Um Caso Raro de Síndrome de Parsonage-turner Envolvendo Apenas o Nervo Radial

A Rare Case of Parsonage–Turner Syndrome Involving Only the Radial Nerve

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Abstract

Parsonage-Turner Syndrome, also known as idiopathic brachial plexopathy or amyotrophic neuralgia, is a rare condition characterized by sudden, severe shoulder pain followed by progressive neurological deficits. This case report aims to highlight an atypical presentation of Parsonage-Turner Syndrome involving the radial nerve exclusively. A 61-year-old right-handed male presented with non-traumatic right shoulder pain evolving over 7 months, followed by paresthesias and sudden loss of wrist and finger extension strength. Electrodiagnostic studies showed recent partial axonal (motor and sensory) injury of the right radial nerve. He was diagnosed with Parsonage-Turner Syndrome based on clinical history and electrophysiological findings and commenced rehabilitation. After two months of rehabilitation, there was significant improvement in wrist and finger extensor strength and hand functionality. This case underscores the importance of considering Parsonage-Turner Syndrome in patients with unexplained upper limb weakness and the necessity of a multidisciplinary approach in managing this condition.

Resumo

A síndrome de Parsonage-Turner é uma patologia rara, caracterizada por dor súbita e intensa no ombro, seguida de défices neurológicos progressivos. Este relato de caso pretende destacar uma apresentação atípica da síndrome de Parsonage-Turner, envolvendo exclusivamente o nervo radial. Um homem de 61 anos iniciou omalgia direita, seguida de parestesias e perda de força na extensão do punho e dedos. Os estudos eletrodiagnósticos evidenciaram uma lesão axonal parcial recente do nervo radial direito. O diagnóstico de síndrome de Parsonage-Turner foi estabelecido com base na história clínica e nos achados eletrofisiológicos, tendo sido iniciado um programa de reabilitação. Após dois meses de reabilitação, verificou-se uma melhoria significativa na força dos extensores do punho e dos dedos, bem como na funcionalidade da mão. Este caso reforça a importância de considerar a síndrome de Parsonage-Turner em doentes com fraqueza inexplicada do membro superior e a necessidade de uma abordagem multidisciplinar desta patologia.

Introduction

Parsonage-Turner Syndrome (PTS), also known as idiopathic brachial plexopathy or amyotrophic neuralgia, is a rare condition that typically presents as sudden, unilateral shoulder pain, followed progressive neurologic deficits, including muscle weakness, atrophy and sensory abnormalities.[1,2] PTS was initially identified in 1879, and it acquired its eponymous designation after a comprehensive account by Parsonage and Turner in 1948.[3]

Although the exact cause and pathophysiology of PTS are complex and incompletely understood, autoimmune

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diseases, genetic factors, post-vaccination status, viral infections, and trauma are among the etiologies cited in the literature.[1,4] According to *Pessa et al.*, PTS is significantly underdiagnosed in everyday clinical practice, with a true annual rate incidence rate of 1 in 1000.[5]

The initial pain in PTS is acute and often severe, localized in the cervical region, shoulder, or upper limb in 96% of patients.[4] In most cases, it manifests as multiple proximal axonal mononeuropathies in the upper limb, involving both motor and sensory nerves, and less commonly affecting mixed and distal nerves.[4] The discomfort may last for a few days or weeks, and once the pain subsides, muscle weakness develops within 24 hours in one-third of patients and within two weeks in most cases.[4,6]

PTS is typically self-limiting, with 80-90% of cases recovering muscle strength within 2-3 years, although residual paresis and exercise intolerance may persist in over 70% of cases.[7,8]

PTS has typically been regarded as primarily a clinical diagnosis, with treatment options limited to conservative measures.[4] These include the early use of corticosteroids, effective pain management, and rehabilitation protocol to address muscle weakness.[4,8]

Shoulder pain frequently prompts individuals to seek emergency, primary, and specialist medical attention.[7] Parsonage-Turner syndrome should be considered in the differential diagnosis of shoulder pain.[8] Understanding the clinical and electrophysiological features of this syndrome may aid in the prevention of iatrogenic conditions.[8]

This case report aims to raise awareness of Parsonage-Turner Syndrome by presenting an atypical case involving a mixed nerve that is rarely affected and even less frequently in isolation. Additionally, it highlights the importance of a multidisciplinary approach to managing this condition.

Case report

A 61-year-old right-handed man, followed in outpatient Neurology clinic for focal epilepsy of vascular aetiology, presented with sudden anterolateral right shoulder pain, described as squeezing and rated 8/10 on the Numeric Pain Rating Scale, which gradually decreased in intensity and began to exhibit neuropathic features over the course of 7 months.

This was accompanied by paresthesia and a sudden loss of motor function in wrist and finger extension, starting one month after the onset of pain. Patient denied preceding trauma, infections or vaccination.

Following symptom onset, he attended a private Neurology consultation, where electrodiagnostic studies showed acute partial axonal (motor and sensory) injury of the right radial nerve, distal to the branch for the brachioradialis muscle. Additionally, cervical radiography, soft tissue ultrasound, computed tomography, and magnetic resonance imaging of the head and neck showed no significant findings. Immunologic studies and viral serologies were negative.

Due to clinical presentation and findings on electrodiagnostic studies, he was referred to a neuromuscular diseases' outpatient clinic. Muscle strength assessment by segments (according to the Medical Research Council scale) showed muscle strength in wrist extension G2/5, finger extension of the right hand G2/5, thumb opposition G5/5, finger abduction G5/5, palmar grip G5/5, and remaining segments of the upper limb G5/5. Additionally, there was hypoesthesia to pinprick on the dorsal side of the first finger and the anatomical snuffbox. He reported no pain during upper limb mobilization. Deep tendon reflexes were normal and symmetrical, except for the styloradial reflex which was absent.

He started rehabilitation program three times a week, achieving a Cochin Hand Functional Scale (CHFS) score of 81/90 at the beginning of treatment. The rehabilitation program focused on strengthening the distal upper limb muscles (with the use of neuromuscular electrostimulation), especially wrist and finger extensors, movement pattern training and proprioceptive reeducation.

After two months of physiatric treatment, there was complete resolution of pain and improvement in the muscle strength of wrist extensors (G4/5) and finger extensors (G3/5), as well as in hand functionality (CHFS 59/90). All these improvements led to greater participation in daily living activities, using the affected hand, namely in basic tasks such as eating, carrying objects, and performing personal hygiene.

Discussion

PTS predominantly affects men between the ages of 20 and 60, with an incidence rate of 1–3 cases per 100,000 people per year.[2,8] The characteristic clinical signs and symptoms include sudden, severe shoulder pain that is non-traumatic and often radiates to the cervical region and the outer part of the arm.[2,4] After a variable period, paresis occurs, which is flaccid, patchy, and progressive, and its distribution does not always match the area of pain.[2] Typically, patients will have sudden severe neuropathic pain that resolves preceding or shortly after the onset of weakness. Most patients will develop atrophy within 5 weeks of onset of symptoms.[2] Multiple peripheral nerves, nerve roots and/or parts of brachial plexus can be affected. In most cases, proximal motor nerves are affected, and less frequently,

sensory or mixed distal nerves. The injuries are typically axonal and incomplete. Approximately one-third of patients develop bilateral and symmetrical symptoms. Diagnosis is based on the patient's medical history, physical examination, and electrodiagnostic studies, which typically shows acute lesion in the aforementioned distribution.[2]

The precise cause and mechanisms underlying PTS are intricate and not fully understood. Factors such as autoimmune reactions, genetic predispositions, infections, and mechanical processes have all been suggested as potential contributors.[8]

Despite reports that 80%–90% of PTS patients show good recovery within 2–3 years, more recent studies have indicated a much less favorable prognosis for most individuals.[8] One extensive cohort study revealed that most patients had a mean Rankin scale score of 2 after several years of follow-up, with only 10% achieving full recovery after more than 3 years.[9] Additionally, over 25% of patients were still unable to work due to PTS.[8] Another detailed follow-up study of a large cohort showed that 60% of PTS patients continued to experience pain after 6 to more than 24 months. The pain was continuous in 56% of cases and restricted daily activities in 54%.[8]

Cabézon et al., reported a case of Parsonage-Turner syndrome in which treatment with analgesics, glucocorticoids, and rehabilitation led to pain resolution and near-complete recovery of fourth finger movement within two months.

In our case, the combination of clinical history and electrodiagnostic study results was consistent with Parsonage-Turner Syndrome. Alternative etiologies for isolated radial nerve injury were considered and excluded: infectious (no history and negative serologies), immune-mediated/inflammatory (negative/normal analytical study), traumatic (no history), and mechanical compression (atypical compression site, distal to the spiral groove, with imaging excluding local pathology).

A limitation of our article is the lack of additional investigations to exclude a local radial nerve lesion, such as magnetic resonance imaging (MRI) of the elbow region adjacent to the area under discussion, or follow-up ultrasound of the peripheral nerves to assess for swelling, edema, or sonographic signs of compression.

To our knowledge, there are no reports of neuralgic amyotrophy of Parsonage-Turner to be presenting with exclusive involvement of the radial nerve.

This case demonstrates an unreported phenotype of PTS involving exclusively the radial nerve. This case is best diagnosed by a careful history and physical exam. Clinicians should have a high index of suspicion for neuralgic amyotrophy of Parsonage Turner in presentation of upper limb weakness, dysesthesias, and parethesias that was preceeded by severe pain, even if the clinical presentation involves the more distal segments.

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