THE ROLE OF ADAMTS7 IN THE ACTIVATION AND PROGRESSION OF REACTIVE ARTHRITIS

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Reactive arthritis (ReA) is a post-infectious inflammatory joint disease that may present as either a mild, self-limiting condition or progress to chronic erosive arthritis. A key mechanism in disease pathogenesis involves the activation of inflammatory cascades mediated by cytokines such as TNF-α and IL-17A, as well as enzymatic systems that damage cartilage and periarticular structures. ADAMTS7, a metalloproteinase, contributes to extracellular matrix degradation and has been associated with joint destruction in various rheumatic diseases.

To evaluate serum ADAMTS7 levels in patients with ReA and assess its association with disease activity and structural joint damage.

Materials and Methods. Sixty patients with confirmed ReA (mean age 36 ± 9 years) were examined. Serum ADAMTS7, TNF-α, and IL-17A levels were measured by enzyme-linked immunosorbent assay (ELISA). Disease activity was assessed using the DAREA and ASDAS indices. Patients were divided into two groups according to ADAMTS7 level: low ($\leq$ 10 ng/mL) and high (> 10 ng/mL). Clinical and radiological joint damage was evaluated.

Results. Patients with elevated ADAMTS7 levels showed significantly higher DAREA (3.8 ± 1.2 vs. 2.1 ± 0.9; $p < 0.001$) and ASDAS (4.1 ± 1.0 vs. 2.5 ± 0.8; $p < 0.001$) scores, higher TNF-α and IL-17A concentrations, and a greater number of affected joints (6.8 ± 2.0 vs. 3.2 ± 1.3; $p < 0.001$). The frequency of enthesitis, dactylitis, and early radiological signs of joint destruction was also higher in the high-ADAMTS7 group. A positive correlation was found between ADAMTS7 levels and markers of inflammation ($r = 0.48–0.55$; $p < 0.001$).

Conclusion. Elevated ADAMTS7 levels are associated with increased clinical activity and early joint damage in patients with ReA. Determination of this biomarker may help predict unfavorable disease progression and stratify the risk of chronic joint involvement.