Current depression and anxiety scores were correlated with disease activity (HADs Depression vs BASDAI $r:0.530$, $p<0.001$; HADs Anxiety vs BASDAI $r:0.500$, $p<0.001$) and function (HADs-Depression vs BASFI $r:0.519$, $p<0.001$; HADs-Anxiety vs BASFI $r:0.391$, $p<0.001$). These relationships were also observed in the pre-pandemic period (HADs-Depression vs BASFI $r:0.326$, $p<0.05$; HADs-Anxiety vs BASDAI $r:0.342$, $p<0.05$).

Conclusion: Depression and anxiety symptoms seems to be comparable before and after the COVID-19 pandemic. Regardless of this period, the presence of both depression and anxiety are associated with disease activity, function and less patient acceptable symptom state.

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Figure 1. Severity of depression and anxiety in AxSpA before and during COVID-19 pandemic

Figure 1.

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AB0670

THE COURSE AND OUTCOMES OF COVID-19 IN PATIENTS WITH ANCA-ASSOCIATED SYSTEMIC VASCULITIS, RECEIVING ANTI-B CELL THERAPY WITH RITUXIMAB

T. Beketova¹, V. Babak¹, M. Suprun¹. ¹V.A. Nasonova Research Institute of Rheumatology, Moscow, Russian Federation

Background: Currently, the issues of the effect of anti-B cell therapy on the risk of severe course of COVID-19 in patients (pts) with ANCA-associated vasculitis (AAV) has not been completely studied.

Objectives: We present an analysis of the COVID-19 incidence and outcomes in 126 AAV pts treated with rituximab (RTX) from one rheumatology center registry.

Methods: Data were collected with telephone/online surveying between 11-15th November 2020 from all 126 AAV pts (58- granulomatosis with polyangiitis, GPA; 38- microscopic polyangiitis, MPA; 22- eosinophilic granulomatosis with polyangiitis, EGPA (54% ANCA-negative); and 8- uncertain AAV). Pts age was 51 (20-81) yr, 62% women. Due to AAV activity or risk of relapse, during the pandemic RTX was prescribed in 48% pts.

Results: The incidence of COVID-19 in AAV pts was 3.5% during the first 3 months of the pandemic, the course was uneventful, and all pts recovered. Since September 2020 a 4-fold increase in the incidence alongside with more severe course of COVID-19 were reported. After 8 months of the pandemic, COVID-19 was diagnosed in 21 (16.6%) pts with median age 55 yr, 57% women. COVID-19 incidence was lower in GPA (15.5%) vs MPA (21.1%) or EGPA (27.7%). The mortality rate was 14.3% (2- MPA, 1- GPA). When analyzing the 5-year survival rate of AAV pts treated with RTX, prognosis worsening was noted; in 2020 there were 3 deaths due to COVID-19, in the previous 5 years- only 2 deaths.

Conclusion: Taking into account the fact the mechanisms of AAV and severe COVID-19 are largely synergistic (Table 1), primarily in the context of obstructive thromboinflammation as manifestation of acute inflammatory syndrome in COVID-19 [1], AAV can potentially contribute to the severe course of COVID-19. Anti-CD20 therapy seems to have a protective effect against severe COVID-19 [2], which, however, can be insufficient in AAV pts with active disease and multiple organ damage. Further analysis of COVID-19 cases in pts with AAV and other rheumatic diseases is exceptionally important.

Table 1. Comparison of AAV and severe COVID-19

Parameter	AAV	Severe COVID-19
Lung damage	High frequency of lung damage	Possibly destructive lung damage
Damage to other organs	Typically affects the upper and lower airways and kidneys; Other organs can be involved	Possible extrapulmonary localization
Vascular involvement	Systemic necrotizing vasculitis of small vessels; In the active phase, hypercoagulation High or very high	Obstructive thromboinflammation of the microcirculation of lungs and extrapulmonary vessels; Increased incidence of Kawasaki syndrome during the pandemic Very high
Inflammation Autoantibodies	ANCA pathogenetic role	Dominant extrafollicular B cell responses; Correlation between SARS-CoV-2 antibody titer and severity of COVID-19; High titers of IFN- α 2/ ω autoantibodies were noted; Netosis, the release of proteases from neutrophils promote the complement system activation, hypercoagulation, endothellitis, production of pro-inflammatory cytokines; An increase of neutrophils in the blood and broncho-alveolar lavage fluid were noted
Neutrophil	Neutrophils are the most important effector cells in the pathogenesis	Changes in the morphology of neutrophils were noted; Netosis, the release of proteases from neutrophils promote the complement system activation, hypercoagulation, endothellitis, production of pro-inflammatory cytokines; An increase of neutrophils in the blood and broncho-alveolar lavage fluid were noted
Complement	Complement is crucial in the pathogenesis; C5a receptor antagonist Avacopan efficacy	Activation of alternative/lectin complement pathways; C5 antagonist Eculizumab has been reported to be effective
Treatment	Immunosuppressants; In the active phase, anticoagulants	Anticoagulants; Glucocorticoids, pro-inflammatory cytokine antagonists, JAK inhibitors, cyclosporine
Prognosis	Serious	High mortality rate

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AB0671

NO EVIDENCE OF SARS-COV-2 IN THE KNEE JOINT: A CADAVER STUDY

M. Grassi¹, V. Giorgi², M. Nebuloni³, P. Zerbi³, M. R. Gismondo⁴, F. Salaffi⁵, P. Sarzi-Puttini², S. G. Rimoldi⁴, A. Manzotti¹. ¹ASST-Fatebenefratelli L. Sacco University Hospital, Orthopaedic and Traumatology Unit, Milan, Italy; ²ASST Fatebenefratelli L. Sacco University Hospital, Rheumatology Unit, Milan, Italy; ³ASST-FBF Sacco, University of Milan, Pathology Unit, Department of Biomedical and Clinical Sciences, Milan, Italy; ⁴ASST-Fatebenefratelli L. Sacco University Hospital, Microbiology Unit, Milan, Italy; ⁵Università Politecnica delle Marche, Rheumatology Department, Jesi, Italy

Background: Despite the considerable research efforts being made to learn more about COVID-19, little is known about the presence of SARS-CoV-2 genetic material in biological fluids other than respiratory droplets, blood, and feces^{1,2}. In particular, little is known about the presence of SARS-CoV-2 in the joints either *post mortem*³ or *in vivo*⁴. To the best of our knowledge, only López-Gonzalez *et al.*⁵ have published a description of acute arthritides occurring during hospitalisation due to COVID-19, and they did not find any SARS-CoV-2 genetic material in the patients' synovial fluid samples.

Objectives: The aim of this *post mortem* study was to assess the presence of SARS-CoV-2 RNA in the knee synovial fluid, synovial tissue, and bone tissue of COVID-19 patients in order to discover whether the joint is a possible route of transmission during orthopaedic surgical procedures, and clarify the possible role of SARS-CoV-2 as a directly arthritogenic virus.

Methods: *Post mortem* synovial fluid, synovial tissue and bone tissue samples were collected from the knees of five patients who died of COVID-19 in our hospital between September and October 2020, and analysed for the presence of SARS-CoV-2 using a commercial real-time polymerase chain reaction (RT-PCR) panel. Quantitative RT-PCR was used to test *post mortem* nasopharyngeal swabs of all of the patients.

Results: No SARS-CoV-2 RNA was detected in any of the knee samples, despite the positivity of the throat swab.

Conclusion: Our findings indicate that SARS-CoV-2 was not detected in knee synovial fluid, synovial membrane or bone. Therefore, our results suggest that all