**A8 CLUES FOR A POSSIBLE ROLE OF SENESCENT T CELLS IN
GIANT CELL ARTERITIS PATHOGENESIS**

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Background and objectives T cells are considered major players in the development of giant cell arteritis (GCA). To date most attention has been focused on Th1 and Th17 cells. However, the contribution of senescent T cells to this strictly ageing-associated vasculitis remains obscure. The purpose of this study was to provide clues whether senescent T cells play a role in GCA pathogenesis. Therefore we assessed frequencies of senescent T cells and their expression of the natural killer marker CD161 in patients with GCA and healthy controls. CD161 has been reported to identify cells with a pro-inflammatory cytokine repertoire (eg, interferon γ and interleukin 17).

Methods Peripheral blood of seven untreated GCA-patients and eight age/sex matched healthy controls was obtained. Using flow cytometry, senescent (or terminally differentiated) T cells were defined as CD45RO-CCR7- cells within the CD4 and CD8 T cell gates. In addition, the naïve (CD45RO-CCR7+), central memory (CD45RO+CCR7+) and effector memory (CD45RO+CCR7-) T cell subsets were identified. Expression of CD161 was assessed in each of these subsets.

Results Frequencies of senescent, naïve and central memory CD4 T cells were similar in GCA-patients and controls, whereas a trend towards decreased CD4 effector memory cells was observed in patients (23% vs 34%). The CD4 senescent T cell subset of GCA patients contained more CD161 cells compared to healthy controls (4,4% vs 1,6%). Also in the CD4 effector memory subset of patients we observed a trend towards more CD161 cells (33% vs 21%).

Frequencies of CD8 naïve, central memory, effector memory and terminally differentiated subsets were similar in patients and controls. In addition no differences were found for CD161 expression in CD8 T cells.

Conclusion We found one clue suggesting a role for senescent T cells in GCA: namely, an increased expression of CD161 by CD4 senescent T cells in GCA patients. This could reflect a natural killer-like phenotype in these cells, associated with high production of IFN γ and cytotoxic effector molecules. The same might be true for the CD4 effector memory subset of GCA patients, since we observed a trend towards more CD161 expression in these cells. In addition we found a trend towards decreased frequencies of circulating CD4 effector memory cells in GCA-patients. This may suggest that these cells preferentially migrate to the site of inflammation, as previously observed in Wegener's granulomatosis.¹

REFERENCES

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