Soft tissue mass around the shoulder

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Clinical history

A previously fit 47 year old female school teacher presented with a six month history of a painful swelling over her right shoulder. There was rapid development of the swelling initially, which then stabilised. On examination she was afebrile with a large, firm, non-tender mass around the right shoulder, which clinically had some cystic features. There was no significant limitation of movement. Other findings on clinical examination included some minor soft tissue capsular swelling of the second and third metacarpophalangeal joints of the right hand. Otherwise the remainder of the locomotor system was normal.

Her erythrocyte sedimentation rate was increased at 51 mm 1st h, but C reactive protein remained normal at <0.6. Full blood count was normal with a haemoglobin of 13.4 g/dl, white blood cell count $7.5 \times 10^9/l$, platelet count $424 \times 10^9/l$. The rheumatoid factor, however, was positive with a titre of 1:1280.

Imaging

QUESTIONS

- (1) What does the plain film (fig 1) show? Suggest a list of diagnoses that would fit this clinical context.
- (2) Which of your diagnoses does ultrasound (fig 2) support?
- (3) Describe the magnetic resonance imaging findings (figs 3-5).
- (4) Using all the evidence what are the most probable diagnoses?

Plain film

There is a large, homogeneous soft tissue mass superolateral to the right shoulder joint, in the region of the subdeltoid bursa. There is no calcification or ossification within the mass. Irregularity of the inferior surface of the acromion and the greater tuberosity suggests some degree of impingement syndrome otherwise there is no bony or joint abnormality.

The diagnostic possibilities in this case can be divided into those conditions arising from the synovium of the joint or subacromial bursa and those arising from muscle or related tissues. Synovial based lesions such as pigmented villonodular synovitis (PVNS) can present as a soft tissue mass, as can synovial osteochondromatosis or soft tissue chondromas. Synovial sarcomas are rare malignant

tumours but characteristically arise adjacent to joints and grow slowly. With the exception of PVNS, all these conditions typically show some degree of calcification on plain film.¹ Most cases of synovial osteochondromatosis show a pattern of coarse calcification and one third of cases of synovial sarcomas show spotty calcification.¹ Cystic lesions such as a ganglion or synovial cyst can occasionally reach this size.

A lipoma would fit the clinical context but fat is of lower density on plain film than muscle and consequently would appear blacker on plain film. Other benign neoplasms such as a haemangioma or an angiolipoma could give this appearance and sarcoma has to be considered.

Ultrasound

The ultrasound examination shows that the soft tissue mass is located inferior to the acromion and deltoid, and superior to the greater tuberosity and supraspinatus tendon. This is the region of the subdeltoid bursa. It contains multiple heterogeneous echoes, indicating that it is either a solid mass or a complex cyst. Ultrasound has the advantage of being a

Figure 1 Anteroposterior radiograph of the right shoulder.

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Figure 2 Ultrasound showing transverse section through the right shoulder.

Figure 3 Coronal T1 weighted image of the right shoulder.

dynamic test and differentiation between a solid or complex cystic lesion can sometimes be made by watching the effect of pressure on its components. In this case, as indeed was suggested clinically, the lesion was thought to be a complex cyst, thus favouring the diagnosis of synovial osteochondromatosis or PVNS. However, the ultrasound does not definitely exclude a synovial sarcoma or a liposarcoma (that is, an extra-bursal lesion).

Fat is characteristically reflective which reinforces the conclusion reached on the plain film that this is not a simple lipoma.

Magnetic resonance imaging (MRI)

Figure 3, a coronal T1 weighted image, shows gross enlargement of the subacromial subdeltoid bursa. The lumen is filled with intermediate signal intensity material, which on the T2 weighted axial images (fig 4) is seen more clearly to represent multiple filling defects outlined by the high signal of fluid within the bursa. The filling defects themselves are intermediate signal on T2 weighting. After gadolinium injection (fig 5) the synovial lining of the bursa enhances but no change occurs in the appearance of the filling defects.

Other important points are that the glenohumeral and acromioclavicular joint, seen in

Figure 4 Axial T2 weighted image of the right shoulder.

Figure 5 Gadolinium DTPA enhanced axial T1 weighted image of the right shoulder.

the coronal plane in figure 3, appear uninvolved and the rotator cuff intact.

Differential diagnoses

SYNOVIAL STEOCHONDROMATOSIS

This is most commonly a monoarticular process, which presents in the third to fifth decade with a 2:1 male preponderance. The condition is thought to be caused by a disorder of the metaplasia of the synovial membrane.¹ Radiologically most cases exhibit calcification on the plain film and therefore this case would be atypical.

PIGMENTED VILLONODULAR SYNOVITIS

This is an unusual proliferative disorder of synovium, which typically presents as a non-painful soft tissue mass arising in the synovial lining of joints, tendon sheaths, fascial planes or ligaments. Calcification is not a feature and the ultrasound appearances would be similar to figure 2. MRI, however, characteristically shows areas of very low signal on T2 weighted images, which represent haemosiderin deposition.²

Operative findings

The subacromial bursa was lined by proliferative synovium and filled with numerous loose bodies (fig 6).

Histological examination showed thickening of the synovial membrane with intimal hyperplasia and a heavy subintimal infiltrate of numerous lymphocytes and plasma cells. Giant cells and lymphoid aggregates were also evident in the deeper subintima.

The loose bodies were composed almost entirely of fibrin with surrounding organising cellular fibrous tissue.

Figure 6 (A) and (B) Operative findings showing multiple rice bodies. One of the largest fragments contained multiple smaller rice bodies.

Final diagnosis

Multiple rice bodies (fibrin bodies) in the sub-acromial bursa caused by rheumatoid disease.

Clinical outcome

The patient made a good recovery after the synovectomy and has had no recurrence of her symptoms. She has not required any further treatment.

Discussion

Rice bodies were originally described by Reise in 1895³ in association with tuberculous arthritis. They were called rice bodies because of their macroscopic similarity to grains of polished white rice. In 1993 Stein *et al*⁴ reported a case that first described the MRI appearances of subacromial bursitis with massive rice body formation.

In 1982, Popert *et al*⁵ studied the frequency of rice bodies occurring in seropositive and seronegative rheumatoid arthritis. In the seropositive group, 72% of 50 joints aspirated using a large bore needle, contained rice bodies. None were seen in the 31 joints of the seronegative group. There are only case reports elsewhere in the literature of rice bodies occurring in seronegative arthritis⁶ and also in hypogammaglobulinaemic arthritis.⁷

Rice bodies vary in size from 2–7 mm (55%) to greater than 7 mm (10%).⁵ In 1977, Berg *et al*⁸ divided rice bodies into two types. Both type 1 and type 2 consist of amorphous or coarsely reticular and condensed fibrin but type 2 also contain a core of mature collagen. It has been suggested that with increasing age, rice bodies undergo a degree of organisation and begin to resemble mature connective tissue.⁵

The precise aetiology of rice bodies remains controversial. In the later stages of rheumatoid arthritis, the total synovial surface area is vastly increased by proliferation and hypertrophy of synovial villi. Fassbender *et al*⁹ suggest that fibrin accumulates in these villous structures, which then become elongated and snap off.

Popert *et al*⁵ argue that it is the precipitation of fibronectin and fibrin, stimulated by the glycosaminoglycans released from eroded cartilage, which is responsible for rice body formation. This theory seems less probable in our case, where the glenohumeral joint was normal both on imaging and at surgery.

A further theory, put forward by McCarty and Cheung, suggests that microcirculatory deficiencies within the synovium and consequent hypoxia, result in synovial microinfarcts.¹⁰ The infarcted fragments then drop off into the joint cavity and their surface becomes covered by fibrin layers.

The final and perhaps most important question is what is the clinical relevance of rice bodies? It is known that fibrin itself is irritant. Therefore rice bodies themselves have been implicated as one of the stimuli for continuing synovial inflammation.¹¹ It is certainly true that their effective removal can produce clinical improvement.⁵ Glynn has gone a step further to suggest that persistent fibrin deposits may provide a continuous stimulus to antibody formation to other changed body constituents.¹²

Conclusion

The imaging characteristics of a huge collection of rice bodies on plain film, ultrasonography, and MRI have been demonstrated. Although uncommon, we suggest that this diagnosis should be borne in mind as a differential of atypical synovial osteochondromatosis.

- 1 Madewell J E, Sweet D E. Tumours and tumour-like lesions in or about joints. In: Resnick D. *Bone and joint imaging*. Philadelphia: W B Saunders, 1989:1185–6.
- 2 Reicher M A. The spectrum of knee joint disorders. In: Mink J H, Reicher M A, Crues J V, Deutsch A L. *MRI of the knee*. New York: Raven Press, 1993:347–50.
- 3 Reise H. Die Reiskörpchen in tuberculösen erkrankten synovialsäcken. *Dtsch Z Chir* 1895; 42:1–99.
- 4 Stein A J, Case J L, Berman J, Levy H. Case report 770. *Skeletal Radiol* 1993;22:71–3.
- 5 Popert A J, Scott D L, Wainwright A C, Walton K W, Williamson N, Chapman J H. Frequency of occurrence, mode of development, and significance of rice bodies in rheumatoid joints. *Ann Rheum Dis* 1982;41:109–17.
- 6 Thevenon A, Cochetoux P, Duquesnoy B, Mestelagh H, Lecomte-Houke M, Delcambre B. Subacromial bursitis with rice bodies as a presenting feature of seronegative rheumatoid arthritis. *Arthritis Rheum* 1987;30:715–16.
- 7 Taborn J D. Rice bodies in hypogammaglobulinaemic arthritis. *J Rheumatol* 1981;8:165–8.
- 8 Berg E, Wainwright R, Barton B, Puchtlen H, McDonald T. On the nature of rheumatoid rice bodies. An immunologic, histochemical and electron microscope study. *Arthritis Rheum* 1977;20:1343–9.
- 9 Fassbender H G. *Pathology of the rheumatic diseases*. Translated by Loewi G. Berlin: Springer-Verlag, 1975:114–15.
- 10 McCarty D J, Cheung H S. Origin and significance of rice bodies in synovial fluid. *Lancet* 1982;ii:715–16.
- 11 Curran R C. Mucopolysaccharides and collagen formation in atherosclerosis. In: Chalmers D J, Gresham G A, eds. *Biological aspects of occlusive vascular disease*. Cambridge: Cambridge University Press, 1964:76–83.
- 12 Glynn L E. The chronicity of inflammation and its significance in RA. *Ann Rheum Dis* 1968;27:105–21.