

ability of hyaluronic-acid levels to predict not only cartilage loss in knee OA, but progression of global damage, too.

**Original article** Bruyere O *et al.* (2006) Osteoarthritis, magnetic resonance imaging, and biochemical markers: a one year prospective study. *Ann Rheum Dis* 65: 1050–1054

## Epratuzumab shows promise in the treatment of primary Sjögren's syndrome

CD22 is thought to have a role in the disruption of B-cell homeostasis seen in primary Sjögren's syndrome, so Steinfeld and colleagues assessed the safety and efficacy of epratuzumab—a humanized antibody against CD22—in an open-label phase I/II study.

Overall, 16 white patients (14 female) with active, primary Sjögren's syndrome were scheduled to receive a 360 mg/m<sup>2</sup> epratuzumab infusion at weeks 0, 2, 4, and 6; two patients did not receive all four infusions because of adverse reactions to treatment. Follow-up was performed at weeks 6, 10, 18, and 32. The authors used a composite end point (the Schirmer-I test, erythrocyte sedimentation rate, and unstimulated whole salivary flow) to assess response to treatment. Patient and physician global assessments, fatigue, and pain were measured with visual analog 0–100 mm scales.

Compared with baseline, patient and physician global assessments and fatigue were markedly improved after treatment with epratuzumab. Over half of the 15 patients who received  $\geq 1$  infusion achieved a  $\geq 20\%$  improvement in two disease activity parameters at week 6; nearly 70% of patients achieved this response at week 32. Within 18 weeks of treatment, mean B-cell levels had decreased; CD22 overexpression in peripheral B cells (observed at baseline) remained downregulated for at least 12 weeks post-treatment. Three patients had slightly elevated human anti-epratuzumab antibody titers, but without apparent clinical effects.

Interestingly, the number of responders was highest at week 32, which the authors suggest might indicate recovery of glandular tissue.

They advise, therefore, that glandular biopsies be taken before and after treatment.

**Original article** Steinfeld SD *et al.* (2006) Epratuzumab (humanised anti-CD22 antibody) in primary Sjögren's syndrome: an open-label phase I/II study. *Arthritis Res Ther* 8: R129

## No need for biopsy in some patients with suspected giant cell arteritis

Color duplex sonography (CDS) of the temporal arteries should be performed before temporal-artery biopsy (TAB; the gold-standard diagnostic method) in patients with giant cell arteritis (GCA), say Greek researchers.

Karahaliou and colleagues investigated whether CDS could replace TAB in a prospective study of 60 consecutive patients aged  $\geq 50$  years with clinically suspected GCA. Baseline CDS was performed in 60 patients and 30 sex-matched, age-matched controls (15 healthy individuals; 15 with diabetes and/or a history of stroke).

Overall, 21 of the 55 participants who completed the 3-month follow-up had presented with a concentric, hypoechogenic halo at baseline CDS, suggestive of arterial-wall inflammation; 18 of these 21 patients were subsequently diagnosed with GCA. A unilateral halo had 82% sensitivity and 91% specificity in diagnosing GCA, and halos of just 0.7 mm diameter were able to predict GCA. Bilateral halos (present in 9 patients with confirmed GCA) had 100% specificity and 41% sensitivity in diagnosing GCA. The presence of blood-flow abnormalities did not improve the diagnostic power of CDS.

The authors recommend that CDS be performed after clinical examination and assessment of laboratory data of patients with suspected GCA. If bilateral halos are present, GCA is confirmed; if a unilateral halo or no halo is present, TAB (directed to the halo area) should be performed. If TAB is positive, GCA is diagnosed; if not, a second TAB or occipital-artery biopsy, and/or imaging of large arteries, might be required to rule out GCA.

**Original article** Karahaliou M *et al.* (2006) Colour duplex sonography of temporal arteries before decision for biopsy: a prospective study in 55 patients with suspected giant cell arteritis. *Arthritis Res Ther* 8: R116