24 DISTURBANCES OF B CELL HOMEOSTASIS IN TAKAYASU ARTERITIS AND GIANT CELL ARTERITIS

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Background Giant cell arteritis (GCA) and Takayasu arteritis (TAK) are an inflammation of the large arteries and their major branches. They have been considered distinct disorders based on their clinical features, age of onset and ethnic distribution. But the histopathology of arterial lesions and imaging studies suggest similar pathogenetic mechanisms in these diseases. Since the authors have recently shown disturbances in the B cell homeostasis in patients with active TAK the aim of this study was to analyse circulating B cell subpopulations in patients with GCA as compared to those with TAK.

Methods Peripheral blood mononuclear cells from six patients with active TAK and six with inactive disease, 16 patients with GCA (eight with active and eight with inactive disease) and 12 healthy controls were analysed by flow cytometry for the expression of CD27, CD19, CD20, MHCII, CD3, CD8 and CD4. Plasma cell population is considered CD20⁺/CD19⁺/CD27⁺⁺⁺. Newly generated plasmablasts express additionally MHC class II. CD19⁺/CD20⁺/CD27⁺ cells were considered as memory B cells and their CD27 low counterpart as naive B cells.

Results Patients with active TAK and GCA showed significantly higher frequencies of plasma cells (among all CD19⁺ B cells) than in inactive patients ($p=0.02$ and 0.029 , respectively) and healthy controls. The absolute plasma cell numbers were increased in TAK patients ($p=0.0015$) but not in GCA. Active TAK patients showed also elevated circulating plasmablasts in comparison with patients in remission ($p=0.0053$). The frequency of plasma cells in the blood correlated significantly with the disease activity according to the NIH criteria ($r=0.73$ and $p=0.069$). In contrast to TAK, active GCA patients exhibited a significant reduction of the memory B cell subset versus

inactive patients ($p=0.028$). Disturbances of T cell subset were not observed.

Conclusions The findings suggest different roles of B cells in the pathogenesis of TAK versus GCA. Circulating plasma cells/plasmablasts may serve as biomarker of disease activity in TAK and provide indication for a B cell and plasma cell directed therapy.