serious infections were 4.6 and 3.5 per 100 pt-years, respectively. Additional results will be presented for original PBO pts.

**Conclusion:** Nearly half the pts treated with TCZ QW maintained CR for the entirety of part 2, though flares still occurred in the remaining pts once they discontinued TCZ treatment. Among pts who maintained CR in part 2, higher proportions of those originally assigned to TCZ were treatment-free compared with those originally assigned to PBO. Retreatment with TCZ restored CR in pts who experienced flare. Cumulative GC doses over 3 years were lower in pts originally assigned to TCZ than in those originally assigned to PBO. No new safety signals were observed with TCZ exposure in GCA pts during the 3-year study.

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**THE IMPACT OF DISEASE EXTENT AND SEVERITY DETECTED BY QUANTITATIVE ULTRASOUND ANALYSIS IN THE DIAGNOSIS AND OUTCOME OF GIANT CELL ARTERITIS: RESULTS FROM THE TEMPORAL ARTERITIS VERSUS ULTRASOUND (TABUL) STUDY AND VALIDATION IN AN INDEPENDENT COHORT**

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**Background:** Colour duplex sonography (CDS) is used in patients with giant cell arteritis (GCA) to detect inflammatory oedema of the vascular wall, known as “halo.” A quantitative analysis of halo characteristics to grade the severity and extension of vascular involvement detected by CDS could improve GCA assessment.

**Objectives:** To develop a quantitative CDS score to improve the diagnosis of GCA, and to correlate the score with histologic findings and clinical outcome. To determine the additional role of clinical signs/symptoms to the CDS score.

**Methods:** We selected patients with a positive CDS and a diagnosis of GCA recruited into the Temporal Artery (TA) Biopsy (TAB) vs Ultrasound in Diagnosis of GCA (TABLE) study. Due to collinearity we fitted 4 different CDS models including combinations of the following items: number of sites and distribution of halos, average and maximum intima-media thickness (IMT) at the level of the TA and axillary arteries (AX) and halos bilaterality. We fitted 4 models with clinical and laboratory findings. We combined the best CDS and clinical models (according to the Akaike Information Criterion) to identify independent correlates of a TAB diagnostic for GCA and of clinical outcome at 6 months (visual loss + VDI ocular + glucocorticoids > 10 mg/day and/or need for immunosuppressants) and performed a 10-fold cross-validation of the model. We validated the clinical outcome model on an independent cohort referred to the fast-track ultrasound clinics of two European rheumatology centres.

**Results:** We included 135 patients with GCA from TABUL (female: 68%, age 73 ±8) and 72 patients from an independent cohort (female: 46%, age 75±7). The

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**Background:** Giant cell arteritis (GCA) is a granulomatous vasculitis of medium and large arteries. In GCA-affected arteries, vascular wall is destroyed by tissue-infiltrating CD4 T cells and macrophages, which leads to intimal neangiogen- esis, intimal hyperplasia and luminal occlusion.

**Objectives:** This study aimed to examine how CD28 signaling plays a role in vasculitis induction and maintenance and which pathogenic processes are depend- ent on CD28-mediated T-cell activation.

**Methods:** We engrafted human arteries into immunodeficient NSG mice and mice engrafted by transfecting GCA immune cells. Human artery-NSG chime- ras were treated with anti-CD28 domain antibody or control Ab. Using tissue section analysis, immunohistochemistry, flow cytometry and immuno- metabolic analysis, treatment effects were examined in vivo and in vitro.

**Results:** Treatment of humanized mice with an anti-CD28 antibody domain pro- foundly reduced tissue-infiltrating T-cells and effectively suppressed vasculitis. Mechanistic studies revealed that CD28 regulated AKT signaling, T-cell prolifera- tion and differentiation of IFN-γ and IL-21-producing effector T-cells. Blocking CD28 signaling disrupted T-cell metabolic fitness; particularly, glucose utilization. Expression of the glucose transporter Glut1 and of glycolytic enzymes as well as mitochondrial oxygen consumption all rely on CD28 signaling. CD28 blockade effectively suppressed vessel wall remodeling processes such as adventitial microvesSEL formation and intimal hyperplasia as well as induction and mainte- nance of CD4⁺/CD103⁺ tissue-resident memory T cells.

**Conclusion:** CD28 stimulation provides a metabolic signal required for patho- genic effector functions in GCA, implicating CD28 signaling as a promising therapeu- tic target.

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