Table 2 Clinical findings in patients of 2 groups

		Early-remission n=41		Long-therapy n=21		p
Demographic, clinical, and laboratory findings at diagnosis						
Woman	n (%)	20	(48.8)	12	(57.1)	0.5
Age: years	mean (SD)	74.2	(7.8)	73.7	(5.8)	0.8
Body weight, kg	mean (SD)	55.3	(11.18)	52.1	(9.23)	0.3
Body mass index, kg/m ²	mean (SD)	22.7	(4.03)	20.7	(3.10)	0.06
Fever	n (%)	10	(24.4)	7	(33.3)	0.5
Body weight loss	n (%)	13	(31.7)	10	(47.6)	0.2
Peripheral edema	n (%)	16	(39.0)	10	(47.6)	0.3
Serum C-reactive protein, mg/dL	mean (SD)	6.4	(4.17)	8.8	(4.06)	0.03
Serum albumin, mg/dL	mean (SD)	3.2	(0.47)	3.2	(0.57)	0.7
Treatment and clinical course						
Starting dose of PSL: mg/day	mean (SD)	12.6	(4.09)	14.5	(3.94)	0.1
mg/kg/day	mean (SD)	0.24	(0.084)	0.28	(0.079)	0.1
Mean duration of GC treatment	month (SD)	14.2	(4.59)	43.4	(21.98)	<0.01
Achieving remission	n (%)	41	(100)	9	(42.9)	
Relapse: times	mean (SD)	0.4	(0.70)	3.1	(2.06)	<0.01
Relapse till 6 months	n (%)	3	(7.3)	8	(38.1)	<0.01
PSL, prednisolone; GC, glucocorticoid; SD, standard deviation						

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THU0294

THE DIFFERENCES IN THE CLINICAL SPECTRUM OF CRYOGLOBULINEMIC VASCULITIS BETWEEN SJÖGREN'S SYNDROME AND HCV HEPATITIS

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Background: Cryoglobulinemic vasculitis (CV) is a serious complication of Sjögren's syndrome (SS) and is closely associated with type II IgMk cryoglobulins. CV has been well documented in HCV patients without SS, and shares common features with CV in SS. So far, few studies have described the clinical picture of CV in HCV negative SS, but the number of studied patients was rather small and CV was not well defined. To better describe the clinical spectrum of CV in SS and explore the differences compared to HCV-related CV, a large cohort of well characterized patients is required.

Objectives: To study the clinical phenotype of CV in HCV-negative SS patients, in a large cohort of well characterized patients, after applying stringent classification CV criteria and in comparison with the classical CV of HCV patients.

Methods: From a total cohort of 1997 consecutive SS patients who fulfill the 2016 EULAR/ACR criteria and are followed up in 5 clinical centers ([Universities of Udine, Pisa and Athens, Harokopio and Ioannina, (UPAH)], those who fulfill the 2011 classification criteria for CV were identified and compared with matched HCV-CV patients according to age and gender. Glandular, extra-glandular manifestations and serologic features were compared between the 2 CV groups. Statistical analysis for categorical variables was performed by Fisher exact or chi-square tests and for continuous variables with t test or Mann-Whitney accordingly.

Results: Among the 1083 SS patients who have been evaluated for cryoglobulins, 114 (9.8%) were found positive. Seventy-one (6.5%) SS patients met the 2011 CV criteria while 44 patients presented with type II IgMk cryoglobulinemia without CV. Sixty nine of 71 (97%) SS related CV patients were females and 2 of 71 (3%) males. Forty eight of 71 (68%) had SS disease onset >35 and <65 years old while 14/71 (19.7%) and 9/71 (12.7%) had SS disease onset <35 and >65 years old respectively. The most common clinical manifestations of CV among SS patients were purpura (90%) followed by arthralgias (70%), fatigue (59%), Raynaud's phenomenon (48%), lymphadenopathy (31%), peripheral neuropathy (22.3%), vasculitic ulcer (11.3%) and glomerulonephritis (11.3%). Interestingly, almost 50% of SS related CV patients developed lymphoma and displayed high frequency of strong predictors including purpura, low C4 complement (88.6%) and salivary gland enlargement (SGE). Compared to HCV-CV patients, SS-CV

patients had higher frequency of sicca manifestations, SGE, fatigue, arthritis, Raynaud's phenomenon, lymphadenopathy, type II IgMk cryoglobulins and lymphoma.

Conclusion: The prevalence of cryoglobulinemia and CV among SS patients is about 10% and 6-7% respectively. SS-CV patients are mainly middle-aged females with purpura as the main clinical manifestation, and up to one half of them may develop lymphoma, which is rarer in HCV-CV. Compared to HCV-CV patients, SS patients with CV have more frequently sicca symptoms, SGE and type II IgMk cryoglobulins.

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THU0295

LOW IMMUNOGENICITY IN PATIENTS WITH GIANT CELL ARTERITIS TREATED WITH TOCILIZUMAB: 3-YEAR RESULTS FROM THE RANDOMIZED CONTROLLED PORTION AND OPEN-LABEL FOLLOW-UP OF A PHASE 3 TRIAL

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Background: Tocilizumab (TCZ) has low immunogenicity in patients with rheumatoid arthritis.¹ The risk for immunogenicity remains to be determined in patients with giant cell arteritis (GCA) treated with TCZ. TCZ administered subcutaneously every week (QW) or every other week (Q2W) with 26-week prednisone tapering was superior to placebo (PBO) plus 26-week (PBO+26) or 52-week (PBO+52) prednisone tapering for the achievement of sustained remission in patients with GCA in the 52-week, double-blind part 1 of the GiACTA trial.² Part 2 was a 2-year open-label, long-term follow-up in which patients were treated at the investigators' discretion; part 2 treatment could include initiation/termination of TCZ QW with or without glucocorticoids or methotrexate.

Objectives: To investigate immunogenicity of TCZ QW and Q2W regimens in patients with GCA in combination with a 26-week prednisone taper regimen versus PBO+26 or PBO+52 over the course of the GiACTA study in the randomized controlled part 1 and long-term follow-up part 2.

Methods: In parts 1 and 2 combined, anti-TCZ antibodies (ADA) and corresponding pharmacokinetic (PK) parameters were assessed in serum samples taken at scheduled times at weeks 0, 8, 24, 36, 52, 76, 100, 136, and 156 or at early withdrawal. Additional assessments were made for patients who interrupted blinded TCZ treatment for ≥4 weeks in part 1 and those who withdrew from the study because of anaphylaxis/hypersensitivity. All samples were tested by screening assay, and samples that were ADA positive were further analyzed by a confirmation assay to verify specificity. If the confirmation assay was positive, 2 additional tests were performed to characterize the detected ADA: a neutralizing assay to test the neutralizing potential of ADAs and an assay to determine whether the detected ADA were of the IgE isotype. Proportions of patients in whom ADA developed were summarized for the safety population.

Results: Among evaluable patients (had baseline and ≥1 postbaseline ADA assessments and received ≥1 dose of study treatment) in part 1, ADA developed in 1 of 95 (1.1%) and 3 of 46 (6.5%) patients after TCZ QW and Q2W dosing, respectively. One of 49 (2.0%) and 1 of 47 (2.1%) in the PBO+26 and PBO+52 groups, respectively, tested positive for ADA but had not received TCZ and were considered false positives. In parts 1 and 2 combined, among 199 patients who received ≥1 dose of TCZ, 193 (97%) were evaluable (Table); TCZ-induced ADA developed in 13 of these patients (6.7%) postbaseline (4 during part 1, 9 during part 2). Of these 13 patients, 8 (4.1%) had ADA with neutralizing potential and 1 (0.5%) had IgE ADA. Most TCZ-induced ADA were transient. There was no clear impact of TCZ-induced ADA on TCZ PK (Figure). No patients with TCZ-induced ADA experienced anaphylaxis, hypersensitivity reactions, or injection site reactions, and none withdrew because of lack of efficacy.

Conclusion: In patients with GCA, treatment-induced ADA developed in a minority of patients and had no impact on TCZ PK, efficacy, or safety. The