Conclusion: Between March and July 2020, IMIDT patients, whether or not taking imeds, did not show an increased risk of reported COVID-like symptoms compared to controls. Continuing immunosuppressant drugs as long as not ill, while following the Dutch COVID rules, appears to be safe.

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AB0668

THE ONSET OF RHEUMATOID ARTHRITIS AFTER COVID-19 – COINCIDENCE OR CONNECTED?

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Background: COVID-19, caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has been suggested to induce autoimmune phenomena. Multiple studies have reported the presence of autoantibodies in patients with COVID-19. Also the presence of anti-citrullinated protein antibodies (ACPA) and flaring of rheumatoid arthritis (RA) after COVID-19 has been described.[1, 2] Furthermore, in rheumatology clinics patients may present with polyarthritis compatible with RA shortly after SARS-CoV-2 infection. However, it is unclear how often ACPA occur after COVID-19 and whether preceding COVID-19 impacts on disease presentation of RA and phenotype of the ACPA response.

Objectives: This study aims to determine the seroprevalence of ACPA after COVID-19 and to investigate the association between preceding COVID-19 infection and disease presentation of new-onset RA, including clinical phenotype and autoantibody response.

Methods: To estimate the prevalence of ACPA after COVID-19 we measured ACPA IgG in samples from 61 patients visiting the specialized post-COVID outpatient clinic of the LUMC 5 weeks after hospitalization, using routine tests or in-house ELISA. Furthermore, we identified 5 patients presenting with polyarthritis compatible with RA after SARS-CoV-2 infection. To study the impact of COVID-19 on disease presentation, we examined clinical phenotype, autoantibody isotype positivity and ACPA IgG variable domain (V-domain) glycosylation of these patients and compared these features to regular RA patients. Autoantibody isotypes, including rheumatoid factor (RF) IgM/IgA, anti-CCP2 IgG/ IgM/IgA and anti-carbamylated protein antibodies (anti-CarP) IgG were measured using in-house ELISAs. The percentage of V-domain glycosylation of purified ACPA IgG was measured with UHPLC.

Results: None of the 61 post-COVID patients tested positive for ACPA 5 weeks after hospitalization, except two patients previously diagnosed with ACPA-positive RA. Thus, we could not observe an increase in ACPA-positivity shortly after COVID-19. Of the 5 patients who developed polyarthritis compatible with RA after SARS-CoV-2 infection, the average age was 63.6 years and 2/5 were female. 4/5 patients had been hospitalized due to severe COVID-19. On average, joint complaints started 6.6 weeks after infection, although two patients reported symptoms before infection. 4/5 patients fulfilled the ACR 2010 criteria for RA. Three patients (patient 1, 4, 5) were phenotypically very similar to regular new-onset RA patients. Patient 3 had a history of seronegative RA and had been in DMARD-free remission for 5 years. She flared 6 weeks after SARS-CoV-2 infection. Patient 2 had a remarkably different presentation. He was admitted with a suspected septic polyarthritis or pneumonia with reactive polyarthritis 6 weeks after COVID-19. ACPA level was low positive. The patient died unexpectedly after two days and autopsy revealed dilating myocarditis of unclear underlying cause. No causative pathogen could be identified.

Previous studies have shown that RA-patients are most often either seronegative or triple-positive for RF, ACPA and anti-CarP antibodies. Autoantibody measurements on sera of the post-COVID polyarthritis patients revealed a similar pattern (Figure 1A) with two patients being completely seronegative, and three patients positive for a range of autoantibodies. In all post-COVID samples, the percentage of ACPA IgG V-domain glycosylation was significantly increased compared to total IgG (Figure 1B), similar as in regular RA.

Conclusion: In conclusion, we found that the seroprevalence of ACPA is not increased post-COVID and that most patients presenting with polyarthritis after COVID-19 resemble regular RA patients, both regarding clinical phenotype and autoantibody characteristics. Although sample size and follow-up was limited, it appears that RA post-COVID may be coincidence rather than connected.

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AB0669

DEPRESSION AND ANXIETY MIGHT NOT BE INCREASED DURING COVID-19 PANDEMIC IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS

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Background: Anxiety and depression are most common psychiatric disorders in chronic inflammatory rheumatic condition as well as axial spondyloarthritis (axSpA) (1). The prevalence of depression has been reported as 11-64% depending on the criteria used. Also self-reported depression and anxiety were found to be associated with disease activity and function in axSpA (1,2). It is observed that mental health is affected among healthy subjects during the COVID-19 pandemic, but this condition has not been systematically reviewed in axSpA patients.

Objectives: We aimed to compare frequency of self-reported depression and anxiety before and during the Covid-19 pandemic in patients with axSpA.

Methods: Seventy-six axSpA patients who were evaluated for the presence of depression and anxiety by using Hospital Anxiety and Depression scale (HADS) before pandemic were included in this study. All participants were classified according to the ASAS axSpA classification criteria. Patients were contacted by phone to participate and complete the HADS questionnaire. Demographic and disease related characteristics including BASDAI, BASFI and Patient Acceptable Symptom State (PASS) were recorded during interview. The HADS cut off value was taken as >7 in both groups to define the presence of anxiety or depression. Before and during pandemic period anxiety and depression scores were statistically compared.

Results: The demographic and disease related characteristics of axSpA patients with and without anxiety/depression were summarized in Table 1. The frequency of anxiety (43.4% vs %43.4; p>0.05) and depression (46.1% vs 44.7%; p>0.05) were found to be similar before and during pandemic period. Patients with anxiety (HADS>7) and depression (HADS>7) had higher BASDAI and BASFI scores and much less PASS positivity (Table 1). Although the frequency of depression was similar between before and during the pandemic period, symptom severity in depression was slightly increased during the pandemic (Figure 1).

Table 1. Patients' demographics and characteristics according to the presence of anxiety and depression

Variables	Presence of depression n:35	Absence of depression n:41	P	Presence of anxiety n:33	Absence of anxiety n:43	P
Age (years) mean ± SD	41.8±11.2	44.1±9.3	>0.05	42.0±10.9	43.6±10.0	>0.05
Male n(%)	21(60.0)	26(63.4)	>0.05	18(54.5)	29(67.4)	>0.05
Education time (years) mean ± SD	9.6±4.8	11.0±4.2	>0.05	9.7±5.0	10.6±4.1	>0.05
Current smoker n(%)	18(51.4)	15(36.6)	>0.05	15(45.5)	18(41.9)	>0.05
Alcohol consumption n(%)	12(34.3)	12(29.3)	>0.05	10(30.3)	14(32.6)	>0.05
Current BMI kg/m ² mean ± SD	26.0±4.8	26.8±4.5	>0.05	26.4±5.0	26.5±4.3	>0.05
Sleep time (hours) mean ± SD	7.6±1.7	7.6±1.3	>0.05	7.5±1.6	7.7±1.4	>0.05
Current BASDAI mean ± SD	2.5±1.6	1.4±1.6	<0.05	2.7±1.8	1.3±1.3	<0.001
Current BASFI mean ± SD	2.4±2.1	1.1±1.3	<0.05	2.4±2.0	1.2±1.4	<0.05
PASS positivity n(%)	16(45.7)	29(70.7)	<0.05	14(42.4)	31(72.1)	<0.05

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

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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

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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