



Current Concept

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Parsonage-Turner syndrome: current perspectives on etiology, diagnosis, and management

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Parsonage-Turner syndrome (PTS) is an underdiagnosed condition that presents with debilitating symptoms in affected patients, with reported incidence rates varying between 1.64 and 3 per 100,000 individuals. Clinical presentation often includes acute shoulder pain associated with neurological deficits that do not follow a regular diagnostic pattern. The cause of this disease is not fully understood, but it is generally triggered by an upper respiratory infection a few weeks prior to presentation. Other associated risk factors include genetic predisposition, vaccines, and surgical intervention. Diagnosis often relies on clinical investigation, holistic physical exam, and appropriate diagnostic studies. Treatment is often conservative, in the form of physical rehabilitation and pain management to avoid muscular atrophy. The condition generally resolves over time, with a recovery rate of 65% at 10 months and a favorable prognosis for most cases. However, recurrences are noted, especially in patients with a genetic predisposition to the condition (75% in the hereditary form vs. 26% in the general form). Future research should explore the pathophysiological processes behind this disease to extrapolate strategies that can achieve an earlier diagnosis and more effective treatment.

Keywords: Brachial plexopathy; Neurologic amyotrophy; Shoulder pain; Upper respiratory infection

INTRODUCTION

Parsonage-Turner syndrome (PTS), also known as idiopathic brachial plexopathy or neurologic amyotrophy, is a rare disorder characterized by acute shoulder pain followed by neurologic symptoms [1]. Parsonage and Turner first described the disease in 1948, after reporting a case series of patients with significant shoulder pain that preceded muscle weakness [2]. The diagnostic complexity of this disorder can lead to delayed recognition and treatment, which can result in significant morbidity.

PTS patients often experience unilateral shoulder pain that can last for a few days to weeks. Progressive neurologic symptoms

follow, such as numbness, motor weakness, and paresthesia typically affecting the proximal upper limb. While the etiology of this condition is largely unknown, it commonly occurs after a precipitating event such as surgery, infection, trauma, or therapy such as vaccination. As such, when patients develop PTS, their symptoms can often be mistakenly attributed to strain from intraoperative positioning, side effects from anesthesia, or even an adverse reaction to vaccine. Comprehensive clinical history, thorough physical examination, and appropriate imaging and testing are required to achieve a correct diagnosis.

Treatment of PTS patients often relies on conservative symptom management. Adequate pain control is essential to prevent

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muscle disuse and atrophy, which can subsequently lead to stiffness. In addition, physical therapy can be prescribed to help re-establish shoulder mobility and joint motion. Given the rarity of this entity and its challenging diagnostic features, knowledge of the clinical, diagnostic, and therapeutic characteristics can be of significant benefit to orthopedic surgeons, particularly shoulder specialists, and patients alike. As such, the purpose of this review is to conduct a focused search of PubMed, Scopus, and Google Scholar using a combination of relevant keywords to explore the clinical findings that characterize PTS and help clinicians better understand how to diagnose this rare condition.

EPIDEMIOLOGY

PTS typically arises in young to middle-aged adults, with a median onset age of 40 years, although cases have been described in children and the elderly [3-6]. From the available studies, the incidence is 1.64 to 3 people per 100,000 in the population every year [5,7]. However, because this disease is often undiagnosed or misdiagnosed, it is hard to determine the true frequency [1], and some studies estimate that the true incidence could be as high as 20-30 per 100,000 [5,8,9]. PTS demonstrates a slight male predominance, with a male-to-female ratio ranging from 2:1 to 3:2 in various studies [3,10]. While the syndrome typically occurs sporadically, familial forms linked to mutations in the SEPTIN9 (SEPT9) gene have been described, following an autosomal dominant inheritance pattern [11]. The syndrome is also known to commonly recur throughout the lifetime [5,6].

Several risk factors have been associated with the development of PTS, including recent infections, vaccinations, surgery, strenuous exercise, and autoimmune conditions. The syndrome has also been reported in association with viral illnesses, particularly following influenza, Epstein-Barr virus, and Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infections. Iatrogenic cases following procedures such as shoulder surgery or regional anesthesia have been documented [12]. PTS is recognized worldwide, with no significant ethnic or geographic predilection. However, due to its often self-limiting nature and variable clinical presentation, true prevalence rates remain difficult to ascertain.

ETIOLOGY AND RISK FACTORS

The etiology of PTS remains incompletely understood, though research suggests a multifactorial origin involving genetic predisposition, immune-mediated mechanisms, and environmental triggers. Studies have linked the condition to preceding infections, vaccinations, and surgical procedures, indicating that an

aberrant immune response contributes to nerve inflammation and damage.

The most common infectious trigger for PTS is an upper respiratory infection, accounting for 20%-52% of cases in one study [13]. Viral microorganisms associated with the development of PTS include cytomegalovirus, coxsackie B virus, Epstein-Barr virus, coronavirus, varicella zoster virus, human immunodeficiency virus (HIV), herpes simplex virus, parvovirus B-19, West Nile virus, and certain hepatitis viruses [14-17]. Hepatitis E Virus, which is present in 10%-15% of patients with acute PTS, has been implicated in a unique phenotype, including elevated liver enzymes, involvement of the phrenic nerve, and a more extensive distribution of impacted nerves [6,15,18,19]. Reported antecedent bacterial triggers include *Borrelia burgdorferi*, *Escherichia coli*, *Staphylococcus aureus*, *Neisseria gonorrhoeae*, *Yersinia enterocolitica*, and *Mycoplasma pneumoniae*, among others [15]. PTS has also been observed after receipt of the coronavirus disease 2019 (COVID-19) vaccine, drugs like nivolumab, and botulinum toxin [19-22].

Additionally, genetic factors, particularly mutations in the SEPT9 gene, have been associated with the disorder, with a hereditary component in approximately 10% of total cases of PTS [15,18,23]. Mutations in SEPT9 on chromosome 17q, which codes for Septin 9, a protein that binds cytoskeletal polymers to microtubules, are inherited in an autosomal dominant fashion [15,18,23]. However, this mutation only accounts for 55% of hereditary PTS, indicating the existence of other genetic predispositions to disease [15]. Hereditary forms of PTS are notable for earlier age at onset, increased recurrence rates, and a more variable distribution of nerves impacted, such as lumbosacral and phrenic [15,18].

Mechanical stress or trauma has also been implicated, as some patients report symptom onset following physical exertion or injury. Types of trauma that have been reported to lead to PTS have ranged from intravenous procedures such as blood draws to exercise to open surgical intervention [1]. It is theorized that, in certain individuals, when nerves of the arm and brachial plexus undergo physical stress and deformation, the blood-nerve barrier is disrupted [15,24]. This allows exposure of peripheral nerve antigens to the immune system, followed by epineural and endoneurial perivascular infiltration of lymphocytes, resulting in ongoing inflammation [6,18]. While these factors offer potential explanations, the exact pathophysiology remains elusive, necessitating further investigation to better define the interplay between immune, genetic, and environmental influences in PTS development.

DIAGNOSIS

Clinical Manifestations

PTS patients typically present with acute onset pain in the shoulder, neck, or proximal arm that lasts for several hours to weeks and is followed by sensory impairment and weakness as the pain subsides [25-27]. The pain is often worse at night and can be exacerbated by movements of the shoulder. Attempts to alleviate the pain with nonsteroidal anti-inflammatory drugs (NSAIDs) or postural changes are often unsuccessful [28]. Unilateral presentation is most common, although bilateral involvement is seen in about one-third of patients [29]. However, even in these unique cases, symptoms tend to be asymmetric, with one side associated with significantly greater severity. The associated pain can take multiple forms, typically starting with sharp and continuous discomfort before transitioning to neuropathic aching.

The ensuing sensory loss and muscle weakness are notable for their occurrence in a patchy distribution that might not align with a single nerve root [27]. Sensory deficits take the form of paresthesia and hypoesthesia, most commonly affecting the skin over the deltoid and lateral arm [26,30]. Muscle weakness affecting the shoulder girdle and upper arm are characteristic. Beyond this typical presentation, there have been reports of additional rare symptoms. Phrenic nerve involvement is uncommon but can result in dyspnea or acute orthopnea if present [25,31]. About 15% of patients experience autonomic dysfunction marked by changes of the arm with respect to temperature, diaphoresis, and skin appearance [13]. Clinical manifestations and diagnostic history features of the disease are summarized in Table 1.

Physical Exam

The patchy quality of sensory and motor symptoms is key to diagnosis. A thorough neurological exam is essential for differentiating PTS from a nerve root pathology, as the former will typically have multiple deficits that are not limited to specific nerve root distributions. On exam, 80% of patients have sensory deficits over the involved extremity, frequently over the lateral aspect of the shoulder [28]. Given that the upper and middle trunks of the brachial plexus are primarily involved, motor deficits tend to affect the suprascapular, long thoracic, axillary, musculocutaneous, and radial nerves, leading to subsequent weakness of the rotator cuff, serratus anterior, deltoid, biceps, and triceps [25,26,32]. Hand and wrist deficiencies can also be seen occasionally, with about one-third of patients experiencing anterior interosseous nerve deficits [33]. Therefore, careful inspection of scapular positioning as well as strength grading of muscles comprising the

shoulder, arm, forearm, and hand are important for diagnosis. Reflexes might also be diminished if there are deficits to the musculocutaneous or radial nerves. Further visual examination of the shoulder can reveal muscle atrophy, an early sign that can appear just weeks after symptom onset [25]. Though it only occurs in about 7% of patients, paradoxical diaphragmatic breathing patterns might be seen if the phrenic nerve is involved [28]. Last, the Valsalva maneuver may be helpful in differentiating from cervical radiculopathy as the pain will be less severe in PTS compared to radiculopathy [34].

Laboratory Findings

Laboratory values can assist with ruling out other differential diagnoses but are not used to confirm the diagnosis of PTS. Cerebrospinal fluid analysis can reveal increased protein and pleocytosis but is usually unremarkable. Complete blood count and erythrocyte sedimentation rate also tend to be within reference ranges. Evidence of elevated inflammatory markers might be found if the presentation was triggered by a recent infection. Histological findings, though not required for diagnosis, have been reported to show epineural perivascular mononuclear infiltrates

Table 1. Clinical manifestations and diagnostic history features of Parsonage-Turner syndrome

Diagnostic feature	Key detail
Onset of pain	Severe, acute Localized in shoulder/neck/proximal arm Lasts hours to weeks
Pain characteristics	Initially sharp and constant later neuropathic eventually aching and dull
Pain behavior	Worse at night Aggravated by movement Poor response to NSAIDs
Neurologic deficits	Sensory loss Muscle weakness occurs after pain subsides
Distribution pattern	Patchy, atypical Does not follow dermatomal pattern
Sensory findings	Paresthesia & hypoesthesia (deltoid region and lateral arm)
Motor findings	Weakness of shoulder girdle and proximal upper arm muscles
Laterality	Usually unilateral Around 1/3 cases are bilateral but severity is asymmetrical
Autonomic symptoms	Present in around 15% of cases Temperature changes, skin color changes, abnormal sweating
Rare features	Phrenic nerve involvement, causing dyspnea or orthopnea

NSAID: nonsteroidal anti-inflammatory drug.

and ischemic type changes, endorsing an immune pathogenesis [1,25]. For the hereditary form of PTS, genetic testing for SEPT9 mutations can be performed to confirm diagnosis [25].

Imaging

Magnetic resonance imaging (MRI) and electromyography (EMG) are the most useful tests for establishing the suspected diagnosis. To optimize evaluation, a dedicated brachial plexus and peripheral nerve protocol can be ordered via a 3.0 T scanner [35]. Early diffuse T2-weighted signal hyperintensity can be seen in the setting of edema and muscle denervation. At a later stage, atrophic changes and fatty infiltration can be evaluated and confirmed by an increased T1-weighted signal (Fig. 1) [36]. A retrospective study of 27 patients found that the muscles innervated by the suprascapular and axillary nerves were most frequently affected in PTS [37]. MRI is also useful in assessing other potential etiologies, such as cervical disc herniation, tears of the rotator cuff or labrum, or impingement [28,38]. Magnetic resonance neurography can identify hourglass-like constrictions in involved nerves, and these have been found in up to 90% of patients with

PTS [39]. High resolution ultrasonography can also be utilized, as affected nerves can demonstrate swelling, constriction, or fascicular entwinement [40]. When performed at least three weeks after symptom onset, EMG findings include the reduction or absence of sensory amplitudes and muscle action potentials [34]. Fibrillation potentials and positive sharp waves can also be seen, but conduction velocities usually remain normal [41]. Last, a shoulder radiograph should be ordered to investigate alternative causes of the pain, such as calcific tendinitis or other trauma to the joint. Chest x-ray is also worthwhile to rule out a Pancoast tumor responsible for compression of the brachial plexus [38].

Differential Diagnosis

As previously mentioned, there is a variety of differential diagnoses that must be considered and evaluated when a patient is presenting with a clinical picture suspicious for PTS. Cervical radiculopathy from herniation, stenosis, or lesion is the most common diagnosis to consider with these symptoms but can often be differentiated by a positive Spurling test and sensory changes that exist in a more specific dermatomal distribution, both of which

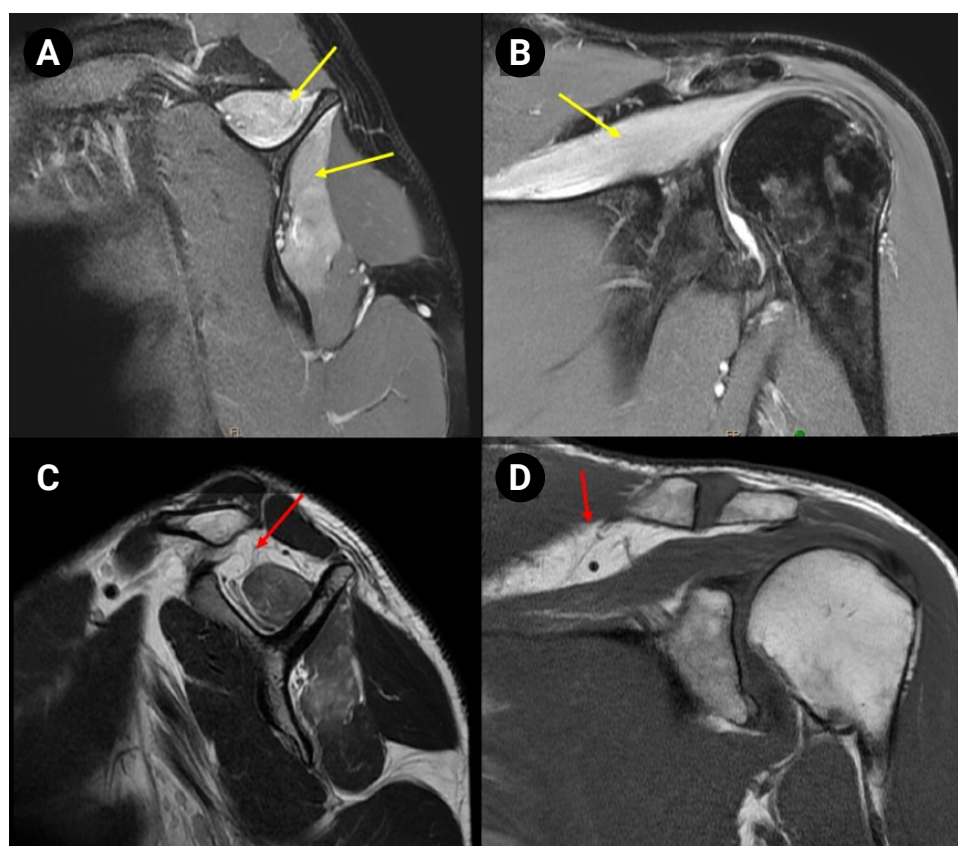


Fig. 1. Magnetic resonance imaging of a 45-year-old male with Parsonage-Turner syndrome. (A) Sagittal and (B) coronal views show T2-weighted signal hyperintensity (yellow arrows) in the supraspinatus and infraspinatus due to muscular edema and inflammation. (C) Sagittal and (D) coronal views show signs of fatty atrophy (red arrows) at the level of the supraspinatus.

will likely be absent in PTS [1]. Other less common neurologic conditions with overlapping features include brachial plexus lesions, transverse myelitis, mononeuritis multiplex, and herpes zoster [26]. Additionally, inflammatory conditions of the shoulder joint such as adhesive capsulitis, subacromial bursitis, and calcific tendonitis can also present with abrupt-onset severe shoulder pain and should be considered. While less likely, it is also important to evaluate for pathologies of the thoracic region that can present with severe referred pain to the shoulder, as in the case of myocardial infarction or Pancoast tumor. Table 2 provides a summary of common differential diagnoses related to PTS.

TREATMENT

Primary management is centered around pain control. A study of 246 patients found that the most effective regimen for pain was a combination of an NSAID and an opiate, such as diclofenac with morphine, though NSAIDs and acetaminophen alone also resulted in some relief [13]. The use of corticosteroids has shown mixed results, as some studies reported improvement in pain reduction and decreased duration of both pain as well as the ensuing paresis [42,43], while others noted an increased risk of future pain flares [13]. So far, the optimal timing, dosage, and duration of corticosteroid therapy are unclear.

Physical therapy and rehabilitation are important for helping patients reestablish fluent motions of the upper extremity and improving the residual strength of affected muscles [28]. Beyond conventional strengthening protocols, targeted physical therapy should focus on motor-control retraining, scapular stabilization, postural correction, and progressive neuromuscular re-education, which has been shown to improve arm function and daily activity scores [15,44,45]. Long term data in the literature have

suggested reduced residual pain and functional limitations with structured rehabilitation programs [46].

In rare instances, when neuromuscular deficits are persistent despite optimized conservative therapy, surgical intervention can be utilized. Imaging studies can reveal hourglass-like constrictions of affected nerves, and these can be amenable to microsurgical procedures, like neurolysis [47,48]. Krishnan et al. [48] reported 24 patients who were randomized to either microsurgical neurolysis or nonsurgical treatment for chronic motor palsy from PTS and noted significant clinical improvement and nerve regeneration in the surgical group compared to the non-surgical group. Other procedures like nerve transfers, nerve grafting, or tendon transfers can also be considered to restore function in chronic cases with irreversible denervation [49,50].

PROGNOSIS

The prognosis of PTS varies significantly among affected individuals, with factors such as severity of nerve involvement, timeliness of diagnosis, and intervention strategies influencing recovery outcomes. The literature describes a latency period of 4–6 weeks from the antecedent trigger to symptomatic onset, although 67% of patients in one study felt pain in the first week (Fig. 2) [6,13,15,18,19,23,24,51]. The natural course of PTS typically follows a biphasic progression, with an initial phase of acute “sharp” neuropathic pain lasting days to weeks, followed by a transient “aching” pain as well as progressive weakness and muscle wasting (Fig. 2) [25]. Studies indicate that prognosis is generally favorable; however, with the majority of patients experiencing substantial improvement over time. In a longitudinal analysis, approximately 65% of patients achieved significant muscle

Table 2. Differential diagnoses of Parsonage-Turner syndrome

Differential diagnosis	Distinguishing clinical features	Key diagnostic findings
Cervical radiculopathy	- Pain radiating to the arm in a dermatomal pattern - Exacerbated by neck movement	- Positive Spurling test - Dermatomal sensory loss - Disc herniation or foraminal stenosis on MRI
Brachial plexus lesion	- History of trauma, surgery, or mass lesion - Persistent or progressive weakness	- Focal plexus injury or mass on MRI or MR neurography - EMG findings corresponding to affected trunks or cords
Adhesive capsulitis/rotator cuff pathology	- Localized shoulder pain - Restricted range of motion - Weakness limited by pain, not denervation	- Capsular thickening or tendon pathology on shoulder MRI or ultrasound - Normal EMG
Herpes zoster (zoster sine herpete)	- Burning or stabbing pain in a dermatomal distribution - Often presents with a rash	- Positive VZV PCR or serology - Strict dermatomal pain distribution

MRI: magnetic resonance imaging, EMG: electromyography, VZV: varicella zoster virus, PCR: polymerase chain reaction.

strength recovery—defined as an improvement to a Medical Research Council grade of 4 or higher—within an average of 10 months following symptom onset [13]. However, full recovery remains challenging for many, with one study showing only 44% of individuals regaining complete pre-morbid function (Fig. 2) [10]. This suggests that, while functional restoration is common, a considerable subset of patients might experience persistent deficits such as muscle weakness, discomfort, and diminished exercise tolerance (Fig. 2) [10].

The severity of a patient's initial symptoms, probability of muscle fiber reinnervation, and early interventions are also key determinants of disease course [6,13,15,18,19,23,24,51]. Individuals presenting with extensive nerve involvement and pronounced weakness tend to have a protracted recovery and higher likelihood of incomplete resolution [6]. Based on rates of axonal advancement and growth, incomplete lesions with short distances for reinnervation have a more favorable prognosis [52]. Lesions that involve distances greater than 20 inches from the denervated muscle fibers or that are complete have a poor prognosis [52]. Moreover, a delayed diagnosis can contribute to worse outcomes, as timely intervention is critical in mitigating pain and preventing secondary complications such as joint stiffness and disuse atrophy [1]. If untreated, PTS can have a debilitating disease course, with one study reporting that 25% of patients were unable to work at 3 years [18]. Corticosteroids, when administered early, have been shown to reduce pain severity and accelerate muscle strength recovery, although their long-term benefits remain a topic of debate [53]. Additionally, structured physical therapy plays a pivotal role in rehabilitation, particularly in preserving joint mobility, maintaining residual strength, and preventing compensatory musculoskeletal dysfunctions [26].

The long-term outcomes of PTS are highly variable. While

many individuals achieve meaningful functional recovery, some continue to experience residual symptoms such as chronic pain, fatigability, and weakness, which can impact quality of life [10]. For example, individuals with involvement of the phrenic nerve can have a protracted recovery of up to 4 years [6]. Furthermore, recurrence is a recognized complication, with estimates suggesting that up to 26% of patients may develop recurrent episodes in either the same or contralateral limb [6]. This rate can be as high as 75% in individuals with the hereditary form of PTS [13,15]. This underscores the importance of long-term follow-up and patient education regarding potential relapse and symptom management strategies. Early diagnosis, targeted pain management, and comprehensive rehabilitation programs are essential in optimizing outcomes and enhancing the quality of life for those affected by this condition.

FUTURE DIRECTIONS

Despite the current advancements in understanding PTS, there remain major gaps in achieving early diagnosis, providing accurate prognostication, and administering individualized treatment. Future studies should focus on identifying biomarkers specific to initial inflammatory activity to help achieve an earlier diagnosis. Moreover, continuing to upgrade advanced imaging modalities can help improve prognostication and better assess the therapeutic direction required for optimizing patient outcomes. Randomized controlled trials exploring the different treatment modalities available can help fine tune treatment guidelines and algorithms, while can improve functional outcomes and recovery. Finally, the establishment of a large multi-center prospective registry with comprehensive patient data can help identify new pathological patterns and develop predictive models for treatment and recovery.

CONCLUSIONS

PTS remains an underrecognized condition with debilitating effects. The disease often presents with acute shoulder pain accompanied by variable sensory and motor neurological deficits that can mimic other entities, leading to delayed diagnosis and treatment. The etiology behind this disease is not completely understood, but one of the most commonly encountered triggers is a recent upper respiratory infection prior to presentation. Other risk factors include surgical interventions, vaccinations, and genetic predispositions, among others. A correct diagnosis often depends on a comprehensive clinical investigation, detailed physical examination, and appropriate diagnostic imaging – particu-

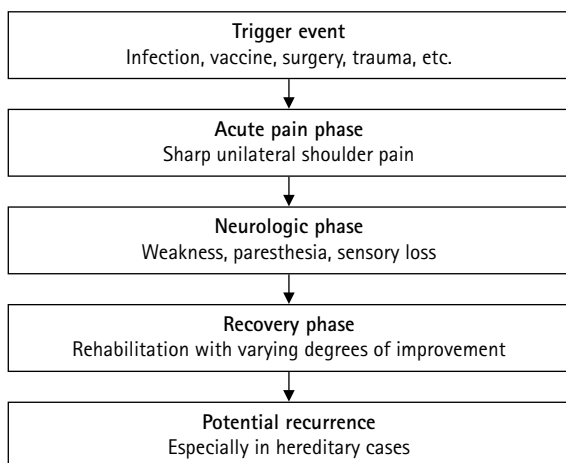


Fig. 2. The clinical progression of Parsonage-Turner syndrome.

larly, MRI and EMG. Treatment of PTS is mainly conservative and focuses on pain control and physical rehabilitation to avoid stiffness and disuse atrophy. Surgical intervention can be helpful in rare cases with persistent and irreversible neuromuscular deficits. Prognosis is often favorable, with most patients regaining significant function over time. However, recurrences are not uncommon, especially in cases with a hereditary pattern. Future research should focus on properly understanding the pathologic processes that govern PTS to extrapolate and deduce effective diagnostic and treatment modalities.

NOTES

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Conflict of interest

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Data availability

Contact the corresponding author for data availability.

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