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CASE REPORT

Parsonage-Turner Syndrome: an Uncommon Cause of Shoulder Pain Diagnosed by Magnetic Resonance Imaging

SG Jakhere, D Yadav, P Narekar

Department of Radiology, BYL Nair Charitable Hospital, Mumbai Central, Mumbai – 400008, India

ABSTRACT

The clinical presentation of Parsonage-Turner syndrome often entails acute-onset shoulder pain, which may be confused with other entities such as rotator cuff tear, acute calcific tendonitis, adhesive capsulitis, cervical spondylosis, peripheral compressive neuropathy, acute poliomyelitis, and amyotrophic lateral sclerosis. Although it is a well-known entity, its appearance on magnetic resonance imaging has rarely been described. Radiologists should be familiar with this entity as radiological features might provide the first clue to the diagnosis. Multiple aetiological factors have been proposed to explain the underlying mechanism including a post-viral or autoimmune process, drugs, injections, burns and anaesthesia, though the exact pathogenesis still remains unclear. We present a case of a patient referred for magnetic resonance imaging of the shoulder to evaluate shoulder pain who was finally diagnosed to have Parsonage-Turner syndrome on the basis of the classical magnetic resonance imaging findings.

Key Words: *Brachial plexus neuritis; Magnetic resonance imaging; Muscle, skeletal; Shoulder*

中文摘要

臂叢神經炎：用磁共振成像診斷肩膊痛的一個罕見病因

SG Jakhere、D Yadav、P Narekar

臂叢神經炎起病通常為急性肩膊疼痛，所以很容易與其他疾病混淆，如肩旋轉袖撕裂裂、急性鈣化性肌腱炎、黏連性關節囊炎、頸椎病、周邊神經壓迫性病變、急性脊髓灰質炎及肌萎縮性脊髓側索硬化症。雖然臂叢神經炎是一種已知的病，文獻中很少提及此病的磁共振影像。由於這些影像可能會為診斷提供第一線索，放射科醫生應熟識這些影像特徵。雖然至今仍未能準確掌握其發病機制，但亦曾提出多個不同的病因去解釋這病，包括病毒感染後或自身免疫過程、藥物、注射、燒傷和麻醉。本文報告一名有肩膊痛的病人被轉介接受磁共振成像檢查，最終因其典型的磁共振影像而被診斷出患有臂叢神經炎。

INTRODUCTION

Parsonage-Turner syndrome (PTS), also called acute brachial neuritis / neuralgic amyotrophy, is an uncommon cause of shoulder pain. It is very often confused with other causes of shoulder pain including rotator cuff tear, acute calcific tendonitis, adhesive capsulitis, cervical spondylosis, peripheral compressive neuropathy, acute poliomyelitis, and amyotrophic lateral sclerosis.[1] Therefore failure to recognise PTS as a cause of shoulder pain may result in inappropriate

Correspondence: *Dr Sandeep G Jakhere, Department of Radiology, BYL Nair Charitable Hospital, Mumbai Central, Mumbai – 400008, India.*
Tel: 9920469297 ; Email: drsandeepjakhere@gmail.com

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treatment or even unnecessary surgery. PTS is a well-known clinical entity in the medical literature. Its magnetic resonance imaging (MRI) appearances, however, have been described in the past 10 years only, which may be related to the late incorporation of fat-saturated sequences in routine shoulder MRI protocols.[2] We present a patient referred for MRI of the shoulder to evaluate acute shoulder pain and was finally diagnosed to have PTS based on his classical clinical and MRI findings.

CASE REPORT

A 40-year-old male presented with increasingly severe left shoulder pain in October 2009 for the last one month. Two months earlier he had fallen on his back, but received no treatment. On examination the patient had full range of shoulder joint movements and there was no tenderness on palpation. Radiographs of the cervical spine and the shoulder were within normal limits. An MRI was requested to rule out rotator cuff pathology and revealed diffusely increased signals within the muscle bellies of supraspinatus and the infraspinatus muscles on the T2 and proton density fat-saturated images, which were consistent with muscle oedema (Figure 1). The rest of the rotator cuff muscles and tendons appeared normal in signal intensity and did not show any evidence of a tear. There was no evidence of bursitis in the peri-articular bursae. The articular cartilage and the bones forming the shoulder joint were normal. There was no evidence of any mass causing extrinsic compression at the suprascapular notch or along the course of the suprascapular nerve. Brachial plexus imaging was unremarkable. On the basis of the findings limited to the supraspinatus and infraspinatus muscles (both supplied by the suprascapular nerve), a diagnosis of viral neuritis affecting the suprascapular nerve (PTS) was made (Figures 2, 3). The patient

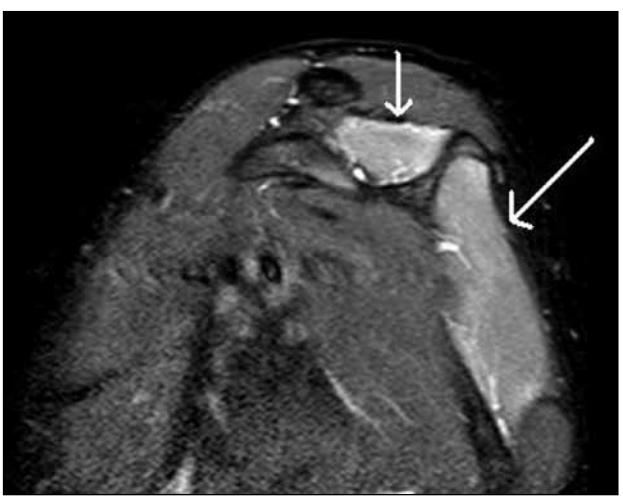

Figure 1. A sagittal T2-weighted fat-saturated image showing hyperintense signal limited to the supraspinatus (short arrow) and the infraspinatus muscle (long arrow). Note that the rest of the rotator cuff muscles show normal signal intensity.

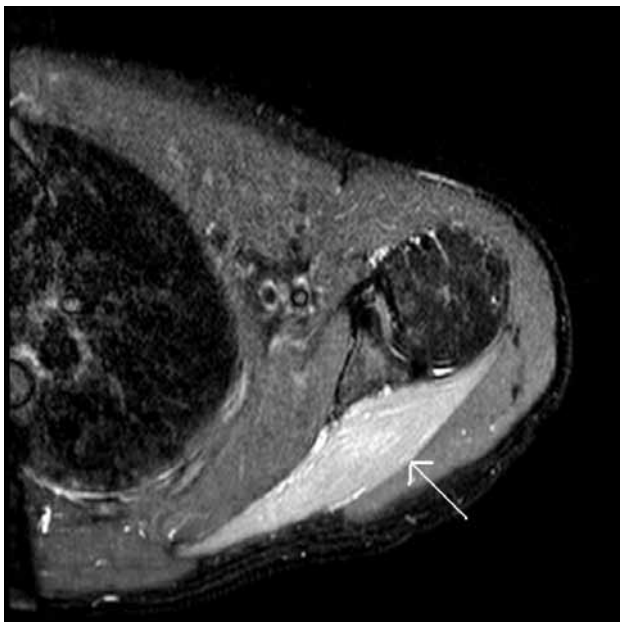

Figure 2. An axial T2-weighted fat-saturated image showing hyperintense signal limited to the infraspinatus muscle (arrow).

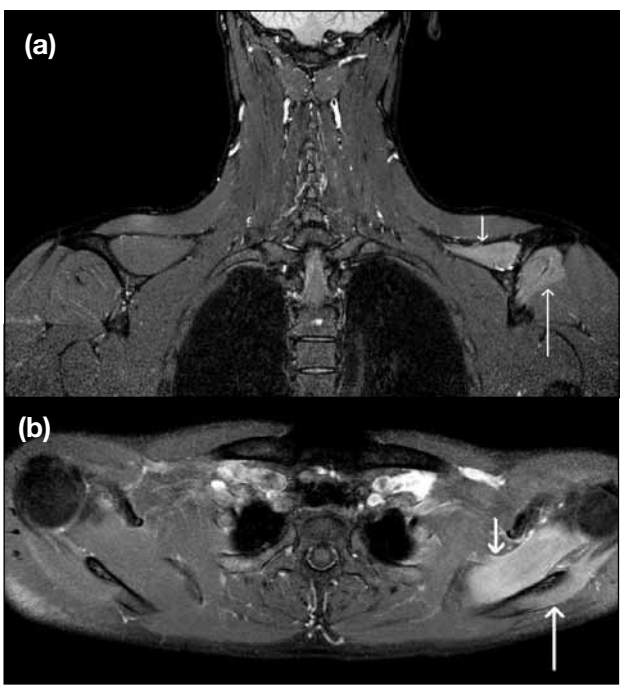

Figure 3. (a) Coronal and (b) axial T2-weighted fat-saturated images showing hyperintense signal limited to the supraspinatus (short arrow) and the infraspinatus muscles (long arrow) in the left shoulder, comparing with the normal signal intensity in the supraspinatus and infraspinatus muscles on the opposite side.

was managed conservatively without any operative intervention. At a follow-up after two months, the pain was considerably less and there was no residual weakness in the affected muscle groups. The patient was offered electromyography, but he declined the test.

DISCUSSION

PTS is a rare clinical entity with an incidence of approximately 1.6 cases per 100,000 inhabitants per year and affects males more than females in a ratio of 2:1. The peak incidence is between third and seventh decade of life.[3] In the largest case series to date, Scalf et al[4] reported involvement of the right shoulder in 46%, the left shoulder in 46%, and bilateral involvement in 8% of the patients. The first case of this uncommon disorder in English literature was reported in 1943 by Spillane,[5] who described the distinct clinical findings in 46 patients with PTS. However, it was only in 1948 that Parsonage and Turner[6] described 136 cases of brachial neuritis and the name for the syndrome that bears their name was coined.

Clinically patients present with sudden-onset severe shoulder pain which lasts hours to weeks and then subsides gradually. Atrophy of shoulder girdle muscles and weakness occurs approximately three to four weeks after the initial symptoms. After the pain subsides, 75% of those afflicted are left with severe weakness in muscles innervated by the upper trunk of brachial plexus. However, full motor recovery can be expected in 36% of the patients at one year, 75% at two years, and 89% at three years.[7] Sensory complaints are usually not prominent but have been noted in 66% of patients in another study.[8] The suprascapular nerve is the commonest peripheral nerve to be involved in the shoulder girdle, 97% of the patients being affected, while the axillary nerve is affected in about 50% of the cases.[2] Involvement of the long thoracic, musculocutaneous, anterior interosseous, ulnar, median, and phrenic nerve has also been described.[9] Rarely the lumbar nerve plexus may also be involved.[10]

The exact cause of the syndrome is unclear, but the commonest aetiology proposed is a post-viral or autoimmune process. In one study, almost 25% of patients had experienced a systemic viral illness prior to the onset of symptoms and 15% had a history of having received vaccination in the recent past.[7] Other putative aetiological factors include drugs,[11] anaesthesia, burns,[12] surgery,[13] injections,[14] rheumatoid arthritis,[15] and giant cell arteritis.[16]

A rare hereditary form of neuralgic amyotrophy has been reported in two siblings who presented with recurrent episodes of unilateral brachial neuritis. Hereditary neuralgic amyotrophy is a rare form of brachial neuritis whose phenotypic spectrum may include hypotelorism, cleft palate, and other dysmorphic features. The condition maps to chromosome 17q25 and is associated with mutations in the *SEPT9* gene. Regrettably, an episode of neuralgic pain was erroneously diagnosed as pronatio dolorosa (radial head subluxation) and treated with epiphysiolysis of the capitellum humeri.[17]

Electromyographic studies are fundamental for the diagnosis of PTS to determine the lesion type (axonal vs. demyelinating) and for evaluation of cases that are subclinical or very early involvement, even though MRI may not reveal the typical denervation oedema pattern.

MRI findings typical of PTS include diffusely increased signal intensity in a group of muscles of the rotator cuff, which are innervated by one or more peripheral nerves originating from the brachial plexus. This diffusely increased signal intensity is best depicted on T2-weighted fat-saturated or short T1 inversion recovery images, although T1-weighted images may also be useful to demonstrate muscle atrophy and fatty infiltration of muscles that appears during the late stage. The increase in the muscle signal intensity has been attributed to increased capillary blood volume in a partially denervated muscle.[18] MRI may show normal signal intensity in the muscle, if imaging is carried out within the first two weeks of symptom onset. In controlled experimental studies, however, subtle increases in signal intensity have been reported as early as 48 hours after symptom onset.[18] Although high signal intensity limited to muscles supplied by one or more peripheral nerves is characteristic of neurogenic oedema, other causes like myositis, myopathy, and tumour need to be excluded by imaging or clinical findings. High MRI signal intensity affecting a specific group of muscles may also be due to entrapment neuropathies, of which the commonest involves the suprascapular nerve. The suprascapular nerve gets compressed in the supraglenoid notch due to external compression by masses like synovial cysts and tumours. A few anatomic variations (thickened superior transverse scapular ligament, hypertrophied subscapularis muscles, an anterior coracoscapular ligament, and a narrow spinoglenoid or suprascapular notch) can also produce similar entrapment.[2] MRI can

show the normal anatomy and any compressive lesions in the course of the affected nerve exquisitely, and thus help exclude other causes of the denervation oedema.

In conclusion, MRI is an important imaging tool for the diagnosis of PTS and to exclude other causes of shoulder pain and denervation oedema.

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