

IL-18 is involved in systemic JIA, but not in oligoarticular or polyarticular forms

Juvenile idiopathic arthritis (JIA) is associated with abnormal production of cytokines. Interleukin (IL)-18 levels are elevated in children with systemic JIA and this cytokine is known to mediate articular damage in patients with rheumatoid arthritis. New data indicates that IL-18 is not involved in the pathogenesis of oligoarticular or polyarticular JIA, but could be a good therapeutic target for systemic JIA.

Jelušić *et al.* analyzed sera obtained from 17 children with systemic JIA, 31 children with oligoarticular JIA, 33 children with polyarticular JIA and 18 randomly selected children who did not suffer from inflammatory diseases. Blood was obtained from patients with JIA during the active phase of the disease and at least 6 months after the onset of clinical remission. Synovial fluid samples were also obtained from the inflamed joints of 16 children with oligoarticular JIA.

Children with systemic JIA had significantly higher levels of serum IL-18 than children with oligoarticular and polyarticular JIA, and than control patients. Serum levels of IL-18 decreased in patients with systemic JIA when the children entered clinical remission, but were still significantly higher than levels detected in those patients suffering from the other forms of this disease. IL-18 was present at comparable levels in synovial fluid and sera collected from patients with oligoarticular JIA, and neither oligoarticular nor polyarticular JIA were associated with higher-than-normal levels of serum IL-18. The authors conclude that IL-18 might be a good target for treatment of systemic JIA.

Original article Jelušić M *et al.* (2007) Interleukin-18 as a mediator of systemic juvenile idiopathic arthritis. *Clin Rheumatol* 26: 1332–1334

Low serum COMP levels predict improved response to anti-TNF treatment in patients with RA

The identification of specific serum biomarkers that represent the response to treatment in patients with rheumatoid arthritis (RA) is a major area of research. Morozzi and colleagues investigated whether cartilage oligomeric matrix

protein (COMP), a serum marker of cartilage turnover, can be used as a prognostic indicator for response to adalimumab treatment in patients with RA.

The study included 35 consecutive patients with RA. Patients received adalimumab 40 mg subcutaneously every 2 weeks for 52 weeks, and serum COMP levels were measured at baseline and after months 3, 6 and 12. The response to therapy was assessed using the ACR20, ACR50, ACR70 and disease activity score 28-joint count (DAS 28) criteria.

The final analysis included 29 patients who completed treatment. Patients were assigned to either the high COMP group (serum COMP >10 U/l; $n=12$) or the low COMP group (serum COMP <10 U/l; $n=17$); overall, the mean serum COMP level did not change significantly throughout treatment. Significantly more patients with low COMP achieved an ACR70 response at 3 months ($P<0.02$) and 6 months ($P<0.02$) than patients with high COMP, and the low COMP group also had significantly lower mean DAS 28 scores at 3 months ($P<0.02$) and 6 months ($P<0.01$) than the high COMP group. No correlation was observed between COMP level and C-reactive protein level or erythrocyte sedimentation rate, or between response scores and anti-cyclic citrullinated peptide antibodies or IgM/IgA rheumatoid factor levels.

The authors conclude that serum COMP levels <10 U/l before anti-TNF therapy is predictive of a rapid ACR70 response in patients with RA.

Original article Morozzi G *et al.* (2007) Low serum level of COMP, a cartilage turnover marker, predicts rapid and high ACR70 response to adalimumab therapy in rheumatoid arthritis. *Clin Rheumatol* 26: 1335–1338

Is plasma concentration of calprotectin a measure of joint inflammation and damage in RA?

Calprotectin is released by activated granulocytes and monocytes/macrophages in arthritic joints. Levels of calprotectin measured in patients' synovial fluid significantly correlate with levels detected in patients' plasma; calprotectin is thought to diffuse from inflamed joints into the circulation.

Hammer *et al.* analyzed 145 patients with rheumatoid arthritis (RA; mean age 59.9 years,