ISSN: 2378-3516

Ketfi et al. Int J Respir Pulm Med 2024, 11:191
DOI: 10.23937/2378-3516/1410191
Volume 11 | Issue 1
Open Access

International Journal of
Respiratory and Pulmonary Medicine

RESEARCH ARTICLE

The Impact of Comorbidities and Obesity on the Severity of COVID-19 and Risk Factors for Mortality, A Prospective Study in Hospitalized Patients

Abdelbassat Ketfi[1*], Rachida Yahiaoui-Sidi Said[2], Ghania Malki[3] and Rama Touahri[1]

[1]*Department of Pneumology, Phthisiology and Allergology, Rouiba Hospital, Faculty of Medicine, University of Algiers, Algiers, Algeria*

[2]*Department of Pneumo-Allergology, Benimessous Hospital, Faculty of Medicine, University of Algiers, Algiers, Algeria*

[3]*Department of Pneumology, Bejaia Hospital, Faculty of Medicine, University of Bejaia, Bejaia, Algeria*

***Corresponding author:** *Abdelbassat Ketfi, Department of Pneumology, Phthisiology and Allergology, Rouiba Hospital, Faculty of Medicine, University of Algiers, Algiers, Algeria*

Abstract

Background: The studies of the clinical and demographic characteristics of COVID-19 patients around the world have made it possible to observe a rich semiology, which implicated obesity as a factor in the severity of COVID-19 pneumonia, and can lead to intensive care or even death. Some biomarkers have been identified as risk factors for mortality. The aim of this study is to verify obesity and the risk factors for mortality of COVID-19 infection.

Methods: This was a single-center prospective study carried out at Rouiba University Hospital, between March 19, 2020 to September 30, 2021. The clinical data were collected: age (year), BMI groups (≥ 30 and < 30 kg/m^2), sex, active smoking, medical history, clinical complaints, Peripheral oxygen saturation (SpO_2) at admission, and the length of hospital stay. A standard laboratory assessment and a chest CT without a contrast agent were performed. The prognostic was verified, and the healing, death, or transfers to intensive care were noted, and the data was analyzed.

Results: Our results showed an obesity rate (26.8%) and a mortality rate (5.3%) and found that obesity increases the risk of severity but not mortality in hospitalized patients. The risk factors for death from COVID-19 were the underlying chronic diseases including diabetes, COPD, renal failure and cardiovascular disease, hypoxia on admission, elevated serum LDH, CRP, and D. Dimer level.

Conclusions: The inclusion of obesity and risk factors in therapeutic management strategy and prognostic scores will be essential to improve the prognosis of hospitalized COVID-19 patients.

Keywords

Obesity, BMI, COVID-19, Severity, Mortality, Risk factors

Introdution

The SARS-CoV-2 coronavirus pneumonia outbreak has rapidly spread globally and COVID-19 was classified as a pandemic by the World Health Organization in March 2020 [1].

COVID-19 is responsible for very broad expressions ranging from mild upper respiratory tract symptoms to acute respiratory distress syndrome (ARDS), hypercoagulability and cytokine storm [2].

However, the analysis of the clinical and demographic characteristics of COVID-19 patients around the world has made it possible to observe a rich semiology, which differs from one region to another and an estimated mortality rate of 3.2% [3]. Current literature suggests that complications from obesity potentially increase the severity of COVID-19, particularly in people under the age of 60 [4]. Previous studies have implicated obesity as a factor in the severity of COVID-19 pneumonia [5] increased hospitalizations [6,7] and the risk of invasive mechanical ventilation [8]. Various mechanisms may be involved, namely restrictive ventilatory deficit,

Citation: Ketfi A, Said RYS, Malki G, Touahri R (2024) The Impact of Comorbidities and Obesity on the Severity of COVID-19 and Risk Factors for Mortality, A Prospective Study in Hospitalized Patients. Int J Respir Pulm Med 11:191. doi.org/10.23937/2378-3516/1410191

Accepted: February 02, 2024; **Published:** February 04, 2024

lipotoxicity and induction of a pro-inflammatory state [9]. Other studies suggest that overweight and obese patients have a higher risk of more serious clinical symptoms during SARS-CoV-2 infection [10-13]. Overweight and obese patients require more frequent hospitalizations in intensive and semi-intensive care units, regardless of age. In addition, overweight and obese patients have a more frequent need for assisted ventilation due to SARS-CoV-2 pneumonia [8].

It suggest that obesity contributes to clinical manifestations and may influence the progression and prognosis of COVID-19 and it is considered as a potential risk factor of the prognosis of COVID-19 [14].

Algeria like the rest of the world is facing the spread of this pathology, and the first patient carrying this virus was detected on February 25, 2020. Obesity is reaching epidemic proportions in Algeria, and weighs heavily on the system of Algerian health. In 2010, the TAHINA study reported a prevalence of total obesity in Algeria of 9.1% in men and 30.1% in women [15,16]. To effectively fight this epidemic, the Algerian health authorities need to identify the risk factors for severe forms of patients hospitalized for Covid-19 [17,18]. Some authors have suggested that overweight and obese patients should be classified as high risk and should be minimally protected from infection and monitored more closely for SARS-CoV-2 pneumonia [10].

Symptoms of COVID-19 can include fever, cough, breathing difficulties, and organ failure, the severity of the disease can lead patients to intensive care or even death [19]. old age, certain biomarkers such as LDH and D-Dimer, have been identified as risk factors for mortality [18,20]. The aim of this study is to evaluate the association between obesity and the clinical, biological, CT, and prognosis profile of Algerian patients hospitalized for COVID-19 and their mortality risk factors.

Patients and Methods

Study design

This study is the third part of a project analyzing clinical, biological and radiological data from Algerian patients with Covid-19. The first part published, consists of a descriptive study [16], the second part concerned the Analysis of the clinical, biological and radiological severity factors of Algerian patients hospitalized for Covid-19: Comparison between patients with normal and low pulsed oxygen saturation of hemoglobin (SpO_2). As a large part of the methodology of this study was previously described [16,21] only the main points of the methodology will be treated in this paper.

This was a single-center prospective study, which was carried out at Rouiba University Hospital, Algiers (Algeria) (period: March 19, 2020 to September 30, 2021). Only patients with a positive diagnosis of Covid-19 [real-time PCR, antigen test [22-25] and pulmonary CT signs compatible with the infection were included in the study. Lack of BMI on admission was a non-inclusion criterion.

Data sources

The following clinical data were collected from a pre-established observation sheet and hospitalized patient records: Age (year), BMI groups (≥ 30 and < 30 kg/m^2), sex, active smoking, notion of contact with a suspected or confirmed case of Covid-19, medical history, clinical complaints, Peripheral oxygen saturation (SpO_2) at admission, length of hospital stay. Body weight was measured, height was self-reported, and indeed it was difficult to measure the height of the patients due to the clinical condition of the patients and to prevent any risk of SARS-CoV-2 transmission. Body mass index (BMI) was calculated using the usual formula (weight [kilograms]/ height squared [square meters]) and categorized into five standard groups on the basis of National Heart, Lung, and Blood Institute criteria [26]: Insufficient BMI, < 18.5 kg/m^2; reference category, 18.5 kg/m^2 at < 25 kg/ m^2; overweight, 25 kg/m^2 to < 30 kg/m^2; and obesity was defined as a BMI ≥ 30 kg/m^2.

A standard laboratory assessment and a chest CT without a contrast agent were performed [16]. The prognostic was verified, and the healing, death, or transfers to intensive care were noted.

Statistical analysis

Quantitative and qualitative data were expressed as means ± standard deviations, and number (%), respectively. Missing data were removed from statistical analyzes [27]. Student's T-test and Pearson's Chi-square test were used to compare quantitative and qualitative data from the two groups, respectively. The results were entered using the Statistica software (Statistica Kernel version 6; Stat Soft. France). A significance level < 5% was retained.

Results

Among the 705 patients hospitalized and were confirmed Covid-19, in the department of pulmonology. Of these, 194 were excluded because anthropometric data was missing at admission. The 511 patients selected were divided into two groups: obese-group (137 patients) and non-obese group (374 patients).

Clinical profile and medical complaints

Table 1 exposes the characteristics and medical background of patients. Compared to the non-obese group, the obese-group included higher percentages of females and patients aged < 50 years. However, the two groups were age matched. Included lower percentage of smokers, have higher percentage of hypertendue and have similar medical background.

Table 2 exposes the clinical complaints and physical

Table 1: Characteristics and medical background of patients.

	Total sample (n = 511)	G_1: BMI ≥ 30 kg/m² (n = 137)	G_2: BMI < 30 kg/m² (n = 374)	p
Characteristics				
Sex (female)	265 (51.9)	87 (64)	178 (47)	< 0.001**
Age ≥ 50 years	335 (65.6)	258 (57)	77 (69)	0.010**
Age (years)	56 ± 15	53 ± 14	57 ± 16	0.002*
Height (m)	1.68 ± 0.09	1.66 ± 0.09	1.69 ± 0.09	0.045*
Weight (kg)	79 ± 15	93 ± 13	74 ± 11	< 0.001*
BMI (kg/m²)	27.8 ± 4.8	33.7 ± 3.5	25.7 ± 3.2	< 0.001*
Corpulence status				
Underweight	11 (2)	-	11 (3)	-
Normal weight	126 (25)	-	126 (34)	-
Overweight	237 (46)	-	237 (63)	-
Obesity level-1	101 (20)	101 (74)	-	-
Obesity level-2	28 (5)	28 (21)	-	-
Obesity level-3	8(2)	8 (6)	-	-
Smokers	69 (14)	10 (8)	59 (16)	0.013*
Contact with a suspected/confirmed case of Covid-19	263 (52)	71 (52)	192 (52)	0.530
Medical background				
Arterial hypertension	169 (33)	56 (41)	113 (30)	0.023*
Mellitus diabetes	132 (26)	35 (26)	97 (26)	0.929
Chronic respiratory disease and allergy	56 (11)	12 (9)	44 (12)	0.348
COPD	9 (2)	1 (1)	8 (2)	0.284
Asthma	28 (5)	8 (6)	20 (5)	0.829
Heart diseases	64 (13)	17 (12)	47 (13)	0.962
Thyroid diseases	46 (9)	17 (13)	29 (8)	0.093
Cancer	14 (3)	2 (1)	12 (3)	0.292

G: Group. BMI: Body Mass Index. Quantitative and categorical data were expressed as mean ± standard deviation and number (%), respectively.

P (probability): $p < 0.05$ (*Student Test, **Two sided Chi-2): G_1 vs. G_2.

Table 2: Clinical complaints and physical exam' data of patients.

	Total sample (n = 511)	G_1: BMI ≥ 30 kg/m² (n = 137)	G_2: BMI < 30 kg/m² (n = 374)	p
Clinical complaints				
Fever	398 (78)	114 (83)	284 (77)	0.344
Cough	385(75)	107 (79)	278 (74)	0.293
Dyspnea	261 (51)	84 (62)	177 (47)	0.004*
Hemoptysis	14 (3)	7 (5)	7 (2)	0.046*
Sore throat	132 (26)	36 (26)	96 (26)	0.867
Ageusia	190 (37)	59 (43)	131 (35)	0.071
Anosmia	176 (35)	60 (44)	116 (31)	0.007*
Abdominal pain	94 (18)	32 (23)	62 (16)	0.071
Vomiting and/or nausea	119 (23)	36 (26)	83 (22)	0.306
Diarrhea	195 (38)	61 (45)	134 (36)	0.060
Myalgia	303 (59)	90 (66)	213 (57)	0.056
Headache	283 (55)	83 (61)	200 (53)	0.122
Skin lesion	19 (4)	3 (2)	16 (4)	0.280

Asthenia	413 (81)	114 (84)	299 (80)	0.294
Anorexia	300 (59)	89 (65)	211 (56)	0.062
Chest pain	142 (28)	39 (29)	103 (27)	0.752
Eye burn	35 (7)	10 (7)	25 (7)	0.770
Fear of heights	117 (23)	33 (24)	84 (22)	0.629
Rhinorrhea	27 (5)	11 (8)	16 (4)	0.084
Physical exam' data				
Temperature at admission	37.3 ± 0.9	37.4 ± 0.9	37.2 ± 0.9	0.318
Respiratory rate (cpm)	24 ± 8	25 ± 10	23 ± 7	0.051
Heart rate (cpm)	90 ± 16	91 ± 15	89 ± 16	0.176
Oxy-sat (%) at admission	90 ± 10	88 ± 11	91 ± 9	0.004*
Fever (temperature ≥ 37.5 °C)	117 (39.4%)	38 (46.91%)	79 (36.57%)	0.105
Tachypnea (respiratory rate > 20 cpm)	210 (60)	60 (69)	150 (57)	0.045*
Tachycardia (heart rate ≥ 100)	84 (18)	24 (21)	60 (17)	0.419
Bradycardia (heart rate ≤ 60)	8 (2)	1 (1)	7 (2)	0.408
Low oxy-sat (< 92%) at admission	315 (62)	69 (50)	246 (66)	0.002*

G: Group; BMI: Body Mass Index. Quantitative and categorical data were expressed as mean ± standard deviation and number (%), respectively.

P (probability): $p < 0.05$ (*Student Test, **Two sided Chi-2): G_1 vs. G_2.

exam data of patients. The two groups have similar clinical complaints and similar physical exam' data (except for dyspnea: Compared to the non-obese group, the obese-group included a higher percentage of patients with dyspnea, with anosmia and her with hemoptysis), and lower oxygen saturation level at admission.

Table 3 presents the biological (ESR and CRP) and hematological data of patients. The two groups had similar biological and hematological data and included similar percentage of patients having anemia, polycythemia, lymphopenia, basocythemia, hyperleukocytosis, thrombocytopenia, thrombocytosis, biological inflammatory syndrome, high CRP or ESR, but a lower level of leucopenia.

Table 4 presents the biochemical data of patients. Compared to the non-obese group, the obese-group have a higher value of LDH, a higher value of sodium, and to a lesser degree AST and creatinine.

Table 5 presents the CTs' data of patients, length of hospitalization and patients' issues.

The two groups have radiological data without significant differences, except for the CT extension of more than 75% more marked in the obese group.

The two groups have a similar duration of hospitalization also have an equal frequency of death and include identical percentages of patients transferred to intensive care.

Table 6 exposes the characteristics, and factors associated with in-hospital mortality.

Compared to the survivor group, the non-survivor group had higher percentages of men, older ages, and more comorbidities (heart disease, hypertension, diabetes COPD, and chronic kidney disease, but the same percentage of asthma. The non-survivor group included a higher frequency of dyspnea and a lower level of saturation. Based on the biological profile, the non-survivor group included a higher level of polynuclear neutrophils, LDH, and C-reactive protein, and a higher number of patients with D-dimers greater than 1600 ng/ml and a higher level of kaliemia. On the other hand, the same group included more patients with an extent of lesions greater than 50%.

Discussion

A few studies were available in the North African environment to assess the risk factors of dying during hospitalizations of patients with COVID-19. Our results could give a model to evaluate risk and use it at convenience in acute care settings.

In this study, we collected data from 511 cases of COVID-19. The cohort was a random group of patients representing the real situation of patients hospitalized in our department with 26.8% of obese patients, although obesity represents according to the tahina study [15] 21.24% of the Algerian population. In a large New York study of patients hospitalized for COVID-19, 41.7% had a body mass index (BMI) > 30 kg/m^2 and 19.0% a BMI > 35 kg/m^2 [28]. Analysis of the data by BMI identified 2 groups with an average BMI of (33.7 versus 25.7) in the whole sample, and of (33.8 versus 25.8) in subjects less than 50-years-old. In our study, the obese vs. non-obese group had higher percentages of women and patients

Table 3: Hematological, ESR and CRP data of patients.

	Total sample (n = 511)	G_1: BMI ≥ 30 kg/m² (n = 136)	G_2: BMI < 30 kg/m² (n = 375)	p
Quantitative data				
Hemoglobin (g/dl)	12.9 ± 1.7	12.8 ± 1.6	12.9 ± 1.8	0.601
Leukocytes (10^3/mm³)	7995 ± 3931	7910 ± 3429	8026 ± 4102	0.779
Leukocyte count				
NPN (10^3/mm³)	5961 ± 3603	5781 ± 3158	6009 ± 3744	0.627
EPN (10^3/mm³)	41 ± 78	30 ± 57	45 ± 84	0.084
BPN (10^3/mm³)	131 ± 130	139 ± 137	138 ± 131	0.416
Lymphocytes (10^3/mm³)	1184 ± 728	1228 ± 751	1168 ± 743	0.430
Monocytes (10^3/mm³)	666 ± 541	688 ± 648	657 ± 514	0.606
Platelets (10^3/mm³)	262 ± 104	266 ± 118	261 ± 101	0.660
ESR (1st h) (mm)	65 ± 37	68 ± 39	64 ± 37	0.386
CRP (mg/L)	55 ± 59	57 ± 59	53 ± 59	0.656
Patients' profile				
Anemia	154 (35)	36 (31)	118 (37)	0.255
Polycythemia	4 (1)	0 (0)	4 (1)	0.227
Leukopenia	44 (10)	6 (5)	38 (12)	0.040*
Lymphopenia	204 (47)	51 (44)	153 (48)	0.435
Basocythemia	128 (30)	38 (34)	90 (29)	0.335
Hyperleukocytosis	103 (23)	28 (24)	75 (23)	0.886
Thrombocytopenia	50 (11)	13 (11)	37 (12)	0.898
Thrombocytosis	26 (6)	9 (7)	17 (5)	0.352
High CRP	248 (63)	66 (63)	182 (63)	0.932
High ESR (1st h)	263 (82)	66 (81)	197 (82)	0.836
Biological inflammatory syndrome	358 (83)	92 (80)	266 (84)	0.377

BPN: Basophilic Polynuclear; BMI: Body Mass Index; CRP: C-Reactive Protein; EPN: Eosinophilic Polynuclear; ESR: Erythrocyte Sedimentation Rate; G: Group; NPN: Neutrophilic Polynuclear. Quantitative and categorical data were expressed as mean ± standard deviation and number (%), respectively.

P (probability): $p < 0.05$ (*Student Test, **Two sided Chi-2): G_1 vs. G_2.

Table 4: Biochemical data of patients.

	Total sample (n = 511)	G_1: BMI ≥ 30 kg/m² (n = 137)	G_2: BMI < 30 kg/m² (n = 374)	p
Quantitative data				
Kidney Function				
Urea (g/L)	0.43 ± 0.31	0.45 ± 0.37	0.42 ± 0.28	0.301
Creatinine (mg/L)	11.81 ± 10.28	13.27 ± 16.16	11.29 ± 7.07	0.072
Liver Function				
ASAT (UI/L)	44.80 ± 30.99	49.39 ± 34.54	43.06 ± 29.54	0.039
ALAT (UI/L)	40.86 ± 39.31	43.08 ± 35.68	40.80 ± 41.52	0.601
ALP (UI/L)	168.19 ± 60.06	161.67 ± 51.11	171.02 ± 61.14	0.156
Serum Electrolytes				
Potassium (mmol/l)	3.90 ± 0.48	3.82 ± 0.53	3.92 ± 0.46	0.063
Sodium (mmol/l)	136.83 ± 4.67	137.70 ± 4.38	136.53 ± 4.73	0.032*
Prothrombin level (%)	84.73 ± 15.41	86.82 ± 15.43	84.54 ± 14.27	0.221
CPK (UI/L)	164.78 ± 317.53	205.56 ± 333.20	142.71 ± 323.20	0.753
LDH (UI/L)	555.42 ± 305.72	647.61 ± 371.44	527.05 ± 277.06	0.001*

ALAT: Alanine Amino-Transferase; ALP: Alkaline Phosphatase; ASAT: Aspartate Amino-Transferase.; BMI: Body Mass Index; CPK: Creatine Phosphokinase; G: Group; LDH: Lactico-Dehydrogenase. Quantitative and categorical data were expressed as mean ± standard deviation and number (%), respectively.

P (probability): $p < 0.05$ (*Student Test, **Two sided Chi-2): G_1 vs. G_2.

Table 5: Computed tomography scan data of patients, length 0f hospitalization, issues of patients.

	Total sample (n = 511)	G_1: BMI ≥ 30 kg/m² (n = 137)	G_2: BMI < 30 kg/m² (n = 374)	p
Radiological signs				
ground-glass	455 (94)	128 (98)	327 (93)	0.078
nodular ground-glass	239 (50)	72 (55)	167 (48)	0.158
diffuse ground-glass opacity	334 (69)	89 (68)	245 (70)	0.778
crazy paving	169 (35)	44 (34)	125 (36)	0.664
condensation	258 (54)	71 (54)	187 (53)	0.860
CT extension of lesions				
< 10%	106 (22)	25 (19)	81 (23)	0.327
10-25%	87 (18)	23 (17)	64 (18)	0.834
25-50%	138 (28)	36 (27)	102 (29)	0.683
50-75%	83 (17)	28 (21)	55 (15)	0.153
> 75%	11 (2)	8(6)	3(1)	< 0.001
Length of hospitalization, issues of patients				
Hospital stay (day)	10.1 ± 6.5	10.0 ± 6.6	10.4 ± 6.2	0.605
Transfer to an intensive care-unit	49 (9.6)	16 (11.7)	33 (8.8)	0.332
Death	27(5.3)	20 (5.3)	7 (5.1)	0.915

BMI: Body Mass Index; G: Group. Data were expressed as number (%). p (probability): p < 0.05 (*Two sided Chi-2): G_1 vs. G_2.

Table 6: Factors associated with in-hospital mortality.

Parameters at admission	Overall (N = 511)	Survivors (N = 484)	Non-survivors (N = 27)	p
BMI, mean ± SD, kg/m²	27.83 ± 4.82	27.85 ± 4.74	27.34 ± 6.11	0,589
Age years	56.2 ± 15.5	55.4 ± 15.2	69.2 ± 14.8	< 0.0001*
Sex (male)	48.1 (246)	47.7(231)	55,6(15)	0.429
Smokers	13.9 (69)	13.4 (63)	23,1 (6)	0.166
Comorbidities				
Heart disease	12.5 (64)	11.2 (54)	37.0 (10)	< 0.0001*
Hypertension	33.1 (169)	32.4 (157)	44.4 (12)	0.197
Diabetes mellitus	25.8 (132)	24.0 (116)	59.3 (16)	< 0.0001*
Asthma	5.5 (28)	5.6 (27)	3.7 (1)	0.678
COPD	1.8 (9)	1.2 (6)	11.1 (3)	0.0001*
Chronic kidney disease	2 (10)	1.5 (7)	11.5 (3)	0.0003*
Symptoms				
Cough	75.5 (385)	75.0 (363)	84.6 (22)	0.268
Dyspnea	51.2 (261)	49.4 (239)	84.6 (22)	0.0008*
SpO_2 > 92%	61.8 (315)	64.3 (311)	15.4 (4)	< 0.0001*
Air ambient Oxy-sat (%)	90.3 ± 9.6	91.2 ± 8.01	72.8 ± 17.1	< 0.0001
Hemoglobin (g/l)	12.86 ± 1.75	12.9 ± 1.7	12.4 ± 2.1	0.162
Polynuclear neutrophils (/mm³)	5961 ± 3603	5845 ± 3557	7898 ± 3879	0.004*
Lymphocytes (/mm³)	1184 ± 729	1187 ± 724	1134 ± 816	0.719
ESR (1st hour) (mm)	65.3 ± 37.6	65.2 ± 37.9	67.0 ± 31.2	0.850
C-reactive protein (mg/l)	54.5 ± 59.0	52.9 ± 57.8	82.5 ± 72.1	0.017*
LDH level (u/l)	555.4 ± 305.7	547.0 ± 294.4	738.4 ± 466.3	0.009*
D-dimers > 1600 ng/ml	25.7 (75)	24.0 (64)	44.0 (11)	0.03*
Kaliemia (mmol/l)	3.90 ± 0.47	3.87 ± 0.46	4.15 ± 0.70	0.017*
Natremia (mmol/l)	136.8 ± 4.67	136.7 ± 4.64	138.6 ± 5.01	0.092

CT extent of covid-19				
< 10%	21.6 (106)	22.3 (104)	8.7 (2)	0.147
10-25%	17.8 (87)	18.2 (85)	8.7 (2)	0.245
25-50%	2.2 (138)	28.1 (131)	30.4 (7)	0.904
50-75	16.9 (83)	15.8 (74)	39.1 (9)	0.004*
> 75	2.2 (11)	1.9 (9)	8.7 (2)	0.032*
Length of stay in the hospital(day)	10.1 ± 6.5	10.0 ± 6.2	11.1 ± 9.9	0.332
Quantitative and categorical data were expressed as mean ± standard deviation and number (%), respectively. p (probability) : p < 0.05 (*Student Test)				

under the age of 50. Comparable results were found in an Italian study, where the relationship between obesity and COVID-19 does not appear evident in the general population, on the other hand it was particularly clear in the youngest subjects [29].

In our study of patients under 50 infected with COVID-19, BMI was similar to the mean BMI of the entire sample, while in other studies in patients of under 50 years infected with SARS-CoV-2 the average BMI was higher, and this index seemed to decrease with age in COVID-19 patients [30-32]. The results suggest that obesity may be more prominent in young patients with COVID-19.

The comorbidities in the high BMI group were particularly less pronounced in the cohort of patients under 50, with fewer underlying illnesses such as hypertension, metabolic disease, diabetes and dysthyroidism, contrary to some studies already reported [6,33-35].

The literature data precise, that the effect of obesity on COVID-19 was independent of comorbidities, such as diabetes, hypertension, and cardiovascular disease. This assumes a significant pathophysiological link between excessive adiposity and severe COVID-19 disease [11,30,36], according to Gao, et al. [37] Obesity has tripled the risk of worsening COVID-19.

Obesity induces T cell depletion through constant low-grade inflammation, which impairs the immune response and the ability to eradicate the virus from the host [37,38]. The lack of activity in obesity may also interfere with the activation of immune cells [39].

Deng, et al. [40] have suggested that visceral, hepatic, epicardial, and perirenal adiposity may predict the risk of severe COVID-19 in young obese patients.

On analysis of chest imaging in obese and non-obese patients, we found that the distribution and frequency of lung lesions were slightly different. The cases of obesity also manifested a higher proportion of specific opacity in ground glass (98% against 93%). Lung damage involvement was slightly more extensive in obese patients, this difference becomes more pronounced, especially for CT lesions greater than 50%. Similar results have been described in the literature [14,41].

Indeed, obesity is the result of abnormal energy metabolism, leading to weight gain and metabolic disturbances, which in turn lead to stress and tissue dysfunction [42,43]. In our study, obese patients showed a slightly high rate of CRP and erythrocytic sedimentation levels than those who were not obese Table 3, these markers of inflammation influence the progression and poor prognosis of COVID-19 disease. Obesity provides a favorable environment for disease pathogenesis and is characterized by a chronic, low-inflammatory condition, which can lead to the production of depleted immune cells, and the body becomes more vulnerable to infections [8,44,45]. The excessive immune response to SARS-CoV-2 is the main reason for the severe forms of illness and the mortality of patients with COVID-19 [2]. The CRP was slightly higher in obese COVID-19 patients compared to non-obese patients in previous studies [46,47]. In addition, significantly higher levels of LDH and hypernatremia in obese patients, are associated with a slight, less pronounced elevation of transaminases mainly AST and hypokalaemia (Table 4).

BMI, ALAT and ASAT were independently and inversely associated with being discharged from hospital in time for these patients. one study found that obesity and abnormal liver function (ALF) predispose patients with COVID-19 to prolonged hospitalization [48], in our study we found the same trend with more patients staying longer than 10 days among obese subjects and subjects with a high ALT level.

In our study, kalaemia was slightly greater in the group of obese patients (3.82 versus 3.92).

Sodium ion disorder, particularly hyponatremia, is a common occurrence in hospitalized patients with COVID-19 in a Chinese study, and is associated with a higher risk of serious illness and increased hospital mortality [49]. In our study, we found a correlation between hyponatremia and the duration of hospitalization, particularly beyond 10 days, and a correlation between hypernatremia high BMI, and mortality (Table 6).

Indeed, the high prevalence of hypokalaemia in patients with COVID-19 in a Mediterranean cohort suggests the presence of a disturbance in the activity of the renin angiotensin system due to severe infection

of SARS-CoV-2 [50,51]. Additionally, this sensitive biomarker may reflect the progression of COVID-19. Hypokalaemia has been shown to be independently associated with the need for invasive mechanical ventilation [50]. Obesity can disrupt immune responses, making obese patients susceptible to infections, both bacterial and viral [52,53]. This increased risk had already been described for infections with the influenza virus, [54] with a longer duration of contagiousness in obese people compared to that in non-obese [50].

In our study, the mean hospital stay was similar to 10 days versus 10.4 days, in fact, the hospital stay was the same in previous studies [35]. In our work, obese patients had lower oxygen saturation on admission (88% versus 91%). In our work, obese patients had lower oxygen saturation on admission (88% versus 91%) [35].

We found no higher mortality or transfer rate to the intensive care unit for obese patients compared to those who were not obese in our sample (5.3 versus 5.1) for mortality, (11.7 versus 8.8) for transfer to the intensive care units. Identical results with a BMI were not found to be an independent predictor of mortality [55].

However in previous viral pandemics, it has been shown that obesity, especially severe obesity (BMI > 40 kg/m^2), is associated with an increased risk of hospitalization, admission to intensive care and death [56,57].

A systematic review and meta-analysis of 22 studies showed that obesity was associated with a poor prognosis for SARS-CoV-2 infection, marked by more cases of severe COVID-19, admission to intensive care, recourse to mechanical ventilation and rapid progression of the disease, especially in younger subjects (OR 3.30 versus 1.72). However, this meta-analysis did not find an association between obesity and hospital mortality [30].

Current literature suggests an association between obesity and increased mortality in patients with COVID-19 pneumonia in the general population, particularly in younger patients [10,13,55,58]. However, a lower BMI ≤ 25 was associated with a decrease in the need for mechanical ventilation [55]. Obesity was significantly associated with a greater likelihood of occurrence of the primary endpoint (death), and a maximum percentage of death (32.61%) was noted in obesity Classes II and III (BMI ≥ 35 Kg/m^2) [59]. Group Class I (BMI 18.5-24.9 Kg/m^2) had the least percentage of meeting the primary endpoint [59]. Severe obesity is a relevant risk factor for COVID-19 hospitalization and severity in young adults, having a magnitude similar to that of aging [60]. Higher BMI in early adulthood was associated with severe COVID-19 many years later with a risk increase starting already at BMI ≥ 22.5 [61]. Study show that having a BMI that is ≥ 30 kg/m^2 is a significant risk factor in COVID-19 morbidity and mortality [62].

Comparing the survivor group with the non-survivor group in Table 6, the latter had higher percentages of males, older ages, and more comorbidities (heart disease, hypertension, diabetes COPD, and chronic kidney disease, but the same percentage of asthma), the results were found by Kyoung Min Kim [63-65]. The non-survivor group had a higher frequency of dyspnea and a lower level of oxygen saturation. Based on the biological profile, the non-survivor group included a higher level of polymorphonuclear neutrophils, LDH, and C-reactive protein, and a higher number of patients with D-dimer greater than 1600 ng/ml, which indicated that D-dimer could be an early marker to improve the management of Covid-19 patients [66], in fact, hospital mortality was significantly higher in patients with high neutrophil count, lower lymphocyte count, elevated CRP, and D-dimer ≥ 2.0 µg/ml than those who had D-dimer < 2.0 µg/ml on admission [67]. On the other hand, the same group included more patients with an CT extent of lesions greater than 50%. The risk of mortality for COVID-19 patients could be evaluated using a lung CT-scan extent cutoff [68,69].

Strengths and Limitations

The strength of this study is prospective study included a single center with the same team of investigators, over 18 months, in a pulmonology department. There were some limitations to the current study. First, our study cannot be considered exhaustive, and might be possible other factors that affect COVID-19 mortality, is the retrieval of clinical data was difficult for severely ill patients.

In summary, obesity contributes to clinical manifestations and can influence the progression and prognosis of COVID-19, with an accumulated risk of serious complications for obese subjects. Our cohort showed an obesity rate (26.8%) and a mortality rate (5.3%), and warned that obesity increases the risk of severity but not mortality in hospitalized patients for COVID-19. Therefore, the inclusion of obesity in prognostic scores and therapeutic management strategy will be essential to improve the prognosis of hospitalized patients with COVID-19. risk factors for death from COVID-19 were a history of underlying chronic diseases including diabetes, COPD, renal failure and cardiovascular disease, hypoxia on admission, elevated serum LDH, CRP and D.Dimer linked to the survival status of COVID-19 patients.

Further studies are needed to assess the association between age, obesity, frailty, and clinical outcome in adults with COVID-19 disease.

What is already know on this topic

- It suggest that obesity contributes to clinical manifestations and may influence the progression and prognosis of COVID-19 and it is considered as a potential risk factor of the prognosis of

COVID-19

- old age, certain biomarkers such as LDH and D-Dimer, have been identified as risk factors for mortality

What this study adds

- Obesity contributes to clinical manifestations and can influence the progression and prognosis of COVID-19, with an accumulated risk of serious complications for obese subjects.
- Our study showed, that obesity increases the risk of severity but not mortality in hospitalized patients for COVID-19.
- The inclusion of obesity in prognostic scores and therapeutic management strategy will be essential to improve the prognosis of hospitalized patients with COVID-19. The risk factors for death from COVID-19 were a history of underlying chronic diseases including diabetes, COPD, renal failure and cardiovascular disease, hypoxia on admission, elevated serum LDH, CRP and D-Dimer linked to the survival status of COVID-19 patients.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Establishment Where the Work was Performed

Department of Pneumology, Phthisiology and Allergology, Rouiba Hospital, University of Algiers, Faculty of Medicine, Algiers, Algeria.

Authors Contributions

KA conceived the study, participated in its design, performed the spirometry tests and the statistical analysis, and helped to draft the manuscript and coordinated the study; RY participated in the lung high-resolution CT scan interpretation, helped to draft the manuscript; GM participated in the diagnosis of connective tissue disease, helped to draft the manuscript; RT participated in its design, helped to draft the manuscript.

Both authors read and approved the final version of the manuscript.

Conflicts of Interest

The authors declare that they have no conflicts of interest concerning this article.

Data Availability Statement

Data are available upon a request from the corresponding author (ketfiabdelbassat@gmail.com).

References

1. Cucinotta D, Vanelli M (2020) WHO Declares COVID-19 a Pandemic. Acta Biomed 91: 157-160.
2. Mehta P, McAuley DF, Brown M, Sanchez E, Tattersall RS, et al. (2020) COVID-19: Consider cytokine storm syndromes and immunosuppression. The Lancet 395: 1033-1034.
3. WHO (2020) Coronavirus disease 2019 (COVID-19): situation report, 73.
4. Caussy C, Pattou F, Wallet F, Simon C, Chalopin S, et al. (2020) Prevalence of obesity among adult inpatients with COVID-19 in France. Lancet Diabetes Endocrinol 8: 562-564.
5. Cai Q, Chen F, Wang T, Luo F, Liu X, et al. (2020) Obesity and COVID-19 severity in a designated hospital in Shenzhen, China. Diabetes Care 43: 1392-1398.
6. Petrilli CM, Jones SA, Yang J, Rajagopalan H, O'Donnell L, et al. (2020) Factors associated with hospitalization and critical illness among 4,103 patients with COVID-19 disease in New York City. MedRxiv.
7. Suleyman G, Fadel RA, Malette KM, Hammond C, Abdulla H, et al. (2020) Clinical characteristics and morbidity associated with coronavirus disease 2019 in a series of patients in metropolitan Detroit. JAMA Netw Open 3: e2012270.
8. Simonnet A, Chetboun M, Poissy J, Raverdy V, Noulette J, et al. (2020) High prevalence of obesity in severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) requiring invasive mechanical ventilation. Obesity (Silver Spring) 28: 1195-1199.
9. Sattar N, McInnes IB, McMurray JJ (2020) Obesity is a risk factor for severe COVID-19 infection: Multiple potential mechanisms. Circulation 142: 4-6.
10. Busetto L, Bettini S, Fabris R, Serra R, Pra CD, et al. (2020) Obesity and COVID□19: an Italian snapshot. Obesity (Silver Sring) 28: 1600-1605.
11. Klang E, Kassim G, Soffer S, Freeman R, Levin MA, et al. (2020) Severe obesity as an independent risk factor for COVID-19 mortality in hospitalized patients younger than 50. Obesity (Silver Spring) 28: 1595-1599.
12. Giacomelli A, Ridolfo AL, Milazzo L, Oreni L, Bernacchia D, et al. (2020) 30-day mortality in patients hospitalized with COVID-19 during the first wave of the Italian epidemic: A prospective cohort study. Pharmacol Res 158: 104931.
13. Palaiodimos L, Kokkinidis DG, Li W, Karamanis D, Ognibene J, et al. (2020) Severe obesity, increasing age and male sex are independently associated with worse in-hospital outcomes, and higher in-hospital mortality, in a cohort of patients with COVID-19 in the Bronx, New York. Metabolism 108: 154262.
14. Kang Z, Luo S, Gui Y, Zhou H, Zhang Z, et al. (2020) Obesity is a potential risk factor contributing to clinical manifestations of COVID-19. International Journal of Obesity 44: 2479-2485.
15. Atek M, Laid Y, Mezimeche N, Boutekdjiret L, Lebcir H, et al. (2010) L'Obésité chez l'adulte de 35 à 70 ans en Algérie. Projet TAHINA. Institut national de santé publique Alger Algérie. 1-93.
16. Ketfi A, Chabati O, Chemali S, Mahjoub M, Gharnaout M, et al. (2020) Profil clinique, biologique et radiologique des patients Algériens hospitalisés pour COVID-19: données préliminaires. Pan Afr Med J 35: 77.
17. Snouber A, Metahri M, Chahraoui S, Benatta D, Boukhari S, et al. (2022) Clinical and computed tomography features of patients suspected of COVID-19 in the university hospital of Oran, Algeria. Tunis Med 100: 374-383.

18. Ketfi A, Touahri R, Chabati O, Chemali S, Mahjoub M, et al. (2021) Severity factors in Algerian patients hospitalized for COVID-19. Tunis Med 99: 734-743.

19. Vardavas CI, Mathioudakis AG, Nikitara K, Stamatelopoulos K, Georgiopoulos G, et al. (2022) Prognostic factors for mortality, intensive care unit and hospital admission due to SARS-CoV-2: a systematic review and meta-analysis of cohort studies in Europe. Eur Respir Rev 31: 220098.

20. Tazerji SS, Shahabinejad F, Tokasi M, Rad MA, Khan MS, et al. (2022) Global data analysis and risk factors associated with morbidity and mortality of COVID-19. Gene Rep 26: 101505.

21. Ketfi A, Touahri R, Chabati O, Chemali S, Mahjoub M, et al. (2021) Facteurs de sévérité chez les patients Algériens hospitalisés pour COVID-19. La Tunisie Médicale 99: 734.

22. Sethuraman N, Jeremiah SS, Ryo A (2020) Interpreting diagnostic tests for SARS-CoV-2. JAMA 323: 2249-2251.

23. Pan Y, Li X, Yang G, Fan J, Tang Y, et al. (2020) Serological immunochromatographic approach in diagnosis with SARS-CoV-2 infected COVID-19 patients. J Infect 81: 28-32.

24. Schwartz KL, McGeer AJ, Bogoch II (2021) Le dépistage des personnes asymptomatiques au moyen de tests antigéniques rapides: un outil de santé publique pour lutter contre la COVID-19. CMAJ 193: 888-891.

25. Yin N, Debuysschere C, Decroly M, Bouazza FZ, Collot V, et al. (2021) SARS-CoV-2 diagnostic tests: Algorithm and field evaluation from the near patient testing to the automated diagnostic platform. Front Med (Lausanne) 8: 380.

26. National Heart, Lung, and Blood Institute (1998) Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: the evidence report.

27. Guan WJ, Ni ZY, Hu Y, Liang WH, Ou CQ, et al. (2020) Clinical characteristics of coronavirus disease 2019 in China. N Engl J Med 382: 1708-1720.

28. Richardson S, Hirsch JS, Narasimhan M, Crawford JM, McGinn T, et al. (2020) Presenting characteristics, comorbidities, and outcomes among 5700 patients hospitalized with COVID-19 in the New York City Area. JAMA 323: 2052-2059.

29. Buscemi S, Buscemi C, Batsis JA (2020) There is a relationship between obesity and coronavirus disease 2019 but more information is needed. Obesity (Silver Spring) 28: 1371-1373.

30. Chu Y, Yang J, Shi J, Zhang P, Wang X, et al. (2020) Obesity is associated with increased severity of disease in COVID-19 pneumonia: A systematic review and meta-analysis. Eur J Med Res 25: 1-15.

31. Kass DA, Duggal P, Cingolani O (2020) Obesity could shift severe COVID-19 disease to younger ages. The Lancet 39: 1544-1545.

32. Bhasin A, Nam H, Yeh C, Lee J, Liebovitz D, et al. (2020) Is BMI higher in younger patients with COVID-19? Association between BMI and COVID-19 hospitalization by age. Obesity (Silver Spring) 28: 1811-1814.

33. Sattar N, Ho FK, Gill JM, Ghouri N, Gray SR, et al. (2020) BMI and future risk for COVID-19 infection and death across sex, age and ethnicity: Preliminary findings from UK biobank. Diabetes Metab Syndr 14: 1149-1151.

34. Nakeshbandi M, Maini R, Daniel P, Rosengarten S, Parmar P, et al. (2020) The impact of obesity on COVID-19 complications: A retrospective cohort study. Int J Obes 44: 1832-1837.

35. Hendren NS, Lemos JA, Ayers C, Das SR, Rao A, et al. (2021) Association of body mass index and age with morbidity and mortality in patients hospitalized with COVID-19: results from the American Heart Association COVID-19 Cardiovascular Disease Registry. Circulation 143: 135-144.

36. Cai SH, Liao W, Chen SW, Liu LL, Liu SY, et al. (2020) Association between obesity and clinical prognosis in patients infected with SARS-CoV-2. Infect Dis Poverty 9: 1-7.

37. Gao F, Zheng KI, Wang XB, Sun QF, Pan KH, et al. (2020) Obesity is a risk factor for greater COVID-19 severity. Diabetes Care 43: 72-74.

38. Mescher MF, Curtsinger JM, Agarwal P, Casey KA, Gerner M, et al. (2006) Signals required for programming effector and memory development by CD8+ T cells. Immunol Rev 211: 81-92.

39. Zheng, Q, Cui G, Chen J, Gao H, Wei Y, et al. (2015) Regular exercise enhances the immune response against microbial antigens through up-regulation of toll-like receptor signaling pathways. Cell Physiol Biochem 37: 735-746.

40. Deng M, Qi Y, Deng L, Wang H, Xu Y, et al. (2020) Obesity as a potential predictor of disease severity in young COVID-19 patients: A retrospective study. Obesity 28: 1815-1825.

41. Luo X, Jiaerken Y, Shen Z, Wang Q, Liu B, et al. (2020) Obese COVID-19 patients show more severe pneumonia lesions on CT. Diabetes Obes Metab 23: 290-293.

42. Guilherme A, Virbasius JV, Puri V, Czech MP, et al. (2008) Adipocyte dysfunctions linking obesity to insulin resistance and type 2 diabetes. Nat Rev Mol Cell Biol 9: 367-377.

43. Bremer AA, Devaraj S, Afify A, Jialal I (2011) Adipose tissue dysregulation in patients with metabolic syndrome. J Clin Endocrinol Metab 96: 1782-1788.

44. Caci G, Albini A, Malerba M, Noonan DM, Pochetti P, et al. (2020) COVID-19 and obesity: Dangerous liaisons. J Clin Med 9: 2511.

45. Dhurandhar NV, Bailey D, Thomas D (2015) Interaction of obesity and infections. Obes Rev 16: 1017-1029.

46. Han W, Quan B, Guo Y, Zhang J, Lu Y, et al. (2020) The course of clinical diagnosis and treatment of a case infected with coronavirus disease 2019. J Med Virol 92: 461-463.

47. Zhang JJ, Dong X, Cao YY, Yuan YD, Yang B, et al. (2020) Clinical characteristics of 140 patients infected with SARS-CoV-2 in Wuhan, China. Allergy 75: 1730-1741.

48. Hu X, Pan X, Zhou W, Gu X, Shen F, et al. (2020) Clinical epidemiological analyses of overweight/obesity and abnormal liver function contributing to prolonged hospitalization in patients infected with COVID-19. Int J Obes 44: 1784-1789.

49. Hu W, Lv X, Li C, Xu T, Qi Y, et al. (2021) Disorders of sodium balance and its clinical implications in COVID-19 patients: A multicenter retrospective study. Intern Emerg Med 16: 853-862.

50. Moreno-Pérez O, Manue J, Ramirez L, Kenneally LF, Perdiguero M, et al. (2020) Hypokalemia as a sensitive biomarker of disease severity and the requirement for invasive mechanical ventilation requirement in COVID-19 pneumonia: A case series of 306 Mediterranean patients. Int J Infect Dis 100: 449-454.

51. Bülent K, Paydaş S, Kuzu1 T, Demiroğlu O, Yücel SP, et al. (2021) Effects of hypokalemia on clinical outcomes in

hospitalized patients with Covid-19 pneumonia. Cukurova Med J 46: 1650-1656.

52. Milner JJ, Beck MA (2012) The impact of obesity on the immune response to infection. Proc Nutr Soc 71: 298-306.

53. Green WD, Beck MA (2017) Obesity altered T cell metabolism and the response to infection. Curr Opin Immunol 46: 1-7.

54. Green WD, Beck MA (2017) Obesity impairs the adaptive immune response to influenza virus. Ann Am Thorac Soc 14: 406-409.

55. Nyabera A, Lakhdar S, Li M, Trandafirescu T, Tall SO, et al. (2020) The association between BMI and Inpatient mortality outcomes in older adults with COVID-19. Cureus 12: 11183.

56. Fezeu L, Julia C, Henegar A, Bitu J, Hu FB, et al. (2011) Obesity is associated with higher risk of intensive care unit admission and death in influenza A (H1N1) patients: a systematic review and meta-analysis. Obes Rev 12: 653-659.

57. Fuhrman C, Bonmarin I, Bitar D, Cardoso T, Duport N, et al. (2011) Adult intensive-care patients with 2009 pandemic influenza A (H1N1) infection. Epidemiol Infect 139: 1202-1209.

58. Hajifathalian K, Kumar S, Newberry C, Shah S, Fortune B, et al. (2020) Obesity is Associated with Worse Outcomes in COVID-19: Analysis of Early Data from New York City. Obesity 28: 1606-1612.

59. Gupta S, Goel V, Nazir N, Srivastava S, Srivastava A, et al. (2022) Impact of body mass index on critically ill COVID-19 patients admitted in a tertiary care hospital: a retrospective observational study. Asian Journal of Medical Sciences 13: 3-7.

60. Fresán U, Guevara M, Elía F, Albéniz E, Burgui C, et al. (2021) Independent Role of Severe Obesity as a Risk Factor for COVID-19 Hospitalization: A Spanish Population-Based Cohort Study. Obesity 29: 29-37.

61. Robertson J, Adiels M, Lissner L, Mehlig K, Geijerstam AA, et al. (2022) BMI in early adulthood is associated with severe COVID-19 later in life–a prospective cohort study of 1.5 million Swedish men. Obesity 30: 779-787.

62. Garlapati PR, Kumar S, Patel M, Sarker B, Tiongson B, et al. (2022) Clinical characteristics and predictors of mortality in obese African-Americans with COVID-19: A single-center retrospective study. J Racial Ethn Health Disparities 10: 160-167.

63. Kim KM, Evans DS, Jacobson J, Jiang X, Browner W, et al. (2022) Rapid prediction of in-hospital mortality among adults with COVID-19 disease. PLoS One 17: e0269813.

64. Munawaroh SM, Prasetya H, Murti BH (2021) Meta-analysis the effect of asthma comorbidity on the mortality of COVID-19 patients. J Epidemiol Public Health 6: 256-267.

65. Kazemi E, Mansoursamaei A, Daliri S, Mansoursamaei M, Rasaf MR, et al. (2023) The effect of comorbid asthma on morbidity, mortality and clinical adverse outcomes in COVID-19 patients. Int J Hosp Res. 11.

66. Farooqi H, Firdous S, Kazmi S, Anwer A, Bashir A, et al. (2022) Elevated D-Dimer levels are strongly associated with High Mortality Rate in COVID-19 patients. An observational Study: Elevated D-Dimers in COVID-19 Patients. Pakistan BioMed J 5: 83-89.

67. Zhang L, Yan X, Fan Q, Liu H, Liu X, et al. (2020) D-dimer levels on admission to predict in-hospital mortality in patients with Covid-19. Journal of Thrombosis and Haemostasis. J Thromb Haemost 18: 1324-1329.

68. Zakariaee SS, Salmanipour H, Naderi N, Arpanahi HK, Shanbehzadeh M, et al. (2022) Association of chest CT severity score with mortality of COVID-19 patients: A systematic review and meta-analysis. Clin Transl Imaging 10: 663-676.

69. Szabó M, Kardos Z, Kostyá L, Tamáska P, Oláh C, et al. (2023) The importance of chest CT severity score and lung CT patterns in risk assessment in COVID-19-associated pneumonia: A comparative study. Front Med 10: 1125530.