Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

247 COVID-19 Severity in Hospitalized Pediatric Patients with Atopic Disease

Dylan Timberlake[1], Rebecca Scherzer, MD FAAAAI[2], Benjamin Prince, MD MCSI FAAAAI[3], Mitchell Grayson, MD[2]; [1]The Ohio State University/Nationwide Children's Hospital, [2]Nationwide Children, [3]Nationwide Children's Hospital.

RATIONALE: Data from the Coronavirus-19 disease (COVID-19) pandemic suggests asthma is not a risk factor for severe disease in adults; it is unclear if this applies to pediatric patients. This study was undertaken to determine if pediatric patients with asthma or atopic disease had altered risk for severe disease when hospitalized with COVID-19.

METHODS: A retrospective chart review was performed of SARS-CoV-2 positive patients admitted to Nationwide Children's Hospital from March 1 to July 31. Charts were evaluated for history of asthma or atopic disease (including asthma) and surrogate markers of COVID-19 severity, including ICU admission, supplemental oxygen requirement, and intubation.

RESULTS: 49 patients were identified as positive for SARS-CoV-2, 22 of whom were admitted for COVID-19 related symptoms. Of the admitted patients, six patients (12%) had asthma and 18 (37%) atopic disease (including those with asthma). ICU admission rate for asthma versus non-asthma was 17% versus 12% ($p=0.78$) and for atopic versus non-atopic was 17% versus 6.4% ($p=0.32$), while supplemental oxygen rates were 17% versus 16% asthma versus non-asthma ($p=0.98$) and 22% versus 13% atopic versus non-atopic ($p=0.43$). Only two patients required intubation and both had atopic dermatitis. Two patients had Multisystem Inflammatory Syndrome in Children – one with allergic rhinitis and atopic dermatitis, the other without any atopic disease.

CONCLUSIONS: Markers of COVID-19 disease severity do not differ based on asthma or atopic status in pediatric patients. In children, like adults, the presence of asthma or atopy does not appear to alter the risk of severe COVID-19.

248 COVID-19 pandemia: Atopy and prospective analysis of the clinical evolution of patients infected with the SARS-CoV-2 virus

Débora S. Guterres[1], Ana Carolina Dos Santos[2], Marina de Almeida Prado Meireles Laubi[2], Marina Laubi[2], Roselene Lourenço[1], Marcus Vinícius[1], Rosana Agondi, MD PhD[3], Ana Karolina Marinho, DO[4], Valéria Brandão[5], Thais Saito[5], Grasiela Lima[5]; [1]Conjunto Hospitalar do Mandaqui, São Paulo, Brazil, [2]Universidade Nove de Julho, São Paulo, Brazil, [3]Hospital das Clínicas Universidade de São Paulo, São Paulo, Brazil, [4]University of Sao Paulo Medical School, [5]Conjunto Hospitalar do Mandaqui.

RATIONALE: A few scientific evidence is now available to date on the clinical evolution of COVID-19 in atopics. The aim of this study was to assess the frequency of atopy in patients hospitalized for COVID-19.

METHODS: A prospective study was carried out with hard descriptive data of 4 months. During this period, 300 patients were admitted with ARS and positive RT-PCR for SARS-CoV-2. Clinical features, history of atopy, total IgE and chest tomography were evaluated.

RESULTS: Of all patients, 212 (70%) were male and the average age was 58 years old. Of the total, 37 patients (12.3%) had a history of atopy according to the following: 19 rhinitis (51.4%), 15 asthma (40.5%) and 3 (1%), atopic dermatitis. Obesity was present in 32% of the evaluated atopics. The mean of atopic patients was aged for 55, 92% O2 Sat, respiratory rate 27 per minute, CT > 50% in 22 patients (7.3%) and 3 patients died (1%). Of these who died, all were elderly (age over 60 years) and had comorbidities such as SH and obesity. Mean IgE total was 538UI/ml, and 3 patients presented IgE above 2000, all of them with clinical suggesting atopy (AD, asthma and rhinitis).

CONCLUSIONS: The literature reports less severe outcomes in atopic patients affected by COVID-19. In the studied group, the frequency of atopy was lower than that observed in the literature for the general population and the tomographic impairment was lower than that of the non-atopic group. The atopic patients who died were elderly and had comorbidities.

249 Characterization of T cell, B cell and Natural Killer Cell Subsets in COVID-19 Patients

Alena Rynda[1], Andrei Hancharou[1], Natalia Antonevich[1], Oxana Timohina[1], Yana Minich[1], Alena Halavach[1], Darya Babrukevich[1], Marina Dotsenko[2], Eduard Dotsenko[2], Lawrence Dubuske, MD FAAAAI[3]; [1]The Institute of Biophysics and Cell Engineering of National Academy of Sciences of Belarus, Minsk, [2]Belarusian State Medical University, Minsk, Belarus, [3]George Washington University School of Medicine, Washington, DC, USA, Immunology Research Institute of New England, Gardner, MA, USA.

RATIONALE: Immune dysregulation is observed in patients with severe COVID-19 associated pneumonia. The aim of the investigation was to perform analysis of immune parameters including cell count, differentiation and activation of lymphocytes from COVID-19 patients.

METHODS: 57 patients with PCR and chest CT confirmed diagnosis of COVID-19 pneumonia were included in the study. Patients were divided into 2 groups: severe/critical (S/C) patients (n=18) which were treated in ICU and required IMV; and moderate (M) patients (n=39). Blood samples from 19 healthy donors (HD) controls were obtained. T cell, B cell, natural killer (NK) cell and innate lymphoid cell (ILC) subsets were assayed using Attune NxT flow cytometer. Nonparametric statistics were used.

RESULTS: A profound decrease of the absolute counts of T-cells, B-cells, NK- and NKT-cells and frequencies (%) of ILC was noted both in S/C and M versus HD ($p<0.01$). Frequency (%) of B-cells in S/C was increased. A decrease in the content of naive T-cells was noted, while the content of TCM, TEM, TEMRA, T-reg and activated HLA-DR$^+$ T-cells did not change significantly in S/C compared with M but both groups had increased exhausted PD-1$^+$ T cells counts.

CONCLUSIONS: T and NK cell lymphopenia was observed in COVID-19 patients, more significant in S/C. No important changes in T-cells differentiation and activation were detected in with S/C or M groups accompanied by increase in exhausted T cells. Immune system anergy may explain the severe course of some COVID 19 patients and cases of reinfection.

250 Effects of Virus Specific Short-Interfering RNAs on Enterovirus D68 Induced Lung Injury

Nicholas Klaiber[1], Michael McVoy[1], Wei Zhao, MD PhD FAAAAI[1]; [1]Department of Pediatrics, Virginia Commonwealth University, Richmond, VA, USA.

RATIONALE: Enterovirus D68 (EV-D68) is an emerging respiratory pathogen. We previously described siRNAs targeting the EV-D68 RNA dependent RNA polymerase, which were able to suppress EV-D68 replication and cytopathic effect *in vitro*. We sought to assess the effects of intranasal application of these siRNAs *in vivo,* utilizing a previously established animal model of EV-D68 infection.

METHODS: 22 female cotton rats were infected i.n. with EV-D68. Two hours p.i. animals were treated intranasally with either EV-D68 specific siRNAs, non-coding siRNAs or PBS. Virus associated lung injury was quantified by a veterinary pathologist utilizing a previously published peribronchiolitis scoring system. Viral VP2 capsid protein expression was measured using indirect immunofluorescence. RT-PCR was used to compare levels of EV-D68 genomic RNA and inflammatory cytokine expression. Viral titers, expression of cytokine genes were calculated as geometric means ± standard error (SE) for all animals in a group at a given time p.i. Student t-test was used to determine statistically significant differences between two groups, using an unpaired, two-tailed test with significance set at $p<0.05$.Pulmonary pathology scores were expressed as the arithmetic means ± SE for all animals in a group.

RESULTS: Peribronchiolitis scores, VP2 expression and inflammatory cytokine expression were all significantly reduced ($p<0.05$) in animals treated with EV-D68 specific siRNA. Viral genome copy number was lower in treated animals, however this difference was not statistically significant ($p=0.06$).

CONCLUSION: I.N. application of EV-D68 specific siRNA was able to decrease virus mediated lung pathology, inflammatory cytokine expression and viral protein expression in a cotton rat model of EV-D68 infection.