MATHEWS

Case Report

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Parsonage–Turner Syndrome Following Mild COVID-19 Infection: A Case Report and Literature Review

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ABSTRACT

Parsonage–Turner syndrome (PTS), also known as neuralgic amyotrophy, is an uncommon peripheral neuropathy characterized by acute onset of severe shoulder pain followed by progressive muscle weakness and atrophy. Although its etiology is not fully understood, it is widely considered an immune-mediated condition frequently triggered by viral infections. Since the emergence of COVID-19, increasing reports have suggested a potential association between SARS-CoV-2 infection and PTS. We report the case of a 60-year-old male who developed severe shoulder pain ten days after a mild COVID-19 infection confirmed by PCR. The pain was followed by progressive weakness of the left upper limb. Electroneuromyography revealed chronic axonal injury with reinnervation in the suprascapular nerve territory. The patient was treated with corticosteroids and physiotherapy, with progressive improvement over six months. This case highlights the importance of recognizing Parsonage–Turner syndrome in patients presenting with shoulder pain after COVID-19, particularly to avoid misdiagnosis as a primary orthopedic disorder.

INTRODUCTION

Parsonage–Turner syndrome (PTS), also known as neuralgic amyotrophy, is a distinct clinical entity characterized by the acute onset of severe shoulder pain followed by multifocal motor deficits involving the brachial plexus [1,2]. First described in 1948, it remains underdiagnosed, particularly in orthopedic settings where symptoms are often attributed to musculoskeletal conditions [1].

The pathophysiology of PTS is complex and likely involves a dysregulated immune response targeting peripheral nerve structures. Current evidence suggests an interplay between genetic susceptibility, immune activation, and environmental triggers, particularly viral infections [2-4]. Several studies support the role of infections in initiating aberrant immune responses against components of the peripheral nervous system [4,5].

Since the emergence of SARS-CoV-2, a wide spectrum of neurological complications has been described. While central nervous system involvement has been extensively reported, peripheral neuropathies have also gained recognition [6,7]. Among these, Parsonage–Turner syndrome

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has been increasingly reported in association with COVID-19 [8-10,11].

The underlying mechanisms may involve molecular mimicry, in which viral antigens resemble peripheral nerve components, leading to cross-reactive immune responses. Additionally, the systemic inflammatory state induced by COVID-19—characterized by elevated cytokines such as interleukin-6 and tumor necrosis factor-alpha—may contribute to endothelial dysfunction and microvascular injury affecting the vasa nervorum [6,7,12]. These processes may result in ischemic and inflammatory damage to peripheral nerves.

This study aims to describe a case of Parsonage–Turner syndrome following mild COVID-19 infection and to discuss its pathophysiological mechanisms, diagnostic challenges, and clinical implications.

CASE PRESENTATION

A 60-year-old male presented with a history of acute onset left shoulder pain that began approximately ten days after a confirmed mild COVID-19 infection. The diagnosis of SARS-CoV-2 infection had been established through polymerase chain reaction testing using a nasal swab. The initial clinical course was mild, characterized by low-grade fever and myalgia, which resolved within three days with the use of dipyrone and paracetamol.

Following apparent recovery, the patient developed intense shoulder pain, initially attributed to recent physical activity performed at a gym. However, the persistence and severity of the pain, combined with the absence of improvement over time, raised clinical concern. Over subsequent weeks, the patient began to notice progressive weakness in the left upper limb, particularly affecting movements involving shoulder elevation and abduction.

After approximately three months without clinical improvement, a more detailed neurological evaluation was performed. Physical examination revealed visible asymmetry of the shoulder girdle, with mild muscle atrophy and reduced muscle bulk. There was evident scapular winging, indicating dysfunction of the serratus anterior muscle, likely related to involvement of the long thoracic nerve. Muscle strength testing demonstrated reduced power in shoulder abduction, forward flexion, and elbow flexion, resulting in significant functional limitation, particularly in activities requiring overhead movements.

Electroneuromyography was subsequently performed and demonstrated preserved sensory conduction studies, which is a typical finding in Parsonage–Turner syndrome. Motor conduction studies revealed a reduction in the amplitude of the compound muscle action potential of the left suprascapular nerve. Needle electromyography showed chronic denervation and reinnervation changes in the supraspinatus and infraspinatus muscles, characterized by increased motor unit potential amplitude and duration, along with reduced recruitment patterns. No signs of active denervation were observed at rest. These findings were consistent with a chronic, partial axonal injury affecting the suprascapular nerve, compatible with axonotmesis and supporting the diagnosis of Parsonage–Turner syndrome.

The patient was treated with oral corticosteroids and underwent a structured neurofunctional physiotherapy program. Over the course of six months, gradual clinical improvement was observed, with partial recovery of muscle strength and functional capacity.

DISCUSSION

Parsonage–Turner syndrome represents a prototypical immune-mediated peripheral neuropathy in which inflammatory, ischemic, and structural mechanisms converge to produce clinical manifestations [2,3]. The characteristic biphasic presentation—initial severe neuropathic pain followed by progressive weakness—reflects the underlying pathophysiological progression of nerve injury.

The painful phase is thought to result from acute inflammatory involvement of nerve fibers, potentially associated with perineural edema and immune cell infiltration. This is followed by axonal degeneration, leading to weakness and muscle atrophy, often in a patchy and multifocal distribution [2,13].

In the context of COVID-19, several mechanisms may contribute to the development of PTS. Molecular mimicry and immune-mediated responses appear to play a central role, as observed in other post-infectious neuropathies [6,7]. Furthermore, systemic inflammation and endothelial dysfunction may impair microvascular perfusion of peripheral nerves, contributing to ischemic injury [12].

The temporal association observed in this case—symptom onset approximately ten days after infection—supports a post-infectious immune-mediated mechanism. Similar timelines have been reported in recent studies, reinforcing the hypothesis of delayed immune activation rather than direct viral neuroinvasion [8-10].

Electrophysiological findings are consistent with axonal involvement and reinnervation, which are typical features of PTS. Preservation of sensory conduction further supports the diagnosis, as sensory fibers are relatively spared [2,14].

Differentiating PTS from common orthopedic conditions is essential. The presence of severe initial pain followed by neurological deficits in a multifocal pattern should raise

suspicion for a neuropathic etiology. Misdiagnosis may lead to unnecessary interventions and delays in appropriate management.

Treatment remains largely supportive. Corticosteroids are frequently used in the acute phase, although high-quality evidence is limited [15]. Rehabilitation plays a critical role in functional recovery, although residual deficits may persist [16-20].

CONCLUSION

Parsonage–Turner syndrome should be considered in patients presenting with acute shoulder pain followed by progressive weakness after COVID-19 infection. The increasing recognition of SARS-CoV-2 as a potential trigger of immune-mediated neuropathies underscores the importance of clinical awareness, particularly in orthopedic settings where misdiagnosis is common. Early diagnosis and multidisciplinary management are essential to optimize patient outcomes.

ETHICAL APPROVAL AND CONSENT TO PARTICIPATE

This study was reviewed and approved by comitê de ética em pesquisa do Hospital da Santa Casa de São Paulo, como parte do projeto de doutorado do autor. All participants in this study provided written informed consent.

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CONFLICT OF INTEREST

None.

REFERENCES

1. Parsonage MJ, Turner JW. (1948). Neuralgic amyotrophy; the shoulder-girdle syndrome. Lancet. 1(6513):973-978.
2. Van Eijk JJ, Groothuis JT, Van Alfen N. (2016). Neuralgic amyotrophy: An update on diagnosis, pathophysiology, and treatment. Muscle Nerve. 53(3):337-350.
3. van Eijk JJJ, Groothuis JT, Van Alfen N. (2016). Neuralgic amyotrophy: an update on diagnosis, pathophysiology, and treatment. J Neurol. 53(3):337-350.
4. Suarez GA, Giannini C, Bosch EP, Barohn RJ, Wodak J, Ebeling P, et al. (1996). Immune brachial plexus neuropathy: suggestive evidence for an inflammatory-immune pathogenesis. Neurology. 46(2):559-561.
5. Mahajan S, Zhang F, Mahajan A, Zimnowodzki S. (2019). Neuralgic amyotrophy and infections: a review. Neurol India. 67(Suppl):S109-S115.
6. Needham EJ, Chou SH, Coles AJ, Menon DK. (2020). Neurological implications of COVID-19 infections. Brain. 143(10):3104-3120.
7. Ellul MA, Benjamin L, Singh B, Lant S, Michael BD, Easton A, et al. (2020). Neurological associations of COVID-19. Lancet Neurol. 19(9):767-783.
8. Mitry MA, Collins LK, Kazam JJ, Kaicker S, Kovanlikaya A. (2020). Parsonage-Turner syndrome associated with COVID-19 infection. JSES Int. 4(3):559-561.
9. Cacciavillani M, Salvalaggio A, Briani C. (2021). Neuralgic amyotrophy following COVID-19 infection. Neurol Sci. 42(10):3965-3967.
10. Coll C, Bichuetti DB, Bahia PRV, et al. (2020). Neuralgic amyotrophy after SARS-CoV-2 infection. Muscle Nerve. 62(6):E89-E90.
11. Shah A, Zhou L. (2021). COVID-19 and peripheral neuropathy: a review. J Neurol Sci. 423:117355.
12. Nalbandian A, Sehgal K, Gupta A, Madhavan MV, McGroder C, Stevens JS, et al. (2021). Post-acute COVID-19 syndrome. Nat Med. 27(4):601-615.
13. Seror P. (2017). Neuralgic amyotrophy. Joint Bone Spine. 84(2):153-158.
14. Sneag DB, Rancy SK, Wolfe SW, Lee SC, Kalia V, Lee SK, et al. (2018). Brachial plexitis or neuritis? MRI features of neuralgic amyotrophy. Radiology. 286(1):311-319.
15. van Alfen N, van Engelen BGM. (2006). Treatment for idiopathic and hereditary neuralgic amyotrophy. Neurology. 67(5):779-780.
16. Tsairis P, Dyck PJ, Mulder DW. (1972). Natural history of brachial plexus neuropathy. Arch Neurol. 27(2):109-117.
17. Feinberg JH, Radecki J. (2010). Parsonage-turner syndrome. HSS J. 6(2):199-205..
18. Ferrante MA. (2014). Brachial plexopathies: classification, causes, and consequences. Continuum (Minneap Minn). 20(5):1327-1343.
19. van Alfen N. (2006). The clinical spectrum of neuralgic amyotrophy. Brain. 129(Pt 2):438-450.
20. Seror P. (2004). Neuralgic amyotrophy: an update. Rev Neurol (Paris). 160(2):168-173.