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The Impact of Obesity on the Prognosis of COVID-19 Patients: Two Pandemics at Once

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ABSTRACT

Objective: This study investigated the impact of obesity on clinical outcomes and mortality among adults hospitalized with COVID-19.

Methods: A total of 258 patients hospitalized and treated in the Pulmonology Department of the University of Health Sciences Sancaktepe Şehit Prof. Dr. İlhan Varank Training and Research Hospital between June 2020 and December 2021 were retrospectively evaluated. Patients were classified into obese (n=125) and non-obese (n=133) groups. Demographic characteristics, laboratory parameters, length of hospital stay, and mortality rates were compared between the groups.

Results: Among the 258 patients included, 148 (54%) were male, with a mean age of 65 ± 15 years. Of these, 125 (45%) were obese. The obese group (63.0 ± 15.7 years) was significantly younger than the non-obese group (68.1 ± 14.2 years) ($p = 0.007$). No significant differences were observed between the groups regarding leukocyte, lymphocyte, CRP, LDH, D-dimer, or ferritin levels. The mean length of hospital stay was comparable between the groups, whereas the mortality rate was significantly higher in the obese group ($p = 0.013$). In addition, ICU admission was significantly more frequent among obese patients than among non-obese patients (36 vs. 16 cases, $p = 0.007$). According to receiver operating characteristic (ROC) analysis, in the obese group, procalcitonin (AUC = 0.862; 95% CI, 0.658–1.000; $p < 0.001$) and D-dimer (AUC = 0.825; 95% CI, 0.733–0.916; $p = 0.001$) exhibited the highest discriminatory ability for predicting mortality. LDH (AUC = 0.739; $p = 0.012$) and ferritin (AUC = 0.716; $p = 0.024$) also demonstrated significant predictive value. In contrast, CRP (AUC = 0.226) and lymphocyte count (AUC = 0.372) did not show discriminative ability. In the non-obese group, only LDH (AUC = 0.776; 95% CI, 0.634–0.918; $p < 0.001$) and D-dimer (AUC = 0.711; $p = 0.005$) demonstrated significant predictive value.

Conclusion: Our findings suggest that obesity is a significant risk factor for mortality in patients with COVID-19 and should be carefully considered in clinical management. Although obese patients were younger, they exhibited higher mortality rates, indicating that obesity may serve as an independent prognostic factor in the course of COVID-19.

Keywords: COVID-19; Obesity; Mortality; Prognosis; Body Mass Index

Introduction

The COVID-19 pandemic emerged at the end of 2019, leading to substantial morbidity and mortality worldwide. SARS-CoV-2 infection manifests across a broad clinical spectrum, ranging from asymptomatic cases to severe pneumonia and fatal acute respiratory distress syndrome (ARDS) [1]. During this period, risk factors such as advanced age, comorbidities, variations in immune response, and obesity have played a critical role in determining disease prognosis [2-8]. Obesity has been recognized by the World Health Organization as a global pandemic, and its prevalence continues to increase worldwide [9]. It is well established that obesity adversely influences not only metabolic and cardiovascular disorders but also the course of infectious diseases. Chronic low-grade inflammation in adipose tissue, excessive production of proinflammatory cytokines (IL-6, TNF-α),

impaired immune cell function, and endothelial dysfunction make obese individuals more vulnerable to infections [2,3,4]. Furthermore, comorbidities commonly associated with obesity—such as type 2 diabetes, hypertension, and cardiovascular disease—further aggravate the prognosis of COVID-19 [2,4,7].

Numerous studies have demonstrated that obesity is associated with an increased requirement for intensive care, higher rates of endotracheal intubation, and greater mortality among patients with COVID-19 [1,7,8,10,11]. However, studies from Turkey remain limited, and data assessing the impact of obesity on the prognosis of COVID-19 in large patient populations are still insufficient [12]. Therefore, clarifying the association between obesity and the clinical course of COVID-19 is essential for identifying high-risk groups and developing optimal management strategies. In this study, the association between obesity and clinical outcomes was retrospectively evaluated in patients hospitalized with a confirmed diagnosis of COVID-19. Mortality, laboratory parameters, and length of hospital stay were compared between groups.

Materials and Methods

The study was designed as a single-center retrospective cohort. Patients who were hospitalized and treated in the Pulmonology Department of a tertiary care training and research hospital with a diagnosis of COVID-19 between June 1, 2020, and December 31, 2021, were evaluated. Patients aged 18 years and older with a confirmed diagnosis of COVID-19, available height and weight data, and complete clinical and laboratory records were included in the study. Patients diagnosed with COVID-19 based solely on clinical and radiological findings, those lacking height or weight information preventing calculation of body mass index (BMI), and those with incomplete laboratory data were excluded from the study.

Patients included in the study were classified according to body mass index (BMI) calculated using height and weight measurements obtained from medical records. Those with a BMI ≥30 kg/m^2 were categorized as the 'obese' group, while those with a BMI <30 kg/m^2 were classified as the 'non-obese' group. Demographic characteristics, comorbidities, laboratory findings (leukocyte, lymphocyte, CRP, procalcitonin, LDH, D-dimer, ferritin), length of hospital stay, and mortality status were recorded through the hospital information management system. This study was approved by the Institutional Ethics Committee of the hospital on September 10, 2025 (Decision No: 302) and was conducted in accordance with the principles of the Declaration of Helsinki.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics for Windows, Version 25.0 (Armonk, NY: IBM Corp.). The distribution of continuous variables was assessed using the Kolmogorov–Smirnov test. Continuous variables with normal distribution were expressed as mean ± standard deviation (SD), whereas non-normally distributed variables were presented as median (interquartile range, IQR). Categorical variables were presented as numbers and percentages (%). For comparisons between groups, the Student's t-test was used for normally distributed continuous variables, the Mann–Whitney U test for non-normally distributed continuous variables, and the Chi-square test or Fisher's exact test, as appropriate, for categorical variables. To evaluate factors associated with mortality, descriptive analyses were performed within age and sex subgroups. Ratio comparisons were used to assess mortality differences between groups. Multivariate regression analysis could not be performed due to insufficient comorbidity data in the medical records. A p-value of <0.05 was considered statistically significant for all tests.

Results

(Figure 1) The gender distribution of the patients included in the study was comparable between the two groups (p = 0.32). The mean age of obese patients was significantly lower than that of non-obese patients (63.0 ± 15.7 vs. 68.1 ± 14.2 years; p = 0.007) (Table 1). Among the entire cohort, the most common comorbidities were hypertension (52.3%), diabetes mellitus (37.6%), and respiratory disease (22.1%). The distribution of comorbidities was similar between obese and non-obese patients, with no statistically significant differences between groups (all p > 0.05) (Table 2). There was no significant difference in gender distribution between the obese and non-obese groups (p = 0.32), whereas the mean age was significantly lower in the obese group (p = 0.007). No significant differences were observed between the groups in leukocyte, lymphocyte, CRP, procalcitonin, LDH, or ferritin levels (all p > 0.05). Although D-dimer levels were higher in the non-obese group (2.38 ± 4.69 vs. 1.52 ± 2.65 µg/mL), the difference did not reach statistical significance (p = 0.08) (Table 3).

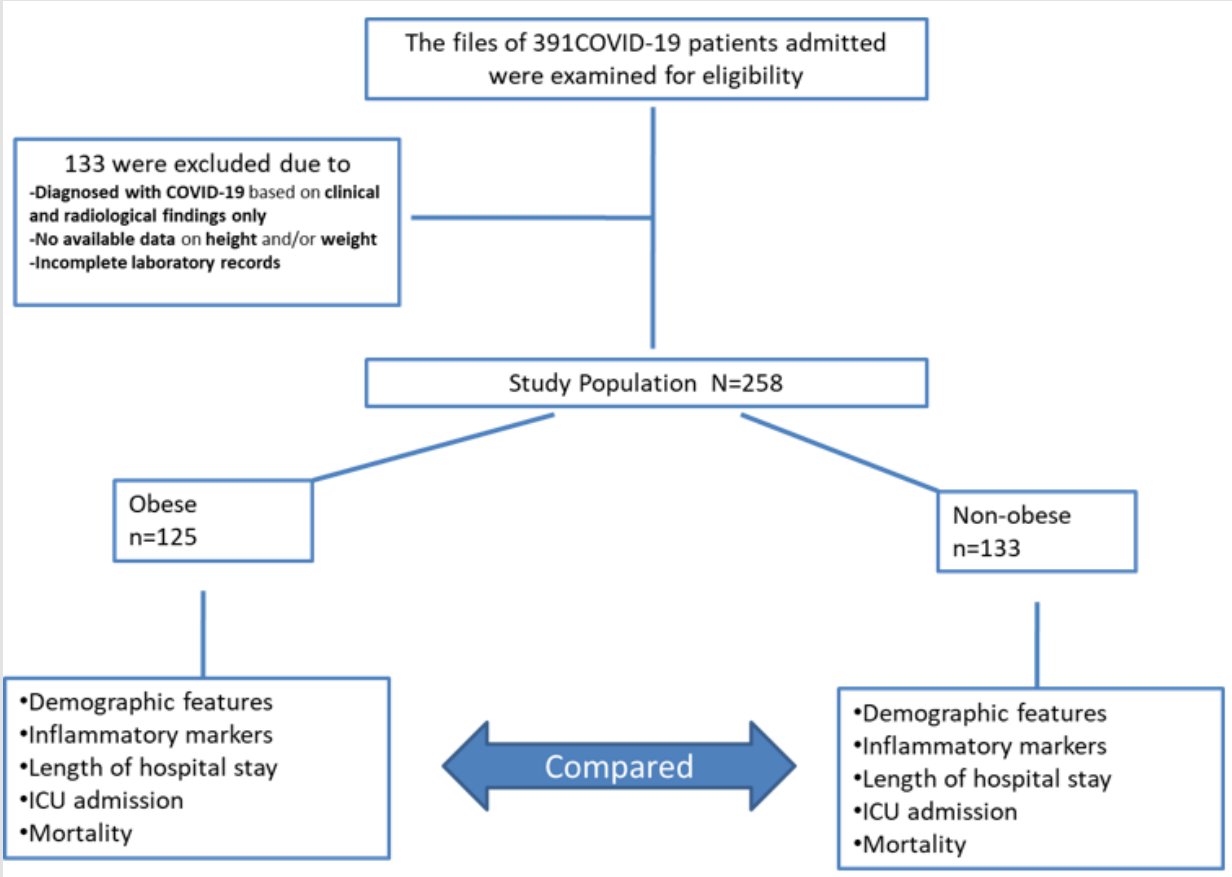

Figure 1: Flow chart of patient selection and study population.

Table 1: Demographic Characteristics.

Variable	Obese (n = 125)	Non-obese (n = 133)	p-value
Female / Male	50 / 75	60 / 73	0.32
Age (mean ± SD), years	63.0 ± 15.7	68.1 ± 14.2	0.007

Table 2: Distribution of Comorbidities in Obese and Non-Obese Patients with COVID-19.

Comorbidity	Total (n = 258)	Obese (n = 125)	Non-obese (n = 133)	p-value
Hypertension	135 (52.3%)	71 (56.8%)	64 (48.1%)	0.163
Diabetes mellitus	97 (37.6%)	49 (39.2%)	48 (36.1%)	0.606
Respiratory disease	57 (22.1%)	31 (24.8%)	26 (19.5%)	0.31
Cardiac disease	46 (17.8%)	20 (16.0%)	26 (19.5%)	0.457
Chronic kidney disease	5 (1.9%)	2 (1.6%)	3 (2.3%)	0.709
Malignancy	16 (6.2%)	8 (6.5%)	8 (6.0%)	0.885

Table 3: Laboratory Parameters and Clinical Outcomes.

Parameter / Outcome	Obese (n = 125)	Non-obese (n = 133)	p-value
Leukocyte (/mm³)	10,013.7 ± 5,449	9,598.6 ± 5,279	0.54
Lymphocyte (/mm³)	1,447.1 ± 3,138	1,052.4 ± 886	0.18
CRP (mg/L)	67.7 ± 61.4	65.1 ± 66.6	0.75
Procalcitonin (ng/mL)	0.26 ± 0.62	0.32 ± 0.96	0.58
LDH (U/L)	392.9 ± 148.1	371.5 ± 163.5	0.28
D-dimer (µg/mL)	1.52 ± 2.65	2.38 ± 4.69	0.08
Ferritin (ng/mL)	685.5 ± 764.2	633.9 ± 691.1	0.58
Length of hospital stay (days)	12.99 ± 6.47	12.42 ± 5.86	0.46
Mortality, n (%)	26 (20.8)	12 (9.0)	0.013

No significant difference was observed between the groups in terms of length of hospital stay (p = 0.46). However, the mortality rate was significantly higher in the obese group (20.8%) compared to the non-obese group (9.0%) (p = 0.013). In addition, ICU admission was significantly more frequent among obese patients than among non-obese patients (36 vs. 16 cases, p = 0.007). In the obese group, procalcitonin and D-dimer were identified as the parameters with the highest discriminatory power for predicting mortality. This finding indicates that inflammatory and coagulation responses play a more prominent role in mortality among obese patients. The high sensitivity of procalcitonin suggests a stronger tendency for infection to progress to a systemic inflammatory response in obese individuals, whereas elevated D-dimer levels highlight the prognostic importance of hypercoagulability and thrombotic complications. The significance of LDH in both groups supports the notion that cellular injury and tissue hypoxia may represent common mechanisms associated with mortality. The association of ferritin with mortality in the obese group further suggests that chronic low-grade inflammation in obesity may contribute additional risk in the course of COVID-19. In contrast, the lack of significant discrimination for CRP and lymphocyte levels implies that these classic inflammatory markers may have reduced prognostic power in the setting of obesity-related chronic inflammation.

Although mean laboratory values did not differ significantly between the groups, ROC analysis demonstrated their predictive power for mortality. In the obese group, procalcitonin (AUC = 0.862; 95% CI, 0.658–1.000; p < 0.001) and D-dimer (AUC = 0.825; 95% CI, 0.733–0.916; p = 0.001) showed the highest discriminatory ability. LDH (AUC = 0.739; p = 0.012) and ferritin (AUC = 0.716; p = 0.024) also demonstrated significant predictive value, while CRP (AUC = 0.226) and lymphocyte count (AUC = 0.372) did not. In the non-obese group, only LDH (AUC = 0.776; 95% CI, 0.634–0.918; p < 0.001) and D-dimer (AUC = 0.711; p = 0.005) were found to have significant predictive value; AUC values of the other parameters remained below 0.6. In the obese group, procalcitonin ≥ 0.175 ng/mL and D-dimer ≥ 1.17 µg/mL were significant predictors of mortality, whereas in the non-obese group, LDH ≥ 400 U/L and D-dimer ≥ 1.4 µg/mL significantly predicted mortality risk (Table 4). These findings indicate that procalcitonin and D-dimer levels play a more prominent role in predicting mortality among obese patients, suggesting that inflammatory and coagulation responses may serve as stronger prognostic markers in this population.

Table 4: ROC Analysis Results: Parameters Predicting Mortality in Obese and Non-obese Groups.

Group	Parameter	AUC	p-value	Cut-off (≥)	Sensitivity	1–Specificity
Obese	Procalcitonin	0.862	<0.001	0.175 ng/mL	0.8	0.2
Obese	D-dimer	0.825	0.001	1.17 µg/mL	1	0.47
Obese	LDH	0.739	0.012	402 U/L	0.6	0.39
Obese	Ferritin	0.716	0.024	687 ng/mL	0.5	0.29
Obese	CRP	0.226	0.004	20.9 mg/L	–	–
Obese	Lymphocyte	0.372	0.181	850 /mm³	–	–
Non-obese	LDH	0.776	<0.001	≈400 U/L	0.6	0.37
Non-obese	D-dimer	0.711	0.005	≈1.4 µg/mL	1	0.53

Discussion

The findings of this study revealed that obesity significantly increases the risk of mortality among patients with COVID-19. The mortality rate in obese patients was nearly twice that observed in non-obese patients (20.8% vs. 9.0%), and this difference was statistically significant. These results are consistent with international literature emphasizing the detrimental effect of obesity on clinical outcomes. For instance, a multicenter intensive care study conducted in Europe reported that the prevalence of obesity among severe COVID-19 cases was unexpectedly high and that the need for invasive mechanical ventilation increased with rising body mass index [11]. Similarly, a study from China demonstrated that obesity may predispose patients to severe disease progression and is associated with critical illness in COVID-19 [13]. A large cohort study conducted in the United States also showed that obesity independently increases the risk of in-hospital mortality from COVID-19, particularly among younger patients [6]. In our study, despite the younger mean age of the obese group compared with the non-obese group, mortality was higher, suggesting that obesity may negate the survival advantage typically associated with younger age. In our cohort, the distribution of comorbidities such as hypertension, diabetes mellitus, respiratory disease, cardiac disease, chronic kidney disease, and malignancy was similar between obese and non-obese patients, with no statistically significant differences.

This finding suggests that the higher mortality observed among obese individuals in our study cannot be explained by a greater burden of comorbid conditions. Therefore, obesity itself may act as an independent risk factor contributing to adverse outcomes in COVID-19 through mechanisms beyond the presence of concurrent diseases, including inflammatory dysregulation, endothelial dysfunction, and altered immune responses. Systematic reviews and meta-analyses published in the literature consistently confirm that a higher body mass index is significantly associated with an increased risk of severe disease and mortality related to COVID-19 [1,8,10]. Considering the limited data reported from Turkey [12], our study provides a valuable contribution to understanding the relationship between obesity and COVID-19 prognosis.

From a pathophysiological perspective, obesity can adversely affect the course of COVID-19 through multiple mechanisms. Adipose tissue in obese individuals has been shown to express higher levels of angiotensin-converting enzyme 2 (ACE2) receptors, which serve as the entry point for SARS-CoV-2, thereby increasing viral entry and dissemination and potentially elevating viral load [2]. In addition, chronic low-grade inflammation associated with obesity and the excessive release of proinflammatory cytokines from adipose tissue (e.g., IL-6, TNF-α) may exacerbate the "cytokine storm," increasing the risk of multiorgan damage [3,4]. Insulin resistance and endothelial dysfunction, both common in obesity, contribute to a hypercoagulable state and greater susceptibility to thrombotic complications. Furthermore, the restrictive effects of excess adipose tissue on respiratory mechanics are clinically important; obesity-induced restrictive lung physiology can reduce pulmonary reserve, facilitate the development of hypoxemia, and exacerbate respiratory failure [10,13]. As a result of these multifactorial pathophysiologic mechanisms, obese individuals tend to experience a more severe course of COVID-19.

In the obese group, procalcitonin and D-dimer were identified as the parameters with the highest discriminatory power for predicting mortality. This finding suggests that inflammatory and coagulation responses play a more prominent role in mortality among obese patients. The high sensitivity of procalcitonin implies a greater tendency for infection to progress into a systemic inflammatory response in obese individuals, whereas elevated D-dimer levels underscore the prognostic significance of hypercoagulability and thrombotic complications. The significance of LDH in both groups supports the hypothesis that cellular injury and tissue hypoxia may represent common mechanisms associated with mortality. The association of ferritin with mortality in the obese group further indicates that chronic low-grade inflammation inherent to obesity may contribute additional risk in the course of COVID-19. In contrast, the lack of significant discrimination for CRP and lymphocyte levels suggests that these classical inflammatory markers may have diminished prognostic utility in the setting of obesity-related chronic inflammation.

In clinical practice, the management of obese patients with COVID-19 presents several challenges due to the aforementioned risk factors. In cases requiring intensive care and mechanical ventilation, intubation and ventilator adaptation can be technically more difficult in patients with high body mass index. The presence of obesity also increases the risk of complications during therapeutic maneuvers such as prone positioning, complicating respiratory support management. Furthermore, pharmacokinetic differences may necessitate dose adjustments for certain medications in obese patients, making effective pharmacotherapy more complex. Additionally, studies have reported that the immune response to vaccination may be weaker in obese individuals compared with those of normal weight [2]. These considerations highlight the need for obesity to be specifically addressed in both treatment and prevention strategies. For example, early and close monitoring of obese COVID-19 patients, as well as careful timing of interventions such as prophylactic anticoagulation and respiratory support, may be warranted. From a preventive standpoint, prioritizing individuals with obesity in vaccination programs and monitoring post-vaccination immune responses may help identify potential gaps in protection. Consistent with previous studies reporting an increased requirement for intensive care among obese patients, our findings also demonstrated a significantly higher rate of ICU admission in the obese group compared with non-obese patients. This observation further supports the association between obesity and a more severe clinical course of COVID-19.

Finally, it is important to acknowledge several limitations of this study. Our research had a retrospective, single-center design, and multivariate analyses that would be necessary to evaluate the independent effect of obesity on mortality could not be performed. Because the influence of additional comorbidities (such as diabetes, hypertension, and cardiovascular diseases) could not be examined in detail, it is not possible to precisely determine the extent to which obesity increased mortality risk independently of other factors. Although the available data suggest that obesity adversely affects the prognosis of COVID-19, our findings are not sufficient to conclusively establish obesity as a direct and independent risk factor. Therefore, larger, prospective, multicenter studies that control for potential confounding variables are needed to clarify the impact of obesity on clinical outcomes in patients with COVID-19. Nonetheless, considering that the World Health Organization has defined obesity as a global pandemic [9], the combined risks arising from the intersection of the COVID-19 and obesity pandemics must be better understood. The convergence of these two pandemics underscores once again the critical importance of combating obesity both at the individual patient level and from a public health perspective. Accordingly, strategies aimed at preventing obesity and effectively managing obese patients will play a key role in reducing morbidity and mortality in future outbreaks similar to COVID-19

Conclusion

This study demonstrates that obesity increases the risk of mortality among patients with COVID-19. The findings are consistent with international literature emphasizing the adverse impact of obesity on disease prognosis. In clinical management and risk assessment, consideration of body mass index (BMI), closer monitoring of obese patients, and implementation of appropriate treatment strategies are essential. Moreover, public health policies aimed at combating obesity may play a critical role in reducing disease burden during pandemics and other public health emergencies [9,12,14,15].

References

1. Földi M, Farkas N, Kiss S, Zádori N, Váncsa S, et al. (2020) Obesity is a risk factor for developing critical condition in COVID-19 patients: A systematic review and meta-analysis. Obes Rev 21(10): e13095.
2. Popkin BM, Du S, Green WD, Beck MA, Algaith T, et al. (2020) Individuals with obesity and COVID-19: A global perspective on the epidemiology and biological relationships. Obes Rev 21(11): e13128.
3. Huang Y, Lu Y, Huang YM, Wang M, Ling W, et al. (2020) Obesity in patients with COVID-19: a systematic review and meta-analysis. Metabolism 113: 154378.
4. Sattar N, McInnes IB, McMurray JJV (2020) Obesity is a risk factor for severe COVID-19 infection: mechanisms and management. BMJ 369: m2423.
5. Gao M, Piernas C, Astbury NM, Hippisley-Cox J, O Rahilly S, et al. (2021) Associations between body-mass index and COVID-19 severity in 6.9 million people in England: a prospective, community-based, cohort study. Lancet Diabetes Endocrinol 9(6): 350-359.
6. Hendren NS, de Lemos JA, Ayers C, Das SR, Rao A, et al. (2021) Association of body mass index and outcomes in patients hospitalized with COVID-19: results from the American Heart Association COVID-19 Cardiovascular Disease Registry. Circulation 143(14): 135-144.
7. Tartof SY, Qian L, Hong V, Wei R, Nadjafi RF, et al. (2020) Obesity and mortality among patients diagnosed with COVID-19: results from an integrated health care organization. Ann Intern Med 173(10): 773-781.
8. Yang J, Tian C, Chen Y, Zhu C, Chi H, et al. (2021) Obesity aggravates COVID-19: an updated systematic review and meta-analysis. J Med Virol 93(5): 2662-2674.
9. (2023) World Health Organization (WHO). Obesity and overweight. Geneva: WHO.
10. Du Y, Lv Y, Zha W, Zhou N, Hong X (2021) Association of body mass index and risk of COVID-19 severity and mortality: a systematic review and dose-response meta-analysis. Obes Res Clin Pract 15(5): 457-463.
11. Simonnet A, Chetboun M, Poissy J, Raverdy V, Noulette J, et al. (2020) High prevalence of obesity in severe SARS-CoV-2 infection: an unexpected finding on intensive care unit admission. Obesity (Silver Spring) 28(7): 1195-1199.
12. TC Sağlık Bakanlığı (2023) COVID-19 Rehberi. Ankara: Sağlık Bakanlığı.
13. Zhang X, Lewis AM, Moley JR, Brestoff JR (2020) A cautionary note on obesity and COVID-19. Obesity (Silver Spring) 28(10): 2040-2046.
14. (2024) Centers for Disease Control and Prevention (CDC). People with certain medical conditions [Internet]. Atlanta (GA): CDC.
15. Fresán U, Guevara M, Tobalina M, Burgui C, Ezpeleta C, et al. (2021) Body mass index, waist circumference and risk of severe COVID-19: prospective analyses in UK Biobank. Obesity (Silver Spring) 29(6): 1220-1229.

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