A127 MAST CELLS CONTRIBUTE TO SYNOVIAL INFLAMMATION IN NON-PSORIATIC AND PSORIATIC SPONDYLOARTHRITIS

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Background and objectives Comparative analyses of synovitis in spondyloarthritis (SpA) versus rheumatoid arthritis (RA) suggested that the innate immune system may play a predominant role in SpA pathogenesis. Based on this recent observation of marked synovial mast cell infiltration in psoriatic arthritis (PsA), the authors aimed to investigate the potential contribution of mast cells to synovial inflammation in SpA.

Materials and methods Synovial tissue and fluid were obtained in non-psoriatic and psoriatic SpA (n=80) and RA (n=50). Synovial tissue was analysed by immunohistochemistry

and double immunofluorescence. Synovial fluid was analysed by ELISA. Synovial tissue biopsies were also used for ex vivo cultures.

Results In comparison with RA, synovial infiltration with c-kit positive mast cells was strongly and specifically increased in SpA independently of the subtype (non-psoriatic vs psoriatic), disease duration, and treatment. Staining of mast cell granules with tryptase and toluidine blue as well as analysis of synovial fluid levels of histamine and tryptase did not indicate increased degranulation in SpA synovitis. Mast cells expressed significantly more interleukin 17 (IL-17) in SpA than RA synovitis and constituted the major IL-17 expressing cell population in SpA synovitis. Targeting mast cells with the c-kit inhibitor imatinib mesylate in ex vivo tissue cultures led to a significant decrease in the production of IL-17 as well as other pro-inflammatory cytokines. Analysis of paired synovial biopsies before and after treatment indicated that this mast cell/IL-17 axis was not modulated by effective tumour necrosis factor (TNF) blockade in SpA.

Conclusion The specific and TNF-independent increase of IL-17 expressing mast cell and the ex vivo targeting of these cells indicate that the mast cell/IL-17 axis may contribute to the synovial inflammation in peripheral SpA.