following rituximab therapy may help guide remission maintenance therapy in these patients. 

**Conclusion**—CD5+ B cells may be a novel immunological biomarker to follow induction of remission and impending flare in patients with ANCA vasculitis before and after treatment with rituximab.

**Reference**

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**Monocyte chemoattractant protein-1 (MCP-1) in antineutrophil cytoplasmic autoantibody (ANCA)-associated vasculitis: Biomarker potential and association with polymorphisms in the MCP-1 and the CC chemokine receptor-2 (CCR2) gene**

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**Introduction**—ANCA-associated small vessel vasculitis (AAV) morbidity and mortality have decreased over the last four decades, but there is still a need of better biomarkers for distinguishing which patients will have a more severe outcome, in order to optimize the treatment. The aim of this study was to confirm our previous results of urinary MCP-1 (uMCP-1) as a prognostic marker, and to explore its potential as a marker of disease activity. Another part of the study was to explore the association of AAV and polymorphism in the MCP-1 gene (-2518A/G) and the CCR2 gene (V64I).

**Patients**—One hundred and fourteen patients with AAV were followed regularly between 2002 and 2009 at Skane University Hospital. Urine samples, blood samples and clinical status were registered. uMCP-1 was analyzed by an in house ELISA. PCR-RFLP was used for the polymorphism analyses.

**Results**—Patients with severe prognosis had significantly higher levels of uMCP-1 compared to patients with non severe prognosis and healthy controls. Patients with renal damage had higher levels compared to patients without renal damage. There was a tendency of higher uMCP-1 levels i active disease compared to remission. AA in the -2518 position of the MCP-1 gene was associated with a more severe outcome compared to the A/G or G/G genotype. The A/A genotype was associated with higher levels of uMCP-1. No significant associations were seen for the CCR2V64I.

**Discussion**—This study confirms the association of high uMCP-1 levels with poor prognosis of AAV. It also suggests an association of the A/A genotype at position -2518 in the MCP-1 gene with poor prognosis.

**Conclusion**—MCP-1 is clearly a candidate biomarker of potential clinical value. The A/A genotype association needs further evaluation.

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**Characterization of regulatory B cells in ANCA-associated vasculitis (AAV)**

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**Introduction**—Phenotypic characterization of the regulatory B cell (Bregs) subset in humans has been proposed based on expression of either CD19+CD24hiCD38hi [1] or CD19+CD24hiCD27+ [2]. Functionally, Bregs exert their suppressive role via production of IL-10. Recently, numerical and/or functional disturbances in Bregs have been associated with autoimmunity. This study aimed to phenotypically and functionally characterize Bregs cells in AAV patients.

**Patients**—Sixty AAV patients (14 active, 46 remission) and 41 healthy controls (HC) were included in the study. B cell subsets were determined in peripheral blood by flow cytometry. Bregs were defined within the CD19+ population as CD24hiCD38hi or CD24hiCD27+. PBMC were stimulated in vitro with CpG-ODN to induce IL-10 production, and the percentage of IL-10+ B cells was analysed by flow cytometry. To examine the suppressive capacity of B cells, sorted CD19+ B cells were co-cultured with monocytes, to evaluate their capacity to inhibit TNF-α production upon LPS activation.

**Results**—The percentage of circulating CD19+CD24hiCD38hi cells was not different in AAV patients in remission compared to HC, but was

**Discussion**—It is reported that MPO-ANCA can induce NETs, and that NETs can induce thrombosis. This study suggests the possibility that the pathogenesis of thrombosis in MPO-AAV could be critically associated with the mechanism via NETs.

**Conclusion**—This study suggests the possibility that the pathogenesis of DVT in MPO-AAV could be critically associated with the mechanism via MPO-ANCA and NETs.

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**Abundant neutrophil extracellular traps in thrombus of patient with microscopic polyangiitis**

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**Introduction**—Patients with MPO-AAV have an increased risk of deep vein thrombosis (DVT). However, the mechanism remains elusive. Recently, it has been reported that aberrant formation and disordered regulation of NETs could be implicated in the development of MPO-AAV. On the other hand, NETs interact with coagulant factors and can induce thrombosis. In this study, we carried out autopsy on an MPO-AAV patient complicated with DVT, and then the association of NETs and DVT was examined.

**Methods**—NETs in the glomeruli and thrombus were examined by immunofluorescent staining. To quantify the NETs volume in the thrombus, immunohistochemistry was performed using anti-citrullinated histone 3 antibody. The area of NETs was quantified by Image J software and then standardized by the numbers of neutrophils counted in the serial sections. The amount of NETs in the thrombus was compared to other thrombi derived from a patient who died of bacterial sepsis and from one who died of post-operative pulmonary embolism.

**Results**—NETs were detected in the glomerular crescents despite the absence of microbes in the MPO-AAV patient. Interestingly, abundant NETs were detected in the thrombus complicated with MPO-AAV, and the amount of NETs was significantly greater compared to other thrombi unrelated to MPO-AAV.

**Discussion**—In the present study, NETs were detected in MPO-AAV. This study suggests the possibility that the mechanism via NETs might be critical for the thrombotic complication in MPO-AAV patients.

**Conclusion**—NETs linked to MPO-AAV may be related to the thrombotic complications in these patients.

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**Pathogenesis**