The diagnostic utility of serum interleukin-22 in patients with suspected axial spondyloarthritis

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Abstract

Background. There is an unmet need for a reliable biomarker for the diagnosis and differentiation of AxSpA from its multiple mimickers. Serum levels of IL-22, which is tightly involved in the pathogenesis of AxSpA, have been previously found significantly elevated in patients with AxSpA, when compared to healthy individuals or persons with osteoarthritis.

Methods. Consecutive patients with established or suspected AxSpA, followed in the outpatient clinic of Bnai Zion Medical Center, Haifa, were enrolled. The demographic and anamnestic data, as well as results of laboratory and imaging studies and estimates of

disease activity were acquired from patients' charts. The final diagnosis of definite or probable SpA, or alternative diagnoses, based on the existing medical data, was determined by the joint opinion of the treating rheumatologist and a senior author (GS), blinded to the estimates of the serum IL-22, which were examined by Quantikine ELISA Human IL-22 Immunoassay.

Results. Serum levels of IL-22 were significantly higher in patients with definite AxSpA (29 patients) compared to patients with alternative diagnoses (14 patients) and healthy volunteers (16 individuals) ($p<0.001$ for both comparisons). Patients with possible AxSpA (7 patients) had a wide range of data distribution, probably reflecting its heterogeneity. The sensitivity and specificity of the serum levels of IL-22 for the AxSpA diagnosis were 0.68 (95% CI 0.49-0.84) and 0.86 (95% CI 0.68-0.95), respectively, for the cut-off of 5 pg/ml, and 0.48 (95% CI 0.29-0.67) and 1 (95% CI 0.85-1) for the cut-off of 10 pg/ml. In patients with AxSpA, serum IL-22 levels did not correlate with mSASSS, BASDAI, ASDAS indices or serum CRP levels, with all results far away from the level of statistical significance.

Conclusion. Serum IL-22 leves are elevated in patients with AxSpA and can serve as an independent biomarker for the differentiation of AxSpA from its non-inflammatory mimickers.