for the expeditious identification and prompt commencement of therapeutic interventions. By embracing this comprehensive approach, healthcare providers can meaningfully contribute to reducing the burden imposed by Parsonage-Turner syndrome, thereby enhancing patient outcomes and overall quality of life.

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