

# Parsonage-Turner Syndrome: MR Imaging Findings and Clinical Information of 27 Patients<sup>1</sup>

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## Purpose:

To review retrospectively the magnetic resonance (MR) imaging findings and clinical information of patients with Parsonage-Turner syndrome (PTS).

## Materials and Methods:

The institutional review board did not require its formal approval or informed patient consent at the time of the study. However, the study was HIPAA compliant. The information in a computerized database of 2875 consecutive shoulder MR examinations was retrospectively reviewed. With use of key terms, the database software identified 81 examinations potentially associated with PTS. Both authors together reviewed the 81 imaging reports and the corresponding patients' medical records. In consensus, they made the diagnosis of PTS in 21 patients (two with bilateral involvement) on the basis of MR findings, electromyographic results, and clinical data. They also examined the data of an additional six patients (one with bilateral involvement) obtained from outside facilities. Ultimately, 30 shoulders of 27 patients (18 male, nine female; age range, 12–81 years; mean age, 41 years) were evaluated. The MR findings and clinical information (ie, regarding atrophy, pain, weakness, electromyographic results, neck and spine history, trauma, excessive overhead activity, recent surgery, vaccination, and illness) of all patients with PTS were reviewed. MR findings of diffuse high T2 signal intensity abnormality and fatty atrophy of muscles were evaluated to assess the pattern of nerve involvement. Structural causes (eg, ganglion cyst or other mass) of neurogenic high T2 signal intensity abnormality were excluded at MR imaging.

## Results:

Twenty-nine (97%) of 30 shoulders had suprascapular nerve involvement; in 15 (50%) shoulders, the involvement was limited to this nerve. Fifteen (50%) shoulders had axillary nerve involvement; in only one (3%) shoulder, the involvement was limited to this nerve. One shoulder (3%) had subscapular nerve involvement. Nine (30%) shoulders demonstrated focal muscular atrophy. Eleven (41%) of 27 patients also underwent electromyography; all of these patients demonstrated neuropathies that matched the patterns of neurogenic high T2 signal intensity abnormality seen at MR imaging.

## Conclusion:

The suprascapular nerve was almost invariably involved (in 97% of shoulders) in patients with PTS. Axillary nerve involvement also was commonly observed (in 50% of shoulders). Subscapular nerve involvement was uncommon (in 3% of shoulders).

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**P**arsonage-Turner syndrome (PTS), also known as acute brachial neuritis and neuralgic amyotrophy, is an uncommon—but not rare—clinical problem. Although this abnormality typically has a characteristic manifestation—namely, acute onset of severe shoulder pain followed shortly thereafter by weakness of at least one shoulder muscle—it is often confused clinically with more well-known disorders such as cervical spondylosis, rotator cuff tear, shoulder impingement syndrome, and acute calcific tendonitis (1–7). Therefore, failure to consider PTS may result in inappropriate treatment or even unnecessary surgery. Although this diagnosis has long been recognized within the medical, neurologic, and orthopedic literature, its characteristic magnetic resonance (MR) imaging appearance has been described only within the past 10 years (1). The delayed description of the MR findings of PTS in the radiologic literature is probably related to the fact that fat suppression (ie, edema-sensitive sequencing) did not become a routine part of shoulder MR imaging protocols until the middle 1990s.

Acute brachial neuritis was recognized as a distinct clinical entity when Spillane (3) reported on 46 patients in 1943. In 1948, Parsonage and Turner (2) reported on a series of 136 patients with a similar condition that they called neuralgic amyotrophy or shoulder-gir-

dle syndrome. Since then, the condition has generally been referred to by the acronym PTS. In 1972, Tsairis et al (4) reported the long-term outcomes of 84 patients with this disorder, although they preferred the term *brachial plexus neuropathy*. In 1998, Helms et al (1) described the MR findings of PTS in three patients, choosing to continue using this name of the disorder until the actual cause is identified. A few additional case reports describing similar characteristic MR findings have followed (7,8).

The MR finding most typical of PTS is that of diffuse high signal intensity involving one or more muscles innervated by the brachial plexus depicted on T2-weighted images. T1-weighted MR images also may show atrophy of the affected muscle(s). The pattern of muscular involvement should match the distribution(s) of one or more peripheral nerves originating from the brachial plexus (1,7). The purpose of our study was to review retrospectively the MR findings and clinical information of the patients with PTS that we have seen.

### Materials and Methods

Our institutional review board did not require its formal approval or informed patient consent at the time of the study. However, the study was Health Insurance Portability and Accountability Act compliant.

### Patient Selection

We retrospectively reviewed the information in a computerized database of 2875 consecutive shoulder MR examinations performed at our institution during a 64-month period (April 1997 through July 2002). During this period, the MR images from these examinations were interpreted by six different attending musculoskeletal radiologists. We searched the database by using the key terms Parsonage, Turner, PTS, neuritis, and neurogenic edema. With use of this set of key terms, we expected to identify all or nearly all potential cases of PTS. The database software identified 81 examinations performed in 79 patients, two of whom underwent bilat-

eral examinations. Both authors together reviewed the imaging reports of these 81 examinations and the corresponding patients' computerized medical records. One of the authors (C.A.H.) was one of the six radiologists who originally interpreted the MR images for clinical purposes. At the start of the 64-month period, this author had 20 years experience with musculoskeletal MR imaging.

Fifty-eight patients were excluded because of insufficient clinical data; a clear temporal relationship between the neurologic disorder and trauma, which suggested traumatic neuropathy; or a morphologic cause of the neurogenic high T2 signal intensity abnormality at MR imaging (eg, an entrapment neuropathy caused by a ganglion or other mass in the suprascapular notch).

The remaining 21 patients (two of whom underwent bilateral examinations) were considered to have PTS, by consensus between the two authors, on the basis of the referring clinician's final diagnosis, the clinical history, the physical examination results, the MR findings, and the electromyographic results.

We also examined the data of an additional six patients with PTS (one with bilateral involvement) that were obtained by means of imaging consultations from outside facilities during the same time period. The MR imaging for these six patients was performed at the various referring institutions. Thus, our study group comprised 27 patients (18 male, nine female; age range, 12–81 years; mean age, 41 years) with PTS, three of whom had bilateral involve-

### Advances in Knowledge

- Of 30 shoulders examined with MR imaging in patients with Parsonage-Turner syndrome, 29 (97%) had involvement of the suprascapular nerve and 15 (50%) had involvement limited to this nerve.
- Of 30 shoulders examined, 15 (50%) had involvement of the axillary nerve; however, involvement was limited to this nerve in only one (3%) shoulder.
- Of 30 shoulders examined, one (3%) had involvement of the upper and lower subscapular nerves (in addition to involvement of the suprascapular and axillary nerves).

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#### Abbreviation:

PTS = Parsonage-Turner syndrome

#### Author contributions:

Guarantors of integrity of entire study, C.M.G., C.A.H.; study concepts/study design or data acquisition or data analysis/interpretation, C.M.G., C.A.H.; manuscript drafting or manuscript revision for important intellectual content, C.M.G., C.A.H.; manuscript final version approval, C.M.G., C.A.H.; literature research, C.M.G.; clinical studies, C.M.G., C.A.H.; and manuscript editing, C.M.G., C.A.H.

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ment, who underwent a total of 30 shoulder MR examinations. They underwent MR imaging 3 days to 1 year (average, 3 months) following the onset of their symptoms. Three of these 27 cases were previously reported in the literature (1).

### Diagnosis

No test can be used to diagnose PTS specifically. Both electromyograms and MR images must be interpreted with consideration of the clinical history. In our study, the diagnosis of PTS was made when (a) MR imaging revealed within the muscle tissue a high T2 signal intensity abnormality distributed in a pattern consistent with that of a peripheral nerve lesion, (b) the clinical findings, including no history of trauma or excessive overhead activity, suggested the diagnosis of PTS, (c) MR imaging revealed the absence of a morphologic cause of the denervation (eg, entrapment neuropathy caused by a mass), and (d) no other diagnosis was offered by the clinicians.

All 27 patients demonstrated a neurogenic high T2 signal intensity abnormality at MR imaging and reported having shoulder pain, which was acute in 18 and chronic in nine patients. In addition, all of these patients either reported having shoulder weakness or were found to have shoulder weakness at physical examination. Eleven (41%) of the 27 patients underwent electromyography, at which all of them demonstrated neuropathies that matched or nearly matched the patterns of neurogenic high T2 signal intensity abnormality seen on the MR images.

### MR Imaging Technique

MR imaging was performed with a 1.5-T magnet (Signa; GE Medical Systems, Milwaukee, Wis) and a commercially available transmit-receive shoulder coil. Patients were placed in the supine position, with the shoulder in external rotation. Images were obtained in the transverse, coronal oblique, and sagittal oblique planes. Pulse sequences included transverse, coronal oblique, and sagittal oblique T2-weighted fast spin echo (3000–4083/60–105 [repetition

time msec/echo time msec]) with frequency-selective fat saturation, sagittal oblique T1-weighted spin echo (400–650/8–20), and coronal oblique intermediate weighted (2000–3200/20–30). Additional technical details are as follows: echo train length of eight for the fast spin-echo sequences and a section thickness of 4 mm, an intersection gap of 1 mm, a field of view of 16 cm, and a matrix of 256 × 192 pixels for all sequences. The outside facility examinations were performed at six different institutions by using varying protocols. However, they all included at least one edema-sensitive sequence: either short-tau inversion-recovery imaging or fast spin-echo T2-weighted imaging with fat saturation.

### Data Collection

The 27 patients' MR examination ( $n = 30$ ) results were reviewed by both authors in consensus. At the time of consensus review, the authors had 25 (C.A.H.) and 4 (C.M.G.) years experience with musculoskeletal MR imaging. The neurogenic high T2 signal intensity abnormality (ie, diffuse high T2 signal intensity throughout muscle) and atrophy patterns seen at each examination were noted by consensus between both authors. Muscles were considered to be atrophied when they had gross volume loss and increased fat content relative to the adjacent muscles. Morphologic causes (eg, a suprascapular ganglion) of the neurogenic high T2 signal intensity abnormality that might mimic PTS at MR imaging were confirmed to be absent by consensus between the authors. There were no discrepancies regarding these findings between our image interpretations and the original diagnostic reports.

We used the pattern of neurogenic high T2 signal intensity abnormality on MR images to determine which nerves were involved. We then compared our MR findings with the electromyographic results, when available (for 11 of 27 patients). Specifically, both authors compared the distributions of muscular high T2 signal intensity abnormality seen on the MR images with the distributions of denervation injury described in the pa-

tients' electromyography reports. We reviewed the conclusions of the electromyography reports to correlate them with the clinicians' impressions and lesion localizations.

We also collected data regarding the concomitant MR findings in the 21 patients (23 examinations) who were treated at our institution. We reviewed all patients' clinical histories specifically for data regarding pain, weakness, trauma or substantial recent physical activity, and history of neck and/or spine evaluation. Because of previously reported clinical associations, we also recorded data regarding recent surgery, vaccination, and febrile illness from the patients' computerized medical records. In addition, we reviewed the given clinical diagnoses associated with the 2875 consecutive shoulder MR examinations that were made before the patients underwent imaging, as well as the final clinical diagnoses.

## Results

### Nerve Involvement

Of the 30 shoulders examined in 27 patients, 29 (97%) had a pattern of neurogenic high T2 signal intensity abnormality (Table) that indicated involvement of the suprascapular nerve. Fifteen of the 30 shoulders had involvement limited to this nerve only (Fig 1).

#### Distribution of Neuropathy in 30 Shoulders

Neuropathy Distribution	No. of Shoulders*
Suprascapular nerve involvement	29 (97)
Isolated suprascapular nerve involvement	15 (50)
Axillary nerve involvement	15 (50)
Isolated axillary nerve involvement†	1 (3)
Subscapular nerve involvement	1 (3)

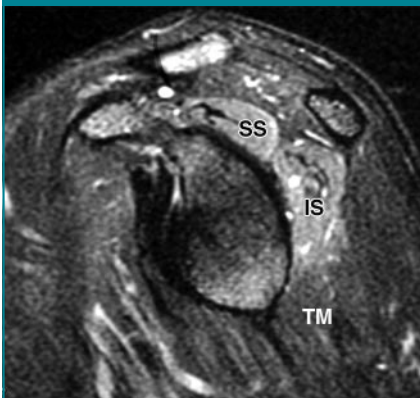
\* Numbers in parentheses are percentages.

† There was suprascapular and axillary nerve involvement on the contralateral side.

The axillary nerve was involved in 15 (50%) of the 30 shoulders (Figs 2 and 3). However, involvement was limited to this nerve in only one (3%) shoulder (Fig 4); this patient also had contralateral involvement of the supra-

scapular and axillary nerves. One shoulder (3%) had involvement of the subscapular nerves—specifically, the upper and lower subscapular nerves innervating the subscapularis muscle—in addition to suprascapular and axillary nerve involvement (Fig 5).

**Figure 1**



**Figure 1:** Isolated involvement of suprascapular nerve. Sagittal oblique T2-weighted fast spin-echo MR image (4000/75) with fat saturation (anterior region is to the left) shows high signal intensity throughout supraspinatus (SS) and infraspinatus (IS) muscles compared with normal signal intensity throughout teres minor (TM) muscle.

#### Side-based Atrophy

Sixteen (59%) of the 27 patients had PTS involvement limited to the right side; eight (30%), involvement limited to the left side; and three (11%), bilateral involvement. Nine (30%) shoulders were noted at MR imaging to have muscular atrophy distributed in a pattern similar to that of the neurogenic high T2 signal intensity abnormality (Figs 2b and 4b). Eleven (41%) patients underwent electromyography also, and all of them demonstrated neuropathies that matched or nearly matched the patterns of neurogenic high T2 signal intensity abnormality seen on the MR images.

#### Additional Data

Two (7%) of the 27 patients were known to have had recent febrile illnesses. Two others (7%) developed symptoms after surgical procedures and anesthesia. Another patient associated

the onset of her symptoms with the birth of her son (in an otherwise uncomplicated spontaneous vaginal delivery).

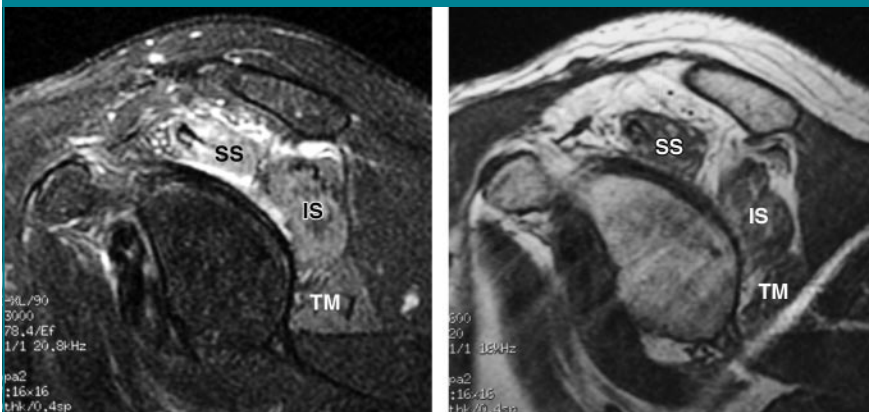
Multiple concomitant findings were appreciated at the 23 shoulder MR examinations performed in 21 patients (two of whom underwent bilateral tests) at our institution, who ultimately received a diagnosis of PTS. According to the MR image interpretations (not surgical correlations), these findings were as follows: rotator cuff tear at two, rotator cuff tendinosis at six, acromioclavicular joint abnormality at 14, biceps tendon abnormality at three, degenerative labra without discrete tears at three, and subacromial-subdeltoid bursitis at nine examinations. Several of these examinations revealed more than one abnormality.

Fourteen of the 2875 consecutive shoulder MR examinations performed at our institution were requested specifically for evaluation of possible PTS. The findings of four of these examinations (ordered by three different orthopedists) actually confirmed the clinical diagnosis. Of the 10 remaining MR examinations, one had normal results. Of the other nine examinations, six had findings suggesting acromioclavicular joint abnormality; two, findings suggesting rotator cuff tear; one, findings suggesting superior labral anteroposterior tear; one, findings suggesting isolated supraspinatus atrophy without rotator cuff tear; and one, findings suggesting rhomboid atrophy. Several of these examinations revealed more than one abnormality. The findings of these 10 examinations could have represented radiologically occult PTS (because imaging was performed too early or too late), or they may have simply represented other abnormalities clinically mimicking PTS.

Six of the 27 patients reported having participated in nontraumatic moderate physical activity the day before the onset of their symptoms. None of these activities involved violent overhead motion.

Eight of the 21 patients with PTS who were treated at our institution also underwent cervical spine MR examinations, which yielded the following results: One examination revealed normal

**Figure 2**



**Figure 2:** Involvement of suprascapular and axillary nerves. (a) Sagittal oblique T2-weighted fast spin-echo MR image (3000/78) with fat saturation (anterior region is to the left) shows high signal intensity throughout supraspinatus (SS), infraspinatus (IS), and teres minor (TM) muscles. (b) Sagittal oblique T1-weighted spin-echo MR image (600/20) shows mild fatty atrophy of supraspinatus, infraspinatus, and teres minor muscles.

Figure 3



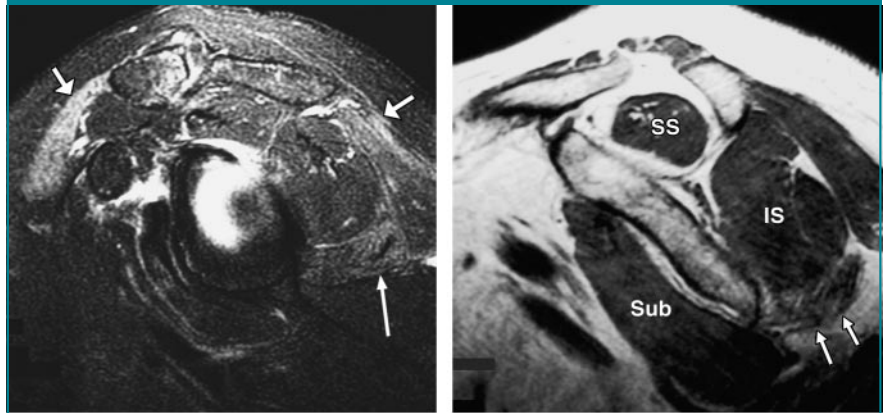
**Figure 3:** Involvement of suprascapular and axillary nerves. Coronal oblique T2-weighted fast spin-echo MR image (4083/63) with fat saturation shows high signal intensity throughout supraspinatus (SS) and deltoid (D) muscles.

findings; six examinations, mild broad-based disk bulges from C3–4 to C5–6 (one with right-sided C3–4 neuroforaminal narrowing); and one examination, moderate central canal stenoses at C3–4 and C4–5 due to broad-based disk bulges (with severe bilateral C4–5 neuroforaminal narrowing). One of the patients evaluated with cervical spine MR imaging had bilateral nerve involvement at shoulder MR imaging. This patient had normal cervical spine examination results, with the exception of a mild C5–6 disk bulge that caused mild central stenosis without foraminal narrowing. The spinal cord was normal in this case.

### Discussion

The MR findings of PTS are thought to reflect denervation injury (1,7). It has been shown that following an acute neurologic insult, denervated muscle initially appears normal. Early study (9–11) results demonstrated increased T2 signal intensity within the affected muscles within 2 weeks after the denervation; the investigators concluded that the signal intensity increase was due to increased extracellular water content. More recently, Wessig et al (12) concluded that the increased T2 signal intensity was more likely to be related to

Figure 4



a.

**Figure 4:** Isolated involvement of axillary nerve in patient with contralateral involvement of suprascapular and axillary nerves. (a) Sagittal oblique T2-weighted fast spin-echo MR image (3000/78) with fat saturation (anterior region is to the left) shows high signal intensity involving teres minor (long arrow) and deltoid (short arrows) muscles. (b) Sagittal oblique T1-weighted spin-echo MR image (650/15) (anterior region is to the left) shows focal fatty atrophy of teres minor muscle (arrows) compared with normal supraspinatus (SS), infraspinatus (IS), and subscapularis (Sub) muscles.

b.

increased capillary blood volume in partially denervated muscle. They also reported high T2 signal intensity abnormality within muscle at only 48 hours following the denervation insult. Neurogenic high T2 signal intensity abnormality in and of itself is not specific for PTS. The high signal intensity has other causes, such as trauma, entrapment neuropathy due to local mass effect (eg, a ganglion in the suprascapular notch), and herniated cervical disks (1,7,13). Quadrilateral space syndrome also can mimic PTS, although it typically has an insidious onset and involves the axillary nerve only (7,14).

Suprascapular neuropathy also has been reported as an uncommon chronic lesion related to a combination of predisposing anatomic conditions and chronic excessive overhead sports activities (particularly volleyball and less particularly baseball pitching). This neuropathy typically presents in athletes who perform high-level overhead sports and have painless infraspinatus atrophy (nerve involvement around the spinoglenoid notch). More proximal nerve involvement (around the suprascapular notch) will include the supraspinatus muscle as well and might include more pain fiber involvement (15–18).

Predisposing anatomic conditions may include a thick or calcified superior transverse scapular ligament, the presence of an anterior coracoscapular ligament, a narrow spinoglenoid or suprascapular notch, and a hypertrophied subscapularis muscle (which may cover the anterior aspect of the suprascapular notch) (15–17). These conditions are generally managed nonsurgically—there is limited surgical correlation in the literature (15–18)—and may mimic PTS at imaging. However, when they do, they generally have a different clinical presentation and do not have axillary or subscapular nerve involvement.

The diagnosis of PTS is suggested when there is an abnormality of the muscles that are innervated by the brachial plexus, without a history of trauma or excessive overhead activity or a morphologic cause identified at MR imaging (7). Other causes of high T2 signal intensity abnormality within muscle, such as myopathy, myositis, and tumor, also should be excluded on the basis of imaging findings and/or the clinical history.

It is unknown how often PTS manifests in the absence of positive MR findings. Given prior experiments that revealed initially normal-appearing muscle following acute denervation and the

onset of muscular high T2 signal intensity abnormality within 2 weeks (9–11), we speculate that MR findings might be falsely negative when the examination is performed early (perhaps within 2 weeks after the onset of symptoms). However, a more recent study revealed subtle high T2 signal intensity abnormality within muscle at only 48 hours following denervation injury in a controlled experimental environment (12).

PTS classically presents with a sudden onset of intense shoulder pain without an apparent cause followed shortly thereafter by substantial local weakness. Typically, the pain subsides over a period of weeks to months, but the weakness may worsen during this time. The long-term prognosis is good: The disorder is self-limited, with gradual recovery usually occurring within months (1–6). Some patients, however, may experience persistent weakness for at least 8 years (5). Tsairis et al (4) noted excellent recovery in 80% of patients by 2 years and in 90% of patients by 3 years. In general, patients with more severe initial symptoms are more likely to have a protracted course (2).

**Figure 5**



**Figure 5:** Involvement of subscapular, suprascapular, and axillary nerves. Sagittal oblique short-tau inversion-recovery MR image (5000/70/150 [repetition time msec/echo time msec/inversion time msec]) (anterior region is to the left) shows marked and increased signal intensity throughout subscapularis (*Sub*), supraspinatus (*SS*), infraspinatus (*IS*), and teres minor (*TM*) muscles.

The current thinking—although it has changed little since 1972—is that PTS is a manifestation of a systemic or localized infectious or immunologic disorder (1,5–7,19). Tsairis et al (4) reported that a systemic viral infection preceded the onset of symptoms in as many as 25% of the patients in their study. In the same series, up to 15% of the patients reportedly received recent vaccinations prior to their initial presentations. Two (7%) of the 27 patients in our series had recent febrile illnesses. Epidemiologic clustering of outbreaks of PTS in association with antecedent viral or febrile illness has been reported in Czechoslovakia (20) and in an American Indian population in the southwestern part of the United States (21).

Prior reports have shown associations of PTS with recent surgery and anesthesia and with childbirth (1–5,22). Two (7%) of our study patients had recently undergone surgery and anesthesia, while another patient (4%) associated the onset of her symptoms with the otherwise uncomplicated vaginal birth of her son.

A genetic component might predispose an individual to PTS: A clinically similar but distinct inherited disorder called hereditary neuralgic amyotrophy has been described. This autosomal dominant disorder is characterized by recurrent, episodic, painful brachial neuropathy in association with mild dysmorphic features (23).

PTS has been reported in patients ranging in age from 3 months to 74 years, with the majority of cases being evenly distributed among patients in the 3rd–7th decades of life. In the two largest (to our knowledge) clinical series of patients with PTS, a male-to-female ratio of approximately 2:1 was observed (2,4). Our imaging study involved patients of a similar age range (12–81 years) and a similar male-to-female ratio (2:1). Similar to the 10%–20% of patients with bilateral PTS in the two large clinical series (2,4), three (11%) of the 27 patients in our study had bilateral PTS.

PTS may involve multiple nerves originating from the brachial plexus. The long thoracic, suprascapular, and

axillary nerves—individually or combined—reportedly have been the most commonly affected, according to clinical evaluation and electromyographic findings (1–6). In our study, the suprascapular nerve was almost invariably involved (in 97% of shoulders), and the axillary nerve was involved in 50% of shoulders. The long thoracic nerve was not found to be involved in any shoulders in this series; however, this nerve innervates the serratus anterior muscle, which is not well evaluated at routine shoulder MR imaging because it is largely excluded from the field of view. The subscapular nerve was involved in one shoulder (3%) in this series.

Eleven (41%) patients underwent electromyography, at which they all demonstrated neuropathies and plexopathies that matched or nearly matched the patterns of neurogenic high T2 signal intensity abnormality seen at MR imaging. Given that only one or two nerves were involved in most of our cases, PTS may actually be a mononeuropathy or polyneuropathy rather than a true plexopathy.

PTS is often confused clinically with more well-known and common disorders such as cervical spondylosis, rotator cuff tear, shoulder impingement syndrome, and acute calcific tendonitis (1,5,6,19). Therefore, knowledge of PTS and its characteristic imaging findings may enable the radiologist to be the first to suggest the correct diagnosis while helping to avert inappropriate therapy or even unnecessary surgery. Treatment of PTS is nonoperative and typically consists of analgesia coupled with physical therapy (1,19).

Our study had limitations. In general, the study was limited by its retrospective nature. Also, there was selection bias: The original imaging reports of the patients chosen for the study described specific findings that were identified by using a key term word search. Owing to the use of this restrictive entry criterion, which led to the exclusion of patients with PTS whose original imaging reports did not include our specific search terms, the occurrence of PTS within our study population may have been underestimated. It is also possible

that some patients with PTS may have been excluded owing to the lack of positive MR findings. Some positive-result examinations may have gone unrecognized at the time of initial interpretation and thus also may have been excluded. All of these limitations suggest that the presence of PTS in our study population might have been underestimated.

The lack of a definite reference standard for the diagnosis of PTS was another limitation. The diagnosis is made by identifying a pattern of muscular high T2 signal intensity abnormality that is consistent with involvement of a peripheral nerve (or nerves) originating from the brachial plexus. Then, other potential causes of this neurogenic high T2 signal intensity abnormality, such as entrapment neuropathy or trauma, must be excluded by performing clinical correlations and searching for additional imaging findings. The diagnosis of PTS in our study was made by consensus between the two authors after they reviewed the MR findings, referring clinicians' diagnoses, clinical histories, and physical examination and electromyographic results.

MR imaging reveals what is probably neurogenic high T2 signal intensity abnormality and atrophy involving the shoulder musculature in patients with PTS. In our study, the muscles innervated by the suprascapular nerve were almost invariably involved (in 97% of shoulders); however, axillary nerve involvement also was frequently observed (in 50% of shoulders). MR imaging can also help to exclude other causes of shoulder pain, such as rotator cuff tear, impingement syndrome, and entrapment neuropathy, all of which may be confused clinically with PTS and might require surgical management. For patients with PTS who undergo shoulder MR imaging, the radiologist may be the

first to suggest the correct diagnosis and subsequently affect both the prognosis and the treatment.

### References

- Helms CA, Martinez S, Speer KP. Acute brachial neuritis (Parsonage-Turner syndrome): MR imaging appearance—report of three cases. *Radiology* 1998;207:255–259.
- Parsonage MJ, Turner JW. Neuralgic amyotrophy: the shoulder-girdle syndrome. *Lancet* 1948;1:973–978.
- Spillane JD. Localised neuritis of the shoulder girdle: a report of 46 patients in the MEF. *Lancet* 1943;2:594–595.
- Tsairis P, Dyck PJ, Mulder DW. Natural history of brachial plexus neuropathy. *Arch Neurol* 1972;27:109–117.
- Misamore GW, Lehman DE. Parsonage-Turner syndrome (acute brachial neuritis). *J Bone Joint Surg Am* 1996;78:1405–1408.
- Dillin L, Hoaglund FT, Scheck M. Brachial neuritis. *J Bone Joint Surg Am* 1985;67:878–880.
- Bredella MA, Tirman PF, Fritz RC, Wischer TK, Stork A, Genant HK. Denervation syndromes of the shoulder girdle: MR imaging with electrophysiologic correlation. *Skeletal Radiol* 1999;28:567–572.
- Ryan M, Twair A, Nelson E, Brennan D, Eustace S. Whole body magnetic resonance imaging in the diagnosis of Parsonage-Turner syndrome. *Acta Radiol* 2004;45(5):534–539.
- Fleckenstein JL, Watumull D, Conner KE, et al. Denervated human skeletal muscle: MR imaging evaluation. *Radiology* 1993;187:213–218.
- Shabas D, Gerard G, Rossi D. Magnetic resonance imaging examination of denervated muscle. *Comput Radiol* 1987;11(1):9–13.
- Polak JF, Jolesz FA, Adams DF. Magnetic resonance imaging of skeletal muscle: prolongation of T1 and T2 subsequent to denervation. *Invest Radiol* 1988;23:365–369.
- Wessig C, Koltzenburg M, Reiners K, Solymsi L, Bendszus M. Muscle magnetic resonance imaging of denervation and reinnervation: correlation with electrophysiology and histology. *Exp Neurol* 2004;185:254–261.
- Fritz RC, Helms CA, Steinbach LS, Genant HK. Suprascapular nerve entrapment: evaluation with MR imaging. *Radiology* 1992;182:437–444.
- Linker CS, Helms CA, Fritz RC. Quadrilateral space syndrome: findings at MR imaging. *Radiology* 1993;188:675–676.
- Bayramoglu A, Demiryurek D, Tuccar E, et al. Variations in anatomy at the suprascapular notch possibly causing suprascapular nerve entrapment: an anatomical study. *Knee Surg Sports Traumatol Arthrosc* 2003;11:393–398.
- Cummins CA, Bowen M, Anderson K, Messer T. Suprascapular nerve entrapment at the spinoglenoid notch in a professional baseball pitcher. *Am J Sports Med* 1999;27(6):810–812.
- Ferretti A, De Carli A, Fontana M. Injury of the suprascapular nerve at the spinoglenoid notch (the natural history of infraspinatus atrophy in volleyball players). *Am J Sports Med* 1998;26(6):759–763.
- Ludig T, Walter F, Chapuis D, Mole D, Roland J, Blum A. MR imaging evaluation of suprascapular nerve entrapment. *Eur Radiol* 2001;11:2161–2169.
- Aymond JK, Goldner JL, Hardaker WT Jr. Neuralgic amyotrophy. *Orthop Rev* 1989;18(12):1275–1279.
- Bardos V, Somodská V. Epidemiologic study of a brachial plexus neuritis outbreak in northeast Czechoslovakia. *World Neurol* 1961;2:973–979.
- Auge WK. Parsonage-Turner syndrome in the native American Indian. *J Shoulder Elbow Surg* 2000;9(2):99–103.
- Simon JP, Fabry G. Parsonage-Turner syndrome after total-hip arthroplasty. *J Arthroplasty* 2001;16(4):518–520.
- Jeannot PY, Watts GD, Bird TD, Chance PF. Craniofacial and cutaneous findings expand the phenotype of hereditary neuralgic amyotrophy. *Neurology* 2001;57(11):1963–1968.