

## Risk of COVID-19 for patients with obesity

Even though there are very few available data on BMI (body mass index) for patients with COVID-19 infections, the role of obesity in the COVID-19 epidemic must not be ignored. Obesity plays an important role in the pathogenesis of COVID-19 infection. In fact, the immune system, which is a key player in the pathogenesis of COVID-19, also plays an important role in obesity-induced adipose tissue inflammation. This inflammation of adipose tissue results in metabolic dysfunction potentially leading to dyslipidaemia, insulin resistance, type 2 diabetes mellitus, hypertension, and cardiovascular disease.

By analogy to other respiratory infections, obesity may play an important role in COVID-19 transmission. For example, in the case of influenza A, obesity increases the duration of virus shedding; symptomatic patients with obesity shed virus 42% longer than adults who do not have obesity.<sup>1</sup> In H1N1 influenza, obesity is an independent risk factor for hospitalization and death.<sup>2</sup>

Since obesity has been shown to increase vulnerability to infections, it may be a risk factor for COVID-19-related mortality.<sup>3</sup> This is all the more so since there are no specific clinical signs that foreshadow the progression from a mild COVID-19 infection to a severe form. Compared with normal patients, BMI was significantly higher in patients with a severe form of COVID-19 infection ( $27.0 \pm 2.5$  [critical group] versus  $22.0 \pm 1.3$  [general group];  $P < 0.001$ ).<sup>4</sup> Peng et al. published a retrospective analysis on 112 patients with COVID-19 infection admitted to the western district of Union Hospital in Wuhan, from 20 January 2020 to 15 February 2020. In this study, the BMI of the critical group ( $25.5$  [23.0, 27.5]  $\text{kg}/\text{m}^2$ ) was significantly higher ( $P = 0.003$ ) than that of the general group ( $22.0$  [20.0, 24.0]  $\text{kg}/\text{m}^2$ ). Patients were further divided into two groups, survivors (84.8%) and non-survivors (15.18%). Among the non-survivors, 88.2% of patients had a BMI  $> 25$   $\text{kg}/\text{m}^2$ , which is a significantly higher proportion ( $P < 0.001$ ) than in survivors (18.9%).<sup>5</sup> The authors concluded that the highest BMI was more often seen in critical cases and non-survivors. Thrombotic events were an aggravating cause of death.<sup>5</sup> Thromboembolic risk is known to be higher in patients with obesity than in the general population.<sup>6</sup> It logically follows that obesity can be an aggravating risk factor for death from COVID-19 infection.

One explanation of the above findings is that COVID-19 has high affinity for human angiotensin converting enzyme 2 (ACE2). ACE2 has been shown to be the putative receptor for the entry of COVID-19 into host cells.<sup>7</sup> Tissue expression of ACE2 differs in kidneys, heart, and lungs of healthy patients and coronavirus-infected patients.<sup>8</sup> The level of ACE2 expression in adipose tissue is higher than that in lung tissue, a major target tissue affected by COVID-19.<sup>9</sup> This is an important finding because adipose tissue might also be vulnerable to COVID-19. It should be noted, however, that there was no difference in the expression of

ACE2 protein by adipocytes and adipose progenitor cells between individuals with obesity and those without.<sup>10</sup> However, individuals with obesity have more adipose tissue and therefore an increased number of ACE2-expressing cells and consequently a larger amount of ACE2.<sup>9</sup> In addition, treatments with specific anti-hypertensive medications 2 (angiotensin-converting enzyme inhibitors [ACEIs] and angiotensin receptor blockers [ARBs]) will increase expression of ACE2 and increase patient susceptibility to viral host cell entry and propagation.<sup>11</sup>

Another factor might also contribute to the increased risk from COVID-19 for patients with obesity. Adipose tissue can serve as a reservoir for human adenovirus Ad-36, influenza A virus, HIV, cytomegalovirus, *Trypanosoma gondii*, and *Mycobacterium tuberculosis*.<sup>12</sup> By analogy, COVID-19 might also infect adipose tissue and then spread to other organs.

Thus, we recommend extra attention and precautions for patients with obesity during this epidemic. Whenever COVID-19 infection is suspected, screening must be systematic, particularly if the patient has obesity. Adipose tissue can be a research model to help understand the pathogenesis of COVID-19 infection and develop an effective treatment.

### KEYWORDS

adipose, BMI, COVID-19, human angiotensin converting enzyme 2, obesity

### CONFLICT OF INTEREST

No conflict of interest was declared.

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