

Challenging Pain Syndromes

Parsonage-Turner Syndrome

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KEYWORDS

- Parsonage-Turner syndrome • Neuralgic amyotrophy • Brachial plexus
- Acute brachial plexitis

KEY POINTS

- The current best approach to Parsonage-Turner syndrome (PTS) is a multidisciplinary approach that includes both physical therapy and pharmacologic treatment, often with multiple agents.
- Corticosteroid treatment may improve pain and hasten recovery.
- Surgical options are available for patients who fail conservative treatment.
- Due to the relatively low incidence of this disorder, further research is needed.

DEFINITION

Parsonage-Turner syndrome (PTS) is a rare disorder typically characterized by an abrupt onset of upper extremity pain followed by progressive neurologic deficits, including weakness, atrophy, and occasionally sensory abnormalities. The cause is unknown. The distribution of the nerves involved as well as the extent of involvement is variable. Any peripheral nerve may be affected but most commonly the upper trunk of the brachial plexus is involved.^{1,2} Recovery is often prolonged and incomplete. A hereditary form of the syndrome, hereditary neuralgic amyotrophy, has also been studied, although it occurs much less frequently. Clinically it presents similarly to PTS, but often at a younger age and has a higher incidence of recurrent attacks.³⁻⁵

Although this clinical condition is most commonly referred to as Parsonage-Turner syndrome, neuralgic amyotrophy, or brachial neuritis, it can be found described in literature under many other names (**Box 1**).⁴⁻⁶

HISTORY

One of the first descriptions of PTS dates back to 1887 when Julius Dreschfeld described 2 cases of recurrent episodes of nontraumatic brachial plexopathy.^{5,6}

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Box 1**Alternative names for PTS**

Acute brachial neuropathy
 Acute brachial plexitis
 Acute multiple brachial neuropathy
 Brachial neuritis
 Brachial plexus neuropathy
 Cryptogenic brachial plexus neuropathy
 Idiopathic brachial neuritis
 Idiopathic brachial plexopathy
 Kiloh-Nevin syndrome
 Localized neuritis of the shoulder girdle
 Multiple neuritis of the shoulder girdle
 Neuralgic amyotrophy
 Paralytic brachial neuritis
 Shoulder girdle neuritis
 Shoulder girdle syndrome

Over the next 60 years, there were several case reports describing similar clinical presentations, but it was not until 1943 that Spillane gave the first full description of the condition in his article, “Localised neuritis of the shoulder girdle.”^{7–9} Spillane described 46 patients with an “acute onset of pain in the shoulder, arm, and side of the neck, over the scapula and down the affected arm” persisting for 7 to 10 days. Several days later, usually after the pain subsided, the patients developed paralysis around the shoulder girdle.¹⁰ In 1948, M.J. Parsonage and John W. Alden Turner published an article titled, “Neuralgic amyotrophy: the shoulder girdle syndrome,” which more firmly established and detailed the clinical aspects of the syndrome.¹¹ This article described a case series of 136 patients who experienced a sudden onset of pain across the shoulder blade lasting from a few hours to 2 weeks, followed by paralysis involving muscles of the shoulder girdle and in some cases patchy numbness along the lateral aspect of the upper arm. In 98 of the cases there was thought to be some precipitating factor, such as surgery, trauma, infection, lumbar puncture, air encephalogram, or antisiphilitic treatment.¹¹

CAUSE AND PATHOPHYSIOLOGY

The exact cause and pathophysiology of PTS are complex and incompletely understood. Autoimmune, genetic, infectious, and mechanical processes have all been implicated.¹² Of the many proposed causes, an infectious or immune-mediated process seems to be the most supported due to the high incidence of preceding infections and immunizations.^{2,5,6} An antecedent event has been identified in 30% to 70% of PTS cases.^{1,11,13,14} It is theorized that an event may trigger an immune-mediated response that incites the development of PTS. In 20% to 52% of cases, infection precedes the development of PTS.^{1,11,14} Approximately 15% of cases occur after immunization.^{11,14}

PTS has been associated with several surgical procedures, including coronary artery bypass surgery, oral surgery, hysterectomy, and a variety of orthopedic

surgeries.^{2,15} Although in surgeries involving the chest or upper extremity, plexopathy may develop from a traction injury because of improper positioning or prolonged pressure over the nerve. However, PTS has been observed following surgical procedures that involve little to no traction on the brachial plexus or surrounding nerves. It may be difficult to distinguish a traction injury from PTS. What distinguishes PTS is nerve involvement without common innervation root, brachial plexus trunk, or cord distribution and muscles with common innervation may be unaffected.² Other less frequently observed antecedent events are listed in **Box 2**.^{1,2,8,9,11,14–16}

Histologic studies have demonstrated evidence of an immune-mediated process. Suarez and colleagues¹⁷ showed the presence of inflammatory cells, particularly T-lymphocytes, within the brachial plexus of a small sample of patients with PTS. In addition, complement fixing antibodies to peripheral nerve myelin have been found elevated in the acute phase of PTS, and levels tended to decrease during the recovery phase.¹⁸

van Alfen¹² suggests that the pathophysiology of the condition involves an interaction between a genetic predisposition, mechanical vulnerability, and an autoimmune trigger. No mutations have been found in patients with idiopathic neuralgic amyotrophy but studies have shown mutations of the septin gene in some families with the hereditary autosomal-dominant form. The septin family of genes is highly expressed in glial cells in neuronal tissue.⁴ In some cases PTS is preceded by increased physical exertion, suggesting that biomechanical factors may also play a role in triggering the attack.¹⁴

EPIDEMIOLOGY

One study suggests that the annual incidence of PTS is 1.64 per 100,000 in the United States.¹⁹ The actual incidence may be higher given the difficulty of recognition. It is described in patients ranging from age 3 months¹⁴ to 81 years¹⁶ but occurs most frequently between the third and seventh decades.¹⁴ PTS occurs more frequently in men than women. Studies have shown the ratio of male-to-female involvement ranges from 1.75:1 to 11.5:1.¹³ The hereditary form is thought to occur 10 times less frequently than the idiopathic form and typically occurs in the second decade of life.^{1,3}

Box 2

Reported antecedent events and associated illnesses

Viral infection—upper respiratory tract infection, flulike syndrome, hepatitis, mononucleosis, malaria, pneumonia, abscess, Typhus, small pox, rheumatic fever, typhoid, poliomyelitis

Immunizations—tetanus toxoid, influenza, horse tetanus antitoxin, DPT

Strenuous exercise

Trauma—fall on shoulder, gunshot wound

Peri-partum

Perioperative—orthopedic, hernia surgery, appendectomy, pilonidal cyst excision, varicocele repair, coronary artery bypass surgery, hysterectomy, tonsillectomy

Medical procedures—lumbar puncture, encephalogram, antisyphilitic treatment

Autoimmune—rheumatoid arthritis, polyarteritis, temporal arteritis

Psychological stress

Data from Refs. ^{1,2,8,9,11,14–16}

PATIENT EVALUATION OVERVIEW

The classic presentation of PTS begins with acute onset of severe pain lasting for several days to weeks, followed by the development of weakness and muscle atrophy. The presentation is highly variable and may present with a wide range of symptoms, including pain, paresthesias, and sensory disturbances. Patients typically do not have constitutional symptoms such as fever or malaise.¹¹

Pain is the predominant presenting symptom in 90% to 95% of patients.^{1,14} The pain is usually worse at night and frequently awakens patients from sleep. It is often described as “constant,” “sharp,” “stabbing,” “throbbing,” or “aching” and in some cases is associated with muscle tenderness.¹⁴ Characteristically the pain is located in the shoulder and often radiates into the arm or neck. The pain is commonly aggravated by movement of the shoulder but not typically by movement of the neck or Valsalva maneuvers.^{20,21} In rare cases, symptoms are localized outside the brachial plexus and in some cases in the lower extremity. The pain is usually unilateral, on the same side in which the weakness develops. Some patients experience bilateral pain followed by unilateral motor symptoms.^{4,14} Although the pain on average, lasts 1 to 2 weeks and then subsides,² the duration of pain is highly variable, ranging from several hours to months.^{1,14} After the acute episode, a portion of patients develops a lingering neuropathic pain in the distribution of the affected nerve and or musculoskeletal pain in the affected muscles or compensating muscles.¹²

Muscle weakness typically begins to develop days to weeks after the onset of pain and often worsens as the pain subsides.^{1,14} In a study of 99 patients, weakness began within the first 2 weeks after the onset of pain in 70% of the cases.¹⁴ The location of the initial pain does not necessarily correlate to the distribution of muscle weakness.²¹ It may be difficult to ascertain the exact onset of weakness because pain may limit the patient’s use of the extremity. In addition, weakness may be hard to recognize if the muscles involved are hard to isolate manually and test.² Atrophy of the involved muscles usually occurs relatively quickly.²¹

A characteristic feature of PTS is the patchy distribution of motor and sensory symptoms. Muscles innervated by one peripheral nerve, multiple peripheral nerves, one or more trunks of the plexus, or a combination of peripheral nerves and trunks may be affected.^{13,21} The most common pattern of weakness involves muscles supplied by the upper part of the brachial plexus, especially the long thoracic nerve. Muscles most commonly affected include the deltoid, supraspinatus, infraspinatus, serratus anterior, biceps, and triceps.⁵ Muscles that have been reported to be affected are listed in **Box 3**.

Individual nerves in isolation or in multiple nerve distributions may be affected. Isolated involvement of the radial, long thoracic, suprascapular, axillary, median, and anterior interosseous nerves are most commonly described.⁵ Less frequently, nerves remote from the brachial plexus are involved, including the lumbosacral plexus, phrenic nerve, lower cranial nerves, and recurrent laryngeal nerve.^{1,6} Involvement of nerves outside the brachial plexus is seen more frequently in the hereditary form of the syndrome, hereditary neuralgic amyotrophy.²³ Nerves that have been reported to be affected are listed in **Box 4**.

Bilateral brachial plexus involvement occurs approximately 30% of the time. In most cases, one side is more affected than the other.^{1,6} In bilateral cases pain and weakness may begin on both sides at the same time or weeks may elapse before involvement of the second side.¹³ Involvement of the clinically unaffected side may be found more often on needle electromyography (EMG) than on clinical examination.⁵

Box 3 Muscles reported affected in PTS
<i>Commonly affected muscles</i>
Infraspinatus
Supraspinatus
Deltoid
Serratus anterior
Biceps
Triceps
Rhomboids
<i>Less commonly affected muscles</i>
Extensor carpi radialis and ulnaris
Pronator teres
Brachioradialis
Trapezius (lower)
Latissimus dorsi
Pectoralis
Flexor carpi radialis and ulnaris
Teres major
Finger extensors
Pronator quadratus
Flexor digitorum profundus
Dorsal interosseus
Adductor pollicis
Extensor pollicis longus
Supinator
Sternocleidomastoid
Paraspinal neck extensors
<i>Data from Refs. 1,11,13,14,22</i>

Sensory symptoms are variable and occur in anywhere from 42% to 78% of cases.^{1,11,13,14} Hypoesthesia and paraesthesias are the most commonly described sensory symptoms.¹ As with weakness, the sensory loss is often patchy in distribution and often corresponds to the sites of plexus or nerve involvement.^{5,21} Most frequently, the sensory loss is incomplete and occurs over the lateral shoulder and upper arm or the radial surface of the forearm.^{5,20,21}

Autonomic symptoms have also been described, although less frequently. These changes include trophic skin changes, edema in the involved extremity, temperature dysregulation, changes in nail or hair growth, and increased sweating. Autonomic findings occur more commonly in patients with involvement of the posterior cord and lower trunk of the brachial plexus than those with upper trunk involvement.^{1,6}

Box 4**Nerves reported to be affected in PTS***Commonly affected nerves*

Axillary nerve

Suprascapular nerve

Long thoracic nerve

Musculocutaneous nerve

Radial nerve

Anterior interosseous nerve

Less commonly affected nerves

Median nerve

Subscapular nerve

Phrenic nerve

Recurrent laryngeal nerve

Spinal accessory nerve

Glossopharyngeal

Hypoglossal nerve

Lateral antebrachial cutaneous

*Data from Refs. 1,2,5,6,16***DIFFERENTIAL DIAGNOSIS**

The diagnosis of PTS is difficult to make, especially in the early stages. PTS may present similarly to other, more common, neurologic and musculoskeletal conditions that cause pain and weakness around the shoulder (**Box 5**). Acute cervical radiculopathies and PTS may both present with a sudden onset of severe pain, although unlike PTS, pain secondary to a cervical radiculopathy may be exacerbated by extension of the neck. In addition, in a cervical radiculopathy, sensory and motor symptoms are usually distributed along a single nerve root. Differentiating between a cervical radiculopathy and PTS may be complicated by the fact that the referred radicular pattern of pain from a cervical radiculopathy, the dynatome, is often different than the traditional dermatomal map.²⁴ Peripheral nerve entrapments may present similarly to PTS; however, the onset of symptoms is generally more insidious and the pain is less severe. Similarly, mononeuritis multiplex has a more progressive onset and typically involves the distal arm and leg, unlike PTS.²³ Many patients with PTS are initially misdiagnosed with shoulder joint pathologic abnormality. In the acute stage, patients with PTS usually have full passive shoulder range of motion, making intrinsic shoulder joint pathology less likely.⁴ Testing passive shoulder range of motion may be difficult in patients with PTS secondary to guarding. To make an accurate diagnosis, it is critical to complete a thorough neurologic and musculoskeletal examination of both the symptomatic and the asymptomatic extremities, including testing manual muscle strength, range of motion, sensation, and reflexes as well as evaluating the shoulder for signs of impingement, adhesive capsulitis, rotator cuff injury, and scapular dyskinesis. It may be challenging to manually test individual muscles surrounding the shoulder. When the scapula is not stabilized, rotator cuff muscles may appear weak even

Box 5**Differential diagnosis for PTS***Neurologic disorders*

Cervical radiculopathy

Mononeuritis multiplex

Multifocal motor neuropathy

Motor neuron disease

Entrapment neuropathies

Transverse myelitis

Complex regional pain syndrome

Brachial plexopathy secondary to trauma, traction, infiltration (ie, Pancoast tumor)

Tumors of the spinal cord or brachial plexus

Herpes zoster

Hereditary neuropathy with liability to pressure palsies

Neurogenic thoracic outlet syndrome

Musculoskeletal disorders

Rotator cuff injury

Calcific tendinitis

Adhesive capsulitis

Bursitis

Shoulder impingement

Myofascial pain syndrome

Neck disorders, such as osteoarthritis, stenosis, facet

when there is no intrinsic weakness. Proper positioning during physical examination helps prevent misdiagnosing rotator cuff weakness.²⁵ Many of the conditions in the differential diagnosis can be eliminated with the classic history of acute severe pain that decreases spontaneously with the onset of weakness.

DIAGNOSTIC STUDIES***EMG/Nerve Conduction Study***

PTS is a clinical diagnosis; however, further diagnostic studies can confirm clinical suspicion and help exclude other causes. Electrodiagnostic studies are particularly useful in localizing the lesion, confirming the diagnosis, and determining the extent of injury. Early EMG testing may be nondiagnostic and therefore should not be performed before 4 to 6 weeks after the onset of symptoms. PTS is thought to be an axonopathy, although findings may vary.^{2,14,21} Routinely tested nerve conduction studies are often normal, although delayed distal latencies, decreased compound muscle action potentials, decreased sensory nerve action potentials, and proximal conduction blocks have all been observed.^{2,4,20} When sensory abnormalities are present, the lateral antebrachial cutaneous nerve is one of the most frequently affected and should be tested. The diagnosis relies on the needle EMG.² Because of the often patchy distribution of involvement, it may be necessary to test a variety of muscles, including

muscles that are not routinely tested. EMG performed 3 to 4 weeks after the onset of symptoms shows evidence of acute denervation (fibrillations and positive sharp waves) and often a reduction in motor unit recruitment in a nonmyotomal pattern. There is selective denervation of muscles of different root levels and different peripheral nerves distributions. Typically the paraspinal muscles are not affected.²¹ EMG studies 4 to 12 months after the onset of symptoms may show old denervation and reinnervation with polyphasic motor unit potentials.² Typical electrodiagnostic findings are summarized in [Table 1](#).

Imaging

Radiographic imaging may be useful in excluding other disorders. A chest radiograph can help rule out a Pancoast tumor if there is a clinical suspicion and will detect an elevated hemidiaphragm caused by involvement of the phrenic nerve.²³ A magnetic resonance imaging (MRI) of the cervical spine may reveal cervical disc disease or nerve root compression. An MRI of the shoulder may identify other causes of shoulder pain, including rotator cuff tears, labral tears, shoulder impingement, nerve entrapment, or mass lesions. Abnormalities signifying denervation may be detected on MRI in cases of PTS.²⁰ MRI findings most characteristic of PTS include diffuse high signal intensity on T2-weighted images, involving one or more muscles innervated by the brachial plexus.^{16,26} Later in the subacute phase, T1-weighted images may demonstrate atrophy and fatty infiltration.¹⁶ MRI may not be sensitive enough to detect early (within the first 2–3 weeks) changes in PTS, although magnetic resonance neurography (MRN) may provide better resolution early on, demonstrating hyperintense thickening of the involved areas of the brachial plexus.²⁷ MRN, however, is currently not readily available at most institutions.

Other Studies

Laboratory studies are generally not helpful in making the diagnosis of PTS. Blood tests are nonspecific and can show elevated liver function levels, mildly elevated creatine kinase, antiganglioside antibodies, and slightly increased cerebrospinal fluid protein and pleocytosis in up to 25% of patients.¹ As mentioned previously, the diagnosis of PTS is ultimately based on clinical history and physical examination.

Pharmacologic Treatment Options

No specific treatments have been proven to reduce neurologic impairment or improve the prognosis of PTS. A *Cochrane Review* concluded that no available studies provided appropriate evidence for a particular form of treatment.¹ There is anecdotal

Table 1	
Electrodiagnostic findings	
Nerve conduction studies	May show reduced amplitude, prolonged distal latency, or conduction block Often are normal
Early EMG findings (3–4 wk after symptom onset)	Acute denervation: Fibrillation potentials, positive sharp waves Reduced motor unit recruitment in muscles innervated by different root levels and peripheral nerves
Late EMG findings (3–6 mo after symptom onset)	Old denervation, early reinnervation: Large duration, large amplitude polyphasic motor units, decreased recruitment

evidence that early corticosteroid therapy may improve pain and hasten recovery in some patients.³ In one study, several patients treated with a 2-week course of oral prednisolone 60 mg in the first week and tapered to 10 mg per day in the second week noted a faster rate of recovery.¹ van Alfen and van Engelen¹ suggests early treatment with oral prednisone 1 mg/kg for the first week and tapering during the second week. Two case reports of PTS showed partial benefit from treatment with intravenous immunoglobulin; however, it has not been reproduced in any larger studies.^{28,29}

Severe pain in the acute stage of PTS is often treated with a combination of nonsteroidal anti-inflammatory medications (NSAIDs) and opioids. Antiepileptic medications, such as gabapentin and carbamazepine, and tricyclic antidepressants, such as amitriptyline, are often used to treat the lingering neuropathic pain that often persists after the acute painful attack. Antiepileptic medications are not as effective at treating the acute severe pain of PTS because of their delayed onset.⁴ Patients with a history of viral infection or when post-herpetic neuralgia is suspected should be treated with antivirals.^{2,4} Strategies used to treat PTS may mimic those used to treat other neuropathic pain conditions such as post-herpetic neuralgia. The American Academy of Neurology recommends gabapentin, lidocaine patch 5%, pregabalin, tricyclic antidepressants, controlled release oxycodone, and morphine sulfate as first-line therapy for the neuropathic pain of post-herpetic neuralgia.³⁰ A recent review of randomized clinical trials on neuropathic pain outlined an evidence-based algorithm for the treatment of peripheral neuropathic pain. The algorithm suggests that tricyclic antidepressants (amitriptyline, nortriptyline, desipramine, imipramine), opioids (CR oxycodone, methadone, morphine), tramadol, gabapentin, and pregabalin are all beneficial in providing pain relief.³¹

Nonpharmacologic Treatment Options

Because PTS is a relatively underrecognized disorder, extensive studies on treatment options have not been performed. In fact, the best treatment of PTS is unknown.⁶ Options for nonpharmacologic treatment are mostly based on small, nonrandomized, nonblinded studies and anecdotal evidence. Currently, nonpharmacologic treatments used in the treatment of PTS include physical therapy, osteopathic manipulation, therapeutic modalities, and acupuncture. Despite a proposed role of physical therapy for preventing loss of range of motion and further disability, physical therapy does not seem to speed up recovery.³

Goals of physical therapy should include maintenance of range of motion and prevention of loss of function. Depending on the level of pain, range-of-motion exercises for the shoulder may be started immediately. The goal of range-of-motion exercises includes preventing secondary loss of function due to conditions such as contractures or adhesive capsulitis.^{2,5} Physical therapy may have a role in pain control. Desensitization exercises may improve allodynia if present. There may also be strategies to reduce traction on involved nerves.⁴

The timing of strengthening exercises depends on several key factors, including level of pain, amount of weakness, and degree of denervation.²² Most authors advise a cautious approach toward strengthening exercises in physical therapy because an overly aggressive approach can overload muscles that are weak or in the early reinnervated stage.² For this reason, authors advise patients to avoid strength training outside of physical therapy sessions during their recovery. Treatment of this condition in high-level athletes should follow the same basic principles. Intensive sport-specific biomechanical training is important before returning high-level athletes to sport. This principle is particularly true in sports that involve throwing, overhead movements (such as basketball, baseball, or tennis), or sports that otherwise involve the shoulder.²

The timing of starting biomechanic training depends on the patient's recovery of motor function. Strength training should not be started until significant weakness has recovered.²

Although the mainstays of therapy include stretching, range-of-motion, and therapeutic exercise, there may be a role for other therapeutic modalities. Transcutaneous electrical nerve stimulation may play a role in pain control. The role of electrical stimulation as a modality to promote recovery is controversial.² There may be a role for acupuncture, particularly in pain control.²

The role of interventional procedures has also not been established. In one study, several patients treated with a corticosteroid shoulder injection reported pain relief.¹ In cases where there is coexisting cervical spine pathologic abnormality, cervical epidural steroid injection may be an important diagnostic and therapeutic modality to distinguish between PTS and cervical radiculopathy.²

Combination Therapies

Most patients with PTS are treated with a multidisciplinary approach that includes both physical therapy and pharmacologic treatment, often with multiple agents.

From anecdotal evidence, combination pharmacologic therapy may be most effective for pain control. To the extent that oral corticosteroids may help improve symptoms but often provide incomplete pain relief, it is reasonable to begin with oral opioids with the oral corticosteroid taper.⁶ As discussed in an earlier section, the recommended dose of oral corticosteroids is a 2-week course of oral prednisolone, 60 mg daily in the first week and tapering to 10 mg per day in the second week.³ After the initial corticosteroid taper, van Alfen and van Engelen¹ recommend that a combination of NSAID with a short-acting or slow-release opioid may be best.

Surgical Treatment Options

In cases that are refractory to conservative pharmacologic and nonpharmacologic treatments, surgery is often considered. Surgical procedures for PTS include neurolysis, nerve grafts, and nerve transfers. Timing of surgical intervention is challenging. Penkert and colleagues³² suggest that delay of treatments beyond 2 to 6 months

Box 6

Complications of PTS

Potential complications

- Shoulder dysfunction
 - Adhesive capsulitis
 - Shoulder subluxation/dislocation due to weakness
- Chronic pain
- Loss of function
 - Weakness in proximal more than distal arm
 - Loss of function due to pain
- Shoulder subluxation/dislocation due to weakness
- Loss of work/disability
 - Need for workplace modification
 - Long-term disability

Box 7**Factors associated with prognosis in PTS***Good prognosis*

- Predominantly upper trunk involvement⁴
- Primarily sensory symptoms³⁴

Poor prognosis

- Prolonged pain
- Prolonged weakness⁴

may result in worse outcomes. Kretschmer and colleagues³³ have reported 60% favorable outcomes after surgical treatment. Surgery is often considered when there are secondary complications. Tendon and muscle transfers are often used to facilitate function in the setting of significant weakness. Surgical intervention is also sometimes performed in the setting of secondary shoulder complications, such as recurrent dislocation.^{4,32,33}

Evaluation of Outcome and Long-Term Recommendations

The disease course of PTS is variable.⁴ In the best possible outcome, pain and weakness can resolve spontaneously in about 1 month²² with conservative treatment alone. Unfortunately, many patients develop long-term complications such as shoulder dysfunction (adhesive capsulitis, shoulder subluxation), chronic pain, loss of function, loss of work, and disability (**Box 6**).

With respect to disease course, 66% of patients begin to show motor function recovery within 1 month of onset of weakness.⁴ A study by Tsairis and colleagues¹⁴ observed the full recovery may follow a more protracted course. The percentage of patients with PTS who reported “excellent” recovery was 36% at 1 year, 75% by 2 years, and 89% by 3 years. A 2006 study by van Alfen and van Engelen¹ observed that although many patients do achieve timely and complete resolutions of symptoms, one-third of patients continue to experience chronic pain and persistent functional deficits (after an average follow-up of greater than 6 years). In patients with greater than 3-years follow-up, mild, moderate, and severe paralysis were seen in 69%, 14%, and 3% of patients, respectively. Factors associated with a good prognosis include predominantly upper trunk involvement and primarily sensory symptoms (**Box 7**).^{4,34} Prolonged pain and weakness are associated with a poor prognosis.⁴ There is no established relationship between prognosis and age.⁴

Another issue that PTS patients face is recurrence of symptoms. Although rare repeat attacks are sometimes severe, subsequent attacks are usually less severe than the first episode.⁴

SUMMARY

PTS is an underrecognized condition. High-quality evidence for the treatment of PTS is lacking. PTS should be considered as a diagnosis in the setting of an abrupt onset of upper extremity pain followed by progressive neurologic deficits including weakness, atrophy, and occasionally sensory abnormalities. Although there are many proposed theories, a common unifying cause has not been established. No specific treatments have been proven to reduce neurologic impairment or improve the prognosis of PTS, although there is anecdotal evidence that early corticosteroid therapy

may improve pain and hasten recovery in some patients. Most patients with PTS syndrome are treated with a multidisciplinary approach that includes both physical therapy and pharmacologic treatment, often with multiple agents. The goals of physical therapy should include maintenance of range of motion and prevention of loss of function. Most authors advise a cautious approach toward strengthening exercises in physical therapy as an overly aggressive approach can overload muscles that are weak or in early reinnervated stages. In cases that are refractory to conservative pharmacologic and nonpharmacologic treatments, surgery is often considered. Further research in treatments to hasten recovery and reduce long-term neurologic impairment is needed.

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