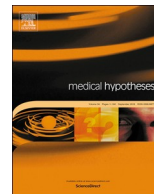




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Obesity, abdominal organ size and COVID-19 severity



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COVID-19 disease is usually more severe in patients with obesity [1]; these patients need more often than normal-weight patients to be mechanically ventilated [2]. The abundant adipose tissue in subjects with obesity overexpresses the receptors and proteases that permit cellular entry of SARS-CoV-2; a role for adipose tissue acting as a virus reservoir and as an accelerator that reinforces violent systemic inflammatory and immune responses has been suggested [3]. In a recent (albeit not yet peer-reviewed) study (n = 750), larger-size abdominal organs (kidneys, pancreas, and liver; assessed with computed tomography) were consistently noted in subjects with obesity versus lean subjects [4]. Abdominal and extrabdominal multiorgan localization of SARS-CoV-2 has been reported [5]. An abdominal-lung connection for SARS-CoV-2 pathogenesis has been proposed in this journal [6].

Based on the above it could be hypothesized that the pathogenesis of the pervasive SARS-CoV-2 infection in patients with obesity may also be a function of the tissue mass – not only of adipose tissue but also of abdominal organs – which is “offered” for infection (we have to note that the assessed viral load has recently been linked with disease mortality [7]). Thus, patients with obesity may carry a relatively larger viral reservoir compared to lean patients, may show overall more extensive infiltration/damage, have a more severe course of COVID-19 infection, and usually take longer to recover. The extent of tissue and organ COVID-19 involvement could be evaluated with biopsy/autopsy/tissue analyses.

Conflict of interest statement

The authors declare no conflict of interest.

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