Obesity

COVID-19 Rise in Younger Adults with Obesity: Visceral Adiposity Can Predict the Risk

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A concerning rise in coronavirus disease 2019 (COVID-19) cases has been recently reported, particularly in the United States. The causes of this increase are likely multifactorial and the object of an ongoing health and socioeconomic debate. However, preliminary data have indicated that the new COVID-19 cases are increasing among younger adults with obesity. Considering this recent spike, the timing of the paper by Deng et al. (1) is of particular importance. Deng et al. (1) not only confirmed that obesity is a major and independent risk factor for COVID-19 complications in young adults (2) but also pointed out ectopic and visceral fat depots as new markers of that risk. The authors found that computed tomography (CT) imaging showed significantly higher fatty liver and epicardial adipose tissue (EAT) in severely and critically ill patients with COVID-19 under 40 years old as compared with those with milder disease.

In the past few months, we learned that obesity is undoubtedly a risk factor for COVID-19 complications and mortality (2). This is particularly important in the United States, where the prevalence of obesity currently exceeds 40%. The higher risk for people with obesity to develop severe COVID-19 cardiac and pulmonary injuries can be attributed to multiple factors, such as the chronic inflammatory status, the delayed immune response, and possibly fat tissue serving as a reservoir for the virus (3). However, there is an additional mechanism that may not be immediately identified, the ectopic and visceral fat accumulation. We now face a different phenotype of the high-risk patient who is much younger and who certainly has obesity but with prominent visceral obesity.

The findings of Deng et al. (1) may provide new insights to untangle the intricate, and still unclear, physiopathologic pathways leading to COVID-19 organ damage. The role of EAT in causing and worsening the COVID-19 cardiac complications recently emerged (4). EAT is an inflammatory depot with dense macrophage infiltrates, highly enriched in proinflammatory cytokines, such as interleukin (IL6), a cytokine that is overexpressed in patients with COVID-19. EAT and the myocardium share the same microcirculation. EAT inflammatory cytokines can reach out to the myocardium via *vasa vasorum* or paracrine pathways (4). Hence EAT is likely implicated in COVID-19 myocardial inflammation and cardiorespiratory failure.

Intrahepatic and perirenal fat infiltration can also play a causative and predictive role of severe COVID-19. Deng et al. (1) found that severely and critically ill young individuals with obesity presented with imaging signs of liver fat deposition. Fatty liver and perirenal fat accumulation can contribute to insulin resistance, hyperglycemia, and hypoalbuminemia, all measurable predictors of COVID-19 complications, as recently highlighted (5).

Remarkably, the paper by Deng et al. (1) reminds us that EAT and intrahepatic fat can be clinically measured with CT or standard ultrasound. CT-measured EAT and liver density could serve as a reliable indicator of inflammation in patients with COVID-19 (4).

We could, therefore, use clinical and imaging tools to better phenotype and stratify younger individuals with visceral obesity, probably asymptomatic or paucisymptomatic but at higher risk for COVID-19 morbidity and mortality. Promisingly, visceral and ectopic fat can be targeted and reduced with lifestyle and pharmacological interventions.

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