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Letter to Editor *Volume 4 Issue 1*

Obesity and Higher Risk for Severe Complications of Covid-19

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Letter to Editor

Since December 2019, the novel coronavirus (nCoV-2019) or severe acute coronavirus 2 (SARS-CoV-2) expanded rapidly from Wuhan, China to all over the globe. By planning for the development of diagnostic devices, issuing hospital monitoring, testing and treatment recommendations, and updating disease information, the World Health Organisation (WHO) has responded promptly [1]. Patients infected with coronavirus disease (COVID-19) have recorded rising numbers of leucocytes, abnormal respiration, and elevated plasma amounts of proinflammatory cytokines [2]. The COVID-19 is 96.5 per cent the same as the RaTG13 bat CoV, suggesting that bats could be the virus-host and passed by the nearby and air droplets. Almost any droplet always drops within a few meters, but there is less risk that it will disperse if it is two or more meters away. Across the world, proper SARS-CoV-2 treatment regimens are under investigation; various medicinal products are recycled and proposed for COVID-19 management [3].

Over the past three decades, the epidemic of obesity has risen dramatically: it is now known to be one of world wide's most extreme health problems as a pandemic. The simplest definition of obesity is a rise in adipose fat deposition. However, in adults BMI is used by WHO, people with a BMI over 30 kg/m^2 are known to be affected by obesity [4]. Obesity in COVID19 patients, which is higher in younger people than older adult patients, has been found to raise the risk of death and intensive care in hospital admission [5]. In the past, obesity has been a dangerous factor in extreme COVID-19 effects, such as mechanical invasion, severe pneumonia, and elevated hospitalizations [6].

The correlation of obesity with different comorbidities safely demonstrates their role in the detection of SARS-CoV-2 patients as an exponentially important feature. Between March 1, 2020, to April 4, 2020, a case report on 5700 COVID-19 patients admitted to 12 New York Hospitals shows that 41.7 per cent of patients were obese, making them one of the most frequent comorbidities [7].

The rise of obesity's abdominal diameter compresses the lungs while positioning tied pressures between the lungs and airways. Lower saturation of oxygen in the lung foundation was combined with impaired breathing with abdominal obesity. The respiratory system has an angiotensin-converting enzyme 2 (ACE2) that acts as an anti-inflammatory. SARS-CoV-2 spike glycoprotein enters human cellulose, including the pulmonary, and the possibility of exaggerating systemic inflammatory and electrolytic imbalance, which can have a potentially lethal effect, is also correlated with ACE2 COVID-19 contagious individuals [8]. Chronic inflammation, following obesity, may lead to the inflammatory cascade of the acute kidney defect and shock to COVID-19 patients. Preliminary findings have shown that obesity is a risk factor for a decrease and that this influence is inflamed by a glomerular filtration rate, albuminuria, and the incidence and development of kidney disease. In around 6% of COVID-19 patients, a related shock is seen, and the inflammatory condition appears to play a major role in the onset [5].

Covid-19 Infection and Risks in People with Obesity

- The tissue of the adipose is rich in receptors ACE2 that function as an entry point to human cells in SARS-CoV-2. The higher number of adipocytes will produce more viral load and extended viremia in people with obesity.

- Increased visceral adiposity contributes to an inflammatory cytokine efflux. This affects systemic cellular functions and is related to low inflammation, which can in some cases lead to the 'cytokine tempest' of COVID-19.
- Obesity has been shown to modify the immune system of multiple pathogens and improve its vulnerability to infection. Higher circulating pro-inflammatory cytokines, as well as lower levels of adiponectin, can affect the immunological response. This contributes to the destruction of the composition of the lymphoid tissue and improvements in the concentrations of leukocytes and inflamed diseases. In persons with obesity, B and T cells are also affected, triggering increased susceptibilities and delays in viral infection resolution.
- Obesity is related to a pro-coagulant profile which may play a role in the COVID-19 thromboembolic complications.
- The decreased pulmonary capacity is associated with obesity, including reduced volume of the expiratory reserve and respiratory systems which could raise the risk of complications of COVID-19 in people with obesity [9].
- Though obesity is not listed in SARS-CoV-2 infection co-morbidities, a potential function of obesity has been hypothesized when assessing the seriousness of COVID-19. Most recently, a French study found high obesity prevalence among SARS-CoV-2-related pneumonia admitted to intensive care with an increase in disease severity with BMI [10]. There are several ways of describing the relation of obesity to the severity of COVID-19. Obesity is a well-known risk factor for diabetes, asthma, and cardiovascular diseases, all of which predict bad COVID-19 outcomes. Obesity may also affect an immune reaction and diaphragm excursion to viral infections (thus causing dysventilation). Besides, the treatment of obese COVID-19 patients can be tougher than routine since the scale of the patient will limit both medical and assistive procedures [11]. The pandemic of COVID-19 poses unparalleled threats to the planet. At Obesity, we have been warned about the obesity crisis and we now need to deal with this dual pandemic threat for our obesity patients.

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