Parsonage-Turner Syndrome in the Pediatric Population: A Case Report

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AUTHORS' CONTRIBUTIONS
Cyrus Safinia: manuscript drafting, clinical data review.
Marko Jakovljevic: writing, editing, references, submission.
Alexandra Lesenskyj DeArias: clinical data acquisition, editing.
Nigel S. Bamford: neurologic consultation, clinical input.
Jason M. Johnson: supervision, editing, corresponding author.

DISCLOSURES
The authors declare no conflicts of interest.

CONSENT
The local institutional review board waived written informed consent for this activity.

HUMAN AND ANIMAL RIGHTS
All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2000 (5).

ABSTRACT

Parsonage-Turner syndrome, a condition rarely seen in the pediatric population, is the focus of our case report. We present the unique case of a 4-year-old boy who developed a painful right brachial plexopathy following a febrile illness. The pain was exacerbated by touch or pressure and subsided when the shoulder was left undisturbed. On examination, he exhibited significant weakness in the right shoulder, with less pronounced weakness in the right hand. Brain and cervical spine magnetic resonance imaging (MRI) were unremarkable, but a MRI of the right brachial plexus revealed nerve root thickening and signal changes in the adjacent muscles, consistent with brachial neuritis. The patient began oral steroids and was referred to outpatient neurology for follow-up. Over several weeks his pain improved, though weakness persisted.

CASE REPORT

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Imaging Findings

MRI of the right brachial plexus performed three weeks after symptom onset demonstrated asymmetric thickening and high T2-weighted short-tau inversion recovery (STIR) signal of the right C5–C7 nerve roots with faint edema of the supraspinatus and infraspinatus muscles and no fatty atrophy—findings characteristic of acute brachial neuritis (Parsonage-Turner syndrome). Brain and cervical-spine MRI were normal, excluding central causes, and there was no enhancement or mass effect on post-contrast sequences.

Management

The child was admitted for diagnostic work-up, including a diagnostic lumbar puncture and extensive infectious/autoimmune studies (all unrevealing). He began oral

prednisolone 1 mg kg^{-1} day^{-1} (6 mL) for two weeks with a three-week taper, plus famotidine for gastrointestinal prophylaxis. Pain improved rapidly; no opioids were required. Discharge instructions included outpatient physiotherapy/occupational therapy and neurology follow-up.

Follow-Up

At nine months the patient no longer reported pain but retained marked proximal right-arm weakness (deltoid 3-/5; shoulder flexion 3-/5) despite preserved hand strength. Sedated EMG confirmed chronic denervation of the biceps, deltoid, supraspinatus and infraspinatus with sparing of triceps and distal forearm muscles, supporting ongoing plexus involvement. Surgical nerve exploration and intra-operative stimulation were discussed; further recovery remains guarded.

DISCUSSION

Parsonage-Turner Syndrome (PTS), also known as acute brachial neuritis or neuralgic amyotrophy, is a rare neurological disorder characterized by the sudden onset of shoulder pain, followed by muscle weakness and atrophy in the upper limb. While PTS predominantly affects adults, cases in the pediatric population are uncommon and often under-recognized due to atypical presentations and limited awareness among clinicians [1]. The etiology of PTS remains unclear, but it is frequently associated with preceding events such as infections, immunizations, or surgeries that may trigger an autoimmune response. Early recognition is crucial for management and improvement of functional outcomes. We present a case of a 4-year-old boy with PTS following a febrile illness, highlighting the importance of considering this diagnosis in pediatric patients presenting with acute brachial plexopathy.

Etiology & Demographics

PTS is a rare disorder, particularly in children. PTS most commonly affects males in the third to seventh decades of life, but it can present across a wide age range (3 months to 75 years) [2, 3]. PTS has been reported in patients anywhere from 3 months to 75 years with a bell curve distribution of cases depending on age [1, 4]. It most commonly affects males with an incidence of 1.64 to 100 cases per 100,000 inhabitants per year depending on geographical location [1-5]. Its rarity in pediatric patients contributes to diagnostic challenges, emphasizing the importance of recognizing its classic features to avoid delay in diagnosis [2, 6].

PTS typically follows an inciting event, such as a febrile illness or immunization, suggesting an immune-mediated mechanism [1-5, 7]. Genetic predisposition is also possible, particularly in children. Epidemics of PTS have been reported in genetically homogenous populations, and mutations in the *SEPT9* gene have been implicated in hereditary cases [3-5]. In this case, the preceding febrile illness and CSF pleocytosis support an immune-mediated etiology.

Clinical & Imaging Findings

The typical PTS presentation involves acute-onset shoulder and arm pain, predominantly in the proximal shoulder girdle, often worsening at night, followed by muscle weakness and, in some cases, sensory deficits [1-6]. Although predominantly unilateral, up to one-third of cases may show bilateral involvement or contralateral electromyography (EMG) abnormalities, even in asymptomatic limbs [2-5]. However, in this case, abnormalities were confined to the symptomatic limb.

Evaluation typically includes MRI and electromyography (EMG). MRI findings in PTS reveal denervation changes such as increased T2 signal intensity in affected muscles, neurogenic edema, and, occasionally, hourglass constrictions of nerves. In this case, MRI demonstrated T2 signal changes in the right supraspinatus and infraspinatus muscles, consistent with acute denervation, along with thickening of the right brachial plexus nerve roots. The absence of fatty atrophy supports an acute-to-subacute presentation (Figure 3). Ancillary testing, including blood work, inflammatory markers, and CSF analysis, was unremarkable except for mild pleocytosis, further supporting an inflammatory etiology.

The axillary, long thoracic, and suprascapular nerves are most commonly involved in PTS, leading to symptoms such as difficulty with arm abduction and winged scapula [1-4, 6]. In this patient, the right shoulder girdle weakness and MRI findings align with this pattern of nerve involvement.

Treatment & Prognosis

The prognosis in pediatric PTS is generally favorable, but some patients may experience incomplete recovery or recurrent episodes [3]. Most cases resolve within 6 months to 3 years [1-7]. Pain typically improves first, while motor recovery often lags due to ongoing muscle denervation. Reinnervation begins within 6 to 12 months, with proximal muscles recovering earlier due to shorter nerve pathways [1]. However, defining recovery endpoints remains complex. Functional strength may improve through compensation by unaffected muscles, even in the presence of persistent EMG abnormalities and ongoing denervation. Thus, reinnervation may serve as a more accurate indicator of true recovery.

In this case, the patient experienced pain resolution but demonstrated persistent weakness—an expected outcome given the slower course of motor recovery. Without timely intervention, ongoing denervation can lead to muscle contractures or atrophy, emphasizing the importance of early management. In chronic or severe cases without spontaneous recovery, surgical intervention might be considered to restore function [4, 5].

Management of PTS is primarily supportive focusing on pain relief, maintaining mobility, and promoting functional recovery. Pain control, often with analgesics or non-steroidal anti-inflammatory drugs (NSAIDs), is critical in the acute

phase to facilitate physical activity and prevent disuse atrophy. Early initiation of physical therapy helps preserve range of motion, strengthen affected muscles, and prevent contractures. Corticosteroids, such as prednisone, are frequently used to reduce inflammation and potentially expedite recovery, though evidence regarding their efficacy remains limited [2]. In severe cases where weakness persists, surgical interventions, such as nerve transfers, may be considered as part of a multidisciplinary treatment approach.

Differential Diagnoses

Diagnostic imaging plays a critical role in confirming PTS and excluding mimics such as nerve entrapment, rotator cuff injury, cervical radiculopathy, or lymphoma. Imaging of the cervical spine may be warranted to exclude cervical radiculopathy; despite this it is important not to overinterpret cervical spine studies because more than 50% of patients with PTS will have unrelated structural irregularities [4]. Clinical presentation may also play a great role in eliminating mimicking disorders. Muscle atrophy within the first few weeks of presentation is a distinguishing symptom that aids in differentiating this syndrome from tendonitis, capsulitis and radiculopathy [4]. Lymphoma and vasculitis can also mimic PTS but these pathologies tend to present with B symptoms and a distinct peripheral nerve distribution, respectively [4]. These etiologies may also present with generalized reduced reflexes unlike the possible localized deep tendon reflex loss in PTS [4].

TEACHING POINT

Parsonage-Turner Syndrome is a rare but important cause of acute brachial plexopathy characterized by sudden shoulder pain followed by weakness, often triggered by a recent illness. MRI plays a crucial role in diagnosis, typically demonstrating asymmetric nerve root thickening, contrast enhancement of the brachial plexus, and T2/STIR hyperintensity in shoulder girdle muscles consistent with denervation.

QUESTIONS

Question 1: Which of the following is a common MRI finding in Parsonage-Turner Syndrome?

1. Vertebral fracture
2. Nerve root thickening (applies)
3. Pneumothorax
4. Hepatosplenomegaly

Question 2: What is the most common triggering factor for Parsonage-Turner Syndrome in pediatric patients?

1. Trauma
2. Febrile illness (applies)
3. Malignancy
4. Medication side effect

Question 3: Which nerve is most commonly involved in Parsonage-Turner Syndrome?

1. Optic nerve
2. Sciatic nerve
3. Axillary nerve (applies)
4. Median nerve

Question 4: What is the typical progression of symptoms in Parsonage-Turner Syndrome?

1. Weakness followed by pain
2. Fever followed by respiratory failure
3. Pain followed by weakness (applies)
4. Seizures followed by hemiparesis

Question 5: Which of the following is NOT part of the differential diagnosis for brachial plexopathy in this case?

1. Cervical radiculopathy
2. Brachial plexus tumor
3. Rotator cuff injury
4. Myocardial infarction (applies)
5. Lymphoma

Explanation: Myocardial infarction does not cause brachial plexopathy, unlike the other listed options [Differential Diagnoses section].

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FIGURES

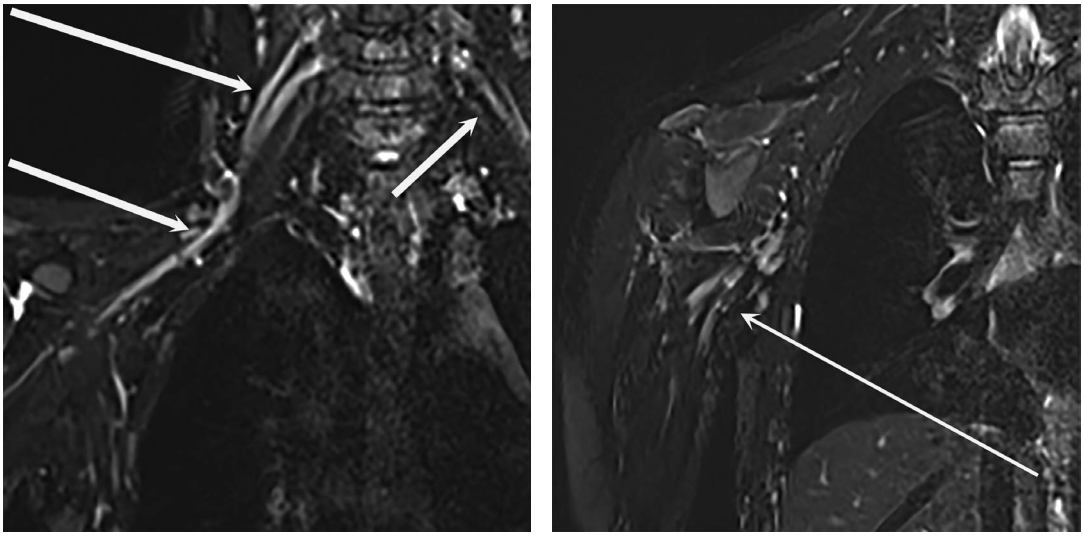

Figure 1a,1b: 4-year-old boy with Parsonage-Turner Syndrome □ Coronal STIR images shows marked enlargement and high STIR signal of the right brachial-plexus nerve roots (arrows) compared with the normal-caliber left side, consistent with acute brachial neuritis □ 3 T MRI, coronal T2 STIR, TR 4917 ms, TE 202 ms, 3-mm slices, no contrast.

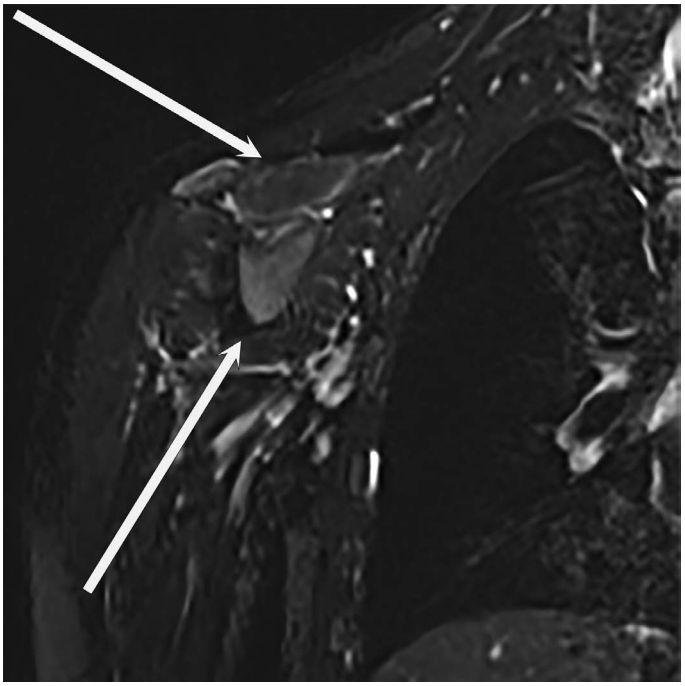

Figure 2: 4-year-old boy with Parsonage-Turner Syndrome □ Coronal STIR image of the shoulder girdle demonstrates diffuse hyperintensity within the right supraspinatus and infraspinatus muscles (asterisks), compatible with acute denervation edema □ 3 T MRI, coronal T2 STIR, TR 4917 ms, TE 202 ms, 3-mm slices, no contrast.

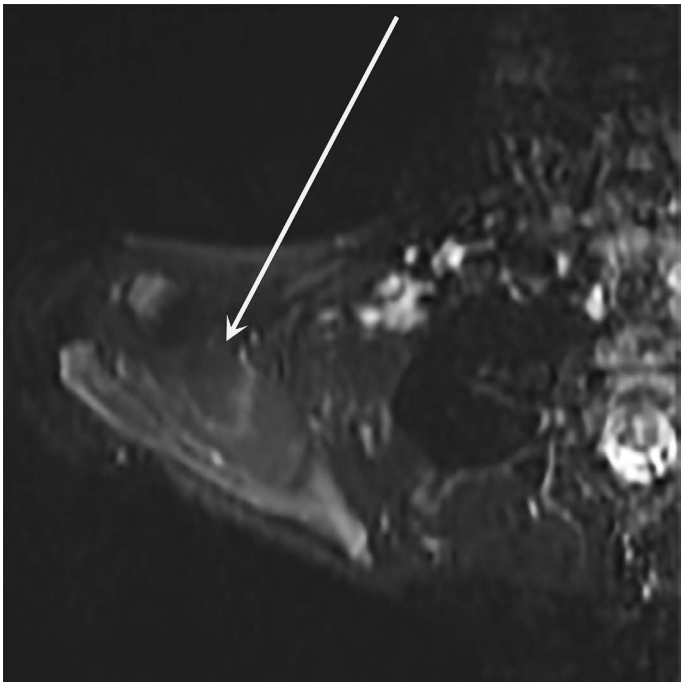

Figure 3: 4-year-old boy with Parsonage-Turner Syndrome □ Axial STIR through the rotator-cuff interval confirms STIR hyperintensity in the supraspinatus and infraspinatus (asterisks) without signal change in the contralateral muscles □ 3 T MRI, axial T2 STIR, TR 4917 ms, TE 202 ms, 3-mm slices, no contrast.

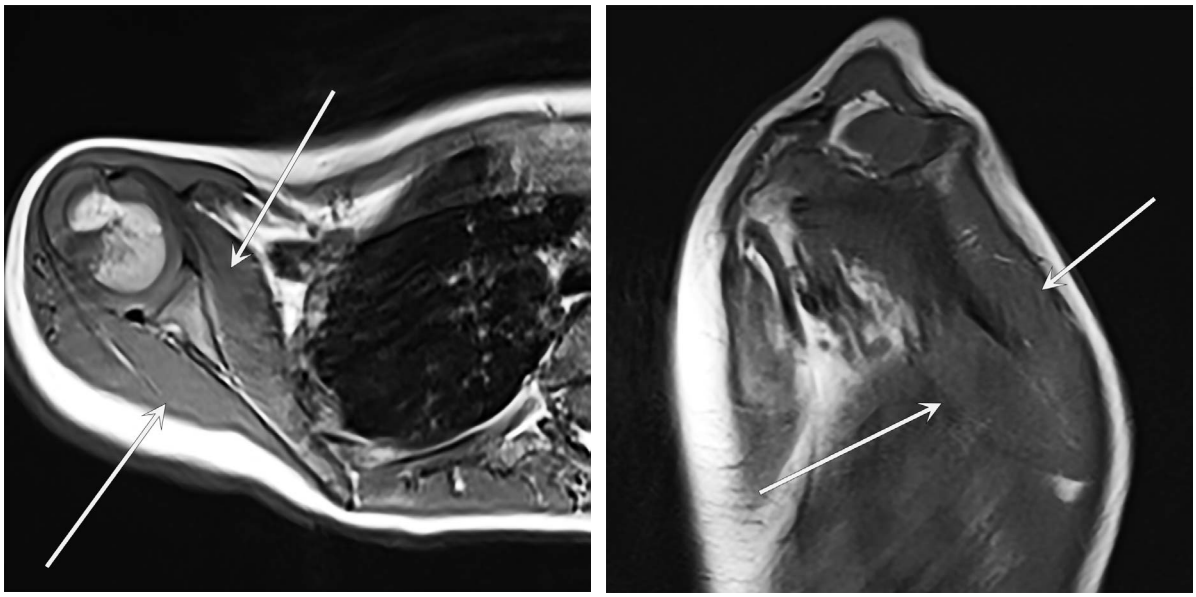

Figure 4 (a,b): 4-year-old boy with Parsonage-Turner Syndrome □
(a) Axial non-fat-sat T1 image of the shoulder shows normal muscle signal without fatty atrophy;
(b) Sagittal T1 Volumetric Interpolated Breath-hold Examination (VIBE) confirms preserved muscle bulk, supporting an acute/subacute denervation process □ 3 T MRI, (a) axial T1 spin echo, TR 700 ms, TE 11 ms; (b) sagittal T1 VIBE, TR 547 ms, TE 3.69 ms; 3-mm slices, no contrast.

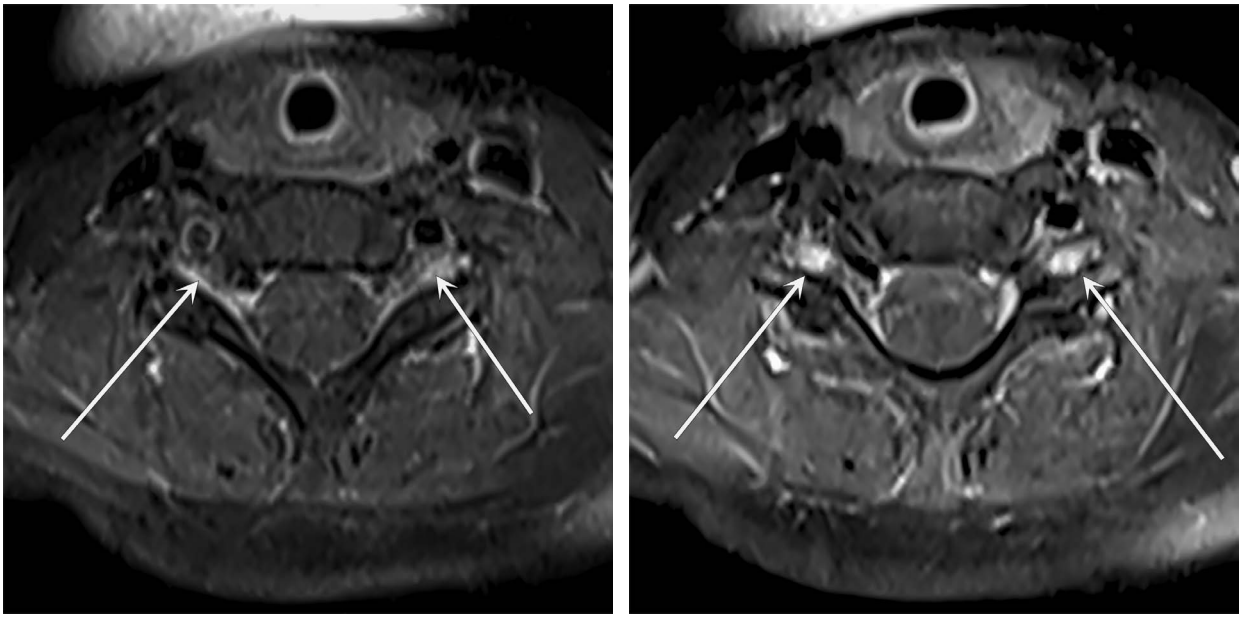

Figure 5a ,5b: 4-year-old boy with Parsonage-Turner Syndrome Axial fat-saturated post-contrast T1 image shows avid enhancement of the bilateral dorsal-root ganglion and exiting nerve roots (arrows), confirming active neuritis 3 T MRI, axial T1 fat-saturated (FS), TR 659 ms, TE 12 ms, 3-mm slices, after 2.5 mL intravenous Dotarem® (gadoterate meglumine, 0.5 mmol mL^{-1}).

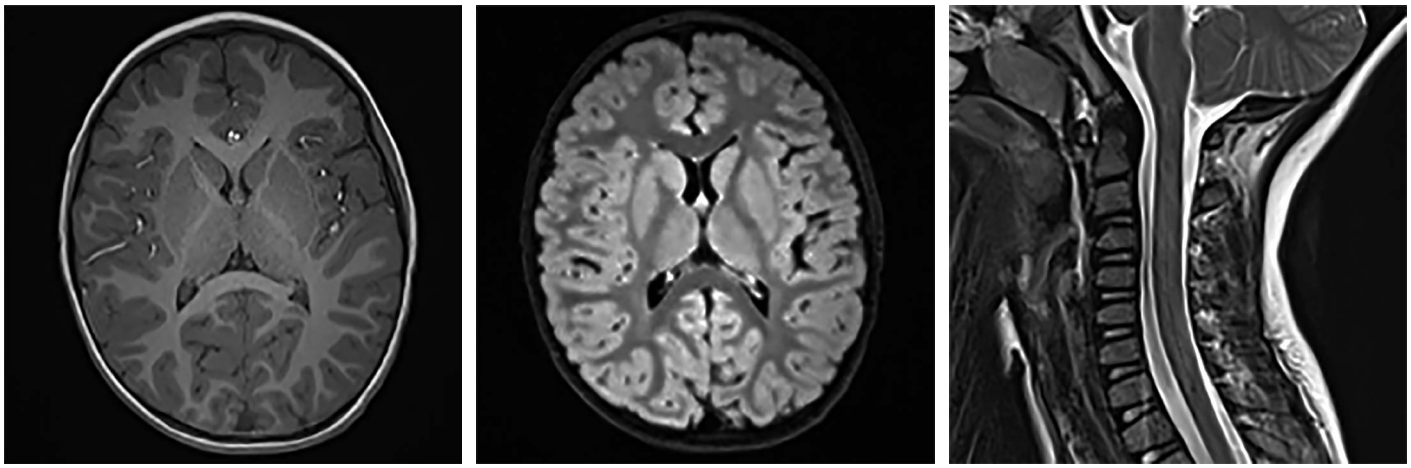

Figure 6a,6b,6c: 4-year-old boy with Parsonage-Turner Syndrome
(A) Axial brain T1 MPRAGE and (B) axial T2 Sampling Perfection with Application-optimized Contrasts using different flip-angle Evolutions (SPACE) demonstrate no intracranial abnormalities;
(C) sagittal cervical-spine T2 TSE shows intact cord and normal vertebral alignment, excluding compressive or demyelinating pathology 3 T MRI, (A) axial T1 MPRAGE, TR 2000 ms, TE 2.49 ms; (B) axial T2 SPACE, TR 7000 ms, TE 380 ms; (C) sagittal T2 TSE, TR 2300 ms, TE 109 ms; 3-mm slices, no contrast.

Table 1: Summary Table

Parameter	Details
Etiology	Immune-mediated, post-infectious, post-vaccination, idiopathic
Incidence	1.64 to 3 per 100,000 annually; extremely rare in pediatrics
Gender Ratio	More common in males (3:1)
Age Predilection	Most common in adults 30-70; pediatric cases rare but reported as young as 3 months
Risk Factors	Recent infection, immunization, genetic predisposition (SEPT9 mutation)
Treatment	Pain management, oral corticosteroids, physical therapy
Prognosis	Pain resolves in weeks; weakness improves over months to years; some may have incomplete recovery
Imaging Findings	MRI: Nerve root thickening, T2/STIR hyperintensity in affected muscles, enhancement of brachial plexus

Table 1: Summary of epidemiology, risk factors, imaging findings and outcomes in pediatric Parsonage-Turner syndrome.

KEYWORDS

Parsonage-Turner; brachial plexopathy; neuralgic amyotrophy; pediatric; MRI; shoulder girdle; denervation

ABBREVIATIONS

CSF = CEREBROSPINAL FLUID
MRI = MAGNETIC RESONANCE IMAGING
PTS = PARSONAGE-TURNER SYNDROME
STIR = SHORT TAU INVERSION RECOVERY
EMG = ELECTROMYOGRAPHY
NSAIDS = NON-STEROIDAL ANTI-INFLAMMATORY DRUGS
FS = FAT-SUPPRESSED
VIBE = VOLUMETRIC INTERPOLATED BREATH-HOLD EXAMINATION.

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