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POSTPARTUM PARSONAGE-TURNER SYNDROME WITH HOURGLASS CONSTRICTIONS AND SPONTANEOUS RECOVERY WITHOUT SURGICAL INTERVENTION

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ABSTRACT

Introduction: Parsonage-Turner syndrome (PTS), also known as neuralgic amyotrophy, is an uncommon neurologic disorder characterised by acute onset upper extremity pain followed by muscle weakness, atrophy and paraesthesias. Severity of symptoms is variable and can involve any peripheral motor nerves of the brachial plexus. Its occurrence in the immediate postpartum period is exceedingly uncommon.

Case description: A 31-year-old G1P1002 with a twin gestation underwent an emergency caesarean delivery for non-reassuring foetal status of twin B following a spontaneous vaginal delivery of twin A. Her pregnancy was complicated by A2 gestational diabetes. Shortly after surgery, the patient experienced right arm numbness, followed by two weeks of severe pain. Upon resolution of pain, she developed profound weakness in the posterior interosseous nerve (PIN) distribution of the right hand. Physical examination revealed loss of finger extension at the metacarpophalangeal joints, weak thumb extension and loss of wrist extension, with decreased sensation to light touch in the radial four digits. Wrist flexion was preserved. Electromyography demonstrated right posterior PIN neuropathy; specialised MR neurography identified multiple hourglass constrictions, pathognomonic for PTS.

Discussion: The patient was managed conservatively with analgesics, a trial of corticosteroids, electrical stimulation and occupational therapy. Eleven months after symptom onset, finger extension returned spontaneously.

Conclusion: This case highlights the presentation of PTS in the immediate postpartum period following caesarean delivery. Recognition of characteristic clinical features and imaging findings is crucial for accurate diagnosis. Conservative management can result in full functional recovery, though symptom resolution may be delayed.

KEYWORDS

Parsonage-Turner syndrome, neuralgic amyotrophy; postpartum neuropathy, hourglass constriction, posterior interosseous nerve palsy

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LEARNING POINTS

- Parsonage-Turner syndrome should be considered in postpartum patients presenting with acute severe upper extremity pain followed by weakness, as the condition may be misdiagnosed as cervical radiculopathy, peripheral nerve compression or carpal tunnel syndrome.
- Hourglass constriction lesions identified on MR neurography are considered pathognomonic for neuralgic amyotrophy, but the presence of these lesions does not necessarily mandate early surgical intervention.
- Spontaneous neurologic recovery may occur even in patients with hourglass constriction lesions, supporting the importance of individualised management and careful longitudinal reassessment before proceeding to operative treatment.

INTRODUCTION

Parsonage-Turner syndrome (PTS), also known as neuralgic amyotrophy, is an uncommon neurologic disorder characterised by acute onset of severe upper extremity pain followed by muscle weakness, atrophy and sensory disturbances. The condition is believed to be immune mediated and has been associated with triggers including infections, surgery, trauma, vaccination and other physiologic stressors. Pregnancy has been described as a potential precipitating factor, although cases occurring specifically in the immediate postpartum period remain poorly characterised. Clinical presentation is highly variable and may involve the brachial plexus or its peripheral branches. Early recognition is important to guide diagnostic evaluation and management. We present a case of postpartum PTS following caesarean delivery with MR neurography demonstrating hourglass constriction lesions and subsequent spontaneous recovery without surgical intervention.

CASE DESCRIPTION

A 31-year-old female (G1P1002) with a twin gestation underwent an emergency caesarean delivery for non-reassuring foetal status of twin B following spontaneous vaginal delivery of twin A. Her pregnancy was complicated by A2 gestational diabetes requiring insulin therapy. She had no prior neurologic or autoimmune disease and no family history of autoimmune conditions.

Shortly after surgery, the patient developed numbness in the right arm followed by severe pain lasting approximately two weeks. After resolution of pain, she developed progressive weakness in the posterior interosseous nerve (PIN) distribution of the right hand.

Motor examination demonstrated loss of finger extension at the metacarpophalangeal joints, weak thumb abduction and loss of wrist extension, while wrist flexion remained preserved. Sensation to light touch was reduced in the radial four digits.

Electromyography demonstrated right posterior interosseous neuropathy; MRI of the right upper extremity showed no evidence of compressive pathology *(Fig. 1)*. Subsequent specialised MR neurography demonstrated characteristic hourglass constrictions of the affected nerve, findings highly suggestive of PTS *(Fig. 2 and 3)*.

The patient was treated conservatively with analgesics and a short course of oral corticosteroids. Rehabilitation included occupational therapy, electrical stimulation and supportive therapies; surgical decompression was planned at 12 months if recovery did not occur. However, spontaneous recovery of finger extension occurred at 11 months and surgery was ultimately avoided.

DISCUSSION

PTS, also known as neuralgic amyotrophy, paralytic brachial neuritis, idiopathic brachial plexopathy, brachial plexus neuropathy and acute brachial radiculitis, is characterised by the abrupt onset of severe upper extremity pain followed by weakness and muscle atrophy, most commonly involving the brachial plexus or its peripheral branches. Although historically considered rare, the estimated incidence may be as high as 1 in 1,000 individuals, suggesting that the condition is frequently underrecognised. The syndrome has been associated with a variety of triggers including infections, vaccinations and surgical interventions. Although pregnancy has been described as a potential risk factor, there is limited literature describing its occurrence in the postpartum period[1-4]. The present case is notable for the occurrence of PTS in the immediate postpartum period following caesarean delivery with documented hourglass constrictions on MR neurography and subsequent spontaneous recovery without surgical intervention.

The pathophysiology of PTS is believed to be immune mediated, with additional contributions from physiologic stressors that may act as precipitating triggers. Neuralgic amyotrophy has been described in the literature as early as 1897; however, the syndrome was formally characterised in 1948 by Maurice Parsonage and John Aldren Turner, who reported a series of 136 patients presenting with sudden onset of severe shoulder pain followed by muscle weakness and atrophy[4]. Early observations of similar clinical presentations were also noted among soldiers during World War II, suggesting a potential association with extreme physical stress and fatigue. In addition to immune-mediated mechanisms, such physiologic stressors may play a contributory role in triggering neuralgic amyotrophy. In this case, while the overall labour course

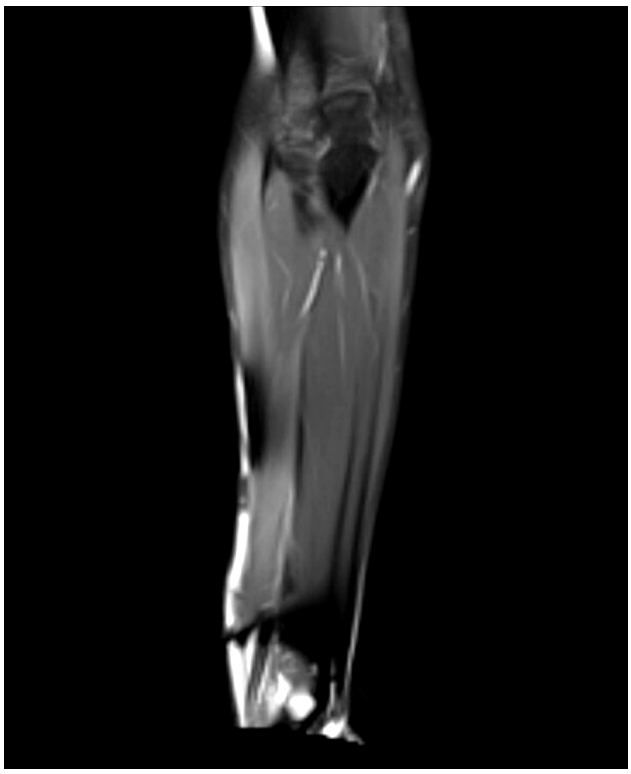


Figure 1. MRI of the right upper extremity demonstrating no evidence of compressive pathology affecting the posterior interosseous nerve.

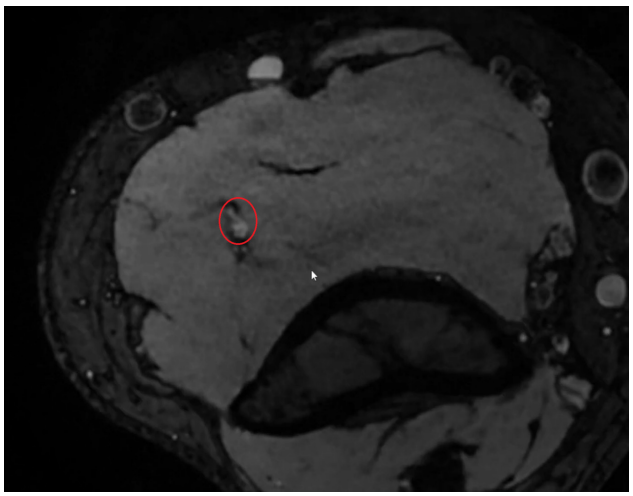


Figure 2. MR neurography of the right upper extremity demonstrating a focal hourglass constriction of the affected nerve.

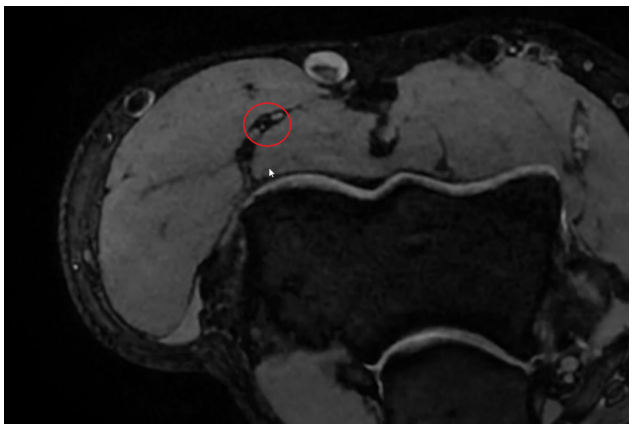


Figure 3. MR neurography demonstrating a second hourglass constriction consistent with neuralgic amyotrophy.

was not prolonged, the patient underwent an extended second stage with approximately four hours of pushing followed by an emergency caesarean delivery, representing a substantial physiologic stressor. While causality cannot be established, it is plausible that cumulative factors including prolonged labour-related fatigue, surgical stress and postpartum immune and hormonal shifts acted synergistically to precipitate symptom onset. A recent case report describes recurrent brachial plexopathy during pregnancy and the puerperium, with neurogenic damage confirmed by electromyography, highlighting the importance of considering immune and metabolic factors in the peripartum period[5]. Existing literature on postpartum neuralgic amyotrophy consists largely of case reports and small case series. In a series of 11 women with postpartum neuralgic amyotrophy, attacks occurred within hours to weeks after delivery. Clinical presentation, degree of weakness and electrodiagnostic findings varied widely, with involvement ranging from a single peripheral nerve to multifocal or bilateral disease. This distribution is consistent with the broader spectrum of neuralgic amyotrophy, in which the most frequently affected nerves include the long thoracic, suprascapular, anterior interosseous, axillary, musculocutaneous and radial nerves. Among these branches, the suprascapular nerve is involved in the majority of cases, while axillary nerve involvement is also common[2]. In contrast, subscapular nerve involvement appears to be relatively uncommon. In postpartum cases, the variability in nerve involvement is particularly notable, and bilateral attacks may be more frequent than in the general population.

Although the incidence of PTS specifically in the postpartum period is unknown, increasing awareness of this diagnosis is important as it is frequently missed. Early recognition allows for appropriate diagnostic evaluation and timely referral for multidisciplinary care. When suspected, adequate analgesia should be provided during the acute phase, as the severity of pain can be debilitating and frequently results in emergency care visits for intravenous analgesia.

Once the diagnosis is suspected or confirmed, appropriate referrals to neurology, pain management, peripheral nerve specialists, and occupational or physical therapy should be considered. Additional referrals, such as genetics or pulmonology, may be appropriate depending on the clinical presentation. A multidisciplinary rehabilitation approach should be instituted early, with a focus on motor relearning, contracture prevention, restoration of functional mechanics, energy conservation strategies and pain management[6,7]. Although most patients experience significant functional recovery over time, some individuals develop persistent weakness or chronic pain. Surgical intervention may be considered in refractory cases or when imaging demonstrates nerve constriction and there is limited evidence of recovery. Surgical options include microneurolysis, nerve grafting or tendon transfers.

Tendon transfer procedures are generally considered after approximately 18 months if meaningful nerve recovery has not occurred.

An additional consideration when evaluating patients with suspected PTS is the possibility of hereditary brachial plexus neuropathy, which has been associated with mutations in the SEPT9 gene[8]. Hereditary forms of the disease are well documented to be triggered by physiologic stressors including surgery or childbirth, with attacks sometimes occurring postpartum. In some cases, prophylactic immunosuppressive therapy including corticosteroids or intravenous immunoglobulin has been used to reduce the risk of recurrent attacks, although the evidence supporting these strategies remains limited[8].

Treatment strategies for idiopathic neuralgic amyotrophy remain largely empirical. Some studies suggest that early treatment with oral corticosteroids during the acute phase may shorten the duration of pain and accelerate motor recovery. In one case series of patients with neuralgic amyotrophy treated with oral prednisolone, a significantly greater proportion experienced early strength recovery compared with untreated controls[9]. Intravenous immunoglobulin therapy has also been described in small case series, but its efficacy remains uncertain and requires further investigation.

Importantly, this case also contributes to the evolving understanding of hourglass constriction lesions in neuralgic amyotrophy. MR neurography studies have increasingly identified these focal nerve constrictions, which are thought to represent structural nerve injury associated with inflammatory processes. Some reports suggest that patients with hourglass constrictions may experience more severe or persistent neurologic deficits and may be considered for surgical decompression when clinical recovery does not occur within several months.

However, as demonstrated in this case, spontaneous recovery may still occur despite the presence of documented hourglass constrictions. Although surgical intervention was planned at 12 months due to persistent weakness, the patient achieved spontaneous grade 5 on the Medical Research Council muscle power scale or near-complete recovery without decompression at 11 months after onset. This observation highlights the variability in the natural history of neuralgic amyotrophy and suggests that the presence of hourglass constriction alone should not automatically prompt early surgical intervention, underscoring the importance of individualised management and careful longitudinal reassessment before proceeding with operative treatment.

The psychosocial impact of PTS should also be considered. The abrupt onset of severe pain and disabling weakness can significantly affect daily activities, employment and quality of life. Patients may experience anxiety, frustration and emotional distress related to the unpredictable disease course and prolonged recovery period. Early recognition, patient education and multidisciplinary care may help mitigate these psychosocial effects and improve long-term outcomes.

CONCLUSION

Parsonage–Turner syndrome represents an underrecognized but important cause of acute upper extremity pain and subsequent motor dysfunction in the postpartum period. This case underscores the diagnostic challenge posed by its variable presentation, particularly in obstetric patients where symptoms may be initially attributed to more common compressive or positional neuropathies. Recognition of the characteristic clinical course, severe pain followed by focal weakness, combined with targeted electrodiagnostic studies and advanced imaging such as MR neurography, is critical to establishing an accurate diagnosis.

Furthermore, this report highlights the evolving understanding of hourglass constriction lesions in neuralgic amyotrophy. Although these findings have been associated with more severe disease and have historically prompted consideration of early surgical intervention, our case demonstrates that meaningful and even complete neurologic recovery may occur with conservative management alone. This reinforces the importance of individualized, longitudinal decision-making and cautions against premature operative intervention in the absence of clear evidence of irreversible nerve injury.

In the postpartum setting, where physiologic stress, immune modulation, and hormonal shifts may converge, heightened clinical awareness is essential. Early diagnosis facilitates appropriate pain control, avoids unnecessary interventions, and enables timely multidisciplinary rehabilitation. Ultimately, this case contributes to the limited body of literature on postpartum Parsonage–Turner syndrome and supports a more nuanced approach to management, emphasizing that the presence of structural nerve abnormalities does not preclude spontaneous recovery.

REFERENCES

1. Ameer MZ, Haiy AU, Bajwa MH, Abeer H, Mustafa B, Ameer F, et al. Association of Parsonage-Turner syndrome with COVID-19 infection and vaccination: a systematic review. *J Int Med Res* 2023;**51**:3000605231187939. doi: 10.1177/03000605231187939
2. Gaskin CM, Helms CA. Parsonage-Turner syndrome: MR imaging findings and clinical information of 27 patients. Radiology 2006;**240**:501–507. doi: 10.1148/radiol.2402050405
3. Smith CC, Bevelaqua AC. Challenging pain syndromes: Parsonage-Turner syndrome. *Phys Med Rehabil Clin N Am* 2014;**25**:265–277. doi: 10.1016/j.pmr.2014.01.001
4. van Alfen N, van Engelen BG. The clinical spectrum of neuralgic amyotrophy in 246 cases. *Brain* 2006;**129**:438–450. doi: 10.1093/brain/awh722
5. Zeng TF, Ding X, Yang D, Wu CC, Xie DD, Zhang WM. Clinical reasoning: A 26-year-old woman with recurrent pain, weakness, and atrophy in bilateral upper limbs during pregnancy and puerperium. *Neurology* 2023;**100**: 631–637. doi: 10.1212/WNL.0000000000201705
6. van Eijk JJ, Groothuis JT, van Alfen N. Neuralgic amyotrophy: An update on diagnosis, pathophysiology, and treatment. *Muscle Nerve* 2016;**53**:337–350. doi: 10.1002/mus.25008
7. Meiling JB, Boon AJ, Niu Z, Howe BM, Hoskote, SS, Spinner RJ, et al. Parsonage-Turner syndrome and hereditary brachial plexus neuropathy. *Mayo Clin Proc* 2024;**99**:124–140. doi: 10.1016/j.mayocp.2023.06.011
8. Klein CJ, Barbara DW, Sprung J, Dyck PJ, Weingarten TN. Surgical and postpartum hereditary brachial plexus attacks and prophylactic immunotherapy. *Muscle Nerve* 2013;**47**:23–27. doi: 10.1002/mus.23462
9. van Eijk JJ, van Alfen N, Berrevoets M, van der Wilt, GJ, Pillen S, van Engelen, B. Evaluation of prednisolone treatment in the acute phase of neuralgic amyotrophy: An observational study. *J Neurol Neurosurg Psychiatry* 2009;**80**:1120–1124. doi: 10.1136/jnnp.2008.163386.