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**Predicting relapse in granulomatosis with polyangiitis: The role of biomarkers**  
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**Introduction.**—Granulomatosis with polyangiitis (GPA) is a small vessel vasculitis which can cause a severe systemic disease. The use of biomarkers to predict disease relapse was explored.  
**Methods.**—Thirty patients with GPA who had experienced at least one relapse, with a total of 55 individual relapse events necessitating potent immunosuppression, and a control group of 11 patients with GPA who had not experienced a relapse were selected. The level of anti-neutrophil cytoplasmic antibodies (cANCA), proteinase 3 (PR3), white cell count (WCC), neutrophil count, lymphocyte count, monocyte count, C-reactive protein (CRP) and creatinine were assessed for any association with relapse. A total of 13,288 tests were analysed, including 1438cANCA results. The clinical yield was calculated for each marker. Remission values were compared between the cohorts. Changes from remission values in the six months prior to relapse were analysed.  
**Results.**—Clinical yields were higher with a greater increase from remission values: a neutrophil count and cANCA titre of more than twice the average remission values were associated with relapse within 6 months in 67 and 32% of cases, respectively, whereas, this was 26 and 19% for a less than two-fold increase. Remission values for most of the biomarkers were higher in the relapse group, for example average WCC was 7.6 (± 2.0) for the relapse group and 6.3 (± 1.7) for the controls.  
**Conclusion.**—A number of the biomarkers showed promise for use in predicting a relapse. None were sufficiently correlated to the relapse events to warrant use as a sole predictor of relapse. They may be of use when trying to evaluate the risk: reward ratio of commencing cytotoxic therapy.  

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P80  
**Circulating gamma delta T cells are significantly reduced in granulomatosis with polyangiitis**  
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**Introduction.**—Granulomatosis with polyangiitis (GPA) is a systemic autoimmune disease affecting small blood vessels leading to tissue damage. The pathogenesis of the disease is unclear and the specific contribution of lymphocytes remains to be clarified. In this study extensive phenotyping of lymphocyte subsets was undertaken and this included investigation of γδ T cells, an innate-like T lymphocyte population.  
**Methods.**—Multicolour flow cytometry was used to phenotype lymphocytes of GPA patients (n = 33) and healthy controls (n = 14). Lymphocyte subsets analysed included γδ T cells and γδ T helper cells, B cells, γδ T cells (including the major γδ T cell subset Vγ9Vδ2), natural killer cells and invariant natural killer T cells. All patients were cANCA positive at time of diagnosis. PBMCs were isolated from peripheral blood and cultured with PR3 (Enzo Bioscience) for six days. T cell proliferation was measured by 3H thymidine incorporation and a stimulation index of two was considered significant.  
**Results.**—γδ T cell reactivity to PR3 was investigated in patients with GPA (n = 18) and healthy control subjects (n = 11). All GPA patients were cANCA positive at time of diagnosis. PBMCs were isolated from peripheral blood and cultured with PR3 (Enzo Bioscience) for six days. T cell proliferation was measured by 3H thymidine incorporation and a stimulation index of two was considered significant.  
**Conclusion.**—These results demonstrate the presence of PR3 reactive γδ T cells in patients with GPA. The role of these cells in disease pathogenesis is unknown. Additional studies focused on HLA-restriction, cytokine analysis and generation of cell lines are underway. PR3-specific T cells are a potential therapeutic target in patient management.  

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P81  
**Proteinase 3 reactive T cells in patients with granulomatosis with polyangiitis**  
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**Introduction.**—Granulomatosis with polyangiitis (GPA) is a systemic autoimmune disease affecting small blood vessels leading to tissue damage. In many patients GPA is characterised by cytoplasmic anti-neutrophil antibodies (cANCA), which target the enzyme proteinase 3 (PR3). The role of these autoantibodies in disease pathogenesis is unknown and their presence suggests that PR3-specific helper T cells are also involved.  
**Methods.**—T cell reactivity to PR3 was investigated in patients with GPA (n = 18) and healthy control subjects (n = 11). All GPA patients were cANCA positive at time of diagnosis. PBMCs were isolated from peripheral blood and cultured with PR3 (Enzo Bioscience) for six days. T cell proliferation was measured by 3H thymidine incorporation and a stimulation index of two was considered significant.  
**Results.**—Two of the 18 GPA patients, but none of the controls, responded to PR3 in culture. The mean stimulation index of responders was 3.28 (range 2.2 to 4.9) and four were cANCA positive at the time of study.  
**Conclusion.**—These results demonstrate the presence of PR3 reactive T cells in patients with GPA. The role of these cells in disease pathogenesis is unknown. Additional studies focused on HLA-restriction, cytokine analysis and generation of cell lines are underway. PR3-specific T cells are a potential therapeutic target in patient management.  

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Large vessel vasculitis

P82  
**Vitamin D level is decreased in patients with Takayasu’s arteritis**  
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**Introduction.**—Takayasu’s arteritis (TAK) is a chronic, inflammatory vasculitis of the aorta and its major branches. Vitamin D (Vit D) is increasingly implicated in the pathogenesis of autoimmune diseases. The immune-regulatory role of Vit D affects both the innate and adaptive immune systems, contributing to the immune-tolerance of