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COVID-19 Patients with Obesity are Autoimmune and not Neutralizing for SARS-Cov-2-Specific Antibodies

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Editorial

Obesity is an inflammatory conditions related with inflammaging and chronic IA, contributing to useful impairment of immune cells, and diminished immunity. Obese men and women have been shown to respond poorly, to infections, vaccination, and remedies for autoimmune conditions. Therefore, weight problems represent risk factor for COVID-19 patients. A strong association between obesity, obesity-associated comorbidities, and extreme effects of COVID-19 has certainly been shown, with adult COVID-19 symptomatic sufferers with Body Mass Index (BMI) > 30 showing higher admission to acute and indispensable care in contrast to lean and obese persons (BMI < 30). These effects have been demonstrated in section in a multi-site prospective cohort of non-hospitalized people in whom weight problems used to be observed to be related with the presence of a couple of COVID-19 symptoms. However, in this cohort weight problems used to be no longer related with expanded risk of infection [1].

The novel single-stranded RNA coronavirus SARS-CoV-2 (severe acute respiratory syndrome corona virus-2) emerged in the remaining months of 2019, brought about the global coronavirus ailment 2019 (COVID-19) pandemic, and used to be accountable for special medical manifestations ranging from moderate disorder to extreme respiratory tract infections, multiorgan failure, and death. The extreme manifestations of the disorder are related with an exuberant inflammatory response and the improvement of a circumstance known as cytokine storm. Published records have indicated that inflammaging, the continual low-grade systemic inflammation, is an important reason of the cell and molecular adjustments prompted with the aid of SARS-CoV-2 and can be accountable for the easiest range of deaths. In addition, inflammaging induces persistent immune activation (IA) and dysfunctional immunity.

We have previously evaluated the results of weight problems on the secretion of SARS-CoV-2-specific immunoglobulin G (IgG) antibodies in the blood of lean and overweight/obese COVID-19 patients, and we have proven that SARS-CoV-2 IgG antibodies are negatively related with BMI in COVID-19 patients, as anticipated based totally on the acknowledged consequences of overweight/obesity on humoral immunity [2]. In this study, we have evaluated the excellent of the antibody response in lean (BMI< 25) and obese (BMI > 30) COVID-19 patients, as compared to age, gender, and BMI-matched uninfected controls, besides preceding records of autoimmunity.

It has been currently proven that the sera of adult COVID-19 sufferers include antibodies with autoimmune specificities. We measured the presence of neutralizing antibodies and autoimmune antibodies precise for malondialdehyde (MDA), which is used as a marker of oxidative stress and lipid peroxidation, and for adipocyte-derived protein antigens (AD), hooked up markers of mobile dying in the overweight adipose tissue (AT). This find out about covered uninfected controls considering the fact that weight problems per se is related with the secretion of autoimmune antibodies, as formerly demonstrated. We hypothesized that SARS-CoV-2 contamination in COVID-19 sufferers with weight problems induces oxidative stress and tissue damage, main to cell demise and launch of intracellular antigens that are now not recognised to be immunogenic autoantigens, and greater in obese than in lean patients [3].

Serum IgG antibodies in opposition to SARS-CoV-2 Spike protein have been measured by using an ELISA developed and standardized in our laboratory. Briefly, 96-well microplates have been lined with recombinant NCP-CoV (2019-nCoV) Spike protein (S1 + S2 ECD) at 2 μg/mL for 1 h at room temperature. Plates have been then washed with Tween-20 0.05% in phosphate-buffered saline (PBS) (PBST) and blocked with assay buffer (1% bovine serum albumin (BSA) in PBS) for 1 h at 37 °C. After blocking, all subsequent steps have been carried out via a DYNEX DS2® Automated ELISA device. First, serum samples diluted at 1:50,000 in assay buffer have been introduced in reproduction and plates have been incubated for 2 h. Next, plates have been washed with PBST and 100 μL per properly of a peroxidase-conjugated goat anti-human IgG, diluted 1:10,000 in assay buffer, had been added. After 1 h incubation, plates have been washed, and a stabilized 3,3′,5,5′-tetramethylbenzidine (TMB) substrate (Sigma) used to be delivered to the wells. The enzymatic response was once stopped after 20 min with a give up answer (1 M sulfuric acid), and absorbance at 450 nm was once study by means of the DYNEX DS2 instrument [4] .

The onset of autoimmunity has been related with viral infections, and it has been suggested that SARS-CoV-2 should be a triggering issue for the improvement of a speedy autoimmune, autoinflammatory sickness in genetically predisposed folks as these with excessive systemic interleukin-6, comparable to what has been determined in SARS-CoV, influenza, and dengue infections. In SARS-CoV patients, excessive stages of serum autoantibodies particular for type-2 pneumocytes have been found, and these antibodies had been especially cytotoxic. In influenza patients, virus-induced autoantibodies towards the alveolar and glomerular basement membrane precipitated the autoimmune sickness known as Good pasture's syndrome. In dengue patients, virus-induced autoantibodies precise for endothelial cells, platelets, and coagulatory molecules lead to odd activation or dysfunction [5]. In all these cases, molecular mimicry between viral and host proteins can also provide an explanation for the cross-reactivity of brought on autoantibodies.

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Conflict of Interest

No potential conflicts of interest relevant to this article were reported.

References

1. Casadevall A, Pirofski LA (2020) The convalescent sera option for containing COVID-19. J Clin Invest 130: 1545-1548.
2. Henderson LA, Canna SW, Schulert GS, Volpi S, Lee PY, et al (2020) On the alert for cytokine storm: immunopathology in COVID-19. Arthritis Rheumatol 72: 1059-1063.
3. Mueller AL, McNamara MS, Sinclair DA (2020) Why does COVID-19 disproportionately affect older people? Aging 12: 9959-9981.
4. Frasca D, Diaz A, Romero M, Garcia D, Blomberg BB (2020) B cell immunosenescence. Annu Rev Cell Dev Biol 36: 551-574.
5. Falagas ME, Kompoti M (2006) Obesity and infection. Lancet Infect Dis 6: 438-446.