

Eye On Imaging

UNCOMMON CAUSES OF SHOULDER PAIN

Most patients with shoulder pain have rotator cuff disease, impingement syndromes, acromioclavicular joint arthropathy and/or cervical spondylosis. There are, however, several other unusual causes of shoulder pain in which imaging plays a critical role in diagnosis.

PARSONAGE-TURNER SYNDROME

Patients with Parsonage-Turner syndrome (PTS), also known as acute brachial plexopathy, present with acute onset of severe shoulder pain and weakness. Ten to twenty-five percent of patients report they had undergone a vaccination or had had an infection prior to the onset of pain, while 20% of patients have bilateral symptoms.

The key MR finding in PTS is a pattern of diffuse high signal intensity (T2 images) in muscles innervated by the affected nerves (Figure 1). In one study, 97% of 30 study patients had high T2 signal indicating involvement of the suprascapular nerve (supraspinatus and infraspinatus muscle edema). Fifty percent of these 30 patients had involvement limited to this single nerve. The axillary, long thoracic and subscapularis nerves have been reported as involved.

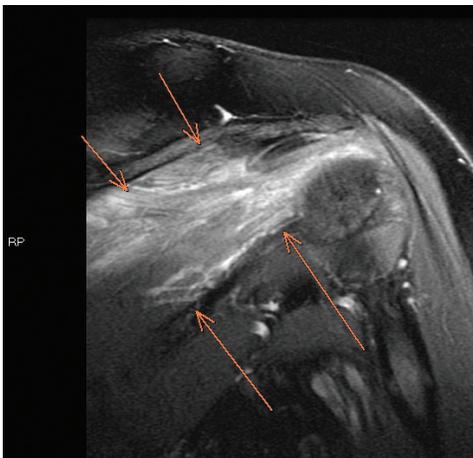


Figure 1 PTS Coronal MR T2 image. The arrows define a markedly edematous infraspinatus muscle. It is the only finding.

T2 weighted, fat saturated sequences will detect intramuscular edema, the earliest finding in PTS, occurring within several days of the onset of symptoms. Atrophy of the involved muscles appears later, characterized by zones of fatty infiltration (T1 images). This combination of edema and atrophy is characteristic of a denervation-type injury.

The long-term prognosis is good. PTS is self-limited with no specific treatment other than palliative measures. Typically, pain subsides over weeks to months, and 80% of patients have fully recovered by two years.

The differential considerations of PTS include suprascapular neuropathy secondary to excessive overhead activities, primary myopathy, tumor or calcific peri-arthritis (see below).

QUADRILATERAL SPACE SYNDROME

Quadrilateral space syndrome (QSS) is a painful disorder of the shoulder affecting both males and females between 20 and 40 years of age. The quadrilateral space is defined by the teres minor above, the teres major below, the long head of the triceps medially and the humeral shaft laterally. Both the posterior humeral circumflex artery and the axillary nerve lie in this space.

The presumed etiology of QSS is the presence of fibrous bands in the quadrilateral space, thought to be a result of prior trauma. The bands produce compression of both the nerve and artery, but most investigators believe that the pain of QSS is due to compression of the axillary nerve, rather than ischemia from the arterial occlusion. The axillary nerve innervates both the teres minor and the deltoid, but it is the teres that is uniformly affected.

The MR findings consist of selective atrophy of the teres minor (or, on rare occasion, the teres minor and the deltoid) (Figure 2). There is no significant edema of the involved muscles. There are no reports of visualization of the fibrous bands by MR.



Figure 2A QSS The arrows point to the teres minor which demonstrates a mild degree of fatty infiltration, accounting for the linear zones of bright fat and the relatively small size of the muscle.

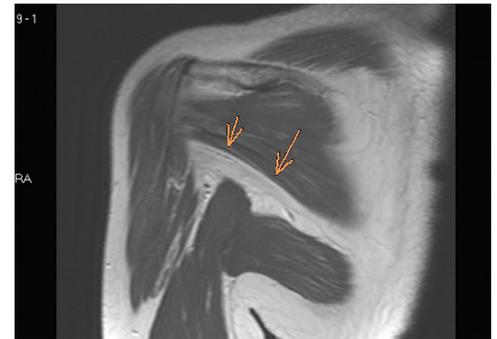


Figure 2B+2C QSS Coronal (B) and Sagittal (C) T1 images in another patient with more advanced disease demonstrate complete fatty replacement of the teres minor muscle (arrows). There is no edema.

Most patients are treated conservatively and only rarely require surgical decompression. Of 18 patients who underwent surgery, eight had dramatic and complete relief of their symptoms, eight improved and two showed no improvement.

BIOGRAPHY

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Jerrold Mink M.D., has written more than forty original articles that have been published in the radiology, orthopedic, sports medicine, and rheumatology literature. In addition, he has co-authored four textbooks on the musculoskeletal applications of MRI including the first specialty texts on the knee and foot and ankle.

Vu Bui M.D., was recruited to Mink Radiology from the University of Colorado Health Sciences Center. Prior to that, he had taken a musculoskeletal fellowship at the Brigham and Women's Hospital in Boston. In addition to Dr. Bui's expertise in musculoskeletal imaging, he has extensive experience with musculoskeletal interventional procedures such as spinal interventional procedures and biopsies.

CALCIUM HYDROXYAPATITE CRYSTAL DEPOSITION DISEASE (HA)

The presence of various crystals within and around joints has been identified; these include urates, calcium pyrophosphate dihydrate (CPPD), and crystalline steroid preparations. Most recently, HA crystals have been identified in periarticular and synovial tissues.

Patients with HA disease typically present with painful calcific deposits in tendons and soft tissues. Such depositions may best be termed calcific periartthritis although a large number of names for the disorder have been used. Men and women between 40 and 70 years of age are typically affected; the shoulder is the most common site of involvement, although the cervical spine, the hip and the elbow have been affected.

Conventional radiographic features of HA include thin cloud-like zones of calcification blending into surrounding soft tissues. With time, they appear denser and more sharply delimited. Such deposits may rapidly enlarge and change shape as a result of extrusion of the material into the subacromial bursa. Spontaneous diminution in size and disappearance of the calcification can occur.

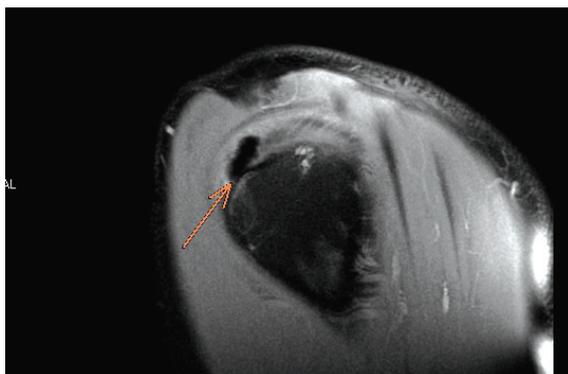


Figure 3A HA In this sagittal T2 image, a large focus of dark calcification (arrow) is present near the footprint of the supraspinatus without significant soft tissue edema. Such lesions are often asymptomatic.

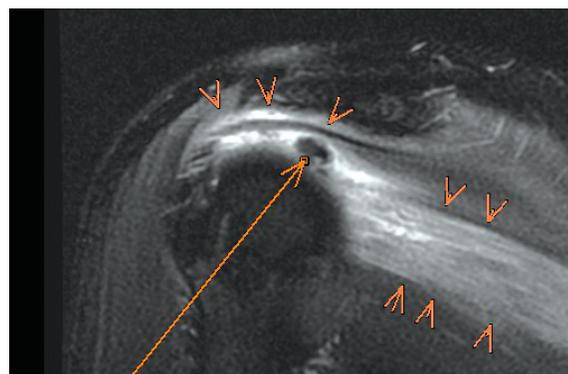


Figure 3B HA Coronal T2 image demonstrates edema (arrowheads) in the infraspinatus muscle, surrounding a large focus of calcification (arrow).



Figure 3C HA In another patient, edema is present around the low signal foci of calcification (arrows), and within the adjacent marrow of the humerus (arrowheads).

The proposed etiology of HA disease is calcium deposition in areas of necrosis or hypovascularity. The typical site of deposition is at the "critical zone" of the supraspinatus, just proximal to the insertion point where the vascular supply is somewhat tenuous. There is a subgroup of patients who demonstrate bilateral and/or polyarticular distribution, suggesting that metabolic (hyperparathyroidism and renal disease) and perhaps genetic factors may be involved in the more "systemic" appearing cases in which multiple sites are affected synchronously.

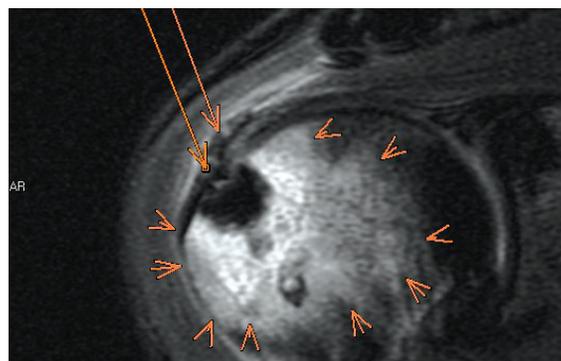


Figure 3D HA On this T2 coronal image, a dark focus of HA (arrows) is present in the greater tuberosity surrounded by intense edema in the adjacent marrow (arrowheads).

The clinical history of HA disease includes a silent phase (Figure 3A), and a mechanical phase in which the calcific deposit increases in size and may rupture into the subacromial bursa. There is typically extensive surrounding soft tissue reaction in muscle and fascia (Figure 3B + C) and it is at this point that percutaneous aspiration and injection of steroid is of greatest value. Intraosseous penetration may result in cystic and calcified lesions (Figure 3D).

Differential considerations of periarticular calcification include collagen vascular disease, renal osteodystrophy, idiopathic tumoral calcinosis, gout, myositis ossificans or trauma.

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2. Gaskin, C. Parsonage Turner syndrome. Radiology August 2006 240:501-507
3. Resnick, D. Diagnosis of Bone and Joint Disorders 2002; 1619