radiological score. In the case where an overlap of both diseases is suspected, the presence of consolidation in the lower lobes may suggest a COVID-19 pneumonia while the presence of fibrosis inside GGO may indicate a SSC-ILD.

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Background: Several reports have shown that severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection may trigger a vigorous immune response that could lead to the appearance of various autoantibodies such as antinuclear antibodies, antiphospholipid antibodies or anti-neutrophil cytoplasmic antibodies among others. Moreover, the pulmonary involvement in SARS-CoV-2 may resemble that of patients with anti-MDA5 positive syndrome or acute form of antisyntethase syndrome.

Objectives: Our aim was to analyse the presence of anti-MDA5 and other myositis-specific autoantibodies such as the antisyntethase antibodies in patients diagnosed with severe acute respiratory syndrome caused by SARS-CoV-2.

Methods: Retrospective observational study performed in a tertiary care center. We included 28 patients admitted to the intensive care unit with severe acute respiratory syndrome, 14 at the onset of the disease (group A) and 14 after 30 days of being treated in an intensive care unit (group B). Chest CT was performed at the admission. We analyzed the presence of anti-MDA5 and antisyntethase antibodies by immunoblot (Euroimmun®) and in those who were positive we performed a confirmatory test by immunoprecipitation.

Results: All chest CT showed bilateral ground glass pattern. Three out of 14 patients of group A (12 males, 86%, mean ± SD age 67.1 ± 12.2) were positive for antisyntethase antibodies (2 anti-PL7, 1 anti-Jo1), and 6 out of 14 patients of the group B (6 males, 48%, mean ± SD age 68.7 ± 8.1) were positive to antisynthetase antibodies (2 anti-PL7, 2 anti-PL12, 1 anti-EJ, 1 anti-OJ+PL7). Immunoblots also show positivity for other myositis-specific or associated antibodies, such as anti-TIF1g, anti-PM75, anti-SAE and anti-SRP. All of these results found by immunoblotting were negative by immunoprecipitation. None of the 28 patients were positive for anti-MDA5 antibodies.

Conclusion: Severe SARS-CoV-2 pneumonia is characterized by ground glass pattern in chest CT, as it is found in anti-MDA5 or antisyntethase syndrome. The positivity of several myositis related autoantibodies shown in immunoblot appears to be more related to the vigorous immune response producing polyclonal immunoglobulins than triggering a real myositis-associated interstitial lung disease. Clinicians must be aware about these false positive results in patients with severe COVID-19 acute respiratory syndrome.

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Background: Patients with anti-neutrophil cytoplasmatic antibody (ANCA)-asso-
ciated vasculitis (AAV) require immunosuppressive therapy for disease control and reduction of disease relapse and may be at risk for complications during Sars-CoV-2 (COVID-19) infection.

Objectives: To analyze the consequences of COVID-19 in a large cohort of AAV patients regarding occurrence, need of hospitalization, treatment at the intensive care units (ICU), or death.

Methods: Data were retrieved from March 2020 to mid-January 2021 from medical records of the AAV cohort (n=233). Patients diagnosed with granulomatosis with polyangiitis (GPA), microscopic polyangiitis (MPA) or eosino-
philic granulomatosis with polyangiitis (EGPA) were included. Data included age, gender, diagnosis, ongoing immunosuppressive medication at onset of COVID-19 or at last follow-up in non-COVID individuals. Renal involvement (ever) and estimated glomerular filtration rate (eGFR) were included. COVID-19 was confirmed either by a positive PCR test in the upper airways or by serology. Severe COVID-19 was defined as need of non-invasive ventilation, ICU care, and/or death.

Results: The cohort comprised of 172 patients with GPA, 50 with MPA and 11 with EGPA. There were 121 females (52%). During the study period, 20 patients (8.6%) were diagnosed with COVID-19. The median age at data retrieval in all patients was 68 years (21-93), in the COVID-19 group 63 (29-93) and 68.5 (21-90) years in the non-COVID patients. Forty-three patients in all (18%) were hospitalized during the study period of which 11 (4.7%) due to COVID-19 infection. In all, 6 deaths occurred of which 3 were related to COVID-19.

At data retrieval, 110 (47%) patients were on prednisolone treatment, 10/20 (50%) in the COVID-19 group and 100 (47%) in the non-COVID-19 group (p=0.5), with significantly higher doses in COVID-19 patients (p<0.001). In patients hospitalized with COVID-19, 6/11 (54.5%) were on prednisolone, median dose 5mg/day (0-50). In the total group 112 (48%) were on disease modifying anti-rheumatic drugs (DMARD) and 64 (27.5%) on rituximab as maintenance therapy. Eight patients were on induction treatment with either cyclophosphamide or rituximab. Of the 20 COVID-19 cases, 8 had severe COVID-19. Of these, 2 were inactive without immunosuppressive treatment, 4 had stable disease with prednisolone (5-7.5mg/day) in combination with DMARDs, and 2 were active treated with high dose prednisolone (25-50mg/day) in combination with cyclophosphamide and rituximab (n=1) or rituximab (n=1).

A higher proportion of patients had active AAV (p=0.03) in the severe COVID-19 then in the non-COVID group (10/213 patients).

In the group with the severe COVID-19, 1/8 (12%) patient had rituximab as main-
tenance therapy, compared to 61/213 (28.6%) in the group of non-COVID patients (p=0.5).

Renal involvement (ever) was present in 144 patients (62%), in 6 patients (30%) with COVID-19, from which 5 (82%) were in the group of severe COVID-19 patients. Median eGFR did not differ between severe COVID-19 and remaining patients with renal involvement independently of COVID-19 infection.

Conclusion: We found a high rate of severe COVID-19 infection in our cohort of AAV patients which indicates risk for serious complications, especially in patients with active disease and intense immunosuppressive therapy. Maintenance ther-
apy with rituximab did not seem to increase the risk for severe COVID-19. The findings stress the need for continued shielding and early vaccination in AAV patients.

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