[1]. Our aim was to examine the role of leflunomide as a corticosteroid-sparing agent in GCA- and PMR-patients.

**Methods.**—Patients with difficult-to-treat GCA and PMR were consecutively recruited from our vasculitis clinic. The dose of corticosteroid and CRP values were recorded before and three months after the initiation of leflunomide. Side effects were also registered. Student’s t-test was used to compare the means. Statistical significance was defined as $P < 0.05$.

**Results.**—Twenty patients were recruited, 10 with PMR (one male, nine females) and ten with GCA (all females). One patient with GCA discontinued treatment before three months due to side effects. The mean value of CRP at starting point was 22 mg/dl (SD 7.9) and 16.9 mg/dl (SD 5.6) for the PMR and GCA group respectively. Staring dose of corticosteroids was 10.5 mg (SD 1.2) for both groups. In the PMR group, a reduction of 4 mg prednisolone between initial evaluation and three months control was registered (95% CI 0.1–3.6) ($P = 0.04$). However, no difference was seen in CRP values ($P = 0.161$). In GCA patients, a reduction of 4 mg (95% CI 0.4–7.5) ($P = 0.03$) prednisolone and a reduction in CRP values of 14 mg/dl (95% CI 0.2–28.0) ($P = 0.05$) were observed.

**Discussion.**—Leflunomide seems to be effective as a corticosteroid-sparing agent in patients with GCA and PMR. In addition, leflunomide is well tolerated. However, no difference was seen in CRP values in PMR patients before and after 3 months of treatment. Randomized controlled studies are warranted to examine the role of leflunomide in treatment of GCA/PMR.

**Conclusion.**—Leflunomide seems to be effective as a corticosteroid-sparing agent in patients with GCA and PMR.

**Reference**


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**P107**

**Tocilizumab for the treatment of giant cell arteritis: Extended follow-up**

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**Introduction.**—Interleukin (IL)-6 contributes to the pathogenesis of giant cell arteritis (GCA) and represents a target for therapy.

**Methods.**—Retrospective study of 12 patients with relapsing GCA treated with monthly infusions of the IL-6 receptor (IL-6R) antagonist tocilizumab (TCZ). Outcomes evaluated were symptoms of disease activity and ability to taper glucocorticoids (GC).

**Results.**—The mean follow-up of this cohort since diagnosis was 37 months (range 17–70). Eight subjects had failed at least one immunosuppressant (methotrexate, azathioprine, cyclophosphamide, infliximab, adalimumab and etanercept), and four had contraindications for use of GC. TCZ (4 mg/kg, n = 3 and 8 mg/kg, n = 9) was given for a mean period of 16 months (range 6–27). Before and during IL-6R blockade, the patients experienced an average of 2.7 (95% CI 2–3.5) and 0.6 (95% CI 0–1.2) disease exacerbations per year, respectively ($P = 0.0006$). The mean daily prednisone dose of the cohort decreased from 24 mg (95% CI 15–33.5) at the time of TCZ initiation to 7.3 mg (95% CI 0.7–14) by the time of last evaluation ($P = 0.01$). On TCZ, 7 subjects maintained disease remission until the end of follow-up for a mean time of 17.5 months (range 8–26), and 5 patients flared after an average of 11 months of therapy (range 2–25). The mean prednisone dose at the time of disease flare in these 5 patients was 4.5 mg/day. One subject relapsed after TCZ discontinuation. Currently, 5 patients take 5 mg/day of prednisone or less, and 3 patients are off GC. Adverse effects attributable in part to TCZ in this series included leucopenia ($n = 5$), transaminits ($n = 8$), and pneumonia ($n = 1$). Autopsy on one patient who died from an unrelated cause revealed persistent vasculitis.

**Discussion.**—TCZ led to significant decrease in the flare rate and GC requirement of a group of patients with highly relapsing GCA.

**Conclusion.**—These findings support the performance of larger, randomized trials of TCZ as a steroid-sparing agent for remission induction and maintenance in GCA.

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**P108**

**Severe relapse of Takayasu arteritis on ongoing treatment with tocilizumab**

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**Introduction.**—Recently, single cases and small patient cohorts have suggested beneficial effect of tocilizumab, a humanized monoclonal anti-IL-6 receptor (IL-6R) antibody, on Takayasu arteritis (TAK). Up-regulation of IL-6 has been shown within inflamed arteries but its role in the pathogenesis of arteritis is currently unclear.

**Methods.**—A female born in 1985 was diagnosed with advanced TAK in 1999. She was operated with aortic grafting and auto-transplantation of the left kidney and treated with steroids in different combinations with; azathioprine, methotrexate, infliximab, mycophenolate mofetil and cyclophosphamide. Due to persistent high disease activity with new vascular lesions, she never could taper Prednisolone below 12.5 mg daily.

**Results.**—Treatment with tocilizumab 8 mg/kg intravenously was initiated and scheduled every fourth week in combination with a fixed Prednisolone dosage. At the time of the fourth tocilizumab course, she got symptoms of relapse with carotidynia, severe musculoskeletal pain, fever and palpable multiple tender skin nodules. An ultrasound showed increased wall thickness in the carotid arteries. An 18F-FDG PET-CT scan showed widespread pathologic arterial wall uptake in both common carotid arteries, in the whole aorta, the brachiocephalic artery, the pulmonary trunk and in the right common iliac artery. Ten days after the fourth tocilizumab treatment the patient was admitted to the hospital because of high disease activity and rising C-reactive protein to 124 mg/L. Infections and malignancy were excluded. After intravenous methylprednisolone she rapidly recovered.

**Conclusion.**—This is to our knowledge the first published case of relapse of Takayasu arteritis on ongoing tocilizumab treatment.

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