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ORIGINAL ARTICLE

Clinical, Laboratory and Radiological Predictors of Outcome of Hospitalized COVID-19 Patients

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ABSTRACT

Background: Clinical symptoms, laboratory markers like C reactive protein (CRP) and D-dimer, and radiological findings such as chest computed topography (CT) could be key predictors of outcomes among hospitalized COVID-19 patients. We aimed to analyze patient comorbidities, symptoms, laboratory findings, and CT chest image findings to compare them with the outcome and predict COVID-19 patient outcomes. **Methods:** This prospective cohort study was carried out on 114 COVID-19 patients who were admitted to Zagazig University Hospitals and Zagazig Chest Hospital, the patients' comorbidities, signs, symptoms, laboratory findings, and CT chest imaging were assessed among all patients to be correlated with outcome. **Results:** It was shown that: Interleukin 6 (IL-6) was able to predict mortality at a cutoff level of > 91.7, with 67.7% sensitivity, 60% specificity (p-value = 0.013). Also CRP, erythrocyte sedimentation rate (ESR), D-Dimer, total leucocyte count (TLC), procalcitonin (PCT) could be used to predict mortality at a cutoff level of > 64, > 77, of > 1.4, > 10.6 with sensitivity of 92.7%, 68.3%, 97.6%, 61%, 43.9% and specificity of 94.5%, 78.1%, 100 %, 58.9 %, 68.5 % with p-value < 0.001 for each). The significant independent predictors of mortality were D-Dimer, CRP, ESR, PH and severity of COVID-19 (p=0.027, < 0.001, < 0.001, 0.041, and 0.012 respectively). **Conclusions**: Early detection of clinical, laboratory, and radiological data can predict COVID-19 patient outcomes, reduce severity, and decrease mortality rates, with potential of hydrogen, inflammatory markers, and CT chest imaging playing crucial roles.

Keywords: Coronavirus; Clinical Laboratory; Inflammatory markers

INTRODUCTION

Cases of pneumonia in Wuhan City, Hubei province, China, with an unknown and extremely dangerous pathogen, quickly escalated into a pandemic, and Chinese authorities notified the World Health Organization of this on December 31, 2019. It was later determined that the infectious agent was a new beta-coronavirus called SARS-CoV-2, which is also known as coronavirus disease 2019 (COVID-19) [1].

A wide range of symptoms and signs have been documented, from no symptoms at all to a severe respiratory tract infection with dyspnea, fever, cough, anosmia, and even respiratory failure or acute respiratory distress syndrome (ARDS) or even death. Diarrhea, vomiting, headache, lethargy, generalized weakness, and thromboembolism are some of the other symptoms that can accompany some presentations [2].

Factors that may predict the outcome include the patient's age, gender, place of residence, any preexisting medical conditions (such as diabetes mellitus, hypertension, obesity, ischemic heart disease, heart failure, COPD), immune response to the COVID-19 virus, results from chest computed tomography (CT), and other laboratory tests. [3].

The COVID-19 pandemic has caused a big burden on health authorities and a large mortality rate in a short time period. Although there was no specific treatment, we hypothesized that early prediction of hospital mortality using clinical data, radiological imaging data, and laboratory data can decrease mortality rates by providing efficient resources and treatment planning. So, the aim of this study was to analyze patient comorbidities, symptoms, laboratory findings, and CT chest image findings to compare them with the outcome and predict COVID-19 patient outcomes.

METHODS

This prospective cohort study was carried out on 114 COVID-19 patients admitted to Zagazig University Hospitals and Zagazig Chest Hospital. After clarifying the study's nature and objectives, informed consent was obtained from the study participants. The research was conducted under the World Medical Association's Code of Ethics (Helsinki Declaration) for human research. This study was carried out after the approval of the Institutional Review Board (IRB) of the Faculty of Medicine, Zagazig University (IRB# 9363/7-3-2022).

Sample Size: Assuming the frequency of WBC >10,000 was 7.06% vs. 28.81% in survival vs. non-survival. At 80% power and 95% CI, the estimated sample was 114 cases using OpenEpi.

Eligibility inclusion criteria involved patients who were aged more than 18 years, hospitalized with mild, moderate, and severe COVID-19 with or without comorbidity, and who had confirmed SARS-CoV-2 infection through real-time PCR testing. Patients with missing data or who were hospitalized <24 h were excluded from the analysis.

Full clinical assessment, including complete history-taking and physical examination, was performed. Data on COVID-19 and the classification of the severity of patients were recorded based on the Egyptian Management Protocol released by the Ministry of Health and Population [4].

Operational Design: The estimated sample size was 114 cases. All patients received treatment and management according to MOHP COVID-19 Protocol 2022 and its revisions. Cases were classified as: Moderate case: Pneumonia without hypoxia. Severe case: Hypoxic pneumonia improves after receiving oxygen treatment. Critically ill case: Hypoxic pneumonia that does not improve with oxygen treatment and/or organ failure [4].

Potential predictive variables included demographic data (age, gender, residence, and time between onset of symptoms to admission), vital signs, associated risk factors (diabetes mellitus, hypertension, ischemic heart diseases, chronic kidney diseases, chronic obstructive lung diseases, pregnancy, immunosuppressive drugs, active malignancy, body mass index >40), and clinical signs and symptoms (cough, sputum, fever, dyspnea, sore throat, diarrhea, anosmia, headache). CT chest findings (ground glass opacities, consolidation, unilateral or bilateral affection, affection of more than 50% of lungs), laboratory findings (complete blood picture, blood group, liver function tests, renal function tests, procalcitonin, interleukin-6, C-reactive protein, erythrocyte sedimentation rate, random blood sugar, arterial blood gases), and electrocardiogram were also included.

Statistical analysis

Coded, inputted, and analyzed using Microsoft Excel software, data was gathered from the patient's history, basic clinical examination, laboratory investigations, and outcome measures. The data was subsequently loaded into SPSS version 20.0, which stands for Statistical Package for the Social Sciences, to do the study. The quantitative data is shown as mean ± SD, while the qualitative data is presented as numbers and percentages. To determine if there were any significant differences, the following tests were utilized: the Chi-square test (X2) for associations and differences in qualitative variables. Results from a t-test, an analysis of variance (ANOVA) for multiple groups, a Pearson correlation for correlation, and logistic regression for independent predictors. For significant results, the P value was set at less than 0.05, and for very significant results, it was set at less than 0.001. Information was gathered and then subjected to statistical examination. Mean,

standard deviation (SD), sensitivity, specificity, predictive value, ROC curve, and the chi-square (x2) test were some of the statistical tests and characteristics utilized.

Ethics Considerations:

This study was ethically approved by the Institutional Reviewer Board (IRB #9363/7-3-2022) in the Faculty of Medicine, Zagazig University Hospital, This prospective cohort study was carried out on 114 COVID-19 patients admitted to Zagazig University Hospitals and Zagazig Chest Hospital. After clarifying the study's nature and objectives, informed consent was obtained from the study participants. The study was done according to The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

RESULTS

Table 1 shows that age was distributed as 62.02±15.24 with minimum 19 and maximum 96 years, males were 47.4%, DM, HTN, IHD, CKD and COPD were distributed as 36%, 35.1%, 18.4%, 3.5% and 9.6% respectively.

Table 2 shows a statistically significant increased time between onset of symptoms to hospital admission in non-survived patients (4.12 ± 1.26 days, p<0.001) when compared with survived patients (2.43 ± 0.85 days).

Table 3 shows statistically significant increased TLC in non-survived patients (13.7 ± 4.5, p=0.041) when compared with survived patients (10.2 ± 3.3). Statistically significant decreased platelets (PLTs) in non-survived patients were revealed (202.09 ± 63.7, p=0.049) when compared with survived patients (236.3 ± 70.7). Statistically significant increased IL-6 was found in non-survived patients (120.9 ± 45.6, p=0.008) when compared with survived patients (74.3 ± 26.5). D-dimer, CRP and ESR show a statistically significant increase in non-survived patients when compared with survived patients (p-value = < 0.001). Statistically significant increased pH in non-survived patients was found (7.43 ± 0.08, p=0.046) when compared with survived patients (7.39 ± 0.08). Statistically significant decreased PaO2 was found in non-survived patients (52.5 ± 9.6, p=0.004) when compared with survived patients (58.4 ± 10.5). High statistically significant increased FiO2 was revealed in non-survived patients (0.76 ± 0.23, p<0.001) when compared with survived patients (0.56 ± 0.18). A high statistically significant decrease in the PaO2/FiO2 ratio in non-survived patients was found (84.03 ± 29.6,p<0.001) when compared with survived patients (140.8 ± 57.6).

Table 4 shows a statistically significant increase percentage of lesion progression among more than 50% of lung fields in non-survived patients (36 patients, 87.8%) when compared with survived patients (38 patients, 52.1%)(p<0.001). Also, a statistically significant increased percentage of consolidation in non-survived patients (31 patients, 75.6%) when compared with survived patients (33 patients, 45.2%) (p=0.002).

Table 5 and Supplementary Figure 1 highlight the predictive performance of several biomarkers for mortality. IL-6 demonstrated a cutoff level of >91.7 with 67.7% sensitivity and 60% specificity (AUC = 0.64; 95% CI: 0.53–0.74; p = 0.013). CRP showed strong predictive accuracy at a cutoff level of >64, with 92.7% sensitivity and 94.5% specificity (AUC = 0.92; 95% CI: 0.85–0.96; p < 0.001). ESR indicated a cutoff level of >77, yielding 68.3% sensitivity and 78.1% specificity (AUC = 0.75; 95% CI: 0.66–0.82; p < 0.001). D-Dimer emerged as the most accurate predictor with a cutoff level of >1.4, achieving 97.6% sensitivity and 100% specificity (AUC = 0.99; 95% CI: 0.96–1.0; p < 0.001). In contrast, TLC (>10.6) and PCT (>0.26) showed lower predictive power, with sensitivities of 61% and 43.9%, specificities of 58.9% and 68.5%, and AUC values of 0.58 (p = 0.128) and 0.51 (p = 0.75), respectively. These results underscore the variability in biomarker effectiveness for mortality prediction.

Table 6 reveals a statistically significant difference in COVID-19 severity between survived and non-survived patients. Among survivors, 8.2% (6 patients) had moderate disease, 65.8% (48 patients) had severe disease, and 26% (19 patients) were classified as critical. Conversely, in the non-survived group, only 2.4% (1 patient) had moderate disease, 29.3% (12 patients) had severe

disease, and the majority, 68.3% (28 patients), were critically ill. These findings underscore the strong correlation between higher disease severity and increased mortality risk.

Table 7 shows: The significant independent predictors of mortality were D-Dimer, CRP, ESR, PH and severity of COVID-19 (p=0.027, < 0.001, < 0.001, 0.041, and 0.012 respectively).

Table (1): Demographic data distribution among studied patients.

Age		**Mean± SD**	**62.02±15..24**	
		Median (Range)	**65.0 (19-96)**	
			N	**%**
Sex		**Female**	**60**	**52.6**
		Male	**54**	**47.4**
Comorbidities	**DM**	**No**	**73**	**64.0**
		Yes	**41**	**36.0**
	HTN	**No**	**74**	**64.9**
		Yes	**40**	**35.1**
	IHD	**No**	**93**	**81.6**
		Yes	**21**	**18.4**
	CKD	**No**	**110**	**96.5**
		Yes	**4**	**3.5**
	COPD	**No**	**103**	**90.4**
		Yes	**11**	**9.6**
	Active malignancy	**No**	**113**	**99.1**
		Yes	**1**	**0.9**
	Obesity BMI>40	**No**	**71**	**62.3**
		Yes	**43**	**37.7**
Pregnant females (1 from 60 female)		**No**	**59**	**98.3**
		Yes	**1**	**1.7**
Total			**114**	**100.0**

DM: Diabetes Mellitus, HTN: Hypertension, IHD: Ischemic Heart Disease, CKD: Chronic Kidney Disease, COPD: Chronic obstructive pulmonary disease.

Table (2): Clinical parameters distribution between studied patients in relation with outcome.

			Outcome		**X^2**	**P**
			Survivors	**Non survivors**		
Time between onset of symptoms to hospital admission in days			**2.43±0.85**	**4.12±1.26**		**0.00****
Cough	**No**	**N**	**0**	**1**	**1.79**	**0.18**
		%	**0.0%**	**2.4%**		
	Yes	**N**	**73**	**40**		
		%	**100.0%**	**97.6%**		
Sputum	**No**	**N**	**18**	**11**	**0.065**	**0.79**
		%	**24.7%**	**26.8%**		
	Yes	**N**	**55**	**30**		
		%	**75.3%**	**73.2%**		
Fever	**No**	**N**	**18**	**16**	**2.58**	**0.108**
		%	**24.7%**	**39.0%**		
	Yes	**N**	**55**	**25**		
		%	**75.3%**	**61.0%**		
Dyspnea	**No**	**N**	**3**	**2**	**0.037**	**0.84**

			Outcome		X^2	P
		%	4.1%	4.9%		
	Yes	N	70	39		
		%	95.9%	95.1%		
Sore Throat	No	N	21	15	0.74	0.38
		%	28.8%	36.6%		
	Yes	N	52	26		
		%	71.2%	63.4%		
Diarrhea	No	N	55	33	0.39	0.53
		%	75.3%	80.5%		
	Yes	N	18	8		
		%	24.7%	19.5%		
Anosmia	No	N	45	27	0.20	0.65
		%	61.6%	65.9%		
	Yes	N	28	14		
		%	38.4%	34.1%		
Headache	No	N	32	26	4.02	0.045*
		%	43.8%	63.4%		
	Yes	N	41	15		
		%	56.2%	36.6%		
Total		N	73	41		
		%	100.0%	100.0%		

Table (3): Laboratory parameters distribution between studied patients in relation with outcome.

	Survivors	Non survivors	t	P
TLC	10.23±3.30	13.71±4.58	2.134	0.041*
PLT	236.37±70.71	202.09±63.70	1.990	0.049*
PCT	0.28±0.071	0.35±0.081	0.853	0.395
IL-6	74.39±26.58	120.90±45.63	2.688	0.008*
D-Dimer	0.82 ± 0.25	2.5 ± 0.66	19.3	0.00**
CRP	44.91±18.3	92.70±27.6	11.080	0.00**
ESR	64.30±19.1	80.78±18.24	4.490	0.00**
Neutrophils	9.3 ± 5.6	14.4 ± 26.6	1.56	0.121
Lymphocytes	0.87 ± 0.6	1.83 ± 5.4	1.5	0.135
Neutrophil/Lymphocyte ratio.	16.9 ± 14.4	22.5 ± 32.7	1.25	0.211
SGPT	47.34±18.63	46.82±17.32	0.075	0.940
SGOT	46.89±14.28	45.97±13.28	0.149	0.882
Direct Bilirubin	0.17±0.065	0.16±0.05	0.541	0.589
Albumin	3.43±0.53	3.28±0.72	1.220	0.225
Serum Creatinine	1.16±0.37	1.07±0.27	1.351	0.179
Blood Urea	48.36±16.32	50.14±17.71	0.439	0.661
RBS	174.39±66.7	195.17±71.3	1.151	0.252
pH	7.39±0.08	7.43±0.08	2.021	0.046*
PaO_2	58.42±10.55	52.58±9.62	2.924	0.004*
$PaCO_2$	44.47±17.65	38.34±12.22	1.974	0.051
HCO_3	25.48±9.82	23.62±4.21	1.155	0.250
FiO_2	0.56±0.18	0.76±0.23	4.173	0.00**
PaO_2/ FiO_2 ratio	140.89±57.6	84.03±29.63	4.691	0.00**

TLC: Total Leucocyte Count, PLT: Platelet Count, PCT: Procalcitonin, IL-6: Interleukin 6, CRP: C-Reactive Protein, ESR: Erythrocyte Sedimentation Rate, SGPT: serum Glutamic Pyruvic Transaminase, SGOT: Serum Glutamic-Oxaloacetic Transaminase, RBS: Random Blood Sugar, pH: potential of hydrogen, PaO_2: partial pressure of oxygen in arterial blood, $PaCO_2$: partial pressure of carbon dioxide in arterial blood HCO_3: Bicarbonate, FiO_2: Fraction of inspired oxygen.

Table (4): Computerized tomography (CT) signs distribution between studied patients in relation with outcome.

	Outcome		X^2	P
	Survivors	Non survivors		
Unilateral infiltration	4 (5.5%)	0 (0.0%)	2.32	0.12
Bilateral infiltration	69 (94.5%)	41 (100.0%)	2.32	0.12
Lesion progression more than 50% of lungs field visual severity scoring of CT chest	38 (52.1%)	36 (87.8%)	14.73	0.00**
Consolidation topography of pneumonia (alveolar infiltration of bronchopneumonia)	33 (45.2%)	31 (75.6%)	9.85	0.002*
Total	73 (100.0%)	(100.0%)		

Table (5): Distribution of mortality predictors as regard inflammatory markers among studied patients.

Area Under the Curve						Sensitivity	Specificity
Test Result Variable (s)	Area	Cutoff	P	95% Confidence Interval			
				Lower Bound	Upper Bound		
IL-6	0.641	>91.7	0.013	0.537	0.745	67.7%	60.0%
CRP	0.92	> 64	0.00	0.85	0.96	92.7%	94.5%
ESR	0.75	> 77	0.00	0.66	0.82	68.3%	78.1%
D-Dimer	0.99	> 1.4	0.00	0.96	1.0	97.6%	100%
TLC	0.58	> 10.6	0.128	0.48	0.67	61%	58.9%
PCT	0.51	> 0.26	0.75	0.42	0.61	43.9%	68.5%

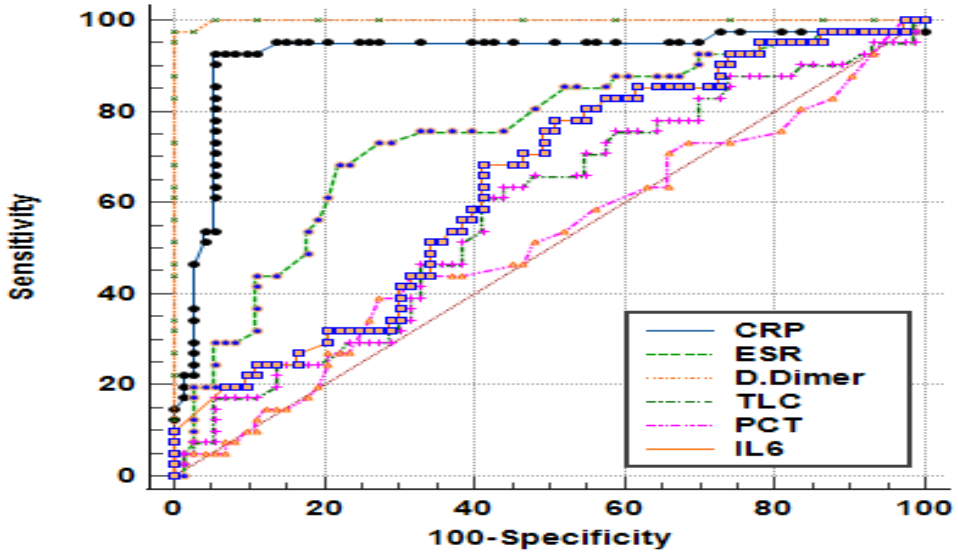
IL-6: Interleukin 6, CRP: C-Reactive Protein, ESR: Erythrocyte Sedimentation Rate, TLC: Total Leucocyte Count, PCT: Procalcitonin.

Table (6): Relation between outcome and severity of COVID-19.

			Outcome		Total	X^2	P
			Survivors	Non survivors			
Severity of COVID-19	Moderate	N	6	1	7		
		%	8.2%	2.4%	6.1%		
	Sever	N	48	12	60		
		%	65.8%	29.3%	52.6%	19.44	0.00**
	Critical	N	19	28	47		
		%	26.0%	68.3%	41.2%		
Total		N	73	41	114		
		%	100.0%	100.0%	100.0%		

Table (7): Multivariate logistic regression for independent predictors regard mortality.

		Wald	P	OR	95% C.I	
					Lower	Upper
	Age	**3.714**	**0.054**	**1.036**	**0.999**	**1.075**
	TLC	**1.833**	**0.176**	**1.050**	**0.978**	**1.128**
	PLT	**3.765**	**0.052**	**0.995**	**0.989**	**1.000**
	IL-6	**1.356**	**0.244**	**1.003**	**0.998**	**1.009**
	D-dimer	**-15.4**	**0.027***	**0.0**	**0.0**	**0.171**
	CRP	**-0.08**	**< 0.001****	**0.92**	**0.89**	**0.948**
	ESR	**-0.049**	**< 0.001****	**0.95**	**0.92**	**0.97**
	pH	**4.195**	**0.041***	**25.535**	**1.283**	**80.120**
	PaO_2	**0.928**	**0.335**	**1.037**	**0.963**	**1.116**
	FiO_2	**1.149**	**0.284**	**10.827**	**0.139**	**842.671**
	PaO_2/ FiO_2 ratio	**0.731**	**0.392**	**0.991**	**0.970**	**1.012**
	Affection of more than 50% in CT chest	**2.514**	**0.102**	**2.913**	**0.714**	**10.745**
	Severity of COVID-19	**5.845**	**0.012***	**3.456**	**1.587**	**10.084**



Supplementary Figure (1): ROC curve for detection of death prediction cutoff regard inflammatory markers.

DISCUSSION

In the early stages of SARS-CoV-2, the infection may have no symptoms, symptoms such as coughing, shortness of breath and even organ failure and death can develop in people of any age [5]. Infection rates, lengths of stay in the hospital, and fatalities from the COVID-19 pandemic have varied considerably throughout and even within regions and countries, raising concerns over the factors that predict the outcome of COVID-19 patients hospitalized [6].

Medical intervention and early diagnosis are essential for reducing mortality rates among

critically ill cases. An important part of controlling COVID-19 is identifying people who are likely to have severe illness or die from the virus. Previous research out of Wuhan, the epicenter of the epidemic, found that hospitalization rates were higher for patients with certain comorbidities, including diabetes, hypertension, and cardiovascular disease [7].

The need for mechanical ventilation, renal dysfunction or liver damage, high C-reactive protein, elevated interleukin-6, lymphopenia, and elevated procalcitonin levels are among the additional factors that have been suggested as indicators of a bad prognosis. The severity of the sickness and fatality rates, however, do not seem to be influenced by any discernible causes. These factors could be used to establish criteria for hospital admission and evaluate the risk of referring patients to the intensive care unit [8].

In our study, age was distributed as 62.02±15.24 with a minimum 19 and a maximum 96 years, males were 47.4%, DM, HTN, IHD, CKD and COPD were distributed as 36%, 35.1%, 18.4%, 3.5% and 9.6%. The results showed a significant difference in the median values of age; the no survivors patients had an older age than the survivors. In the line of the present study, Richardson et al. [9] and Zhang et al. [10] found a higher disease severity associated with age. The older patients had more severity and morbidity and less improvement during follow-up.

In addition, Cao et al. [11] found that older age was a risk factor associated with severity of disease in in Shanghai, China. In our study, we showed that the risk of death was significantly higher in patients who had increased time between onset of symptoms to hospital admission. Researchers in China found that the longer duration since onset of symptoms till patients seek for medical advice and are diagnosed with COVID-19, the more severe the disease was. A similar explanation was advanced for the low COVID-19 mortality rate in South Korea: people went to the hospital the moment the symptoms of the virus began to appears [12,13].

The increased duration between the appearances of symptoms and hospital admission was associated with higher ICU admission. A potential confounding factor is the fact that patient variables (such as age and the presence or absence of comorbidities like cardiovascular disease) differed depending on the time it took from the start of symptoms to hospital admission. The clinical course and prognosis of a disease are closely related to variations in patient characteristics, vital signs, or imaging procedures, such as age, inflammatory levels, or computed tomography detected lung lesions, as determined by the delay between the onset of symptoms and hospital admission [14,15].

Also, the results of our study revealed that cough, sputum, fever, dyspnea, sore throat, diarrhea and anosmia were not good predictors for death in COVID-19 patients in our cohort study. Our data are by Hesam-Shariati et al. [16] and Alimohamadi et al. [17], who demonstrated through their meta-analysis that COVID-19 is mostly characterized by a high fever, coughing, and difficulty breathing. The severity of the illness determined the clinical findings in COVID-19, which were independent of case outcomes. In our study regarding complete blood picture, the non-survived patients showed a significant increase in TLC. While platelets count was significantly decreased. That would be due to superimposed bacterial infection and sepsis. When comparing lymphocyte counts between survivors and non-survivors, no statistically significant changes were found.

In their study, El-Kassas et al. [18] demonstrated that comorbidities in COVID-19 patients were associated with a substantial drop in platelet count. An infection-induced cytokine storm may explain why TLC levels rise despite lymphopenia. [19]. Also, our data in accordance with Huang et al. [20] and Siddiqi et al. [11] studies, increased concentration of serum procalcitonin was relatively less common in all patients. Upon admission, the majority of patients had procalcitonin levels in their serum that were within the normal range (<0.1 ng/mL). Patients whose infections progressed to the intensive care unit had procalcitonin levels above 0.5 ng/mL.

Plasmatic level of CRP, in our study, was found to be a good predictor of hospitalized COVID-19 mortality and was significantly higher in critical and severe patients and significantly lower in moderate patients. Increased levels of inflammatory biomarkers in plasma, such as C-reactive protein, were linked to a more severe case of COVID-19, and hyper inflammation is known to occur throughout the disease's clinical course. [21,22].

Our research found that arterial blood gases measurements taken at admission showed significantly higher potential of hydrogen (pH) and fraction of inspired oxygen (fio2) in non-survived patients compared to survived patients. On the other hand, survived patients had considerably higher partial pressures of oxygen in the arterial blood (pao2) and in the alveoli (pao2/fio2) ratios (p-value = 0.00). The more lung injury in individuals who did not survive compared to those who survived might be responsible for that.

In contrast to our study, there was no specific pattern of ABG analysis in COVID-19 patients who were treated in the ICU. However, the most common result was respiratory alkalosis and there was no significant relationship between ABG analysis and the outcome of COVID-19 patients. Further research is recommended with a larger sample to see the profile of ABG analysis in COVID-19 patients by considering the risk factors [23].

Our results demonstrated that, in comparison to survivors, patients who did not survive had a significantly higher percentage of lesion progression affecting more than 50% of the lungs fields. Consolidation percentages were also much higher among patients who did not survive than in those who survived. Regarding unilateral and bilateral infiltration, there was no discernible difference or correlation between patients who survived and those who did not survive.

Similarly, Hesam-Shariati et al. [16] found that CT scans can reveal the degree of illness. In order to better diagnose and treat patients, it may be helpful to study clinical characteristics with CT scan findings to better understand how diseases manifest across age groups. In our study, multivariate analysis was done using a logistic regression test. The results showed that D-dimer, ESR, CRP, blood pH and severity of COVID-19 have significant impacts on COVID-19 patients' mortality. Meanwhile, age and TLC, PLT, IL-6, PaO2, FiO2 and, PaO2/FiO2 ratio did not have significant impacts COVID-19 patients' mortality. In agreement with us, El-Kassas et al. [18] stated that the mortality rate for patients with severe COVID-19 was 50%, but it was just 0.5% for moderate cases. Also, the multivariate regression of potential predictors of COVID-19 severity in the study of Zayed et al. [24] who revealed the following predictors: age, mean platelet volume, serum ferritin, and IHD.

CONCLUSIONS

Early detection of clinical, laboratory, and radiological data and findings can predict outcome of hospitalized COVID-19 patients and can be useful in decrease severity of COVID-19 and decrease mortality rate. Potential of hydrogen (PH), inflammatory markers like (ESR, CRP& D-dimer) and severity of COVID-19 were recommended to be used as independent predictors of mortality. CT chest image plays a significant role in diagnosing the pulmonary changes associated with COVID-19 patients.

Recommendations

We could recommend: Integrated of D-Dimer, CRP, ESR, PH and severity of COVID-19 as a score to predict outcome of COVID-19 patients. Include high sample size and more investigations in future studies

Conflicts of Interest

The authors report no conflicts of interest.

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None declared

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