Ultrasound detection of knee patellar enthesitis: a comparison with magnetic resonance imaging

M Kamel, H Eid, R Mansour

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The diagnosis of enthesitis in clinical practice is difficult and usually based on conventional radiographic findings, which are not helpful in most cases.[1] We previously reported that ultrasound (US) was sensitive in detecting peculiar pathological features of enthesitis around the heel.[2] Furthermore, we have continued to study the efficacy of ultrasonographic diagnosis of enthesitis of other tendon and ligament insertion sites.

METHODS AND RESULTS

Sixteen patients (10 male, six female, mean age 45.6 years) with a diagnosis of seronegative arthropathy were recruited from the population for the study. Their mean disease duration was 6.3 years. They had seronegative arthropathy and knee enthesopathy without typical conventional radiographic evidence. An HDI 3000 ATL US machine (Advanced Technology Laboratories, USA), equipped with a 12 MHz linear transducer, was used to examine the knee patellar enthesis.

The present study produced interesting findings (figs 1A and B).The US images of the knee patellar enthesis showed loss of the normal fibrillar echo texture of the patellar tendon, no homogeneous pattern, blurring of the patellar tendon margins, irregular focal or generalised increased tendon thickness, and focal ill-defined tendon defects, with loss of their tightly packed echogenic dots. The US images clearly showed the definition of the patellar tendon margins, which were more precise and anatomically defined than the magnetic resonance (MR) images (figs 1C and D).

DISCUSSION

The US examination of the knee joint clearly detected the early calcification foci of the patellar tendons. However, the calcification process of the knee patellar ligament developed less often in the patients than the calcification of the Achilles tendon found in a previous report.[2 3]

The process of fatty degeneration of the patellar tendon was detected early in US images, and appears as hyperechoic intratendinous lesions.[4 5]

This study detected a significant thickening of the patellar tendon, which can be measured by US. We believe that this US feature is more sensitive and reliable in diagnosing early enthesitis than a classical MR high signal intensity image

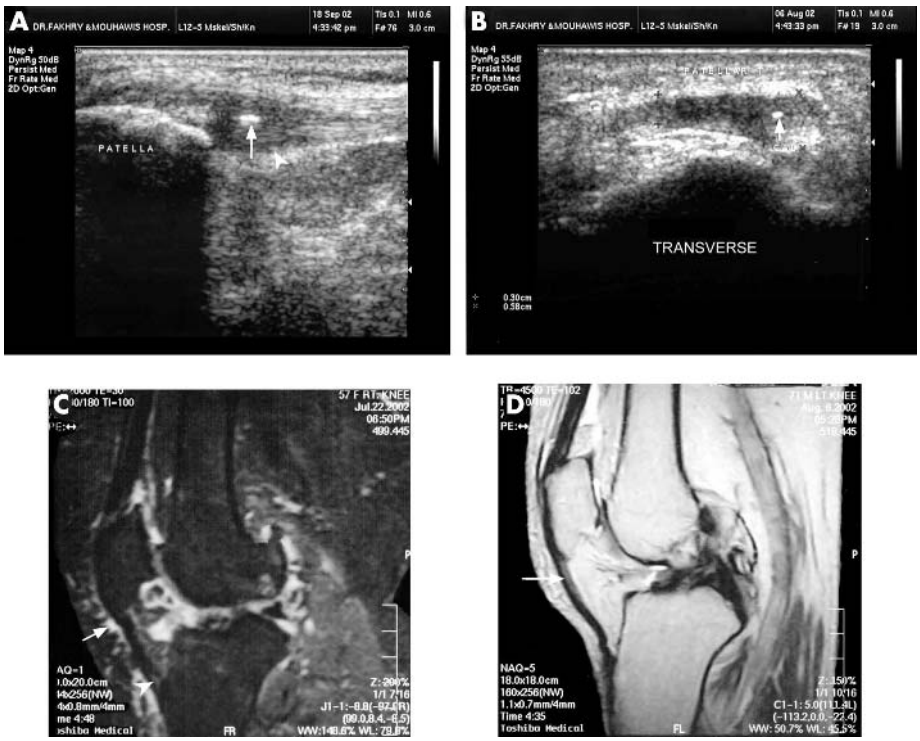

Figure 1 (A) A sagittal US scan shows thickened proximal entheses of the patellar ligament with loss of its fibrillar echo pattern, loss of the sharp definition of its posterior aspect compared with the distal portion (arrow head), calcific foci (arrow). (B) A transverse US scan of the same patient shows the thickened medial part of the patellar ligament with calcific focus (arrow). (C) A sagittal T_2 fat suppression image shows the thickened distal part of the patellar tendon with altered signal intensity (arrow head) and prepatellar bursitis (arrow). (D) A sagittal Pd weighted image shows high intensity signals of the proximal patellar tendon.

within the superior medial and central aspects of the patellar tendon at its proximal attachment. This interesting observation has been confirmed in other related studies.[6 7]

In conclusion, we found several pathological differences between the pattern of patellar enthesitis and that of Achilles tendon and plantar fascia of the heel. The entheseal changes of the patellar tendon occurred at the tibial or patellar insertion either on its medial or lateral aspect, but in the case of the Achilles tendon, the entheseal changes were detected only in the calcaneal insertion. The presences of Calcific foci were found more often in the Achilles tendon than in the patellar tendon. US is a valuable and sensitive diagnostic method in patients with seronegative spondyloarthropathy and knee enthesopathy who have normal findings with conventional radiological images of the knee joint.

Authors' affiliations
M Kamel, Al-Azhar University, Cairo, Egypt
R Mansour, Dr Fakhry and Almuhawis Hospital, Alkhobar, Saudi Arabia
H Eid, Menofyia University, Egypt

Correspondence to: Dr M Kamel; mkamel56@hotmail.com

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REFERENCES

1 **Danda D**, Shyam Kumar NK, Cherian R, Cherian AM. Enthesopathy: clinical recognition and significance. *Nat Med J India* Mar 2001;**14**:90–2.
2 **Kamel M**, Eid H, Mansour R. Ultrasonographic detection of enthesitis: a comparison with MRI. *J Rheumatol* 2003;**30**:774–8.
3 **Kamel M**, Moghazy K. Ultrasonographic detection of enthesitis: a comparison with MRI [abstract]. *Arthritis Rheum* 2001;**44**(suppl 9):95.
4 **Rawool Nm**, Nazarian LN. Ultrasound of the ankle and foot. *Semin Ultrasound CT MR* 2000;**21**:275–85.
5 **Gibbon WW**, Cooper JR, Radcliffe GS. Distribution of sonographically detected tendon abnormalities in patients with a clinical diagnosis of chronic achilles tendinosis. *J Clin Ultrasound* 2000;**28**:61–6.
6 **Reiff DB**, Heenan SD, Heron CW. MRI appearances of the asymptomatic patellar tendon on gradient echo imaging. *Skeletal Radiol* 1995;**24**:123–6.
7 **Yu JS**, Popp JE, Kaeding CC, Lucas J. Correlation of MR imaging and pathologic findings in athletes undergoing surgery for chronic patellar tendinitis. *AJR Am J Roentgenol* 1995;**165**:115–18.

Longlasting effects of immunoadsorption in severe Sjögren's syndrome

M Böhm, T Dörner, F Knebel, A Bruns, N Jochmann, G Baumann

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Despite advances in understanding the immunopathogenesis of Sjögren's syndrome, successful therapeutic interventions are extremely limited.

CASE REPORT

With this in mind we began a immunoadsorption treatment of a 38 year old woman, diagnosed 1.5 years ago with Sjögren's syndrome. She reported dry eyes and mouth for several months and intermittent arthralgias, especially of the small finger joints and elbows, as well as swollen joints of the hands, elbows, and ankles for six years. During the six months before treatment her joint symptoms had increased significantly and considerably impaired everyday activities. A Schirmer's test was positive. The antinuclear antibodies and rheumatoid factor were raised, autoantibodies against Ro/SSA and La/SSB were positive. Thus, the patient fulfilled four of six revised criteria of primary Sjögren's syndrome.[1]

Previous corticoid treatment (prednisolone 20 mg a day for four weeks) had led to oropharyngeal candida mycosis, and methotrexate (25 mg a week) did not have a therapeutic effect. Because chloroquine had side effects in her family, the patient refused to take hydroxychloroquine. Upon presentation in our clinic, the patient took 5 mg a day of prednisolone. Because the severe arthralgias and sicca symptoms did not respond to conventional treatment we started to treat the patient with immunoadsorption, to improve the symptoms by reducing IgG to 10–20% of its initial level.

Approval of the ethics committee and informed consent by the patient were obtained, and we started immunoadsorption therapy according to a previous protocol used for patients with dilated cardiomyopathy.[2 3]

Immunoadsorption treatment took place in two consecutive cycles with an interval of four weeks. The first cycle comprised three treatment days and the second, two. The plasma filtration was similar to haemodialysis. The IgG-Therasorb Adsorber and a Mirosorb treatment unit (Plasmaselect, Teterow, Germany) treated 7 litres plasma a day. Plasma IgG, antibody complexes, and fragments of antibodies were bound to the Fc fragment of polyclonal sheep antihuman antibodies, bound in turn to Sepharose.[4]

After the first treatment cycle, the patient showed remarkable clinical improvement, with lessening of arthalgias and articular swelling, and subsequent increased joint mobility. The score for tender/swollen joints[5] reduced from a value of 29 at the beginning to 0 at the end of the study. Complement factors C3 and C4 fell to 67% and 78% of baseline (0.8–0.6 g/l and 0.19–0.15 g/l), respectively. In addition, circulating immune complexes reduced to 29% (3.3–1.0 g/l) and the rheumatoid factor to 55% of initial value (2.5–1.4 g/l). The treatment reduced IgG from 24.31 to 4.88 g/l after the first cycle and from 22.72 to 10.04 g/l after the second cycle. The IgG level increased within 16 months to 34.70 g/l.

The changes during the second treatment cycle were less striking in their effect on circulating immune complexes (reduced to 53% (1.55–0.83 g/l)) and the rheumatoid factor (reduced to 92% of the initial level (6.56–6.09 g/l)). Increases were seen in the values of circulating immune complexes (from 0.83 to 0.96 g/l) and of rheumatoid factor (from 6.09 to 6.24 g/l) after 16 months.

As a result of the striking and sustained clinical benefit achieved, the treatment was discontinued after the second cycle of immunoadsorption.

DISCUSSION

To prevent a rebound and infection after immunoadsorption, the treatment protocol requires intravenous IgG substitution (0.5 g per kg body weight Venimmun) after each cycle, indicating that the patient's IgG has a significant role in the disease. We cannot exclude the possibility that immunoglobulin infusion may have had some effect on the outcome. This needs to be examined by additional studies.