

Letter to the editor

MRI outcomes in prolotherapy for lateral epicondylitis

In *IMM* volume 32, number 3 your readers were presented with the result of a study by Rabago *et al.*, presented as a randomized controlled trial (RCT), showing non-association between the clinical result of prolotherapy and the pre- and post-treatment magnetic resonance imaging (MRI) findings. How come, with this non-association? Can we rely on that result? Well, one simply has to use one's seventh sense – the critical sense before taking the result for granted and before thinking about why function does not always follow structure.

A RCT must be based on a hypothesis, and even though the authors do present a problem formulation they do not present a hypothesis *per se*. However, let us suppose that they have formulated a hypothesis, which they wish to test in a RCT, a hypothesis that is meaningful according to the information given in the Introduction. To test a possible association between the effect of a treatment on a pathologic condition and the pre- and post-treatment results of a paraclinical examination, there must be some correlation between the pathologic condition – the diagnosis – and the result of the paraclinical examination. Do we have any indications of such a correlation? No, at least not according to the following sentence by the authors: 'No validated metric correlates MRI findings with the clinical disease severity either at baseline or in response to clinical improvement'.

However, first of all we must have a reliable diagnosis, i.e. a reproducible and valid diagnostic process, and this has not been demonstrated either in the present paper or in the primary paper to which they refer. According to the primary paper, inclusion criteria were 'diagnosis of lateral epicondylitis (LE) and elbow pain for at least 6 months'. But how the authors came to the diagnosis – the diagnostic criteria – are not described: What were their demands for the clinical history – except for the pain duration – and what were their demands for the results of the clinical examination? Without knowing the reliability, we cannot rely on the homogeneity of the study population.

Secondly, is the paraclinical method used in the study relevant and are the results of the method reproducible and valid?

The authors use a 0.2 T Artoscan clinical MR scanner to study soft tissue pathology. They do not state whether it is a wholebody MR scanner or a dedicated extremity-imaging machine. For the imaging procedure T1- and T2-weighted spin-echo pulse sequences were used. The authors also state that the images were extracted from a 'damaged' optical disc and viewed using Efilm software. Furthermore the authors made up a three-level grading scale for evaluation of the T1- and T2-weighted spin-echo MR images, since they were unaware of any grading scale for MRI of LE. MRI was performed before treatment and 8/16 weeks following the injection. The authors do not explain why these specific examination intervals were chosen.

LE involves the soft tissues around the bony humeral condyle with clinical signs of inflammation. MRI depicts this soft tissue inflammation as unspecific tissue edema and tendon changes. Edema is best detected on MR by the use of STIR (short tau inversion recovery) images, which has higher sensitivity for edema compared to T2-weighted images. Furthermore, high-field MRI (field strength above 1.0 T) does detect soft tissue edema much better than low-field MRI.

In conclusion, imaging of soft tissue pathology should be by high-field MRI with use of STIR images and application of intravenous contrast (gadolinium). In our opinion, the MRI equipment as well as the imaging method used in the present study is inadequate for examination of soft tissue pathology by modern standards.

MRI of bone marrow edema has been studied in detail and it is well known that the changes seen in the MRI images do not correlate with the clinical condition of the patient. As far as we know, this applies for inflammation of soft tissue edema too. So at the present state, the temporal relation between changes in MRI-depicted edema and the patients' condition is not known at all. Furthermore, the exact pathophysiology of pain in LE is also poorly understood. Hence, a three-level grading scale for MRI – based upon pure speculation and not tested for reliability – and evaluating only the signal intensity of the soft tissues with no causal knowledge of the etiology of clinical pain, is of poor scientific value.

However, let us suppose that we have had a reliable diagnosis and a relevant and reliable paraclinical method that furthermore correlated with the disease severity; can we then say that the study is performed *lege artis*?

We are sorry to say: no we do not think so. Looking at the primary article, recruitment of participants is not described as being consecutive, and furthermore the authors do not explain why eight patients, who met the initial inclusion criteria, were found ineligible for the study. After randomization another four subjects dropped out; again no reasons are given. The lead author performed the recruitment and all the injections, with blinded syringes. In spite of that, we find the patient-blinding dubious, as injection with a sclerosant

will introduce inflammation in contrast to isotonic saline. Finally, the reader is not informed about who is doing the 52-week telephone follow-up. Was it the lead author who included the patients or was it a blinded observer?

So to summarize: the premises are not present to take up a discussion on the interesting aspect that function does not always follow structure.

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