Abstract

Background: Axial spondyloarthritis (axSpA) is a chronic inflammatory rheumatic disease affecting primarily the spine and its adjacent structures. The disease is characterized not only by destructive joint changes but also by excessive osteoproduction, which can lead to gradual ankylosis of the spine and thus significantly reduce the mobility and quality of life. The pathogenesis of the disease is not yet fully understood, but a strong genetic background is suggested, along with dysregulation of tissue metabolism resulting from an imbalance of pro- and anti-inflammatory immune mechanisms. We are still lacking biomarker with sufficient sensitivity and specificity which could help to identify early diagnosis, to monitor subchondral damage, and to differentiate rapidly progressing patients. The aim of this work was to determine the levels of potential biomarkers of connective tissue metabolism, fat metabolism and new promising biomarkers for both disease subtypes, their relationship to disease activity and progressive structural changes.

Results: We have shown increased serum/plasma levels of connective tissue metabolism biomarkers (especially matrix metalloproteinase mediated metabolites), which were able to differentiate patients with early and late forms of axSpA from healthy individuals (HC), were related to disease activity and radiographic progression. Furthermore, we demonstrated the relationship between adipokines and axSpA patients. Specifically, visfatin serum levels were significantly higher in axSpA patients compared to HC and were associated with spinal involvement. We were the first to report the possible relationship between microRNA in pathogenesis of the disease and its progression. Furthermore, we presented higher plasma levels of heat shock protein 90 (Hsp90) in patients with both forms of disease and their association to the primary subchondral changes in a form of bone edema.

Conclusion: The results of our studies support recent findings of dysregulation in connective and adipose tissue metabolism and showed a possible association with subchondral damage of early and late forms of axSpA. In addition, we pointed out new, yet not fully studied biomarkers that could bring additional information into the complex pathogenesis of the axSpA.