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## Magnetic resonance imaging in the evaluation of treatment response of lateral epicondylitis of the elbow

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**Abstract** The purpose of this study was to investigate the treatment response in lateral epicondylitis (tennis elbow) by MRI. Magnetic resonance imaging was obtained in 30 patients with clinical symptoms of lateral epicondylitis of the elbow using T1-, T2- and T2-weighted fat-saturated (FS) sequences. The patients were randomised to either i.m. corticosteroid injection ( $n=16$ ) or immobilisation in a wrist splint ( $n=14$ ). Magnetic resonance imaging of the elbow was performed on a 1.5-T MR system at baseline and after 6 weeks. The extensor carpi radialis (ECRB) tendon, the radial collateral ligament, lateral humerus epicondyle at tendon insertion site, joint fluid and signal intensity changes within brachio-radialis and anconeus muscles were evaluated on the MR unit's workstation before and after 6 weeks of treatment. The MRI was performed once in 22 healthy controls for comparison and all images evaluated by an investigator blinded to the clinical status of the subjects. The MR images showed

thickening with separation of the ECRB tendon from the radial collateral ligament and abnormal signal change in 25 of the 30 patients on the T1-weighted sequences at inclusion. The signal intensity of the ECRB tendon was increased in 24 of the 30 patients with lateral epicondylitis of the elbow on the T2-weighted FS sequences. In the patients there were no associations between pathologically signal intensity within the ECRB tendon on T1- and T2-weighted sequences and the degree of self-reported pain (Dumbbells test) at inclusion. In general, the MRI changes persisted in the patients at follow-up after 6 weeks despite clinical remission. The increased signal intensity within the extensor tendon is indicative of lateral epicondylitis humeri. The changes in signal intensity and morphology of ECRB tendon seem to be chronic and may persist despite clinical improvement.

**Keywords** Epicondylitis · Treatment control · MRI

### Introduction

Lateral epicondylitis, also referred to as tennis elbow, is probably caused by degeneration, tendinosis and tearing most commonly of the extensor carpi radialis brevis (ECRB) tendon [1, 2, 3], but a variety of processes have been implicated in lateral epicondylitis, including affection of the extensor digitorum communis tendon [4].

The condition often occurs as a result of repetitive sports-related trauma but may be seen in non-athletes [5, 6]. Previous studies of histopathology have described fibrovascular proliferation, intratendinous calcification and cartilage formation, and fibrofatty degeneration and partial tendon rupture may be found [2, 7, 8, 9]. The same forces or mechanisms that lead to tendon derangement may also produce associated lateral collateral liga-

ment sprain or tear and scarring or degeneration [2, 7] and oedema of the anconeus muscle was found extensively in one study [10].

Magnetic resonance imaging is not needed initially but is indicated for differentiating the various contributing factors, assessing their severity and guiding further management [9, 11]. The MRI is more sensitive than ultrasonography in diagnosing lateral epicondylitis, but initially ultrasonography can be used as an imaging tool [11]. Sonographic evaluation of the elbow can include evaluation of joint fluid, guidance for aspiration, evaluation of the supporting tendons and ligaments, and limited evaluation of the articular cartilage.

The most frequent MRI findings in lateral epicondylitis is a abnormal signal change in the ECRB tendon and thickening with separation of the extensor carpi radialis tendon from the radial collateral ligament [2, 12, 13], caused by fibrosis, scarring and degeneration, including myxoid change, in the ECRB tendon origin [14]. Thickening or thinning and abnormal signal change of the extensor tendon was reported to be related to intratendinous oedema or partial or complete tears [2, 7, 15, 16]. Alterations in signal intensity in the extensor tendons, particularly on T2-weighted sequences, have been described in cases of lateral epicondylitis [7] with discrete fluid signal on T2-weighted sequences corresponding to focal tears [7, 15].

No single intervention has been universally efficacious [17]. No work has been published yet investigating the role of MRI in the evaluation of a treatment response.

The purpose of this study was to determine the value of MRI in lateral epicondylitis in a randomised-controlled design with either wrist immobilisation or i.m. corticosteroid injection.

## Materials and methods

A total of 30 patients with lateral epicondylitis (median age 46 years, age range 28–60 years) were included and randomised to treatment with either wrist immobilisation (splint/wrist brace;  $n=14$ ) or i.m. corticosteroid injection (20 mg methylprednisolone+10 mg lignocaine;  $n=16$ ). The patients had local tenderness over the lateral elbow region and increased pain in the extensor muscles induced by weight-bearing contraction of the extensor muscles at clinical examination. Duration of symptoms varied from 0.3 to 8 months. None of the patients had other causes of elbow pain at baseline examination and underwent steroid or local anaesthesia injections within 2 months prior to the MR examination. Clinical examination was performed prior to MRI, which was performed within a week. The MRI of the elbow region was obtained before and 6 weeks after treatment.

The MRI was performed once in 22 healthy controls (median age 40 years, age range 23–61 years) without symptoms of lateral epicondylitis.

The study was conducted in accordance with Helsinki Declaration II and approved by the local ethics committee. Informed written consent was obtained from all patients and healthy volunteers.

## MRI

Magnetic resonance imaging of the elbow was obtained on whole-body 1.5-T Gyroscan ACS-NT (Philips, Best, The Netherlands) using a 12-cm receive-only surface coil placed over the lateral elbow region. Coronal T1-weighted spin-echo sequences (TR/TE: 500 ms/15 ms), coronal and axial T2-weighted turbo spin-echo (TSE) sequences (TR/TE: 2000 ms/80–100 ms) and axial and coronal T2-weighted fat-saturated (FS; TR/TE: 1277–2922 ms/70–100 ms) sequences of the elbow were obtained. In 4 patients axial T2-weighted fast-field echo (FFE) sequences (TR/TE: 600 ms/15 ms) with a flip angle of 35° were obtained instead of T2-weighted FS sequences. Matrix size was 256×256, FOV was 160 mm and slice thickness was 3–4 mm with 0.3- to 0.4-mm gap. The patients were positioned supine with the arm along the side of the body and the elbow extended facing the lateral aspect of the femur.

## Image evaluation

Abnormal signal intensities of the extensor tendon were assessed mainly on the axial T2-weighted FS or FFE sequences on the MR-unit workstation. Thickening of the ECRB tendon, as well as an abnormal separation between the extensor tendon and the radial collateral ligament, were best identified on coronal T1-weighted sequences (Figs. 1a, 2a). The signal intensities of the ECRB tendon, brachio-radialis muscle and anconeus muscle were measured on the T2-weighted or FFE sequences with software installed on Philips workstation.

Two experienced investigators blinded to clinical status analysed independently all images obtained on MRI in 30 patients and 22 healthy controls.

## Clinical examination

All patients were examined by a rheumatologist and evaluated with a visual analogue 11-point pain box scale (VAS), grip strength, patient's global assessment on a scale 0–3 and Dumbbells test.

## Statistical analysis

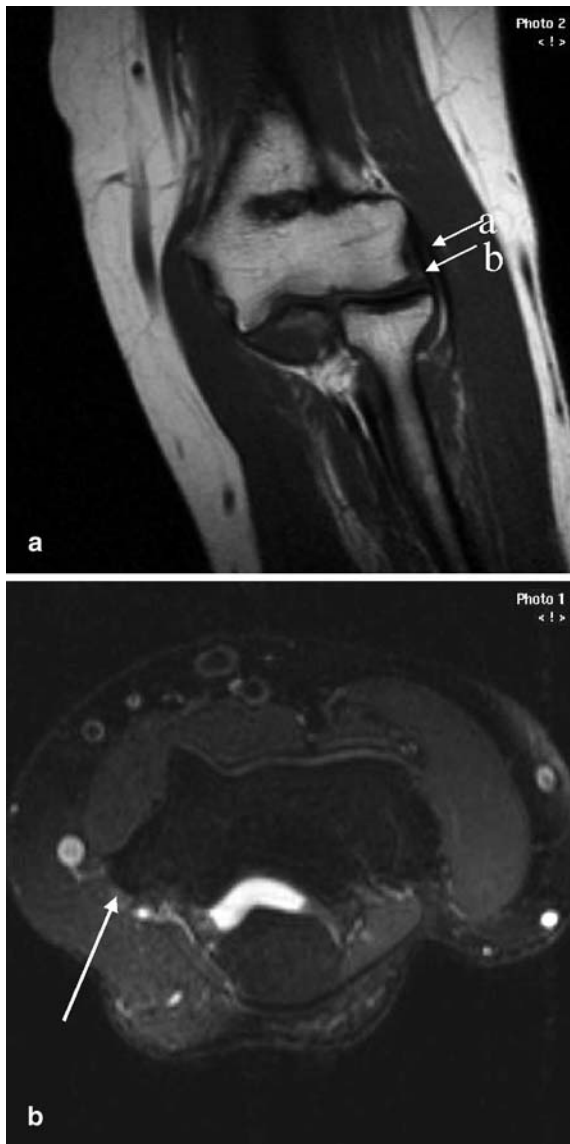
Both parametric and non-parametric tests were applied. The material was dichotomised according to the evaluation of the signal intensity (normal or abnormal) and the groups were formed and compared with respect to pain on VAS by Mann-Whitney test. Having tested the material by the Kolmogorov-Smirnov test, the signal intensities of ECRB were compared with un-paired (patients vs controls) and paired (before/after treatment) *t* test using SPSS software (version 10). Level of significance was set at 0.05.

## Results

### MRI at inclusion

At inclusion, thickening of the tendon with separation of the ECRB tendon from the radial collateral ligament and abnormal signal change was found in 25 of the 30 patients on the T1-weighted sequences (Figs. 1, 2a) and in 3 of the 22 controls (Fig. 3c).

On the T2-weighted FS images, abnormally high signal intensity was found in 24 of 30 patients (Figs. 2b,



**Fig. 1** **a** Coronal T1-weighted sequence in a 32-year-old female healthy control. *Arrows* indicate the extensor carpi radialis brevis tendon (*a arrow*) and the radial collateral ligament (*b arrow*). **b** Axial T2-weighted fat-saturated sequence (*arrow*)

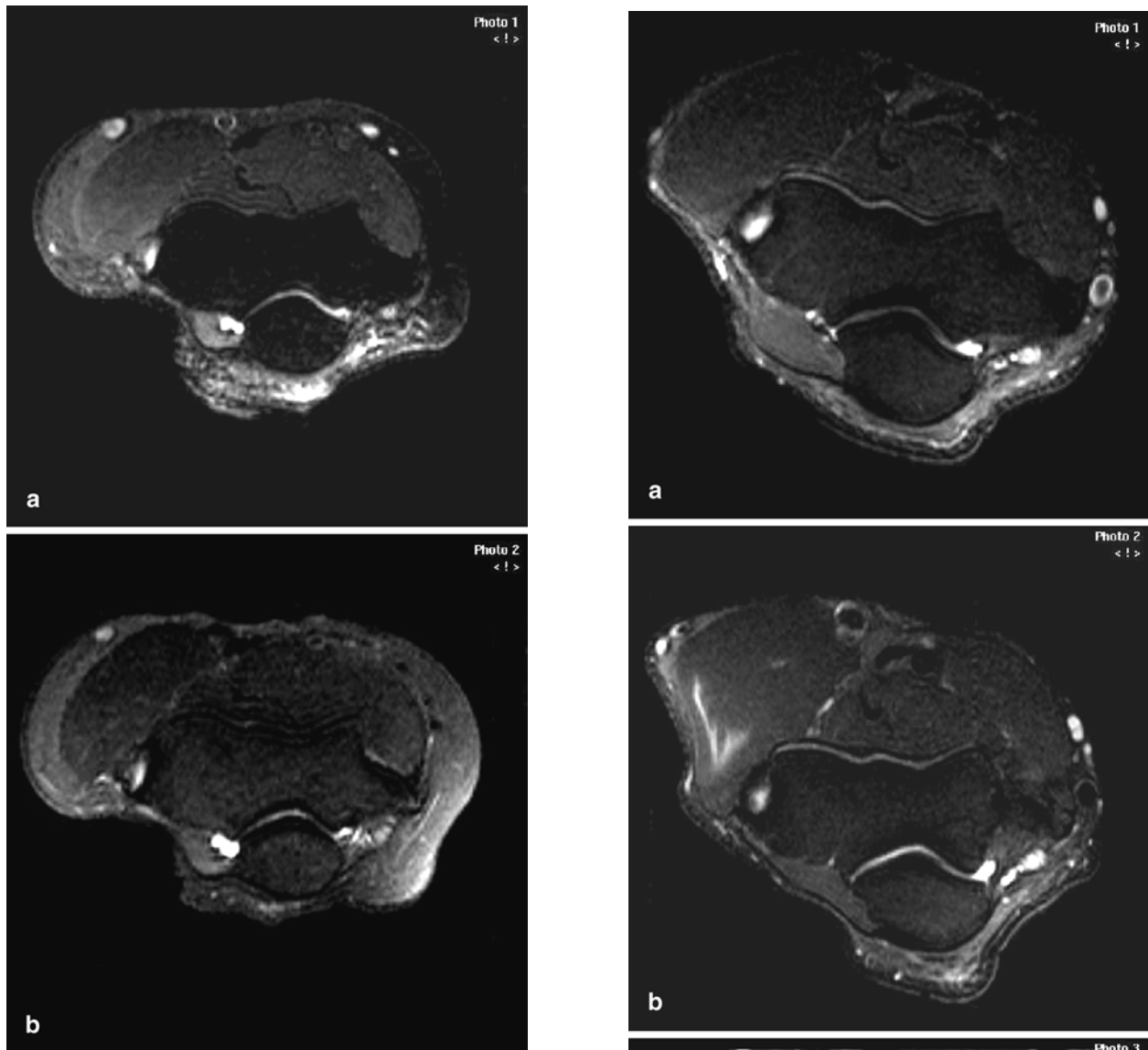
4b) and in 2 of 22 controls. In 2 patients with increased signal intensity of the ECRB tendon on the T2-weighted images the signal intensity changes of the ECRB tendon could not be reconfirmed on the T1-weighted sequences. Within the patient group, there was no indication of differences in pain level in the patients with and without abnormal signal intensity (Mann-Whiney test,  $p > 0.05$ ).



**Fig. 2** **a** Coronal T1-weighted sequence in a 60-year-old man with 6 months duration of symptoms. The signal intensity of the extensor carpi radialis brevis (ECRB) tendon is increased and is thickened. A separation is seen between the ECRB tendon and the radial collateral ligament (*arrow*). **b** Axial T2-weighted fat-saturated image with increased signal intensity at the origin of the ECRB tendon adjacent to humerus (*arrow*)

The signal intensity of the anconeus and brachio-radialis muscle in patients was not abnormal compared with controls (*t* test;  $p > 0.5$ ).

Using T1-weighted sequences in two of the patients, the ECRB tendon could not be identified at origin site. In 3 patients, similarly, the radial collateral ligament origin was not identified. Bone oedema, defined as increased signal intensity on T2-weighted FS images, was not



**Fig. 3a, b** Axial T2-weighted fat-saturated image in a 53-year-old woman with 3 months duration of symptoms. **a** Baseline MRI. The signal intensity of the ECRB tendon is increased at the origin adjacent to the humerus. **b** A 6-week MRI follow-up after splint treatment. The signal intensity of the ECRB tendon remained increased despite clinical improvement



**Fig. 4a–c** A 36-year-old man with 6 months duration of symptoms. **a** Baseline axial T2-weighted fat-saturated image. The signal intensity of the ECRB tendon is increased. **b** The 6-week MRI follow-up after steroid injection. The increased signal intensity of the ECRB tendon is persistent. **c** Coronal T1-weighted sequence at 6-week MRI follow-up with increased signal intensity of the ECRB tendon and thickened fibres. An abnormal separation is seen between the ECRB tendon and the radial collateral ligament (arrow)

found within the humerus bone at origin site or new bone formation in patients or in controls.

#### Six-week MRI follow-up

At 6-week MRI the morphological changes of the ECRB tendon was normalised in 2 patients (wrist splint  $n=1$  and glucocorticoid injection  $n=1$ ) and 3 patients developed pathological changes within the ECRB tendon on coronal T1-weighted sequences after 6-week follow-up. At the T2-weighted FS sequences, the increased signal intensity within the ECRB tendon persisted at 6-week MRI follow-up in 9 patients of 14 patients in the group treated with wrist immobilisation and in 11 of 16 patients in the group treated with steroid injection (Fischer's exact test;  $p>0.05$ ).

### Discussion

We found an increased signal intensity and thickening of the extensor carpi radialis brevis tendon on MRI in the majority of the patients before and after intervention, although the duration of disease symptoms was shorter in our patients compared with previous MR studies [2, 9, 10].

The normal tendon composed of well-ordered collagen bundles should in absence of the so-called magic-angle effect produce no MR imaging signal and should appear black and smoothly marginated and general of uniform thickness on all sequences (Fig. 1a). With overuse or receptive micro-trauma, stress produced on the tendon may produce interstitial and collagen failure [18].

These findings have been substantiated on MRI findings as the signal intensity changes in ECRB tendon on MRI has been reported to be correlated with fibrovascular proliferation and fatty degeneration [9] mucoid degeneration without inflammation and correlating MR imaging with surgical findings further strengthened the theory that lateral epicondylitis occurs from micro-tears and mucoid degeneration of the tendon [2].

In contrast to a previous study, we did not find signal intensity changes of the postero-lateral anconeus muscle [10, 15]. The oedema of anconeus muscle has been proposed as a secondary sign of extensor tendon muscle injury. This association has not proved to be very frequent in our experience, maybe due to the differences in sequences or the shorter duration of symptoms in our patients.

Abnormal signal intensity and/or thickening of the ECRB tendon were in this study also seen in 3 of 22 controls, although they did not clinically have symptoms of lateral epicondylitis. This is in accordance with previous studies, in which lateral epicondylitis MRI features in healthy controls was found [9, 15]. It can be speculated that the changes in the controls could reflect a subclinical tendinosis.

Magnetic resonance imaging features as, for example, abnormal or altered signal intensity of the ECRB tendon and separation of the extensor carpi radialis tendon from the radial collateral ligament, were found in the majority of our patients with lateral epicondylitis of the elbow. As the increased signal intensity is related to fibrovascular proliferation, contrast-enhanced imaging might be helpful in the differentiation between highly proliferative tissue and ruptured tendon. Administration of contrast media may increase the signal intensity but had not been found to provide additional information [9, 19]. In our study no indication was found of differences in pain level between patients with or without abnormal MRI findings; however, no firm conclusion may be drawn as to this point due to the relatively low number of patients, especially with normal findings.

The ECRB tendon was invisible in 2 patients. This could indicate a tendon rupture but could also be explained by the fact that slice thickness was 3–4 mm and tendon thickness was 2–3 mm. The ECRB tendon was identified in all controls.

Different interventions have been used in the treatment of lateral epicondylitis, but to date, no specific intervention has proved universally efficacious. The existing evidence of the treatment efficacy of steroid injection or splinting for the treatment of epicondylitis lateralis is not conclusive.

The MRI may have a role in evaluating a treatment response in order to plan a more specified therapy planning among the variety of treatment modalities. Although a limited number of patients was included in each treatment group, in our study it seems that wrist brace was as effective as injection with corticosteroid at 6-week follow-up examination [20]. The MRI did not demonstrate different treatment response within the two treatment groups and the MRI findings persisted at 6-week MRI follow-up, regardless of the improvement reported by most patients.

A possible explanation for this observation could be that changes on MRI need a longer period after treatment to be normalised. A longer MRI follow-up period of the patients with epicondylitis lateralis may have demonstrated regression in the MRI since these MRI features are caused by degenerative changes, which probably will persist more than 6 weeks on MRI.

### Conclusion

The MRI was able to demonstrate pathological changes of the ECRB tendon and soft tissue abnormalities in patients with lateral epicondylitis of the elbow. Although the patients improved clinically at 6-week clinical follow-up examination, MRI demonstrated that the degenerative changes persisted. Future studies may clarify the relation treatment response and duration of pathological changes on MRI.

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