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**AB0068**

**TOCILIZUMAB TREATMENT FOR LARGE VESSELS VASCULITIS: REAL LIFE PREDILEMME EXPERIENCES**

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**Background:** IL-6r targeting therapy has been proven to be effective and safe in Giant Cell Arteritis (GCA) as well in Takayasu Arteritis (TA) in RCTs, probably because of the similar pathologic findings and vessel size (Large Vessel Vasculitis, LVV). However, real world data are scarce.

**Objectives:** The aim of the study was to evaluate the effectiveness of Tocilizumab in LVV in real life settings.

**Methods:** We retrospectively evaluated, from 2011 to 2017, the outcomes (including glucocorticoid dosage) in patients affected by LVV (according to 1990 ACR classification criteria) who received 8 mg/kg iv Tocilizumab (TCZ) monthly, due to the inadequate response to immunosuppressant. Demographic and clinical characteristics and laboratory findings were collected at baseline and consecutive follow up visits, over 52 weeks. Statistical analysis was performed using the GraphPad Software ver. 6.0 (San Diego CA USA) using appropriate tests.

**Results:** We analyzed n.10 patients (6/10 GCA, 4/10 TA) with mean age (± SD) 56 ± 21 years and mean disease duration 41±38 months. Eight out of 10 patients were female. Over the entire observation period, 7/10 also received concomitant methotrexate (mean dose 12.6 ± 2.7 mg/week) while 2/10 received azathioprine (100 mg/day). Median (IQR) prednisone equivalent dose at baseline was 22.5 (10-25) mg/day. At baseline, median ESR was 49 (31-58) mm/h and median CRP level was 15.4 (1.95-28.53) mg/l. Upon TCZ treatment we observed a good disease control in absence of headache, fever and other LVV clinical signs through 52-week follow up. At 52 weeks, we observed a significant reduction of ESR down to 6.5 (2.75-12.25) mm/h as well as of CRP level, down to 1.5 (0.63-5.37) mg/l (p<0.001). A meaningful steroid sparing effect was observed between the groups. Patients with active vasculitis demonstrated higher rate of “rolling”, 74.1% ±27 vs. 12.5%±22 vs. 6.5%±12, p<0.001; microhemorrhages or PCS 30.1%±26 vs. 1.5%±4.5 vs. 1.9%±1, p<0.05, which reversely correlated with disease duration r=-0.4, P=0.05,avascular areas 76±19.5 vs. 23.4% ±28 vs. 26±18.5, p<0.01 and neoangiogenesis 57%±23% vs 26.5% ±22.5 vs. 6% ±10.5, p<0.01. PCS was observed exclusively in 5 of 17 patients with active vasculitis. "rolling", microhemorrhages, avascular areas and neoangiogenesis. PCS may be a specific sign of active vasculitis. NVC is an easy, readily available additive tool in the management of vasculitis. Further studies are needed to ascertain the role of NVC in vasculitis.

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**AB0069**

**NAILFOLD VIDEO CAPILLAROSCOPY AS A POTENTIAL DIAGNOSTIC TOOL IN SYSTEMIC VASCULITIS**

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**Background:** Vasculitides are primarily classified by artery size: large, medium or small, yet some overlap is evident as in Takayasu, a large vessel vasculitis manifesting also in retinal arterioles. Nailfold videocapillaroscopy (NVC) enables us to inspect changes in microvasculature. Only several small uncontrolled case series of light capillary microscopy in adult patients with vasculitis were reported in the literature, describing avascular areas and microhemorrhages in granulomatosis with polyangiitis (GPA) patients [1-2] and in [4] Behcet disease, and thin and tortuous capillaries in Takayasu arteritis [5].

**Objectives:** To characterize nailfold capillary changes by NVC in patients with autoimmune vasculitis compared to healthy controls.

**Methods:** Consecutive autoimmune vasculitis patients fulfilling the ACR criteria and age and gender matched healthy controls were evaluated by NVC using Optika Mediscope with a magnification of X200. Patients with peripheral artery disease and ischemic heart disease were excluded. Capillaroscopy images were centrally analyzed. NVC was analyzed, noting: architecture, number of capillaries per field, capillary width, capillary morphology, microhemorrhages, peri-capillary stippling (PCS)- hemosiderin deposits probably representing former capillary leak, slow capillary flow (“rolling” or sludging of red blood cells) and avascularity. Continuous data are presented as the mean ± SD. Categorical variables are presented as frequencies and percentages. Comparisons of continues variables were made using 2-tailed t-tests and differences between groups by a 1-Way ANOVA.

**Results:** Seventeen patients with active vasculitis, 8 patients with vasculitis in remission (11 polyarteritis nodosum, 2 GPA, 3 eosinophilic granulomatosis with polyangiitis, 2 microscopic polyangiitis, 2 Takayasu, 3 Sjogren vasculitis (one with cryoglobulinemia), 1 primary central nervous system vasculitis and 1 lupus vasculitis) were compared to 25 age and sex matched healthy controls. The mean age (59 ±18 vs. 51±19 vs. 52 ±15), and the percent of females (53%, 50%, 60%), were similar between the groups. Patients with active vasculitis demonstrated higher rate of “rolling”, 74.1% ±27 vs. 12.5%±22 vs. 6.5%±12, p<0.001; microhemorrhages or PCS 30.1%±26 vs. 1.5%±4.5 vs. 1.9%±1, p<0.05, which reversely correlated with disease duration r=-0.4, P=0.05,avascular areas 76±19.5 vs. 23.4% ±28 vs. 26±18.5, p<0.01 and neoangiogenesis 57%±23% vs 26.5% ±22.5 vs. 6% ±10.5, p<0.01. PCS was observed exclusively in 5 of 17 patients with active vasculitis. “rolling”, microhemorrhages, avascular areas and neoangiogenesis. PCS may be a specific sign of active vasculitis. NVC is an easy, readily available additive tool in the management of vasculitis. Further studies are needed to ascertain the role of NVC in vasculitis.

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