Background: Recently evolved from a monochromic flu-like disease to a poly-symmetric “spectrum of disease,” our understanding of coronavirus disease 2019 (COVID-19) is still far from being complete [1]. Hyperinflammation involving not only the lungs but also the musculoskeletal system, skin, cardiovascular, genitourinary systems is immune-mediated resembling the flares of a full-blown rheumatic disease [2,3].

Objectives: To describe the prevalence and type of rheumatic manifestations in a cohort of COVID-19 patients hospitalized in the COVID-19 rheumatology department in University Hospital St. Marina, Varna, Bulgaria.

Methods: In the present single-center cohort study, a retrospective database analysis was performed among all COVID-19 patients hospitalized from 1 Dec 2020 to 22 Jan 2021. All 243 patients (age 19 - 93 years) were treated for moderate or severe SARS-CoV-2 infection confirmed by laboratory tests, including positive polymerase chain reaction (PCR) test, and imaging modality. Inpatient treatment included antibiotics, dexamethasone, anticoagulants, and antiviral drug remdesivir (optional). Detailed disease history and clinical examination were carried out by a fully certified rheumatologist and/or specialist in internal medicine.

Results: Among all 243 COVID-19 patients, those with prominent self-reported myalgia and arthralgia were 26% (n = 63) and 21.3% (n = 52), respectively. We had 4 (1.6%) cases of newly developed cutaneous vasculitis and 2 (0.8%) cases of severe Raynaud’s phenomenon after SARS-CoV-2 infection onset. Two patients experienced severe muscle weakness, had elevated creatine phosphokinase, and were diagnosed with inflammatory myopathy secondary to COVID-19. Lupus-like syndrome was observed in 2 (0.8%) patients.

Conclusion: Rheumatic manifestations are part of the heterogeneous spectrum of COVID-19 disease. Amidst the COVID-19 crisis, each newly onset rheumatic manifestation warrants exclusion of SARS-CoV-2 infection. Therefore, a rheumatologist should be a part of a multidisciplinary approach towards the COVID-19 treatment.

REFERENCES:

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Information about the course of COVID-19 in patients with certain rheumatic diseases is still lacking.

**Objectives:** To examine clinical course of COVID-19 in patients with Sjögren’s syndrome treated with anti-CD20 monoclonal antibody (rituximab).

**Methods:** Single center observational study. Diagnosis of SjS was based on ECR/EULAR 2016 criteria. COVID-19 diagnosis was based on positive PCR test even without clinical symptoms and/or typical clinical features (CT signs, fever and anosmia). Rituximab was administrated in two 1000 mg infusions 14 days apart for the 1st course, then 500 mg every 6 months.

**Results:** 19 patients were included, 18 women and 1 man. Median age was 55 years (29-70 years), and median rituximab treatment duration was 24 months (1-48 months). Five patients had concomitant RA (2 patients), SLAE (1 pt), Systemic sclerosis (2 patients). Patients with RA took baricitinib and methotrexate as well. Three patients had chronic lymphocytic heart disease and/or arterial hypertension. 12 patients were PCR positive, 6 negative and in 1 test the result was not done. 11 patients had full and 4 partial B-cell depletion in peripheral blood. Five patients had <20% lung involvement on CT, 2 patients – 20%-40% and 4 patients – 40-60%. Three patients with 40-60% lung involvement required hospitalization due to marked shortness of breath and long febrile period, 2 of them received anti-IL6 treatment and neither of them required mechanical lung ventilation (either non-invasive or invasive). Seventeen patients were treated at home and recovered in 10-24 days. Anti-SARS-CoV-2 IgG were measured in 9 patients, 6 (66.7%) of them were positive.

**Conclusion:** It seems that neither SjS itself nor anti-CD20 therapy predisposes patients to severe course of COVID-19. Presumably risk factors such as age, diabetes or anamnesis of cardiovascular diseases have far more significant impact on COVID-19 severity. Data hints that anti-CD20 therapy might negatively affect the formation of specific anti-SARS-CoV-2 humoral immunity, but further investigation is required to determine that with any degree of certainty.

**Disclosure of Interests:** None declared

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