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# Evaluation of Clinical Outcomes in Patients with COVID-19

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ORIGINAL ARTICLE

# Evaluation of Clinical Outcomes in Patients with COVID-19☆

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**Abstract**

*Background*: **COVID-19 clinical manifestations are diverse, ranging from asymptomatic and moderate symptoms to severe diseases. It is difficult to anticipate the clinical course or identify people at risk of worsening.**

*Aim of the work*: **To estimate the prevalence of morbidity and mortality in COVID-19-infected patients and to evaluate risk factors associated with the severity of COVID-19 infection.**

*Patients and methods*: **In 200 patients with COVID-19, 100 patients had comorbid disease (cardiac, renal, or hepatic), and 100 age and sex-matched patients had no comorbid disease. Detailed history, clinical examination, radiological investigations, and evaluation of illness severity were recorded for all patients.**

*Results*: **Non-survivors had significant increase in hypertension (HTN), chronic obstructive pulmonary disease, chronic kidney disease (CKD), and stroke in comparison to survivors. Dead cases had significant increase in respiratory, cardiovascular, neurological, renal, hematological, endocrinal, and dermatological complications in comparison to alive cases. Dead cases had significant increase in ICU admission, higher incidence of need for more advanced modes of mechanical ventilation (MV), higher incidence of severe and critical hypoxia, and higher incidence of need for MV.**

*Conclusion*: **COVID-19-infected patients of old age, associated comorbidities, and specific laboratory and imaging criteria were found to be associated with death. Moreover, higher incidences of respiratory, cardiovascular, neurological, renal, hematological, endocrinal, and dermatological complications as well as severe and critical hypoxia and ICU admission and MV were associated with death among Covid-19 patients.**

*Keywords:* Clinical outcomes, COVID-19, Morbidity, Mortality

## 1. Introduction

A cluster of individuals with pneumonia of unknown cause was identified in December 2019 in Wuhan, Hubei, China.[1] Consequently, a new coronavirus (SARS-CoV-2) was discovered from samples of afflicted individuals' lower respiratory tracts.[2]

Clinical manifestations of COVID-19 are diverse, ranging from asymptomatic and moderate symptoms of the upper respiratory tract to severe diseases with multiorgan failure and death. It is difficult to anticipate the clinical course or identify people at risk of worsening.[3]

Substantial disparities in clinical and demographic characteristics of COVID-19 individuals in different locations of the world have been identified.[4] Individuals with comorbidities are susceptible to COVID-19 pneumonia. In addition, biomarkers in the blood of COVID- 19 individuals with varying disease severity vary considerably.[5]

In elderly individuals infected with the COVID-19 virus, high risks of morbidity and death have been frequently documented, a result that has been consistent across different nations. It is believed that medical interventions, especially transplant, and chronic illnesses such as HTN, obesity, diabetes

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mellitus (DM), cardiovascular disease, pulmonary disease, and other chronic, noncommunicable diseases worsen outcomes in COVID-19 patients, hence increasing death rates.[6]

The goal of the research was to determine the prevalence of mortality and morbidity among COVID-positive cases and to assess the risk factors linked with the severity of COVID infection.

## 2. Patients and methods

This retrospective and prospective observational study was carried out in Al-Azhar University Assiut specialized hospital and other quarantine hospitals during the study period from November 2021 to May 2022.

### 2.1. Patients

In total, 200 cases who were diagnosed as COVID-19-infected cases were subjected to this study. Diagnosis was reached by using real-time reverse transcript-ion polymerase chain reaction (rRT-PCR) on respiratory tract swab samples.

**Inclusion criteria were** laboratory confirmation of SARS-CoV-2 (rRT-PCR) analysis of nasal and pharyngeal swab specimens which denoted a positive result[1]; clinical and computerized tomography results were gathered from medical records on admission and from all ages.

**Exclusion criteria were** missing clinical or CT records on admission.

### 2.2. Methods

All patients were subjected to complete history taking including demographic and clinical data, full clinical examination including assessment of general condition and vital signs (pulse, blood pressure, respiratory rate, and temperature). Abdominal, chest, and heart examination were assessed. Height and weight were measured, and body mass index (BMI) was calculated.

Laboratory Investigations included complete blood picture (CBC), hemoglobin concentration (Hb %), red blood cells (RBCs), white blood cells (WBCs), neutrophil, lymphocyte, and platelet counts, fasting blood glucose level, random blood glucose and HbA1c. The diagnosis of DM was diagnosed when the fasting blood glucose is $^{3}$126 mg/dl and/or glycosylated hemoglobin is >6.5 % or by the use of hypoglycemic agents or by self-reported history of diabetes. Kidney profile including serum urea and creatinine, liver profile such as alanine aminotransferase (ALT), aspartate aminotransferase (AST), serum total bilirubin, and serum albumin, CRP, ferritin, and D dimer were performed.

Radiological investigations in the form of chest radiograph and chest CT were reported by a radiologist for all cases. On CT chest, each of the five pulmonary lobes was visually rated on a scale of 0–5 as follows: 0, no engagement; 1, <5 % involvement; 2, 25 % involvement; 3, 26–49 % involvement; 4, 50–75 % involvement; and 5, >75 % involvement.[7] Evaluation of illness severity: The degree of disease on admission was assessed in accordance with the WHO—China Joint Mission on SARS-CoV-2 report (WHO, 2020). Mild (confirmed with rRT-PCR, without clinical symptoms), moderate (laboratory confirmed, with pneumonia), severe (oxygen saturation $\leq$93 % at rest; respiratory distress with a respiratory rate $^{3}$30 breaths/min and/or pulmonary infiltration >50 % of the pulmonary parenchyma within 24–48 h), and critical (respiratory failure requiring mechanical ventilation (MV), shock, or other organ failure that requires ICU).

All patients received medical treatment according to protocols of the Ministry of Health and Population, Egypt.

The study was approved by the local Ethics Committee, Faculty of Medicine Al-Azhar Assiut University. An informed written consent was obtained from clinically stable cases or from the responsible relatives of critically ill cases.

### 2.3. Statistical analysis

We analyzed the data using the Statistical Package for the Social Sciences (SPSS), version 24 for Windows. If numerical data was regularly distributed, it was described using means and standard deviations. For verifying the normality of the distribution of numerical variables, the Kolmogorov–Smirnov test was applied. The Chi-square test was used to examine the relationship between categorical variables. In the event of a breach of assumptions, Fisher's exact test was used. Independent sample *t*-test was performed to examine the difference between two groups regarding numerical parameters. A $P$ value of less than 0.05 was statistically significant.

## 3. Results

We found that regarding sociodemographic data, dead cases had significantly older age (64.77 ± 12.43 vs 57.46 ± 13.69 years; $P < 0.001$) and had higher body mass index (BMI) (30.9 ± 10.94 vs 26.37 ± 7.09 kg/m$^2$; $P = 0.004$) in comparison to alive cases. However, there was no significant difference

*Table 1. Difference between both groups concerning sociodemographic characteristics. F, Fisher's exact test.*

| | Discharged ($n = 138$) | Died ($n = 62$) | $P$ value |
|---|---|---|---|
| Age (years) | 57.46 ± 13.69 | 64.77 ± 12.43 | <0.001 |
| Sex | | | |
| Male | 64 (46.4) | 28 (45.2) | 0.880 |
| Female | 74 (53.6) | 34 (54.8) | |
| BMI (kg/m$^2$) | 26.37 ± 7.09 | 30.9 ± 10.94 | 0.004 |
| Residence | | | |
| Rural | 74 (53.6) | 40 (64.5) | 0.167 |
| Urban | 64 (46.4) | 22 (35.5) | |
| Smoking | 67 (48.9) | 43 (70.5) | 0.005 |
| Cough | 130 (94.2) | 56 (90.3) | 0.372 F |
| Dyspnea | 136 (98.6) | 60 (96.8) | 0.589 F |
| Sore throat | 40 (29) | 18 (30) | 1.00 |
| Fever | 98 (71) | 50 (80.6) | 0.167 |
| Diarrhea | 34 (24.6) | 12 (19.4) | 0.471 |

between both groups regarding Sex, residence, smoking, or symptoms (Table 1).

Laboratory data revealed that dead cases had significantly higher total leukocyte count ($P = 0.009$), and lower lymphocytic count ($P = 0.015$) in comparison to alive cases. Dead cases had significantly higher CRP levels, higher ESR levels, higher ferritin, higher total bilirubin levels, higher serum urea and creatinine levels and higher HbA1C and fasting blood sugar levels in comparison to alive cases ($P < 0.001$ for all). (Table 2).

Regarding radiological findings, dead cases had significantly higher incidences of bilateral severe opacity on Radiograph ($P < 0.001$) and significant increase in bilateral ground glass opacity ($P = 0.005$) and crazy paving on CT scan ($P < 0.001$). Moreover,

*Table 2. Difference between study groups concerning laboratory finding outcomes. Independent sample t-test.*

| | Discharged ($n = 138$) | Died ($n = 62$) | $P$ value |
|---|---|---|---|
| Hb (mg/dl) | 12.13 ± 1.99 | 12.11 ± 2.52 | 0.935 |
| WBCs (*10$^3$ cmm) | 9.56 ± 5.21 | 11.77 ± 6.08 | 0.009 |
| Neutrophil count (*10$^3$ cm m) | 63.96 ± 19.45 | 66.05 ± 20.52 | 0.489 |
| Lymphocyte count (*10$^3$ cm m) | 16.77 ± 8.99 | 13.59 ± 7.22 | 0.015 |
| Platelet count (*10$^3$ cmm) | 240.19 ± 105.29 | 211.13 ± 103.89 | 0.072 |
| CRP (mg/dl) | 23.17 ± 23.98 | 81.82 ± 46.15 | <0.001 |
| ESR (mg/dl) | 39.96 ± 23.64 | 78 ± 29.2 | <0.001 |
| Ferritin | 386.59 ± 329.66 | 915.85 ± 587.01 | <0.001 |
| D dimer | 3.81 ± 1.68 | 3.73 ± 2.51 | 0.968 |
| ALT | 49.13 ± 15.04 | 53.42 ± 17.18 | 0.787 |
| AST | 40.61 ± 5.84 | 49.68 ± 42.03 | 0.221 |
| Total bilirubin | 0.91 ± 0.32 | 1.54 ± 1.18 | <0.001 |
| Albumin | 3.18 ± 0.53 | 3.14 ± 0.52 | 0.678 |
| Creatinine | 1.29 ± 0.59 | 1.95 ± 1.31 | <0.001 |
| Urea | 47.89 ± 29.91 | 65.03 ± 23.09 | <0.001 |
| HbA1C | 6.52 ± 1.94 | 8.25 ± 3.03 | <0.001 |
| FBS | 119.07 ± 49.31 | 167.48 ± 82.27 | <0.001 |

*Table 3. Radiological findings and patient outcomes. F, Fisher's exact test.*

| | Discharged ($n = 138$) | Died ($n = 62$) | $P$ value |
|---|---|---|---|
| **Radiograph findings** | | | |
| Normal | 16 (11.6) | 0 | |
| Bilateral mild opacity | 80 (57.97) | 6 (9.7) | |
| Bilateral moderate opacity | 42 (30.43) | 24 (38.7) | <0.001 F |
| Bilateral severe opacity | 0 | 32 (51.6) | |
| **CT findings** | | | |
| Bilateral GGO | 100 (72.5) | 56 (90.3) | 0.005 |
| Grazy paving | 28 (20.3) | 52 (83.9) | <0.001 |
| Consolidation | 50 (36.2) | 24 (38.7) | 0.753 |
| Cardiomegaly | 0 | 2 (3.2) | 0.095 |
| Bilateral effusion | 0 | 2 (3.2) | 0.095 |
| **CT score** | | | |
| 1 | 38 (27.5) | 0 | |
| 2 | 62 (44.9) | 2 (3.2) | |
| 3 | 36 (26.1) | 16 (25.8) | <0.001 |
| 4 | 2 (1.4) | 16 (25.8) | |
| 5 | 0 | 28 (45.2) | |
| **Duration of treatment** | 10.36 ± 4.49 | 11.19 ± 4.52 | 0.229 |

dead cases had significantly higher CT scores in comparison to alive cases ($P < 0.001$). (Table 3).

We found that dead cases had significant increase in HTN ($P = 0.001$), chronic obstructive pulmonary disease (COPD) ($P = 0.02$), CKD ($P = 0.037$), and stroke ($P < 0.001$) in comparison to alive cases (Table 4).

Dead cases had significant increase in respiratory ($P = 0.001$), cardiovascular ($P < 0.001$), neurological ($P = 0.004$), renal ($P < 0.001$), hematological ($P = 0.001$), endocrinal ($P = 0.009$), and dermatological complications ($P < 0.001$) in comparison to alive cases (Table 5).

Dead cases had significant increase in ICU admission ($P < 0.001$) and higher incidence of need for more advanced modes of mechanical ventilation (volume control and SIMV modes) ($P < 0.001$). (Table 6).

Regarding severity outcomes, we found that non-alive cases had significant increase in severe and critical hypoxia ($P < 0.001$) and higher incidence of

*Table 4. Association between associated comorbidities and patients' outcomes. C; Chi square test. F; Fisher's exact test.*

| Comorbidities | Discharged ($n = 138$) | Died ($n = 62$) | $P$ value |
|---|---|---|---|
| HTN | 40 (29) | 34 (54.8) | 0.001C |
| DM | 46 (33.3) | 26 (41.9) | 0.267C |
| COPD | 20 (14.5) | 18 (29) | 0.02C |
| CKD | 6 (4.3) | 8 (12.9) | 0.037 F |
| Bronchial asthma | 12 (8.7) | 4 (6.5) | 0.780 F |
| Bronchiectasis | 0 | 2 (3.2) | 0.095 F |
| Chronic liver disease | 2 (1.4) | 4 (6.4) | 0.589 F |
| IHD | 12 (8.7) | 8 (12.9) | 0.445C |
| HF | 2 (1.4) | 2 (3.2) | 0.589 F |
| AF | 2 (1.4) | 2 (3.2) | 0.589 F |
| Stroke | 0 | 8 (12.9) | <0.001 F |

*Table 5. Association between overall complications and patients' outcomes. F; Fisher's exact test.*

| | Discharged ($n$ = 138) | Died ($n$ = 62) | $P$ value |
|---|---|---|---|
| Respiratory complications: | 4 (2.8) | 44 (69) | <0.001 F |
| CVS complications | 18 (13) | 34 (54.9) | <0.001 F |
| Neurological complications | 6 (4.3) | 10 (16.2) | 0.004 F |
| Renal complications | 18 (13) | 26 (41.9) | <0.001 |
| GIT complications | 14 (10.1) | 10 (16.1) | 0.245 |
| Hematological complications | 8 (5.7) | 10 (18) | 0.001 F |
| Musculoskeletal | 6 (4.3) | 6 (9.7) | 0.196 F |
| Endocrinal complications | 2 (1.4) | 4 (6.5) | 0.009 F |
| Dermatological complications | 0 | 26 (41.9) | <0.001 F |

*Table 6. Outcomes of ICU characteristics and main mood of mechanical ventilation among patients admitted to ICU. F; Fisher's exact test.*

| Variable | Discharged ($n$ = 138) | Died ($n$ = 62) | $P$ value |
|---|---|---|---|
| **Admission to ICU** | 54 (39.1) | 62 (100) | <0.001 |
| **Mood of mechanical ventilation** | | | |
| AC | 2 (1.4) | 8 (12.9) | |
| CPAP + PS | 0 | 4 (6.5) | |
| NIV-PC | 6 (4.3) | 6 (9.7) | |
| NIV-PS | 0 | 2 (3.2) | |
| SIMV | 2 (1.4) | 26 (41.9) | <0.001 F |
| VC | 2 (1.4) | 14 (20.8) | |

need for mechanical ventilation ($P$ < 0.001) in comparison to alive cases (Table 7).

## 4. Discussion

According to their outcome, cases in this research were classified as alive cases or dead cases, with 62 (31 %) cases who died (non-survivors) and 138 (67 %) cases who were released (alive cases). Similar results were observed by Zhou et al.[3] (28.3 %). However, Guan et al.[5] determined that SARS-CoV-2 mortality was 1.4 %. In the research conducted by Huyut et al.,[8] the death rate among treated cases (n = 4597) was 5.07 % (233/4597). Different sample sizes and case inclusion criteria may have contributed to these disparate mortality outcomes.

*Table 7. Outcomes of severity and oxygen support among included patients. F; Fisher's exact test.*

| Variable | Discharged ($n$ = 138) | Died ($n$ = 62) | $P$ value |
|---|---|---|---|
| **Severity** | | | |
| Mild | 22 (15.9) | 0 | |
| Moderate | 12 (8.7) | 0 | |
| Severe | 92 (66.7) | 2 (3.2) | <0.001 |
| Critical | 12 (8.7) | 60 (96.8) | |
| **Oxygen support** | | | |
| NIV mechanical ventilation | 8 (5.8) | 12 (19.4) | |
| Invasive mechanical ventilation | 4 (2.9) | 48 (77.4) | |
| Nasal prong | 14 (10.1) | 0 | <0.001 |
| Venturi mask | 92 (66.7) | 2 (3.2) | |
| No | 22 (11) | 0 | |

Dead cases were substantially older (64.77 12.43 vs. 57.46 13.69 years; $P$ 0.001) in terms of sociodemographic data in the present study in comparison to alive cases (64.77 12.43 vs. 57.46 13.69 years; $P$ 0.001). Similarly, Salinas-Escudero et al.[9] showed that the risk of death during the follow-up period was considerably higher for older persons. Furthermore, Wei et al.[10] observed that being older than 50 years increases the likelihood of catastrophic illness complications. Moreover, according to data from 79,394 verified cases in a Chinese research by J. T. Wu et al.,[11] cases younger than 30 and older than 59 years were 0.6 and 5.1 times more likely to die following the onset of symptoms, respectively.

According to laboratory data from the current investigation, dead cases had a substantially higher total leukocyte count ($P$ = 0.009) than alive cases. In agreement with our findings, Wu et al.[11] and Yang et al.[12] found that leukocytosis was an independent predictor of in-hospital mortality.

A prolonged drop in the number of lymphocytes in the peripheral blood is an early sign of severe/critical illness in SARS-CoV-2 cases. There is an abundance of evidence describing lymphopenia in a substantial number of SARS-C0V-2 cases. Zhang et al.[13] Laboratory results from this investigation indicated that dead cases had a considerably lower lymphocyte count than alive cases ($P$ = 0.015). Similarly, Wu et al.[11] and Yang et al.[12] found that lymphopenia was an independent predictor of in-hospital mortality. The lower lymphocyte numbers may be due to viral attachment, immunological damage from inflammatory mediators, or lymphocyte exudation into inflammatory pulmonary tissues. Wang et al.[14]

Dead cases in the current research showed substantially higher CRP levels, ESR levels, and ferritin levels in comparison to alive cases ($P$ 0.001 for all). In agreement with our findings, Huyut et al.[8] found that CRP, ESR, and ferritin levels were greater in SARS-CoV-2 cases who died than in those who survived. In a separate investigation, Ardestani and Azizi[15] found that COVID cases who died had greater CRP levels than infected cases who were still alive. In addition, the study by Tang et al.[16] revealed that ferritin levels were elevated in SARS-CoV-2 cases who did not survive and were severely affected.

According to Sun et al.[17] and Wang et al.,[14] the rise in these values was caused by a secondary bacterial infection or an enhanced inflammatory response characterized as the cytokine storm resulting from SARS-CoV-2 infection.

This study found that dead cases had substantially higher total bilirubin levels than alive cases ($P = 0.001$). Similarly, Huyut et al.[8] discovered that bilirubin levels were greater in cases who did not survive than in those who did. In addition, Guan et al.[5] discovered that the amount of bilirubin was greater in severe instances of SARS-CoV-2 than in less severe cases.

The current investigation found that dead cases had considerably higher levels of serum urea and creatinine than alive cases ($P$ 0.001 for both). These data suggested that renal failure may have resulted from significant kidney involvement in COVID fatalities. In agreement, Huyut et al.[8] discovered that urea and creatinine levels were greater in cases who did not survive in comparison to those who did. In addition, Mertoglu et al.[18] discovered that severe COVID cases had greater blood levels of urea and creatinine than slightly infected individuals.

HTN and diabetes were the most prevalent comorbidities in the current research, occurring in 74 (37 %) and 72 (36 %) individuals, respectively. Likewise, Ou et al.[19] observed in their meta-analysis that diabetes is a prevalent comorbidity among COVID cases. The current investigation found that dead cases had substantially higher levels of HbA1C and fasting blood sugar than alive cases ($P$ 0.001 for both). Williamson et al.[20] observed that diabetic cases infected with SARS-CoV-2 who had high HbA1c levels before hospital admission had an increased risk of mortality.

Zhang et al.[21] found that cases with DM at admission had an increased risk of composite outcomes (ICU hospitalization, MV, and mortality). Moreover, Russell et al.[22] found that hyperglycemia during hospital therapy was a risk factor for mortality in cases with severe COVID.

The expression of ACE2, the entrance receptor of SARS-CoV-2, is elevated in the lungs and other organs of type 2 DM cases.[23] This increase is related to chronic inflammation, endothelial cell activation, and insulin resistance, all of which exacerbate the inflammatory response and contribute to failure of the alveolar–capillary barrier, according to Hayden.[24]

Dead cases in the current research exhibited a considerably greater incidence of bilateral ground-glass opacity ($P = 0.005$) and crazy paving on CT scan ($P$ 0.001) in comparison to alive cases. In addition, the CT scores of dead cases were substantially higher than those of alive cases ($P$ 0.001). Zhang et al.[13] showed a correlation between the number of damaged lobes on CT scans and the severity of illness. In addition, Chen et al.[25] and Huang et al.[1] showed that bilateral pulmonary involvement was more prevalent in cases with aberrant chest CT scans, exhibited mostly as numerous ground-glass opacities and subpleural lesions.

On the basis of these findings, continual monitoring of chest CT scans is effective for assessing the progression of illness. However, it is difficult to acquire a second chest CT scan in hospitalized severely sick cases, especially intubated and ventilated cases. In this circumstance, a bedside radigraph is a viable alternative. In our research, dead cases exhibited considerably higher rates of bilateral severe radiograph opacity ($P$ 0.001). Few studies have evaluated the predictive usefulness of CXR for COVID results. Toussie et al.[26] discovered that a pulmonary zone severity score on the baseline CXR was linked with the requirement for intubation in COVID cases between the ages of 21 and 50 years. In addition, Toussie et al.[26] discovered that the proportion of lungs affected was a predictor of the requirement for ventilatory assistance.

In terms of related comorbidities, dead cases in the current research showed a considerably greater incidence of HTN ($P = 0.001$) in comparison to alive cases. In agreement with our findings, Li et al.[27] determined that HTN was an independent risk factor for COVID severity. In addition, S. Huang et al.[28] demonstrated that HTN is an important predictor of death. Moreover, Li et al.,[27] Wang et al.,[14] Wu et al.,[11] and Yang et al.[12] revealed that HTN was an independent predictor of in-hospital mortality. The imbalance between the two primary renin–angiotensin–aldosterone system pathways, namely downregulated angiotensin-converting enzyme (ACE) 2/angiotensin-(1–7) and upregulated ACE/Angiotensin II, may contribute to the higher risk of COVID cases with comorbidities and advanced age. Inadequate blood pressure management is a significant risk factor for all cardiovascular fatalities and a confounding factor for COVID deaths. Gao et al.[29]

In the current study, dead cases had a considerably greater incidence of COPD than alive cases ($P = 0.02$). Similarly, in the Zhang et al.[13] cohort of 289 hospitalized COVID cases, 6.1 % of dead cases had COPD comorbidity, which was significantly greater than the incidence of COPD among non-severe cases (0.6 %). In addition, in the Zhou et al.[3] multicenter cohort of 191 COVID cases, dead cases showed a greater prevalence of COPD than alive cases (7 % vs. 1 %). In addition, Guan et al.[5] found that COPD (hazard ratio [HR]: 2.681) was a risk factor for admission to ICU, IMV, and mortality after adjusting for age and smoking in a Chinese national COVID study.

In comparison to alive cases, dead cases had a substantially greater incidence of CKD ($P = 0.037$). Consistent with our findings, Williamson et al.[20] observed that individuals with coexisting CKD had a greater risk of mortality than those without CKD, and that this risk increases as CKD progresses. In addition, of the 10,482 cases in the Ng et al.[30] research with COVID, 419 had end-stage renal disease and a greater in-hospital mortality rate than those without COVID (31.7 % vs. 25 %). It is well known that cases with CKD had a high frequency of comorbidities, such as HTN, cardiovascular disease, and DM, which may have contributed to the worse outcomes among COVID cases. Moreover, Salinas-Escudero et al.[9] observed that persons with chronic renal disease had a considerably increased chance of dying during follow-up.

In comparison to alive cases, dead cases in our research had a considerably greater incidence of stroke ($P$ 0.001). This was consistent with what Harrison et al.[31] hypothesized, namely that cases with ischemic stroke and COVID had a greater risk of 60-day death in comparison to those with ischemic stroke and no COVID. This is also congruent with findings from Yaghi et al.[32]'s research of cases with ischemic stroke in New York, which has shown that COVID carriers had considerably higher fatality rates than controls. The results of the study by Ntaios et al.[33] have shown that COVID-associated ischemic strokes were more severe with worse functional outcomes and a greater risk of death, in comparison to historical controls with ischemic strokes that did not include COVID.

In comparison to alive cases, dead cases had a substantially greater incidence of respiratory ($P = 0.001$), cardiovascular ($P$ 0.001), neurological ($P = 0.004$), renal ($P$ 0.001), hematological ($P = 0.001$), endocrine ($P = 0.009$), and dermatological problems ($P$ 0.001). In accordance with our findings, Gonzalez-Fajardo et al.[34] found that arterial thrombosis was related to elevated fatality rates in COVID cases. Moreover, Santoso et al.[35] discovered a correlation between heart damage and death. In addition, Wang et al.[14] showed a link between acute renal injuries and severe COVID, as well as a high death rate among COVID cases.

Dead cases had a considerably higher incidence of ICU admission ($P$ 0.001) and a significant increase in the necessity for more sophisticated modes of mechanical breathing (volume control and SIMV modes) ($P$ 0.001). Zareifopoulos et al.[36] observed, in keeping with our findings, that ICU admission and intubation are markers of high-level illness severity and indicate an elevated risk of mortality. In the study by Salinas-Escudero et al.,[9] intubation was identified as a significant risk factor as it more than doubles the chance of mortality in both men and women.

In comparison to alive cases, dead cases in the current research exhibited a considerably greater incidence of severe and critical hypoxia ($P$ 0.001) and a higher incidence of the requirement for mechanical breathing ($P$ 0.001). In a similar vein, Li et al.,[27] Wang et al.,[14] Wu et al.,[11] and Yang et al.[12] showed that hypoxia was an independent predictor of in-hospital mortality. In addition, Namendys-Silva et al.[37] observed that the death rate of cases with severe coronavirus rises when IMV is necessary.

### *4.1. Conclusion*

Covid-19-infected individuals with advanced age, obesity, and related comorbidities showed a considerably increased risk of mortality. Some laboratory and imaging criteria were shown to be related with mortality in Covid-19 cases. In addition, increased rates of respiratory, cardiovascular, neurological, renal, hematological, endocrine, and dermatological problems, as well as severe and critical hypoxia, ICU hospitalization, and mechanical breathing, were linked with mortality in Covid-19 cases.

## Authors' contribution

All authors contributed to the study conception and design. Material preparation, data collection, and analysis were performed by Mohammed Bhagat Abd El-Hafiz and Hossam Abd El-Moez Mohammed. The first draft of the manuscript was written by Hamdy Melegy Abdullah, and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

## Presentation meeting

Nil.

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The authors have no financial interest to declare in relation to the content of this article.

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## Conflicts of interest

The authors declared that there were no conflicts of interest.

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