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The Role of Positional Release Therapy in Treating Recalcitrant Brachial Plexus Neuritis: A Case Report

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A 17-year-old female soccer player presented with severe right shoulder pain and scapular winging due to brachial plexus neuritis. The patient was diagnosed with Parsonage-Turner syndrome, a rare condition often resistant to traditional physical therapy, which typically persists for 6 months to years, at times requiring surgical intervention. Over the course of 6 weeks, the patient received positional release therapy once a week coupled with electrical modalities, massage, and a daily home exercise program. This case report is unique because we believe we were the first to use positional release therapy for treatment and the patient's condition resolved more quickly than is typically reported.

Key Words: neuralgic amyotrophy, Parsonage-Turner syndrome, manual therapy

Key Points

- Recalcitrant brachial neuritis is a rare condition, often resistant to traditional therapies.
- Positional release therapy may be an invaluable tool for treating patients with acute or recalcitrant forms of brachial neuritis or other neuroinflammatory conditions.

Positional release therapy (PRT) is a gentle and pain-free manual therapy that can be used across the life span for all ages and most conditions,[1] making it an excellent therapy for brachial plexus neuritis. Cases of brachial plexus neuritis often involve a sudden traumatic force to the patient's shoulder girdle, neck, or both, such as in sport when an athlete's arm is suddenly pulled or pushed backward, producing a "stinger."[2] However, other mechanisms may also produce brachial neuritis and affect its duration and severity. Cases of brachial plexus neuritis have been reported in numerous clinical situations that involve some sort of antecedent impact to the patient, whether it be surgical, infectious, traumatic, or even therapeutic (eg, vaccinations or antibiotic treatments[3,4]), yet the condition can also be inherited.[3]

Patients with brachial plexus neuritis are in severe pain and using therapies that cause more pain is not ideal for calming the patient's neurologic system.[5] Simply described, PRT is the opposite of stretching and is pain free in its application. Instead of stretching tissues or forcing them to give way, tissues are pushed together, twisted, compressed, and manipulated, much like when one is trying to unknot of a chain necklace. By unkinking the knot or tissue restriction, pressure is taken off entrapped vessels and nerves, which improved the major symptoms of patients with brachial plexus neuritis[3,5,19,20] by reducing pain and spasm,[6,7] improving blood flow,[8] restoring strength,[9–13] increasing range of motion,[9–17] improving quality of life,[9,10,15,18] and enhancing sport performance.[6,9,10]

Because of its ease of use, application in a variety of environments, and utility in treating resistant tissue restrictions, PRT is an excellent therapy for immediate and lasting reduction of the tonic tissue restrictions often found in such neurologically based conditions as brachial plexus neuritis. It may be the ideal treatment for neuroinflammatory conditions in reducing the sustained stretch reflex through muscle spindle manipulation[21,22] and reducing γ gain or drive,[14,21] which may lessen sensitivity of the spindle,[21] thereby reducing muscular contraction and overall neurologic upregulation. Moreover, PRT has been proposed to improve blood flow to tissues.[1,8] In patients with brachial plexus neuritis presenting with tonic contraction, blood and lymph flow would be reduced because of the constrictive pressure placed on the lymph and blood vessels by the sustained muscular contraction. Using diagnostic ultrasound, Sikdar et al[23] found that blood flow was dramatically reduced in active and latent trigger points. Speicher et al[8] provided preliminary evidence that the use of PRT increased blood flow at the lateral elbow common extensor tendon. Patients with brachial plexus neuritis may experience reductions in strength, range of motion, sport performance, and quality of life.[1,5,7,10] Improving blood flow by reducing the constriction of vessels and nerves with PRT is an important therapeutic goal: improved tissue perfusion may help restore the adenosine triphosphate energy cycle that assists in mechanical coupling and uncoupling of the fibrils that power muscular contraction but may also foster adequate neurochemical balance to

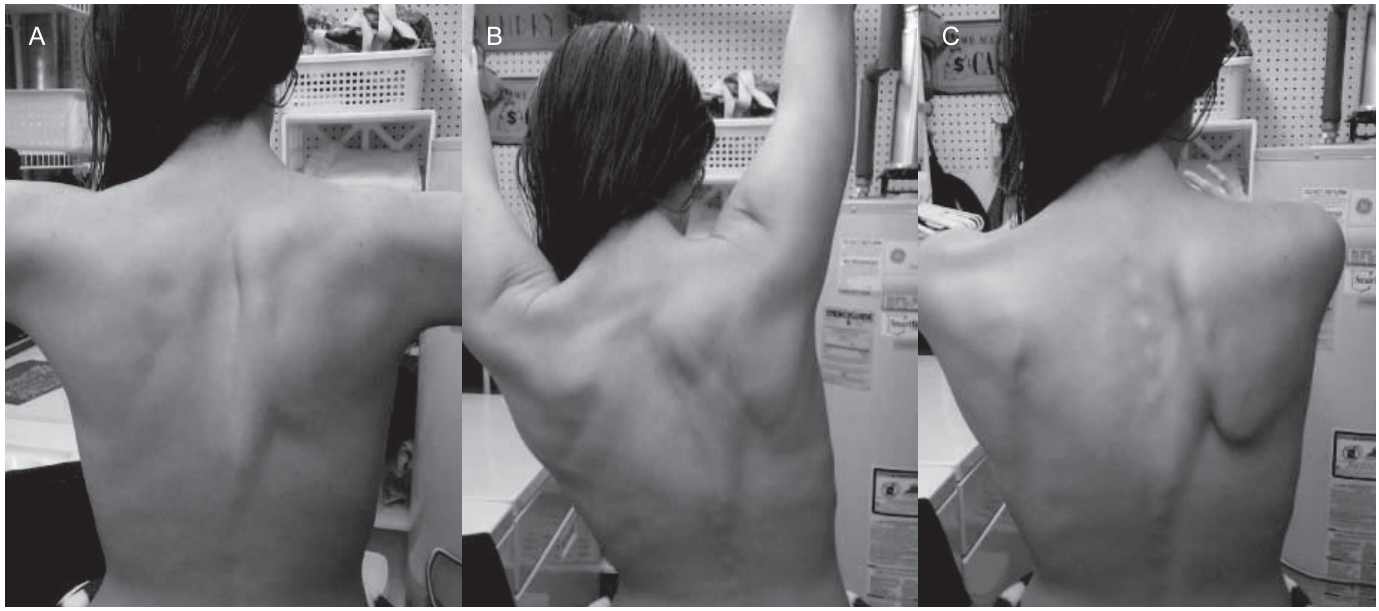

Figure 1. Pretreatment presentation of patient with Parsonage-Turner syndrome. A, Arms abducted to 90°. B, Arms abducted to approximately 170°. C, Arms flexed to approximately 90°.

restore tissue homeostasis,[1,24] which is critically needed in this condition.[19,25,26]

The purpose of our case report was to (1) to present the case of a 17-year-old White female high school soccer player who presented with recalcitrant brachial plexus neuritis and (2) discuss the successful management and treatment of her condition using PRT. We provide practicing clinicians with a novel therapeutic approach for treating patients with brachial plexus neuritis. Additionally, this case study lays the groundwork for future investigations into how PRT can be used for other neuroinflammatory conditions that produce hard-to-resolve tissue and myofascial restrictions, such as scapular dyskinesis, thoracic outlet syndrome, cervicogenic headache, chronic low back pain, and postconcussion syndrome.

CASE PRESENTATION

Patient

A 17-year-old female soccer player presented to our clinic with severe right shoulder pain and scapular winging. The patient sought our services for her symptoms because traditional physical and medication therapy were unsuccessful in resolving her condition.

Medical History

The patient's mother reported that her daughter started to experience severe left scapular pain shortly after a diagnosis of mononucleosis. The patient sought chiropractic manipulation for her left shoulder pain, which resolved with treatment. The following day, she started to experience moderate right shoulder pain and again sought chiropractic manipulation. Immediately upon manipulation of the right shoulder, she felt complete arm numbness accompanied by severe pain and burning radiating from the right shoulder into her hand; shortly thereafter, she experienced a significant muscle spasm and pain in her right scapular musculature.

At the time of the patient's first visit to our clinic, she was undergoing treatment for a low potassium level and had recently completed treatment for streptococcal pharyngitis. The patient and her mother denied any other previous trauma or condition involving the neck or shoulders.

Two months after she first noticed the left shoulder pain, the patient presented to our clinic with severe (9/10) right shoulder girdle pain, rated on a numeric pain rating scale (0 = *no pain* and 10 = *worst imaginable pain*), with unilateral right tonic muscular spasm, scapular winging, decreased shoulder mobility, antalgic gait, peripheral paresthesia, and allodynia. The patient also reported that after the onset of her right extremity pain and numbness, she started to experience significant winging of her scapula.

Physical Evaluation

Before our evaluation, the patient had seen several physicians for her condition. Bloodwork showed a low potassium level but no evidence of ongoing mononucleosis or any other condition that might correlate with her symptoms. The physicians suspected that her symptoms might be related to a cervical or shoulder condition; therefore, magnetic resonance imaging (MRI) was conducted to rule out injury to these regions, but the findings were unremarkable. Because of the lack of diagnostic findings, the patient was prescribed pain medication, muscle relaxers, and conventional physical therapy, which she reported were unsuccessful in resolving her symptoms.

Two months after the onset of pain, the patient presented to us with severe resting pain throughout her right shoulder girdle that was focally intense at the medial border of the scapula. Significant winging of the right scapula was present at rest and exacerbated with shoulder abduction (pain increased to 10/10; Figure 1). The patient was in obvious distress, brought to tears with any movement of her shoulder, active or passive, or with light touch to the shoulder girdle. Her gait and resting posture were dramatically altered due to the tonic contracture of her

Table 1. Electromyographic Findings of Patient Case With Parsonage-Turner Syndrome[a]

Side	Muscle	Nerve	Root	Insertion Activity	Fibrillation Potential	Positive Sharp Waves	Amplitude	Duration	Polyphasic Potentials	Recruitment Ratio	Interference Pattern
Right	Infraspinatus	Suprascapular	C5-6	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml
Right	Flexor carpi radialis	Median	C6-7	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml
Right	**Biceps**	**Musculocutaneous**	**C5-6**	**Nml**	**Nml**	**1+[a]**	**Nml**	**Nml**	**0**	**Nml**	**Nml**
Right	Trapezius	Spinal accessory	Cranial nerve XI, C3-4	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml
Right	Rhomboid Major	Dorsal scapular	C5	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml
Right	Supraspinatus	Suprascapular	C5-6	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml
Right	Latissimus dorsi	Thoracodorsal	C6-8	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml
Right	**Serratus anterior**	**Long thoracic**	**C5-7**	**Nml**	**Nml**	**2+[a]**	**Nml**	**Nml**	**0**	**Reduced**	**Nml**
Right	1st Dorsal interosseous	Ulnar	C8-T1	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml
Right	Triceps	Radial	C6-7-8	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml
Right	Deltoid	Axillary	C5-6	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml

Abbreviation: Nml, normal.

[a] Positive sharp waves (fibrillations) of the musculocutaneous and long thoracic nerves and reduced recruitment of the serratus anterior are denoted in bold.

right shoulder girdle and spinal musculature. She was no longer able to participate in soccer or any athletic activity and had taken a medical leave from school due to her condition. At the time of her evaluation with us, she also reported full right arm numbness and burning and weakness with any action of her right arm, elbow, or hand. She stated that she could not dress herself due to pain and weakness throughout her entire right extremity.

We conducted a history and brief assessment at her initial visit. However, the goal of that visit evolved into reducing the patient's pain and spasm to obtain a more accurate evaluation. The Disablement in the Physically Active Scale[27] was used to assess her functional ability and quality of life. Based on her overall score of 34/64, the patient was deemed moderately disabled, with the heaviest factor weight on pain and range-of-motion impairment. Although we did not directly assess resistive range of motion at this time due to her pain level, we evaluated active and passive range of motion and performed a palpatory examination. The patient was able to actively abduct her right arm to 90°, but doing so increased her winging and muscular spasm and elevated her pain to 10/10, limiting any further movement. Passive abduction produced the same result. Multiple trigger and tender points were present throughout the scapular, thoracic, cervical, and shoulder regions, with predominance on the right; palpation of most points resulted in ratings of 9 or 10 out of 10. The empty can test for assessing rotator cuff integrity was positive for pain and weakness of the right extremity. Based on the patient's history and evaluative findings, we suspected she was suffering from a type of brachial plexus neuritis, either long thoracic nerve palsy or Parsonage-Turner syndrome (PTS); therefore, an electromyographic (EMG) study was ordered, which occurred approximately 3 months after the onset of her symptoms.

A type of brachial neuritis, PTS is typically diagnosed based on clinical findings but is considered an axonal process that can be confirmed by abnormal EMG findings,[28] which allows the clinician to rule out other possible neurologic conditions.[5] As seen in Table 1, our patient's EMG revealed abnormal positive sharp waves of the musculocutaneous and long thoracic peripheral nerves with reduced recruitment of the serratus anterior muscle on the involved side, which coupled with her symptom presentation, led to the diagnosis of PTS.

Treatment and Clinical Course

The initial goal for the patient's therapy was to reduce pain and muscular spasm. Then the primary aim of treatment was to restore upper extremity and shoulder girdle strength and kinematics. From the time of our initial encounter with the patient, the course of therapy lasted approximately 3 months (Table 2). The therapeutic interventions were applied to the right and left upper quarters except for electrical stimulation, which was applied to only the right shoulder girdle. Both the involved and noninvolved shoulders were treated because pain and tissue guarding are often not localized. Therefore, to optimize tissue release, both upper quarters were treated with the intervention. The treatment approach comprised nine 1-hour positional release therapy sessions (Figure 2), electrical stimulation, effleurage massage, and a daily home exercise program to restore upper extremity kinematics and strength. The treatment timeline (Table 2) provides a roadmap for clinicians and researchers to replicate the patient outcomes we achieved with our approach to this patient with brachial plexus neuritis. Clinicians can address impairment of the structures listed in Table 3 by consulting PRT texts for instructions.

The patient presented with severe pain on the first visit, which initially prevented the application of PRT or other manual therapy techniques; thus, moist heat coupled with interferential current (IFC) was first applied to the right shoulder girdle, which then permitted the use of PRT and light massage to further reduce pain and spasm. Pretreatment, the patient presented with 9/10 shoulder girdle pain and severe muscular spasm. After treatment, her spasm and pain were reduced to 0/10, which lasted 4 hours, but her symptoms increased to 8/10 until she returned for therapy 2 days later.

Table 2. Treatment Timeline and Patient Outcomes

Time, wk	Treatment Interventions	Numerical Pain Rating Scale Score[a]	Disablement in the Physically Active Scale Score	Active Range of Motion (Abduction), °
1–8	Symptoms develop; conventional physical therapy, chiropractic treatment, and medication administered	Not available	Not available	Not available
9	Initial application of PRT and IFC stimulation (sensory) and massage	9/10	34/64 (pretreatment)	90
10	PRT, moist heat, and massage with home portable IFC unit (motor)	7/10		100
11	PRT, moist heat, massage, and discontinuation of portable IFC unit	2/10		112
12	PRT and home exercise program	2/10		130
13	PRT and home exercise program	0/10		180
14	PRT and return to soccer	0/10		180
15–17	PRT	0/10	1/64 (posttreatment)	180

Abbreviations: IFC, interferential current; PRT, positional release therapy.

[a] 0 = *no pain*, 10 = *worst imaginable pain*.

On the second visit, the patient did not have severe pain, which permitted a more comprehensive application of PRT to the right and left upper extremities, torso, and cervical spine. Moist heat and massage were also applied after PRT. She was provided with a portable IFC unit for home use to control pain. Pain and muscular spasm fluctuated between 2/10 and 7/10 during the time span for the next 2 treatments, which consisted of PRT, moist heat, massage, and use of the portable IFC unit at home as needed. The patient was now able to brush her teeth and hair.

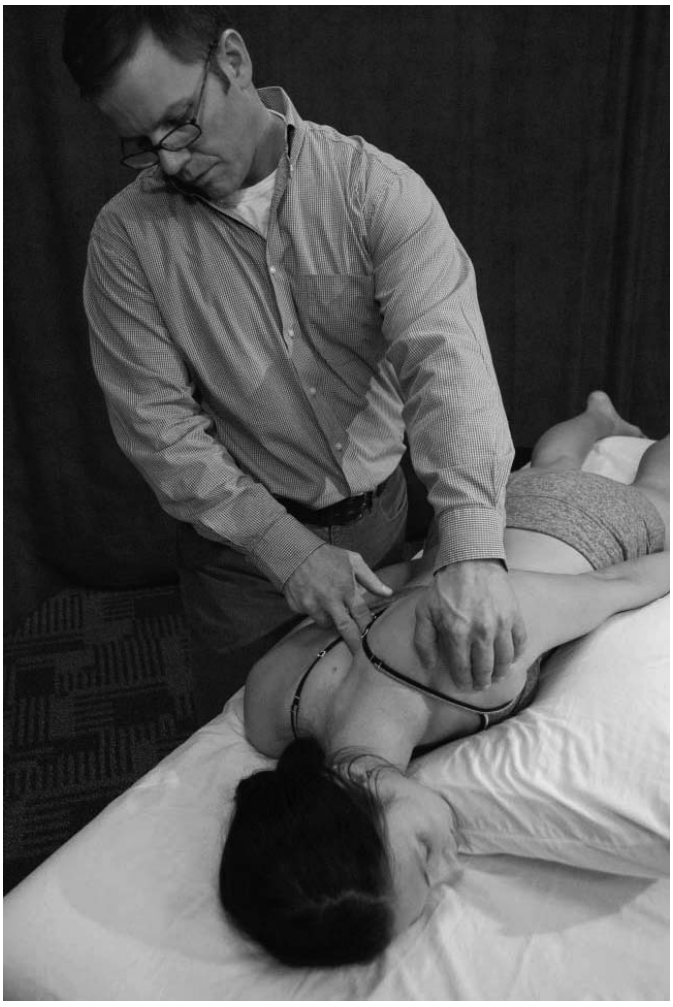

Figure 2. Positional release of rhomboid muscles.

After her fourth treatment, she was able to run a 5K road race with her right shoulder pain rated at 2/10. At this point in the treatment, tonic muscular spasm was absent, and motion was nearly restored to a functional range. A daily home exercise program to restore upper extremity strength and kinematics was prescribed.

After 6 PRT sessions combined with traditional modalities and 2 weeks of a daily home exercise program (Table 4), the patient was able to return to competitive soccer without limitation or complication. After 3 additional PRT sessions, she was discharged pain free and without strength or range-of-motion deficits. At 6-month and 1-year follow-ups, her physical examination was unremarkable, and she continued to participate in competitive soccer and rock climbing without limitation.

Intervention

Our patient presented with significant pressure insensitivity, pain, muscular spasm, and reduced strength and range of motion. We chose PRT as the primary treatment modality because it can reduce pressure sensitivity,[14–16] pain,[6,7,14,29] and muscular spasm[7,9] and can restore strength[11–13] and range of motion.[6,7,14–17] The initial goal of therapy was to reduce the patient's pain and muscular spasm, which was accomplished primarily through the application of PRT. Each positional release (Table 3) lasted 30 seconds to several minutes, but the positioning and duration of the releases were determined according to the fasciculatory response method, first introduced for clinicians using PRT by Speicher and Draper in 2006.[30,31] With the fasciculatory response method, the painful or hypertonic tissue is placed in a shortened position; when a "tissue twitch" or fasciculation is felt, the clinician manipulates the tissue into the position that produces the strongest fasciculation and holds this position until the fasciculation

Table 3. Muscles Treated Bilaterally With Positional Release Therapy

Shoulder	Thorax	Cervical
Infraspinatus	Levator scapulae	Levator scapulae
Supraspinatus	Lower trapezium	Anterior scalene
Teres minor	Serratus posterior	Middle scalene
Teres major	Serratus anterior	Upper trapezium
Long head of biceps	Rhomboid major	Subclavius
Triceps	Rhomboid minor	
Pectoralis minor		

Table 4. Daily Home Exercise Program

Therapeutic Exercise	Sets and Repetitions
Isometric YTWLs[a]	5 sets each, 10-s hold
Empty can resistive band	3 sets, 12–15 repetitions
Proprioceptive neuromuscular facilitation stretch of the trapezium, levator scapulae, and rhomboids	3 sets, 20-s stretch and 10-s push
Supine cane shoulder flexion and abduction	3 sets, 10 repetitions each

[a] Therapeutic rehabilitation technique in which the patient stands against a wall and positions the arms in the shape of the letters, pushing isometrically against the wall to facilitate activation of the scapulothoracic muscles to restore shoulder kinematics.

abates. Typically, once the fasciculation subsides, the tissue unwinds and becomes pain free.

Once the patient's pain and spasm were reduced, the primary treatment goal was to restore shoulder girdle strength and kinematics through a basic daily home exercise program (Table 4) that lasted for several weeks after the fourth treatment session. The exercise program resolved the patient's remaining shoulder girdle kinematic and strength deficits.

Comparative Outcomes

The classic description of PTS is a patient who suddenly develops constant, severe unilateral shoulder girdle pain that may extend to the trapezius, upper arm, forearm, and hand.[32] Scapular winging may be seen in cases where the long thoracic nerve innervating the serratus anterior muscle is involved; however, the condition may involve any of the nerves or sections of the brachial plexus,[20,25,32] which often challenges clinicians in their diagnosis.[32] The hallmark clinical signs and symptoms suggestive of PTS and criteria for excluding the condition are shown in Table 5. The lumbosacral plexus, phrenic, recurrent laryngeal, and cranial nerves can also be affected but this occurs more rarely.[18] The most commonly affected nerves are the suprascapular, axillary, musculocutaneous, long thoracic, and radial nerves.[18] Also known as neuralgic amyotrophy or brachial neuritis, PTS has been reported in numerous clinical situations that involve an antecedent impact on the patient, whether surgical, infectious, traumatic, or even therapeutic, such as cases involving vaccinations or antibiotic treatments[4,18]; however, the condition can also be inherited.

Hereditary neuralgic amyotrophy (HNA) has been identified in 300 families worldwide.[18] An autosomal-dominant condition, HNA is associated with mutations of gene septin 9 on chromosome 17q23.[18,25] In contrast to patients with idiopathic neuralgic amyotrophy or PTS, patients with HNA often present with dysmorphic features (eg, facial asymmetry, cleft palate, short distance between the eyes) and typically display severe paresis and signs and symptoms earlier in life, often starting in childhood.[18] Also, the painful episodes occur with greater frequency in hereditary than idiopathic cases.[18] Regardless of whether the patient has HNA or idiopathic PTS, Seror[5] reported that the condition usually involves 3 sequential phases: the painful phase, the weakness and sensory phase, and the recovery phase. Uniquely, our patient presented with all 3 phases simultaneously.

After 4 weeks of treatment, with PRT as the primary modality, the patient was able to run a 5K road race with 2/10 right shoulder pain. At this point, tonic muscular spasm was absent, and range of motion was nearly restored to a functional range—a remarkable feat considering the etiology and prognosis of PTS.[5,18] The painful aspect of PTS usually resolves within 6 months to 3 years, but the time frame for recovery depends on the amount of axonal loss and the speed of nerve growth.[18] Though our patient showed nerve conduction deficits at 3 months, she was pain free within 6 weeks after PRT was implemented and did not demonstrate functional or sensory deficits.

A daily home therapy program to restore upper extremity strength and kinematics was prescribed. After 6 treatments and 2 weeks of the daily home exercise program, the patient was able to return to competition soccer without limitation or complication. She was discharged with no pain and no strength or range-of-motion deficits. At 6-month and 1-year follow-ups, her physical examination was unremarkable, and she continued to participate in competition soccer and rock climbing without limitation. In contrast, up to 89% of patients with PTS experience atrophy and sensation abnormalities at the 3-year mark, and 33% have chronic pain and functional deficits 6 years after the initial insult.[19]

DISCUSSION

Positional release therapy was an effective modality for the expeditious resolution of our patient's brachial neuritis. Also known as neuralgic amyotrophy, acute brachial plexus neuropathy, or brachial plexus neuritis,[18] PTS is considered a rare syndrome.[18,19,25,33] It presents with severe, abrupt, unilateral shoulder pain that may begin insidiously but quickly amplifies in severity and intensity with ensuing muscle weakness, scapular winging, and possible sensory disturbance and altered scapular kinematics.[19] The condition can affect the general population across the life span, with its greatest incidence in the third and seventh decades

Table 5. Diagnosis of Parsonage-Turner Syndrome[a]

Probable Presentation	Potential Additional Signs and Symptoms	Exclusion Criteria
Recent onset of severe (≥7 on a scale of 0/10) shoulder pain (unilateral or bilateral)	Upper extremity weakness without severe initial pain	Absence of abnormal compensatory shoulder movements
Glenohumeral, scapulothoracic, or both, movement abnormality with abduction-flexion	Extensive multifocal paresis of upper extremity	Pain, weakness, or both continue to progress
Paresis of long thoracic, scapular, and anterior interosseous nerves 3 wk after symptom onset	Involvement of other peripheral nerves: lumbosacral plexus, phrenic, recurrent laryngeal	Only glenohumeral passive range-of-motion deficits present
		Symmetric weakness
		Horner syndrome
		Diabetes mellitus

[a] Adapted from Seror.[5]

of life.[18] The annual reported incidence is 1.6 to 3 cases per 100 000, and it is 2 times more likely to occur in men than women; it typically manifests in the right extremity and 97% of the time is unilateral in nature.[18] Several authors[18,33,34] believed the incidence of PTS was likely underreported due to a lack of proper diagnosis and recognition. Currently, no data are available on how often this condition presents in the athletic population, but many sports medicine professionals are aware of other types of neuroinflammatory conditions that occur in contact sports, such as "burners" and "stingers."[2] Monteiro Dos Santos et al[20] reported that Dreschfeld was the first to describe the condition in 1887, and then in 1948, Parsonage and Turner[34] presented 136 cases, which they termed *Parsonage-Turner Syndrome*. Since 1948, the condition has been studied in depth, yet to date, no definitive mechanism has been identified as the primary cause, nor has any therapy approach or intervention been found to be superior to another.[18] The lack of consensus regarding the cause and ideal treatment of this condition probably reflects the variable presentations clinicians may encounter among patients.

Based on the evaluative and diagnostic EMG findings, we diagnosed our patient with PTS. After chiropractic manipulation of the right shoulder, the patient presented with severe right shoulder pain, muscle spasm, and scapular winging. However, she had recently been diagnosed with mononucleosis, so whether the manipulation or underlying illness was the trigger for the onset of PTS, a similar presentation should prompt the clinician to consider the possibility of PTS.

Besides the classic evaluative findings associated with PTS (Table 5), abnormal EMG findings should also serve as a red flag and may be a useful tool for evaluating the effectiveness of PRT as an intervention for this condition and other forms of brachial neuritis seen in athletes. Valls-Solé[28] recommends an EMG be done at least 14 days after onset. Among patients with PTS, abnormal EMG findings are most often identified in the peripheral nerves of the upper trunk of the brachial plexus (suprascapular, long thoracic, axillary) but the anterior interosseous, musculocutaneous, and spinal accessory nerves can also be affected.[35] An EMG performed several weeks after symptom onset may reveal acute denervation of the peripheral nerves and nerve roots, whereas disc conditions show denervation of the specific nerve roots affected.[18] Positive sharp wave fibrillations are seen with PTS, as observed in our patient (Table 1)[18,25]; however, motor and sensory nerve conduction velocities remain normal[18,28] in contrast to the increased distal latencies as seen with long thoracic nerve palsy due to compression or traction injury,[32] which may also be present in unresolved stingers or burners.[2] Tjoumakaris et al[18] reported that at 3 to 4 months after onset, patients "may show chronic denervation and early signs of reinnervation with polyphasic motor unit potentials." However, these findings were not demonstrated in our patient's EMG results, although testing occurred 3 months after symptom onset. Therefore, early diagnosis is critical to avoid long-term complications, which may be seen on MRI.[35]

Unlike the typical prolonged recovery time (6 months to years) and resultant persistent deficits described in the literature with conservative interventions,[19] the introduction of PRT in this case was, we believe, the driving factor in facilitating full recovery within 6 weeks without persistent deficits. Up to 90% of patients who present with PTS experience pain and sensory abnormalities for as long as 3 years and one third for as long as 6 years.[19] Our patient might have been in the 10% who recover within 3 years, yet she did not improve until PRT was introduced, which raises the question of whether PRT may also be helpful in expediting recovery from other types of brachial neuritis seen in athletes, such as stingers and burners. Nonetheless, it is still important to consider other possible underlying conditions and interventions for a patient with a suspected case of PTS or another form of brachial neuritis.

In this case, the MRI showed no other shoulder injuries. Gaskin and Helms[35] reported that MRI findings may help to rule out other shoulder conditions (eg, calcific tendinitis and tissue trauma) and that a high T2 signal intensity visible more than 2 weeks after symptom onset may demonstrate denervation of the muscles innervated by the brachial plexus in the absence of a history of acute trauma; when correlated with the physical examination, this could suggest PTS. In our patient, the MRI was initially used to examine the muscles, including those of the rotator cuff, but no muscle denervation was present.

Due to the varied clinical presentations and symptoms associated with PTS,[18,36] multiple clinicians were unsuccessful in the early treatment of our patient, resulting in limitation of activities of daily living and cessation of sport activity. What made this case unique, along with the use of PRT as the primary treatment modality, was her rapid recovery after 2 months of persistent symptoms despite traditional care.

Even though PRT appeared to play a significant role in the treatment process and expeditious recovery of our patient, we also used other physical and therapeutic modalities that have been shown to play important roles in treating this condition.[14,18,26,35] However, to our knowledge, this is the first case of PTS to be treated with PRT as the primary treatment method, which has broad clinical implications. Based on the clinical outcomes of this case, we suggest that sports medicine professionals consider the use of PRT as a first-line intervention for acute and recalcitrant forms of brachial neuritis and other neuroinflammatory conditions that require pain relief, spasm reduction, and improvement in strength and range of motion.

CLINICAL BOTTOM LINE

A rare form of brachial neuritis,[18,33] PTS is often diagnosed late in its clinical course[20] and has few treatment options[18] to alleviate the severe pain or expedite return to functional activity and sport. Although PRT appeared to accomplish both of these outcomes when combined with traditional and physical modalities, comparative studies using PRT for acute and recalcitrant brachial neuritis should be conducted. A clinician who is a novice at PRT should still try this intervention for patients with acute or recalcitrant brachial neuritis due to its potent effects in reducing the severe pain and hypertonicity. Most importantly, PRT is a passive, nonpainful therapy that should not cause any additional trauma because the nerves are not being put on tension; in fact, just the opposite is true. The

clinician simply shortens the hypertonic tissue and waits for it to relax or for the twitch response (fasciculation) to dissipate. Yet as with most manual therapies, greater proficiency will be gained through formal instruction and practice. Regardless of the treatment paradigm or approach used to treat PTS, clinicians should be aware that the condition is often brought on by infection and trauma, which can occur frequently in the high school and collegiate populations, particularly in those contact-sport athletes. Clinicians will be well served in the early treatment of patients with PTS using PRT if they form their diagnosis by considering their evaluation findings coupled with EMG and possibly MRI results.

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