ASPI | Afarand Scholarly Publishing Institute; Iran
ISSN: 2008-2630 Iranian Journal of War & Public Health 2022;14(2):125-131 DOI: 10.29252/ijwph.14.2.125

Comorbidity Affecting the Severity of COVID-19 Infection in Iraq

ARTICLE INFO

Article Type
Descriptive study

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How to cite this article
Ghafil N Y, Jawad Kadhim Z, Dananah F M, Abdiljaleel Alkaabi Y S, Hassoun Y L. Comorbidity Affecting the Severity of COVID-19 Infection in Iraq. Iranian Journal of War & Public Health. 2022;14(2):125-131.

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Article History
Received: March 6, 2022
Accepted: March 26, 2022
ePublished: May 30, 2022

ABSTRACT

Aims COIVD-19 Patients suffering from other diseases such as diabetic mellitus, asthma and obstructive pulmonary disease have increased morbidity and mortality rates resulting in more hospitalization and intensive therapy unit admissions. The study aimed to characterise and investigate the clinical pattern and the severity of symptoms in COVID-19 patients and to study its relationship with the coexisted morbidities.

Instrument & Methods The data was collected from COVID-19 patients (N=202) who attended the Al-Shefaa center in Al-Najaf city, Iraq, during the period from 6 September 2020 to 20 January 2021. Demographical features, comorbidities, chronic diseases, and more prevalent symptoms were studied. The data was collected anonymously and processed using IBM SPSS 25 software. The descriptive statistics were performed.

Findings 121 patients (59.9%) had a body mass index (BMI) of less than 18, while 38 patients (18.8%) have BMI ranged 18.5 to 24.9. Furthermore, 16 patients (7.9%) have BMI between 30-34.9 and only 6 (3%) have a BMI of 35-39.9. Patients with hypertension 9 (4.5%), respiratory diseases 7 (3.5%), atherosclerosis 6 (3%), diabetic Mellitus 6 (3%). Patients with moderate Covid-19 symptoms account for 54% of the cases while only 2% of the patients developed severe manifestations. Hair fall happened in 105 patients (52%), the severity can range from mild (43.6%) to severe (2.5%), and fever 61 (30%). Eighty-five patients (42.1%) of admitted patients with an incubation period of 2 weeks while 6 (3%) for 4 weeks. The requirement for artificial oxygen therapy was seen in 19 (10%) of cases, whereas 183 patients (90%) recovered without artificial oxygen support. Obesity was observed in more than 10% of infected patients, whereas more than 40% of cases have coexisted chronic diseases such as hypertension, diabetes mellitus, respiratory tract disorders and other chronic diseases. The duration of the disease ranged from a few days (15.3%) to 2 weeks (41%) to 4 weeks (3%).

Conclusion The study highlights the possible role of obesity and concurrent chronic diseases on the magnitude and duration of COVID-19 illness.

Keywords COVID-19; Comorbidities; Severity; Obesity; Diabetes Mellitus

CITATION LINKS

[1] Coronavirus-2019 (Covid 19): A review ... [2] Characteristics of SARS-CoV-2 and ... [3] Coronavirus impacts on ... [4] Molecular pathogenesis ... [5] A familial cluster of pneumonia ... [6] Barriers and facilitators to participation in physical activity for children ... [7] Structure, function, antigenicity ... [8] SARS-CoV-2 cell entry depends on ACE2 ... [9] Cryo-EM structure of the 2019-nCoV ... [10] The discovery of the ... [11] Angiotensin II and angiotensin 1-7 ... [12] Good ACE, bad ACE do battle ... [13] Angiotensin-converting enzyme 2 ... [14] Clinical and immunological features ... [15] A pneumonia outbreak associated ... [16] Hypertension in patients with ... [17] Prevalence of venous thromboembolism ... [18] Clinical features of patients infected ... [19] Structural insights into ... [20] Detection of 2019 novel ... [21] COVID-19 in persons with ... [22] COVID-19 pandemic, coronaviruses ... [23] Kidney disease is associated with ... [24] Association of higher body mass ... [25] Factors associated with COVID-19-related ... [26] Obesity could shift severe ... [27] Obesity and mortality among ... [28] Are patients with hypertension and ... [29] Longitudinal changes of inflammatory ... [30] Early enhanced expression of ... [31] Endothelial dysfunction contributes to ... [32] Clinical characteristics of 138 hospitalized ... [33] Complications of COVID-19 ... [34] Neurologic manifestations of hospitalized ... [35] Concern coronavirus may trigger ... [36] Characteristics of peripheral lymphocyte ... [37] Nervous system involvement after ... [38] Contact tracing assessment of ... [39] Virological assessment of hospitalized patients ... [40] Duration of Culturable SARS-CoV-2 in Hospitalized ... [41] Management of post-acute covid-19 ...

Introduction

Coronavirus (COVID-19) is a global pandemic infectious disease as an RNA virus belonging to the family Coronaviridae. It is highly communicable with a small size (65_125 nm in diameter), and many spikes on the outer surface. Several types of designated coronavirus such as alpha and beta coronaviruses (HCoV-OC43 and HCoV-HKU1, respectively), and newly gamma and delta cause severe acute respiratory syndrome (SARS) described in late 2019 in Wuhan, China, and spread quickly to more than 180 countries [1, 2]. Most elderly patients with diabetic and obstructive pulmonary disease (COPD) reflect a higher risk for severity of COVID-19 and increased morbidity and mortality rates due to muscle atrophy and a decrease in lung reserve [3]. Fever and headache are the most common symptom that appeared in 90% of infected cases; cough is present in two-thirds of patients, anosmia (70.2%), nasal obstruction (67.8%), and dysgeusia (alteration of the sense of taste) are looked in (49.8%) of cases [4].

Several pandemic studies indicated that the vital host of COVID is Raccoon dogs, while palm civets and bats of the genus Rhinolophus as secondary hosts [5]. The modes of transmission for COVID-19 involve the respiratory transmission by spreading of droplets carrying the virus via talking and coughing; typically, SARS-CoV-2 can survive 2–9 days on the surfaces of metal and wood; in addition, bio-aerosols involve small droplet virus nuclei [6]. The molecular structure of SAR-COV2 is characterized by structural proteins and enzymes; each type enhances a specific function: (A) Structural proteins: include spike glycoprotein (S) proteins marking all coronaviruses and binding to its receptors on the host cell membrane and enhancing penetration, while membrane protein, envelop E protein, and nucleocapsid (N) protein are comprised "matrix protein" to protect the genetic information of the virus. Also, Hemagglutinin-esterase (HE) is a dimer protein that binds to sialic acids on surface glycoproteins to facilitate virus spread through the mucosa [7]. (B) Enzyme protease; or called non-structural proteins such as 3-chymotrypsin-like protease, papain-like protease, and RNA-dependent RNA polymerase, help the virus to replicate [8].

The main pathogenesis of COVID-19 is related to its molecular structure-induced inflammatory response, primarily the binding of S-glycoprotein and ACE2 receptor on the surface of lower respiratory tract cells is the first step for virus entry. The angiotensin-converting enzyme is an enzyme located mainly in the endothelial capillaries of the lungs and kidney epithelial cells; it converts the inactive decapeptide angiotensin-I into the octapeptide angiotensin-II; those have vasodilator, anti-proliferative and protective functions by activating the MAS/G receptor (G-protein-coupled receptor) through which Angiotensin (1-7) signal [9, 10].

Furthermore, angiotensin-converting enzyme2 (ACE2) is a monocarboxypeptidase membrane enzyme located on ciliated cells of upper airway epithelia and type 2 pneumocytes of alveoli. ACE2 counters the activities of the related angiotensin-converting enzyme (ACE) by reducing the amount of angiotensin-II and increasing angiotensin-I; ACE2 activation prevents the deleterious effects of angiotensin II on the cells and organisms, such as cell death, fibrosis, angiogenesis, and thrombosis [11]. In addition, ACE2 acts as a ligand through its identified MAS1 receptor, a G-protein–coupled receptor. It serves as the main entry point into cells for SARS-CoV-2. [12]. When viral particles reach pulmonary cells, it binds with their membrane ACE2 receptors and decreases ACE2 activity; this binding contributes to an elevated level of ANG-ll to damaging tissues of the lungs [13].

When SARS-CoV-2 particles invade the respiratory mucosa, they start inducing a sequence of immune reactions (depending on the severity of covid-19), an elevation of the level of pro-inflammatory cytokines (IL)1-β, IL2, IL4, IL7, IL8, IL9, IL10, IL12, IL13, IL17, hematopoietic growth factor (HGF), interferon-gamma (IFNγ), tumor necrosis factor-alpha (TNFα), macrophage inflammatory proteins (MIP1α and MIP1β), granulocyte colony-stimulating factor (GCSF), granulocyte-macrophage colony-stimulating factor (GMCSF), monocyte chemotactic protein 1 (MCP1) have been observed in the blood of COVID-19 patients; the viral material are now ready to attack the adjacent epithelial cells and acts as infectious products on-going for community transmission via respiratory droplets [14].

Coronavirus has a high negative impact on the respiratory system**;** in some critical cases, it can be associated with acute respiratory distress syndrome (ARDS) and acute renal failure; the mortality rate increases with age over 80 due to severe pneumonia fever [15]. Many epidemic studies reported different comorbidities such as smokers, persons with organs transplantation, people with uncontrolled diabetics, hypertension, pulmonary, hepatic, and kidney disease. Those with a tumor on chemotherapy are associated with a high risk of developing severe illness and death [16]**.** Concerning the blood pressure BP; some experts believe that uncontrolled BP results in chronic inflammation throughout the body, damages blood vessels, dysregulation of the immune system, and may be associated with an up to 2.5-fold higher risk of severe and fatal COVID-19, especially among older people [16].

The reports from hospitalized patients in Wuhan, China, demonstrate a coronavirus can produce deep vein thrombosis, heart attack and myocardial injury through the increased levels of interleukin_6 (IL_6), Lactate dehydrogenase (LDH), ferritin, and D-dimer;

also an echocardiographic disorder occurs in 7% of patients and about 22% in patients who need intensive therapy unit. [17, 18]. A subset analysis of hospitalized patients with sickle cell anemia confirmed SARS- CoV-2 showed that most deaths occur in those aged more than 50 years, especially those who have progressive renal insufficiency, hypertension, and chronic lung disease [19, 20].

Moreover, COVID-19 is particularly life-threatening in patients with leukemia, lymphoma myeloma, and allogeneic hematopoietic stem cell transplantation. The reports showed those patients had a higher risk of intensive care unit (ICU) admission, invasive ventilation, or mortality rate reach to 32% to 40% compared to patients without malignancy; the severity due to their humeral and cellular immunosuppression status resulted mainly from the chemotherapies, radiotherapy, or immunosuppressive drugs; besides, the coronavirus infection can induce both innate and adaptive immune response and allow excessive infiltration of many immune cells such as T cells and macrophages resulted in the liberation of an excessive amount of pro-inflammatory cytokines IL-1B and IL-6, also fast exhaustion of the NK cells and cytotoxic T cells and may lead to the fast progression of the viral infection and devastation of the immune system [21].

Regarding comorbidities, a correlation between hyperglycemia/diabetic Mellitus and COVID-19 infection has been proposed, reinforced by the notion that impaired immunity and inflammatory state are associated with both conditions. A previous clinical study in Italy illustrated that the patients with DM have an increased risk of requiring rigorous care unit, invasive mechanical ventilation, or mortality after the invasion of SARS-CoV-2 due to compromised innate immune responses to infection, higher levels of inflammatory mediators such as interleukin-6, C-reactive protein and rapid deterioration in patient condition. It has been demonstrated that two-thirds of patients requiring hospitalization and later died from novel coronavirus infection had a past medical history of diabetes mellitus [22].

Consequently, severe COVID-19 infection may induce impaired kidney function, which is represented by acute tubular necrosis associated with elevated serum creatinine, proteinuria, and hematuria in patients with chronic renal syndrome. The renal tubular cells are more likely to be attacked and damaged by SARS-CoV-2 because they are rich in ACE2 receptors. In addition, the kidney is comprised of a network of capillaries and small arteries which are venerable to damage by this novel virus as the whole volume of blood runs through the kidneys multiple times in one day, exposing the kidney to inflammatory cytokines circulating in the blood which may injure the renal blood vessels. Autopsy specimens showed that there might be a direct cytopathic consequence for SARS-CoV-2 invasion on renal cells. Data collected from transmission electron microscopy of renal tissue after 12-hr of the autopsy showed small particles with the typical appearance of coronavirus bodies in kidney podocytes; consequently, the injured glomerular epithelium can be detected in the acute stage and diabetes. Also, the impaired capillary function in the glomeruli, leakage of protein and RBCs in urine, inappropriate coagulation responses, and worsening of edema can be envisioned [23]. The present study is designed to examine the spectrum of symptoms associated with COVID-19 infection, the duration of disease, and the severity grades, then links the variables with patient demographic data and the presence of comorbidities.

Instrument and Methods

A cross-sectional study involved 202 patients infected with coronavirus 19 and stayed in the Al-Shefaa center at Al-Hakeem hospital in Al-Najaf city. The study extended from 6 September 2020 to 20 January 2021. The verbal consent was taken from all included patients after understanding the idea of such research work for the health and the society. The data were obtained from 160 male and 42 female infected patients recruited into Al-Shefaa center using a pretested self-report questionnaire prepared for this research and divided into three sections according to the information taken from the patients themselves or their case reports in the emergency department. The self-description questionnaire contained:

Section one (demographic features): It included name, age, gender, address, job, and body mass index (BMI).

Section two: It included the history and duration of co-morbidities and related diseases such as any chronic disease, respiratory, immune, and metabolic and blood disorders. Section three: It involved the clinical severity for covid19, which comprised duration and severity of infection, clinical symptoms such as fever, cough, insomnia, thrombosis, breathing difficulties, fatigue, need for supplemental oxygen.

The data was collected anonymously and processed using IBM SPSS 25 software. The descriptive statistics were performed to analyze the results expressed as a percentage and frequency presented in tables as appropriate.

Findings

A total of 202 patients with COVID-19 had been concerned in this cross-sectional study; they were 160 males and 42 females.

The results showed 121 (59.9%) patients with a BMI of <18. About 77.2% of infected people do not have comorbidities; about 54% with moderate illness, and about 0.5% person had dyspnoea and headache. There were 85 admitted patients for prolonged 2

weeks (Table 1).

Table 1) Results of patients characteristics

Variables	Number	Percent
BMI (kg/m²)		
< 18	121	59.9
18.5-24.9	38	18.8
25-29.9	21	10.4
30-34.9	16	7.9
35-39.9	6	3.0
Comorbidities		
Without comorbidities	156	77.2
Hypertension	9	4.5
Joints Diseases	2	1.0
Diabetic Mellitus	6	3.0
Polycystic ovary syndrome	1	0.5
Respiratory Diseases	7	3.5
Immunity Diseases	2	1.0
Cancer	1	0.5
Atherosclerosis	6	3
Thyroid Diseases	1	0.5
Hematologic Diseases	3	1.5
GIT Diseases	2	1
other chronic Diseases	6	3
Severity of disease		
Mild	88	43.6
Moderate	109	54
Sever	5	2.5
Total	202	100
Symptoms during infection		
Anosmia	6	3
Cough	7	3.5
Dyspnoea	1	0.5
Fatigue	2	1
Hair Fall	105	52
Headache	1	0.5
Myalgia	17	8.4
Fever	61	30.2
Duration of disease (days)		
3-4	31	15.3
7	50	24.8
14	85	42.1
21	30	14.9
28	6	3.0
Supplemental an artificial oxygen		
No	183	90
Yes	19	10

Discussion

The results of our cross-sectional study showed the variation in body mass index BMI among 202 patients admitted to the Al-shefaa center, and about 10% of them were obese. An analogous investigation study evaluated the association between BMI and suspected ability to infect with COVID-19 and observed that patients aged<60 years with BMI≥35 are (2.2-3.6) times more admitted to intensive care unit compared with patients of the same age who had BMI≤30 and it considers the obesity as an epidemiologic risk factor that contributes increasing morbidity rate in United states and may aggravate pathogenesis of diabetes and hypertension. However, an estimate of the association between obesity and severity of infection should be deduced with caution as it is unclear whether these were based on individual responses to infection and may contribute to age, gender, ethnicity, and the existence of co-morbidities [24].

According to a report study from the United State, it was found that at least approximately 68% of the hospitalized patients possessed at least one comorbidity, among which obesity represents (48.3%) of them [25]. In a meta-analysis, 6916 patients who tested positive for COVID-19 were involved. The percentage of patients requiring intensive care unit (ICU) admission or receiving invasive mechanical ventilation (IMV) was more than 60%. The analysis showed a strong association between the mortality rate and obesity in younger men. However, in another study conducted in Paris involving in hospitalized patients with COVID-19, it was found that obesity and mortality were similarly associated across the different age groups [26, 27].

Our cross-sectional study showed different comorbidities among 202 patients; about 9 (4.5%) have hypertension, (3.5%) with respiratory diseases, 6 (3%) have diabetes, atherosclerosis, or chronic diseases, and about (1.5%) have hematological disorders. The present study is consistent with several correspondent studies that found persons testing positive for covid19 had at least one comorbidity: hypertension, respiratory diseases, diabetes; or these associated with increased risk of severe illness and mortality rate of hospitalized patients. A parallel meta-analysis study showed hypertension was present in 10-20% of infected patients; the mechanism by which hypertension leads to increased risk of COVID-19 infection is uncovered yet, and it may be multiplexed with other underlying comorbidities. For instance, hypertension complicated by myocardial injury worsen the prognosis of COVID-19 disease since cardiovascular accidents or end-organ damage are usually correlated with low-grade control of elevated blood pressure. In contrast, the mean blood pressure increases with age [28].

Almost two-thirds of infected people over 60 have high blood pressure, therefore, an older age, poorly controlled blood pressure and cardiovascular events can explain the observed relationship between age, hypertension and severity of COVID-19 symptoms. The reduced level of immunity and decreased ability to fight foreign invaders may contribute to this situation. Alternately, using ACE inhibitors and angiotensin receptor blockers (ARBs) as a treatment for hypertension possibly elevates the levels of angiotensin-converting enzyme-2 [28].

The relationship between COVID-19 infection and diabetes mellitus has been addressed in several publications. They showed that the severity of COVID-19 disease and the mortality rate are independently associated with pre-existing diabetic conditions or even with a certain degree of hyperglycemia. Nevertheless, the complications of diabetes mellitus (chronic kidney disease and heart failure) elevate COVID-19 mortality. On the other side, in human monocyte, studies have shown that SARS-CoV-2 replication increases directly in high

glucose environment, and SARS-CoV-2 infection induces the switch to glycolysis as a result of enhanced production of mitochondrial reactive oxygen species and activation of hypoxia-inducible factor 1α, dysregulation of the immune response, glycemic deterioration. Moreover, insulin requests are associated with increased secretion of inflammatory cytokines, especially IL-6, via promoting oxidative stress. IL-6 can alter DNA, proteins and lipids of structural cells, and these changes might lead to faster deteriorations in COVID-19 patients suffering from diabetes mellitus [29]. A study demonstrated the biological evaluation of immunity in diabetic patients with severe COVID-19 which observed enhanced levels of bacterial DNA and lipopolysaccharide as a bacterial by-product of lung origin in elevating the production of IL-6 as a biomarker of pulmonary injury. IL6 causes increased affinity cellular binding to the virus, diminished T-cell function; increased susceptibility to intense inflammation and cytokine storm in severe COVID-19 [22]. Data obtained from an epidemiological study involving patients with COVID-19 in China reported that the prevalence of concurrent illnesses such as chronic respiratory disease and chronic obstructive pulmonary disease (COPD) was 1.4% and 2.4%, respectively. Different bacterial species, such as *Pseudomonas aeruginosa* and *Staphylococcus aureus*; and other respiratory viruses influence the mortality rate caused by chronic pulmonary diseases via triggering disease aggravations. Additionally, in vitro studies from SARS-CoV, clinical observations and radiological investigations indicated that the ciliated airway epithelium might serve as a primary site for viral inertance and express inflammatory mediators and interferons. On the other hand, microscopic findings showed diffuse alveolar damage accompanied by hyaline membrane formation, inflammation, and activation of lymphocytes and pneumocytes, microvascular thrombi with proteinaceous edema. These findings may somewhat explain why patients with chronic obstructive pulmonary disease seem more susceptible to death from severe COVID-19 infection [30].

Several studies indicated that inflammation is one of the atherogenic constituents developed in vascular injury and lipid metabolism disturbance. The vascular endothelial cells are critical in regulating inflammation and blood clotting factors. Likewise, coronavirus replication causes the damage of endothelial cells and accumulation of inflammatory cells, deposition and oxidation of low-density lipoprotein (LDL), which lead to endothelitis. Moreover, it stimulates releasing cytokines and chemokines, resulting in coagulative abnormalities accompanied by elevated fibrinogen, antithrombin, activated partial thromboplastin time (aPTT), and increased prothrombin (PT) and D-dimers. Lipid oxidation causes upregulation of vascular cell adhesion molecule-1 (VCAM-1), formation of foam cells and infiltration by CD4 T cells and resulting is plaque building which is at high incidence of thrombosis in lungs, prostate and kidneys [31]. Furthermore, our results showed 88(43.6%) patients with mild illness, 109 (54%) moderate and 5 (2.5%) of patients were severe cases. According to primary data from a study in China, 81% of people with Covid-19 had mild or moderate illness (including people without and mild pneumonia), 14% had severe, and 5% had a critical illness. However, some patients who have mild symptoms initially will subsequently have precipitous clinical deterioration that occurs approximately 1 week after symptoms onset. Also, the data suggests that people who use immunosuppressive therapy, pregnancy, smoking, liver disease, pulmonary fibrosis, and thalassemia may have a higher risk of serious illness [32].

Moreover, our results indicated most prevalent symptoms at 2–14 days after exposure to the coronavirus 2019 were hair fall that appeared in 105 (52%) patients during infection. Recent studies made in Wuhan, China also showed about 48.5% of patients developed hair loss after SARS-CoV-2 infection due to acute illness, fever, weight loss, associated anxiety, sudden hormonal changes, and persistent post-COVID-19 inflammatory reactions, physical and emotional stress can force more hairs into the shedding phase [33]. In addition, myalgia appears in 17 (8.4%) of patients. An analogous study in China exposed that myalgia occurs in 16.9% of patients; Myalgia occurs through covid19 infection and is usually mediated by up-regulated interleukin-6. Assessment of Creatine kinase (CK) and lactate dehydrogenase (LDH) levels can determine skeletal muscle injury. Also, it occurs when cytokines evoke the production of prostaglandin E2 which stimulates pain through peripheral pain receptors; Moreover, SARS-CoV-2 can invade the nervous system by engaging with ACE2 to cause skeletal muscle damage [34]. Our study showed that 1% of patients have fatigue; a comparative study reported it in 28.16% of patients. It may be related to an increase in viral load and immune response to the infectious process. In addition, insufficient energy production to meet the required metabolic demands relates fatigue to other symptoms [35] The present study found that fever occurred in 30% of patients, other study showed intermittent fever was observed among 58.66% patients, which signals the organism's response to viral particles that affect temperature-regulating center, it may be due to the proportion of lymphocytes [36]. Also, headaches and dyspnoea were seen in 0.5% of patients. A study in Australia showed dyspnea seen in 30.82% and 36% reported headaches of infected patients; both are generally related to greater severity of covid19. Also, tumor necrosis factor, interleukin 2 released by immune cells in response to viral infections may

cause headache; in addition, when SARS-CoV-2 invades lung tissue, it may cause alveolar gas exchange disorders, leading to hypoxia in the brain, increasing the anaerobic metabolism of mitochondria in brain cell and accumulation of acid metabolites. It will obstruct cerebral blood flow, swelling of brain cells, cerebrovascular dilatation, and headache due to ischemia and congestion [37].

The disease duration or persistent symptoms were reported in 15% of patients for 4 days, and 24% for a week. In another study that involved over 2500 close contacts of 100 individuals infected with COVID-19 in Taiwan, all the 22 secondary cases had their first exposure within six days of symptom appearance. They showed that specimens collected from the upper respiratory tract had a higher level of viral RNA due to viral invasion of the alveolar epithelium cell accompanied by infiltration of immune cells [38]. Our study found that 42% of patients with symptoms onset for two weeks. Related study of academic publications has estimated the illness in 10–20% of patients. Additionally, isolation of infectious virus from upper respiratory specimens more than 10 days after illness onset has only rarely been documented in patients who had nonsevere infection [39]. Furthermore, 14.9% of symptoms persist for 3 weeks is similar to our result that the symptoms are estimated after 3 weeks is 14.9 %. In a recent review of 28 published studies, the pooled median duration of viral RNA detection in respiratory specimens was 18 days following the onset of symptoms. If viral RNA is still detectable in upper respiratory specimens, the RNA concentrations are generally at or below the levels at which replication-competent virus can be reliably isolated [40].

Oxygen therapy is reserved for the most severe and critical COVID-19 cases, our study showed that 90.6% of patients do not require O2 therapy (90.6%), While 19 Patients from the total number require O2 therapy (9.4%). A recent study showed the needed care could be approximated to meet the oxygen therapy demand for patients suffering from dyspnea, hypoxia or respiratory failure in severe and critical hospitalized cases, accounting for 20% of cases. About 75% of the COVID-19 cases requiring hospital admission will be reported as “severe”, and 25% as “critical”. Thus, the overall delivery of medical oxygen can be measured according to the appropriate flow rates for each patient category of severity [41].

Conclusion

Obesity may play a role in the pathogenesis of COVID-19 disease and recorded more than 10% of the patients are overweight. Moreover, patients who have coexisted diseases such as hypertension, diabetes, respiratory tract disorders and other chronic diseases represent more than 40% of infected people which might contribute to the poorer prognosis of COVID-19 victims. The degree of illness ranged from mild, moderate, to severe, whereas the disease duration among the affected individuals varies from a few days to four weeks. Whereas few patients required artificial oxygen supplementation. Finally, a range of symptoms affected the infected people including cough, anosmia, fever, and hair fall.

Acknowledgments: None declared by the authors.
Ethical Permissions: None declared by the authors.
Conflicts of Interests: The authors declare no conflicts of interests.
Authors’ Contribution: Ghafil NY (First Author), Introduction Writer/Main Researcher (40%); Jawad Kadhim Z (Second Author), Methodologist/Statistical Analyst (15%); Dananah FM (Third Author), Discussion Writer/Methodologist (15%); Abdiljaleel Alkaabi YS (Fourth Author), Discussion Writer (15%); Hassoun YL (Fifth Author), Introduction Writer/Assistant Researcher (15%)
Funding/Support: None declared.

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