

Clinical, Laboratory and Tomographic characteristics associated with obesity and BMI at Hospital admission in adult patients with COVID-19: a cross-sectional study

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ABSTRACT

Introduction: The pathological status of obesity can influence COVID-19 from its initial clinical presentation, therefore, the identification of clinical and laboratory parameters most affected in the presence of obesity can contribute to improving the treatment of the disease. **Objective:** To identify the clinical, laboratory, and tomographic characteristics associated with obesity and BMI at t hospital admission in adult patients with COVID-19. **Methods:** This is a cross-sectional observational study with a total of 315 participants with COVID-19 confirmed by rt-PCR. The participants were divided into non-Obese (n=203) and Obese (n=112). Physical examinations, laboratory tests, and computed tomography of the chest were performed during the first 2 days of hospitalization. **Results:** Patients with obesity were younger, and they had higher systolic and diastolic blood pressure, higher frequency of alcoholism, fever, cough, and headache, higher ALT, LDH, and red blood cell count (RBC), hemoglobin, hematocrit, and percentage of lymphocytes. Also, they presented a lower value of leukocyte count and Neutrophil/Lymphocyte Ratio (RNL). The parameters positively correlated with BMI were alcoholism, systolic and diastolic blood pressure, fever, cough, sore throat, number of symptoms, ALT in men, LDH, magnesium, RBC, hemoglobin, hematocrit, and percentage of lymphocytes. The parameters negatively correlated with the BMI were: age and RNL. **Conclusion:** Several parameters were associated with obesity at hospital admission, revealing better than expected results. However, these results should be interpreted with great caution, as there may be some influence of a phenomenon called the Obesity Paradox that can distort the severity and prognosis of the patient.

Keywords: COVID-19; obesity; signs and symptoms; Biomarkers; Tomography.

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INTRODUCTION

Obesity started to be considered a disease for the first time in 2013, by the American Medical Association, and since then it was realized that it was a disease with high prevalence in many countries, reaching pandemic proportions¹. Worldwide, in 2016, 39% of adults were overweight and 13% were obese². In Brazil, in 2018, 55.4% were overweight and 20.3% were obese³. The problem of the high and increasing prevalence of obesity in the world is due to the risk for other non-communicable diseases and chronic conditions, including type 2 diabetes mellitus, non-alcoholic fatty liver disease, systemic arterial hypertension (SAH), myocardial infarction, stroke, dementia, osteoarthritis, obstructive sleep apnea and various types of cancer⁴.

In addition to the influence on non-communicable diseases, obesity has also been considered an important factor associated with complications of the disease caused by the new coronavirus (COVID-19), with a negative influence on the general hospitalization rate, hospitalization in the intensive care unit, disease severity, and death, also contributing to the development of more virulent strains⁵. Among the possible justifications for the worse prognosis of patients with obesity and COVID-19 are the chronic and intrinsic changes in obesity, mainly metabolic changes and in the endocrine, nervous, immune, cardiovascular, respiratory, and hematological systems⁶. In addition, there is still no specific guidance on the best approach for a patient with obesity and COVID-19⁷.

The pathological status of obesity can influence COVID-19 from its initial clinical presentation, therefore, the identification of clinical and laboratory parameters most affected in the presence of obesity can contribute to improving the treatment of the disease⁸.

This study aims to identify the clinical, laboratory, and tomographic characteristics associated with obesity and BMI at hospital admission in adult patients with COVID-19.

METHODS

Ethical aspects

This study was conducted following the Helsinki Declaration. The study was approved by the Research Ethics Committee of the University Hospital of the Federal University of Piauí (Approval Number: 4.276.844). All research participants agreed to participate and signed the Free and Informed Consent Form.

Study design, setting, and population

This is a cross-sectional, descriptive, and single-center study, with data analysis of the first two days of admission of all patients admitted between June 2, 2020, and August 18, 2020, at the State Field Hospital, located in the city of Teresina, capital of the state

of Piauí, Brazil. All consecutive patients with a diagnosis of SARS-CoV-2 infection were included and confirmed by reverse transcriptase polymerase chain reaction (rt-PCR) from a nasopharyngeal sample. Participants whose weight and height data were not collected during hospitalization were excluded. The definition of obesity was performed according to the global nutritional status classification criterion by the body mass index (BMI) recommended by WHO⁹ for adults, that is, $BMI \geq 30 \text{ kg/m}^2$.

Data collection

The State Field Hospital of Piauí was planned since its creation to be a health institution for research purposes. Therefore, the data collection instruments used were specific to this work, where the hospital staff was previously trained. All data were collected in the first contact with the patient, with complementary exams performed within 48 hours after admission.

The following clinical data were self-reported and recorded in standardized electronic forms pre-defined by the research group: Age, sex, days of symptoms until hospitalization, weight, height, diseases, signs, symptoms, presence of smoking, and alcohol consumption. Smoking was defined as the continuous use of cigarettes and similar products in the last 6 months. Alcoholism was defined as the weekly ingestion of any alcoholic product in moderate to large doses in the last 6 months, according to the patient's perception. Vital signs were measured by the nursing team using standardized hospital instruments and recorded on specific forms: axillary temperature, heart rate, respiratory rate, systolic and diastolic blood pressure, oxygen saturation, and capillary blood glucose. Laboratory tests and computed tomography (CT) of the chest were under the responsibility of a single company experienced in the market and all the information was transferred in full to a database with double entry, to avoid typing errors, with subsequent review by a team of experienced doctors. Laboratory data collected were ALT, AST, creatinine, LDH, lactate, ferritin, CRP, sodium, potassium, magnesium, red cell count, hemoglobin, hematocrit, white cell count, and platelet count. Chest CT data were collected from a specific form with result categories and ground-glass opacification levels in case there was this change.

Statistical analysis

After applying the normality test, the quantitative variables of the obese and non-obese groups were expressed as a median and interquartile range, and comparisons between groups were performed using the Mann-Whitney U test. Qualitative variables were presented as absolute frequency and percentages, and the associations were tested using Pearson's Chi-Square Test. To test the correlation of variables with the body mass index, the Spearman correlation coefficient (ρ) was used for the quantitative variables, and the point-biserial correlation coefficient (r) for the

dichotomous qualitative nominal variables. No imputation was made for missing data. The level of significance was set at $p < 0.05$. The study data were processed using the Statistical Package for the Social Sciences software, version 27 (IBM Inc., Chicago, IL, USA).

RESULTS

The sample consisted of 313 patients, 35.56% of whom were obese. Of patients with obesity, 57.1% were male, with a median of 58.65 (IQR: 47-73.2) years and 10 (8-13) days of symptoms until hospitalization. At least 50% of patients with obesity had some comorbidity, with 58.6% having systemic arterial hypertension (SAH) and 34.2% having diabetes. A sample of this population was assessed according to habits 50% were smokers and 67.9% were alcoholics (Table 1).

Regarding general clinical parameters, patients with obesity and COVID-19 were younger ($p < 0.01$), had a higher frequency of drinking ($p < 0.01$), had higher systolic blood pressure (SBP) ($p < 0.05$) and had higher diastolic blood pressure (DBP) ($p < 0.01$). Likewise, the body mass index (BMI) showed a weak negative correlation with age ($r = -0.215$; $p < 0.01$) and a weak positive correlation with alcoholism ($\rho = 0.229$; $p < 0.01$), SBP ($r = 0.150$; $p < 0.01$) and DBP ($r = 0.198$; $p < 0.01$). The presence of obesity was not associated with severity, duration of symptoms until hospitalization, length of stay, SAH, diabetes, smoking, number of comorbidities, temperature, heart rate, respiratory rate, and oxygen saturation (Table 1).

Patients with obesity and COVID-19 more often presented fever ($p < 0.05$), cough ($p < 0.05$), and pain in the head ($p < 0.05$). BMI was also associated with these parameters through a weak positive correlation with fever ($r = 0.121$; $p < 0.05$), cough ($r = 0.131$; $p < 0.05$), headache ($r = 0.116$; $p < 0.05$), and, in addition to these, with the number of symptoms at hospital admission ($\rho = 0.158$; $p < 0.01$). However, obesity and BMI were not associated with fatigue, dyspnea, myalgia, anorexia, sore throat, chest pain, anosmia, ageusia, diarrhea, and nausea/vomiting (Table 2).

Regarding laboratory parameters (Table 3), patients with obesity and COVID-19 had significantly higher values of: ALT ($p < 0.05$); lactic dehydrogenase (LDH) ($p < 0.01$), red blood cell (RBC) count ($p < 0.01$), hemoglobin ($p < 0.01$), hematocrit ($p < 0.01$) and percentage of lymphocytes ($p < 0.01$). In addition, patients with obesity had lower white blood cells (WBC) count ($p < 0.01$), lower neutrophil count ($p < 0.05$), and higher and lower neutrophil/lymphocyte ratio (NLR) ($p < 0.01$). Some parameters were stratified by sex due to the recommendation of the biomedical analysis laboratory: ALT, AST, hemoglobin, and ferritin. Of these, only hemoglobin showed a difference between the groups, with the highest values in men ($p < 0.01$) and women ($p < 0.05$) with obesity. There was a weak positive correlation of BMI with ALT in men ($\rho = 0.176$; $p < 0.05$); weak positive correlation with LDH ($\rho = 0.164$; $p < 0.01$), moderate positive correlation with magnesium ($\rho = 0.482$; $p < 0.05$), weak positive correlation with red cell count ($\rho = 0.293$; $p < 0.01$), weak positive correlation with hemoglobin ($\rho = 0.208$; $p < 0.01$) and its subgroups stratified by male ($\rho = 0.253$; $p < 0.01$) and female

Table 1: Influence of obesity and BMI on general clinical parameters of COVID-19 in adults at hospital admission

Parameter	Comparison between groups				Correlation with BMI r or ρ
	Non-obese	n	Obese	n	
Age (years)	68 (52.4-79.6)	203	58,65 (47-73.2)**	112	-0.215**
Male	120 (59.1%)	203	64 (57.1%)	112	-0.049
Days of symptoms until hospitalization	10 (7-12)	159	10 (8-13)	112	0.118#
Severe illness	114 (64.4%)	177	68 (68.0%)	100	0.073
SAH	103 (51.0%)	202	65 (58.6%)	111	0.092
Diabetes	62 (30.7%)	202	38 (34.2%)	111	0.055
Smoking	36 (42.9%)	84	28 (50.0%)	56	0.031
Alcoholism	36 (42.9%)	84	38 (67.9%)**	56	0.229**
Number of comorbidities	1 (0-2)	203	1 (0-2)	112	0,011
Axillary temperature	36 (35.6-36.4)	184	36 (35.8-36.4)	109	0.058
Heart Rate (bpm)	84 (72-99)	191	84 (74.8-95.3)	109	-0.005
Respiratory frequency (RI/min)	20 (18-22)	166	20 (18-22)	93	0.055
Systolic blood pressure	130 (120-140)	192	134 (120-150)*	107	0.150**
Diastolic blood pressure	80 (70-83)	192	80 (76.8-90)**	105	0.198**
Oxygen saturation (%)	96 (94-97.8)	189	96 (94-98)	109	-0.033

The comparison data were presented using the median and interquartile range and compared using the Mann-Whitney U test or presented using the absolute frequency and percentage and compared using Pearson's chi-square test. The correlation data were presented as a point-biserial correlation coefficient (r) or Spearman's (ρ), as appropriate. For all tests, results were considered statistically significant when $p < 0.05$. * $p < 0.10$; * $p < 0.05$; ** $p < .01$; SAH: systemic arterial hypertension; n: total number of observations per group.

Table 2: Influence of obesity and BMI on COVID-19 symptoms in adults at hospital admission

Parameter	Comparison between groups				Correlation with BMI
	Non-obese	n	Obese	n	r or ρ
Fever	128 (63.7%)	201	85 (75.9%)*	112	0.121*
Cough	147 (73.1%)	201	94 (83.9%)*	112	0.131*
Fatigue	37 (18.4%)	201	23 (20.5%)	112	0.078
Dyspnea	162 (80.6%)	201	92 (82.1%)	112	0.079
Myalgia	111 (55.2%)	201	60 (53.6%)	112	0.039
Anorexia	46 (22.9%)	201	20 (17.9%)	112	-0.067
Sore throat	42 (20.9%)	201	27 (24.1%)	112	0.116*
Headache	63 (31.5%)	200	49 (43.8%)*	112	0.091
Chest pain	38 (19.0%)	200	27 (24.1%)	112	0.037
Anosmia	33 (16.5%)	200	23 (20.5%)	112	0.097 [#]
Ageusia	40 (20.0%)	200	28 (25.0%)	112	0.087
Diarrhea	56 (28.0%)	200	39 (34.8%)	112	0.038
Nausea/vomiting	25 (12.5%)	200	9 (8.0%)	112	-0.097
Number of symptoms	4 (3-6)	203	5 (3-7) [#]	112	0.158**

The comparison data were presented using the median and interquartile range and compared using the Mann-Whitney U test or presented using the absolute frequency and percentage and compared using Pearson's chi-square test. The correlation data were presented as a point-biserial correlation coefficient (r) or Spearman's (ρ), as appropriate. For all tests, results were considered statistically significant when $p < 0.05$. [#] $p < 0.10$; * $p < 0.05$; ** $p < 0.01$; n: total number of observations per group.

($\rho = 0.184$; $p < 0.05$), hematocrit ($\rho = 0.247$; $p < 0.01$), weak negative correlation with basophil count ($\rho = -0.154$; $p < 0.01$) and its relative value ($\rho = -0.145$; $p < 0.05$) weak positive correlation with relative value of lymphocytes ($\rho = 0.154$; $p < 0.01$) and weak negative correlation with NLR ($\rho = -0.142$; $p < 0.05$). Ground-glass opacification showed no difference between the obese and non-obese groups and also did not correlate with BMI (Table 3).

DISCUSSION

In this study, obese patients with SARS-CoV-2 infection were younger than those without obesity. Excess weight has been considered an independent risk factor for hospitalization. Although young adults are considered to be at lower risk for hospitalization and disease severity, they became more prevalent when obese⁵. Furthermore, we found an inverse relationship between BMI and age that was also found in similar studies⁹⁻¹². COVID-19 was not consistent with Brazilian epidemiology, where obesity is more prevalent in older adults³. An important study with data from Brazil pointed out that patients with obesity who died from COVID-19 are younger than patients without obesity. The unfavorable interaction between pathogen, host, and environment in the younger adult with obesity is not yet elucidated¹³.

The association between alcoholism and obesity found in this study can be justified by some complementary hypotheses: 1) alcohol consumption is usually higher among people with obesity; 2) there was an increase in alcohol consumption during confinement resulting from the pandemic and 3) concomitance between

obesity and alcoholism causes overexpression of type I angiotensin II receptors, impairs the immune system and leaves the patient more likely to develop symptomatic COVID-19^{14,15}.

The median systolic and diastolic blood pressure of the obese and non-obese groups fit as stage 1 systemic arterial hypertension, according to the American College of Cardiology and the American Heart Association¹⁶, meaning that at least 50% of all patients had SAH. When observing the prevalence of self-reported SAH, we found an agreement in the results, since 51% of the non-obese group and 58.6% of the obese group reported having the disorder. The study by Ritter et al.¹⁷ listed a series of biological mechanisms that occur in obesity and influence the development of SAH: systemic inflammation, endothelial damage, higher plasma concentrations of renin, angiotensinogen, angiotensin II conversion enzyme, angiotensin II and aldosterone, with greater activation of the renin-angiotensin-aldosterone system, among others. Taing's cross-sectional study¹⁸ with the general population of India found a considerable positive correlation between BMI and systolic and diastolic blood pressure, as observed in this study. Therefore, obesity is an independent factor for the increase in blood pressure and its influence is proportional to the increase in BMI.

Excess adipose tissue may be responsible for a more symptomatic presentation of COVID-19^{7,17}. We found in this study a higher prevalence of fever, cough, and headache in patients with obesity, in addition, fever, cough, sore throat and the number of self-reported symptoms were directly proportional to the increase in BMI. Although fever was self-reported by more than 60% of

Table 3: Influence of obesity and BMI on laboratory and tomographic parameters of COVID-19 in adults at hospital admission

Parameter	Comparison between groups				Correlation with BMI r or p
	Non-obese	n	Obese	n	
Capillary glycemia (mg/dL)	146 (117.5-256.5)	89	157.5 (120.5-217.5)	54	0.034
ALT (IU/L)	37 (24-63.8)	181	45 (27-72)*	97	0.096
ALT in men (IU/L)	41 (25-90.5)	108	54.5 (42.5-105.5)#	54	0.176*
ALT in women (IU/L)	36 (22-53.5)	73	34 (21-64)	43	-0.002
AST (IU/L)	44 (31-66.8)	181	47 (34-65)	97	0.032
AST in men (IU/L)	47.5 (31-72)	108	39 (52.5-68.5)	54	0.029
AST in women (IU/L)	41 (30.5-63.5)	73	46 (31-58)	43	0.040
Creatinine (mg/dL)	0.8 (0.6-1.2)	198	0.8 (0.6-0.9)	99	-0.003
LDH (UI/L)	323 (274.8-436.3)	185	356.5 (292.3-440.8)**	97	0.164**
Lactic acid, Lactate (mg/dL)	4.1 (3.3-5.4)	154	4.25 (3.2-5.4)	90	-0.027
Ferritin (ng/dL)	961.6 (557.7-1 508)	182	861.4 (475.6-1 526)	98	0.062
Ferritin in men (ng/dL)	1 162 (646.6-1 683)	107	1 321 (811.1 - 1 934)	53	0.138*
Ferritin in women (ng/dL)	623.7 (344.4-1 225)	75	592.5 (365.7 - 1 061)	45	0.049
CRP (mg/L)	6.5 (3.4-9)	186	7.3 (3.7-9)	98	0.092
Sodium (mmol/L)	138 (135-141)	185	138 (135.3-140)	103	0.036
Potassium (mmol/L)	4.8 (4.3-5.1)	186	4.9 (4.3-5.2)	103	0.093
Magnesium (mg/dL)	2 (1.8-2.3)	17	2.05 (1.9-2.3)	11	0.482*
Red Blood Cells (10 ³ cells/ mm ³)	4.5 (4.1-4.9)	188	4.8 (4.4-5.2)**	103	0.293**
Hemoglobin (g/dL)	13.3 (12.2-14.4)	188	13.8 (12.63-14.6)**	103	0.208**
Hemoglobin in men	13.6 (12.7-14.8)	188	14.6 (13.8-15.4)**	103	0.253**
Hemoglobin in women	12.8 (11.5-13.8)	188	13.3 (12.4-14.2)*	103	0,184*
Hematocrit (%)	40.1 (36.5-43.2)	188	41.75 (38.6-44.2)**	103	0.247**
RDW	12.30 (11.6-13.3)	188	12.33 (11.7-12.9)	103	-0.093
White Blood Cells (cells/mm ³)	9 344 (6 446-12 740)	188	8 208 (6 549 - 11 645)*	103	-0.092
Neutrophils (cells/mm ³)	7 386 (5 008-10 492)	188	6 357 (4 436 - 9 504)*	103	-0.111*
Neutrophils (%)	80 (76-85)	188	80 (72-84)*	103	-0.097*
Lymphocytes (cells/mm ³)	931 (585-1 295)	188	994 (670.3-1 451)#	103	0.109#
Lymphocytes (%)	10 (6-16)	188	11.5 (8-20)**	103	0.154**
Monocytes (cells/mm ³)	458 (285-718)	188	463 (302-652.8)	103	-0.048
Monocytes (%)	5 (3-7)	188	5 (4-7)	103	0.014
Neutrophil/Lymphocyte Ratio	8.25 (4.3-12.2)	188	7.55 (4.6-11.9)**	103	-0.142*
Platelets (10 ³ cells/mm ³)	224.8 (181.8-302.3)	188	221.6 (169.3-304.9)	103	-0.025
Ground glass opacity >50%	14 (38.9%)	36	13 (44.8%)	29	-0.019

The comparison data were presented using the median and interquartile range and compared using the Mann-Whitney U test or presented using the absolute frequency and percentage and compared using Pearson's chi-square test. The correlation data were presented as a point-biserial correlation coefficient (r) or Spearman's (p), as appropriate. For all tests, results were considered statistically significant when $p < 0.05$. * $p < 0.10$; * $p < 0.05$; ** $p < 0.01$; ALT: alanine aminotransferase; AST: aspartate aminotransferase; LDH: lactic dehydrogenase; CRP: C-reactive protein; RDW: red cell distribution width; n: total number of observations per group.

patients, the temperature was normal in more than 75%, probably due to self-medication. The two symptoms that were more frequent in patients with obesity were also the two most frequent in the general population¹⁹. The prospective cohort study by Nilles et al.²⁰, which collected BMI information, socio-demographic data, comorbidities, and symptoms from patients with COVID-19, showed that obesity was associated with an increase in the reporting of symptoms, mainly self-reported fever, but not

measured. Another interesting finding from the work of Nilles et al.²⁰ was that the clinical condition is strongly influenced by obesity among younger age groups. The scientific literature says that patients with obesity have more expressive clinical conditions since the onset of symptoms and this is a possible explanation to justify the greater risk of hospitalization⁵. What can justify the most symptomatic illness process of patients with obesity are the following changes resulting from obesity: chronic inflammatory

state, diffuse cardiovascular involvement, reduced respiratory capacity, impaired immune response, overexpression of the angiotensin conversion enzyme (ACE2), and greater virulence^{17,21}.

The patient with obesity has several altered laboratory parameters due to the overlapping pathophysiological molecular mechanisms of obesity and COVID-19¹⁷. In the men in this study, we observed that ALT is proportional to the increase in BMI, which may reveal a predisposition for this association in males. The association between obesity and higher ALT may be before the disease caused by SARS-CoV-2 since patients with obesity have a higher risk of having an abnormal liver function, or it may be a consequence of the acute inflammation of the current disease^{22,23}.

The acute phase inflammatory markers are commonly altered in patients with obesity and COVID-19, and LDH is one of the most frequently elevated acute phase inflammatory factors and has the potential to predict prognosis²⁴. Our study found significance for a higher median of DHL in patients with obesity and a positive correlation with BMI. The study by Mostaghim et al.²⁵ compared laboratory parameters of patients with obesity (BMI>30) with patients without obesity in the first two days of hospitalization and observed an increase in LDH on both days. LDH is released into the circulation when cell membrane necrosis occurs, due to viral infection or lung injury. So, increased DHL values in patients with obesity suggest greater lung injury²⁶.

Serum magnesium levels were higher in patients with obesity and also showed a positive linear correlation with BMI. Hypermagnesemia is common in patients with obesity in general²⁷, however, when associated with insulin resistance of diabetes or metabolic syndrome, there is serum hypomagnesemia, due to intracellular changes²⁸. As the postprandial capillary glycemia measured in the two groups did not show a statistical difference between them, as well as the absolute frequency of self-reported diabetes, we can assume that the glucose metabolism also influenced the magnesium metabolism of both groups and the final result was a higher level of magnesium in patients with obesity, but within normal limits.

Studies that associate obesity and hematological changes in humans are scarce²⁹. No articles were found on the association between hematological changes and patients with obesity and COVID-19. Therefore, this work aims to create hypotheses that justify the changes found in the complete blood count.

The red blood cell, hemoglobin, and hematocrit values were higher in patients with obesity. Obesity and increased waist circumference increase the production of red cells by several different mechanisms^{29,30}. In general, inflammation is associated with the suppression of erythropoiesis, but even with chronic inflammation caused by obesity, there is an increase in erythropoiesis in patients with obesity. The study by Vuong et al.³⁰ listed mechanisms that explain the increase in red blood cells, hematocrit, and hemoglobin in patients with obesity: increased insulin and

insulin-like growth factors in people with metabolic syndrome; chronic hypoxia resulting from the pathophysiology of obesity, which can be aggravated by the common association with obstructive apnea; and, finally, leptin and adiponectin levels may be related to erythrocyte metabolism. In patients with non-severe COVID-19 regardless of BMI, there is an increase in erythrocytes, an increase in hemoglobin, and a reduction in RDW, so we can conclude that our patients with obesity and COVID-19 had less severity, based on the blood count parameters³¹.

Regarding the leukogram, obesity causes a series of pathophysiological changes that result in the alteration of leukocytes. In people with obesity, adipose tissue has hypertrophied adipocytes with lipid engorgement, cell stress, and frequent apoptosis that result in the abundant release of inflammatory mediators. These inflammatory mediators affect the immune system in different ways. Studies with rodents have shown thymic involution, reduction of the size and cellularity of the thymus, reduction of hematopoiesis, increase of myeloid progenitor cells, reduction of lymphoid progenitor cells, and reduction of the output of virgin T cells³². However, studies with human beings have shown that patients with obesity normally had higher values of total leukocytes, neutrophils, and lymphocytes than patients without obesity, these elevations being proportional to the BMI^{29,33}. There is still no consensus on the influence of obesity on the immune system in response to an infection. According to Green and Beck's review³⁴, most authors argue that obesity impairs the immune response, while some argue that there are protective mechanisms involved, depending on the acquired infectious disease. People with obesity and infectious diseases generally have high leukocyte and neutrophil values¹⁷, and a reduction in the production and circulation of T lymphocytes³⁴. About COVID-19, it was found that patients with obesity with more severe disease had an increase in the total leukocyte count, a reduction in lymphocytes, and an increase in the neutrophil/lymphocyte ratio (RNL) and a lower percentage of basophils, monocytes, and eosinophils³⁵.

The absolute values of total leukocytes and neutrophils in people with obesity found in this work were curiously lower. The only parameter of the comparatively high leukogram in this study was the relative value of leukocytes, which also showed an upward trend in the absolute value, but remains lower than the lower limit of normality. Therefore, all parameters presented an increase or decrease opposite to that expected in patients with obesity and infection. The unexpected change in leukocytes in patients with obesity and COVID-19 may be a peculiarity related to SARS-CoV-2. The meta-analysis by Zeng et al.³⁶ evaluated the influence of the leukogram parameters on the severity of COVID-19. Their results showed that higher leukocyte and neutrophil counts are related to greater disease severity, as well as higher neutrophil/lymphocyte ratio (NLR) and lower lymphocyte ratio, with no difference in monocyte values³⁶. With that, we can conclude that the

leukogram values of patients with obesity were associated with less severity.

The patients with obesity observed in this study did not present different tomographic findings of the chest from the patients without obesity, despite high LDH values, chronic inflammation, and impaired respiratory physiology characteristic of obesity¹⁷. The tomographic presentation of patients with COVID-19 and what can influence them is not fully elucidated. Some authors suggest that there is an association between greater pulmonary impairment on chest CT and inflammatory markers, however, the relationship with the severity and prognosis of the disease is not entirely clear^{26,37}. As no reliable correlation has yet been found between chest CT and COVID-19 severity/prognosis, the WHO recommends that this imaging test be performed only as a diagnostic resource where there is no laboratory test and to assist in making decisions about the location of hospitalization, whether in the infirmary or the intensive care unit and not as a parameter to weigh prognosis³⁸.

This study has some limitations that should be highlighted, with emphasis on 1) cross-sectional research design, with limitation of causal inference from the results; 2) descriptive nature of the analyses; 3) impossibility of controlling the analyzes for potential confounding factors; 4) size and representativeness of the sample. Also, due to hospital dynamics, not all intended parameters of all patients were not recorded, with small variations in sample size for each analyzed parameter. However, it is important to highlight that the results of the present study contribute information about the hospitalization profile of obese patients with COVID-19 and hematological parameters at patient admission as well as to generate hypotheses about the relationship between obesity and BMI with clinical and laboratory parameters not yet studied in COVID-19.

In this study, we observed that all laboratory parameters at the admission of patients with obesity indicated less or equal severity to those without obesity. This observation is consistent with the initial risk classification, which identified a similar percentage of severe cases between groups. It has been suggested that a phenomenon

called the "Obesity Paradox" refers to the lower severity of some respiratory, cardiovascular, and renal diseases in patients with obesity in the short and medium term³⁹. Regarding COVID-19, the current literature shows that the paradox does not apply, as obesity worsens the overall prognosis^{5,6}. However, the studies that reached this conclusion evaluated the risk of hospitalization, hospitalization in an intensive care, need for intubation, and death. This work evaluated something different: the clinical, laboratory, and tomographic findings in the first days of hospitalization, which configures an approach that has not yet been explored. In addition, there is a possibility of involvement, at least in part, of the Obesity Paradox in the results of the less severe laboratory parameters found in the first days of hospitalization. However, as mentioned earlier, this work has a limitation related to the statistical power of the sample, whose size can influence the statistics found. Thus, further studies are needed to confirm this possibility.

Finally, we concluded that patients with obesity and COVID-19 at hospital admission were younger, had a higher frequency of alcoholism, fever, cough, and headache, and had higher levels of DPB, SPB, ALT, LDH, red blood cells, hemoglobin, hematocrit and relative lymphocytes, and lower values of leukocytes, neutrophils, and NLR. In addition, BMI was positively correlated with alcoholism, DBP, SBP, fever, cough, sore throat, number of symptoms, ALT in men, LDH, magnesium, red blood cells, hemoglobin, hematocrit, and relative lymphocytes; and negative correlation with age and NLR. In this way, it is concluded that several parameters were associated with obesity at hospital admission, revealing better than expected results in the patients with obesity and COVID-19. However, these results should be interpreted with great caution, as there may be some influence of a phenomenon called the Obesity Paradox that can distort the severity and prognosis of the patient.

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