Survival Analysis and Risk Factors for in-hospital mortality in COVID-19 Patients: A retrospective cohort study

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Abstract

Purpose:

Since the outbreak in late December 2019 in Wuhan, China, coronavirus disease-2019 (COVID-19) has become a global pandemic. We analyzed and compared the clinical, laboratory, and radiological characteristics between survivors and non-survivors and identify risk factors for mortality.

Methods:

This single-center study was conducted at Hospital for COVID-19 patients in Kashan, Iran. Inpatients with confirmed COVID-19 were included. Clinical and laboratory variables, radiological features, complications were collected. Patients were classified as the discharged or survivor group and the death or non-survivor group based on their outcome (improvement or death). Clinical, Epidemiological characteristics, as well as laboratory parameters, were extracted from electronic medical records. Cox regression analysis was conducted to identify the risk factors for mortality. Significant level was set as 0.05 in all analyses.

Results:

Multiple Cox regression showed age (HR 1.028; 95% CI 1.016–1.039), LDH (HR 1.0004; 95% CI 1.0002–1.001), AST (HR 1.002; 95% CI 1.001–1.004) and BUN (HR 1.008; 95% CI 1.004–1.012) as factors associated with an increased risk of in-hospital mortality.

Conclusion:

The current study showed that higher mortality was significantly associated with age, LDH, AST and BUN. Assessing risk factors of the disease could be helpful for clinicians to detect the risk of disease progression, to perform proper intervention earlier to get the best therapeutic outcome.

Introduction

Coronaviruses (CoV) are single-stranded RNA viruses belonging to the Coronaviridae family and are classified into four genera based on their genetic characteristics: Alphacoronavirus, Betacoronavirus, Gammacoronavirus, and Deltacronavirus [1]. Coronaviruses are a broad family of viruses that cause a wide range of diseases in mammals and birds. Infection with this viral family causes a wide range of clinical symptoms, from the common cold to chronic respiratory diseases [2]. Coronavirus disease 2019 (COVID-19) is a member of the family, which was observed firstly in Wuhan, China in December 2019. To date, the disease has been spread around the world and put an extra burden on healthcare workers in every society. Since the human immune system is not already prepared to fight with SARS-CoV-2 (the cause of Covid-19 disease), the rate of spread of this deadly virus in human communities is very high.

Like the other two viruses in this family, the Middle East Respiratory Syndrome (MERS) and the Severe Acute Respiratory Syndrome (SARS), SARS-CoV-2 originated from animals. The main path of transmission of the virus is through respiratory droplets or contact with contaminated surfaces and objects [3].

Fever, cough, and shortness of breath are the most common symptoms of the COVID-19 disease. Muscle ache, headache, confusion, chest pain, and diarrhea can be considered as the following manifestations [3, 4]. Severe disease can expand dyspnea as well as hypoxemia during a week after the first appearance of the symptoms, which is possible to develop acute respiratory distress syndrome (ARDS) or end-organ failure [4]. Senescence, neutrophilia, and organ as well as coagulation dysfunction, for instance, high level of LDH and D-dimer can be considered as several significant risk factors in association with Acute respiratory distress syndrome (ARDS) and its progression from mild symptoms to death [5]. Besides, there were different factors, such as comorbidities, lymphocyte counts, CD3 and CD4T-cell counts, AST, pre albumin, creatinine, glucose, low-density lipoprotein, serum ferritin, PT, which were only related to the expansion of ARDS, not death [5]. A report shows that lymphocytopenia was identified in 83.2%, thrombocytopenia in 36.2%, and leukopenia in 33.7% of the cases. In addition, upregulating of C-reactive protein in the vast majority of the patients along with less frequent increasing of alanine aminotransferase, aspartate aminotransferase, creatine kinase, and d-dimer were observed in the cases [6]. This study, described clinical characteristics, laboratory data and epidemiologic factors between surviving and no-surviving hospitalized COVID-19 patients. Potential risk factors for death on admission were determined. We showed some valuable data to forecast the death of COVID-19 patients through this retrospective cohort study of 528 cases in Dr. Beheshti Hospital in Kashan, Iran.

Methods

Study population

This retrospective cohort study was done on 528 patients in Dr. Beheshti Hospital in Kashan, Iran, and aged 17–99 years who were diagnosed with COVID-19. According to World Health Organization interim guidance [7], the COVID-19 pneumonia of all patients was confirmed through SARS-CoV-2 reverse-transcriptase polymerase chain reaction test and by nasopharyngeal/ oropharyngeal swab or sputum specimen [8]. As reported by hospital data, patients were admitted from March 25, 2020, to September 26, 2020.

Data collection

The epidemiological, clinical and outcomes were gathered from medical records of patients by physicians and medical students. Following up the patients was done until September 26, 2020. All clinical outcomes and their definitions were checked by 3 authors. Patients' data were kept secretly in a locked and password protected computer. The SARS-CoV-2 infection of patients was confirmed by taking

throat swab samples and real-time reverse transcriptase–polymerase chain reaction [9]. More tests including blood routine tests, coagulation, biochemical tests and computed tomography was done for every patient. These clinical data were gathered from the first days of hospitalization. The self-reported temperature of patients was considered as the highest temperature in order to minimizing the interference of patients' treatment during hospitalization. Classification of age was defied as < 60, and 60 ≤ years [10]. For body temperature were classified as, ≤ 37.3°C, 37.3°C -39°C and ≥ 39°C [5].

Statistical Analysis

Data were collected through SPSS version 26 using descriptive statistics (frequency (%)/ Mean ± SD/Median (IQR)) and inferential statistics (Chi-square, independent t-test and Mann-Whitney U test to compare clinical features and laboratory findings between surviving and deceased groups as well as Cox proportional hazards regression model to identify factors associated with death from COVID-19 patients) were analyzed.

Results

Demographic Characteristics and Laboratory Parameters

The findings of Table 1 showed that the mean age of the deceased patients was significantly higher than the surviving patients (71.09 ± 15.71 vs 54.20 ± 15.81, p < .001). But there was no significant difference between the two groups in terms of gender, BMI, blood type, underlying disease (comorbidity) and clinical symptoms (p > .05). Also, laboratory parameters (except for RBC, HB, HCT and total bilirubin) showed a significant difference between the dead and surviving groups (Table 2).

Survival Analysis

Multiple Cox (proportional-hazard) regression showed age (HR 1.028; 95% CI 1.016–1.039), LDH (HR 1.0004; 95% CI 1.0002–1.001), AST (HR 1.002; 95% CI 1.001–1.004) and BUN (HR 1.008; 95% CI 1.004–1.012) as factors associated with an increased risk of mortality. The survival curves based on risk factors (age, LDH, AST and BUN) are shown in Fig. 1.

Table 1
Demographic and clinical characteristics of patients

Variable		Survivor	Non-survivor	p-value
Gender	Male	168 (50.1)	80 (41.5)	.054
	Female	167 (49.9)	113 (58.5)	
Age (year)	Mean ± SD (min-max)	54.20 ± 15.81 (19–96)	71.09 ± 15.71 (17–99)	.000
	≥ 60	121 (36.1)	151 (78.2)	
BMI	Mean ± SD (min-max)	28.69 ± 4.12 (22.1–46.3)	29.27 ± 4.04 (22.2–46.3)	.117
	Normal (< 25)	78 (23.3)	29 (15)	
	Overweight (25–30)	130 (38.8)	81 (42)	
	Obesity (> 30)	127 (37.9)	83 (43)	
Blood type	A+	106 (31.6)	67 (34.7)	.886
	A-	11 (3.3)	4 (2.1)	
	B+	77 (23)	46 (23.8)	
	B-	8 (2.4)	6 (3.1)	
	AB+	36 (10.7)	17 (8.8)	
	AB-	4 (1.2)	3 (1.6)	
	O+	85 (25.4)	43 (22.3)	
	O-	8 (2.4)	7 (3.6)	
Comorbidity	Diabetic	122 (36.4)	74 (38.3)	.659
	Hypertension	98 (29.3)	66 (34.2)	.237
	Cardiovascular disease	46 (13.7)	29 (15)	.682
	Asthma	47 (14)	31 (16.1)	.526
	Malignancy	4 (1.2)	2 (1)	.869
	Autoimmune disease	17 (5.1)	11 (5.7)	.758
	Hematonosis	14 (4.2)	5 (2.6)	.345
	Organ failure	81 (24.2)	53 (27.5)	.404
Symptom	Fever	263 (78.5)	150 (77.7)	.833

Variable		Survivor	Non-survivor	p-value
	Chills	264 (78.8)	153 (79.3)	.899
	dyspnea	210 (62.7)	127 (65.8)	.473
	Cough	229 (68.4)	132 (68.4)	.993
	Chest pain	161 (48.1)	101 (52.3)	.344
	Headache	174 (51.9)	108 (56)	.373
	Fatigue	293 (87.5)	166 (86)	.633
	Myalgia	263 (78.5)	143 (74.1)	.247
	Sore throat	117 (34.9)	70 (36.3)	.756
	Abdominal pain	76 (22.7)	42 (21.8)	.806
	Nausea	124 (37)	64 (33.2)	.373
	vomiting	64 (19.1)	33 (17.1)	.566
	diarrhea	128 (38.2)	66 (34.2)	.357
	Odor disorder	172 (51.3)	100 (51.8)	.917
	Taste disorder	128 (38.2)	68 (35.2)	.495
	Anorexia	199 (59.4)	111 (57.5)	.671
	Loss of consciousness	51 (15.2)	26 (13.5)	.583
	Visual impairment	66 (19.7)	43 (22.3)	.481

Table 2
Laboratory findings of patients on admission

Variables		survivor	Non-survivor	p-value
WBC (×109/L)	Median (Q1-Q3)	6.5 (4.6–11.3)	13.62 (8.78–20.72)	.000
	Normal (4–11)	204 (60.9)	69 (35.8)	
RBC (×109/L)	Median (Q1-Q3)	5 (4–5)	5 (4–5)	.966
	Normal (4-5.5)	305 (91)	155 (80.3)	
HB (gr/dL)	Median (Q1-Q3)	13.4 (12.3–14.7)	13.4 (11.65–14.8)	.576
	Normal (12–18)	271 (80.9)	135 (69.9)	
HCT	Median (Q1-Q3)	39.5 (36.5–42.4)	40.1 (35.2–44)	.204
	Normal (34–45)	268 (80)	123 (63.7)	
Platelets (×109/L)	Median (Q1-Q3)	207 (153–269)	137 (82.5–200)	.000
	Normal (150–450)	243 (72.5)	77 (39.9)	
AST (AU/L)	Median (Q1-Q3)	35 (25–55)	60 (35.5–101)	.000
	Normal (5–40)	207 (61.8)	63 (32.6)	
ALT (AU/L)	Median (Q1-Q3)	28 (18–49)	38 (22.5–62.5)	.001
	Normal (5–40)	215 (64.2)	102 (52.8)	
ALP (AU/L)	Median (Q1-Q3)	174 (138–214)	226 (172.5–311)	.000
	Normal (65–306)	310 (92.5)	142 (73.6)	
CPK (AU/L)	Median (Q1-Q3)	102 (58–176)	133 (61.5–295)	.005
	Normal (0-243)	275 (82.1)	138 (71.5)	
LDH (AU/L)	Median (Q1-Q3)	530 (406–725)	768 (582.5-1075.5)	.000
	Normal (0-550)	180 (53.7)	42 (21.8)	
PT (s)	Median (Q1-Q3)	12 (11–13)	13 (11–16)	.000
	Normal (10.5–13.5)	212 (63.3)	85 (44)	
APTT (s)	Median (Q1-Q3)	25 (23–27)	28 (25–39)	.000
	Normal (21–37)	307 (91.6)	137 (71)	
D-dimer (µg/mL)	Median (Q1-Q3)	556 (300–1069)	1320 (572–4769)	.000
	Normal (0-1.5)	154 (46)	42 (21.8)	

Variables		survivor	Non-survivor	p-value
Fibrinogen (s)	Median (Q1-Q3)	626 (479–800)	700 (536–851)	.013
	Normal (200–400)	37 (11)	13 (6.7)	
CRP (mgr/L)	Median (Q1-Q3)	39 (14–63)	127 (61–197)	.000
	Normal (< 10)	56 (16.7)	8 (4.1)	
ESR (mM)	Median (Q1-Q3)	33 (18–53)	57 (35–89)	.000
	Normal (< 22)	96 (28.7)	28 (14.5)	
BUN (mgr/dL)	Median (Q1-Q3)	15 (11–21)	58 (32–87)	.000
	Normal (8–25)	263 (78.5)	23 (11.9)	
CR (mgr/dL)	Median (Q1-Q3)	1 (0.9–1.2)	1.8 (1.2–2.7)	.000
	Normal (0.4–1.5)	304 (90.7)	74 (38.3)	
NA (mM/L)	Median (Q1-Q3)	137 (134–140)	140 (135.5-143.5)	.000
	Normal (135–145)	242 (72.2)	131 (67.9)	
K (mM/L)	Median (Q1-Q3)	4.1 (3.8–4.4)	4.9 (4.4–5.6)	.000
	Normal (3.5–5.5)	315 (94)	140 (72.5)	

Table 3
Multiple Cox regression of mortality risk factors

Variable	HR (95%CI)	p-value
Age	1.028 (1.016–1.039)	.000
LDH	1.0004 (1.0002–1.001)	.000
AST	1.002 (1.001–1.004)	.013
BUN	1.008 (1.004–1.012)	.000

Discussion

This current study, investigated the relationship between demographic clinical data of COVID-19 patients and the rate of mortality in these patients. At the early stage in 2020, the mortality rate of COVID-19 was nearly 3.7–5.4% [11–13]. The mortality rate increased up to 52.4% in patients suffering from ARDS [5] and 61.5% among severe and critical cases [14]. The study revealed that there is no difference in symptoms of survival and non-survival groups that these results were confirmed with previous study [15]. Fatigue, fever and chill were the most common symptoms in both survival and non-survival groups that these results were similar to finding by other research [16]. Whereas other study found that higher

respiratory rate and lower puls oximetry in non-survival group were more common and non-respiratory symptoms such as fever and myalgia/arthralgia were more frequent in survivor patients [17].

The data revealed that older ages potentially have higher risk of mortality which is consistent with other studies [5, 12, 18–22]. Interestingly, no significant difference was seen between the mortality rate of COVID and comorbidities such as diabetes, cardiovascular disease, hypertension and asthma in patients. These findings are in contradiction with previous results [22] which reported the significant increase in mortality rate of cardiovascular, hypertension and diabetic patients. A strong correlation between older ages accompanied with comorbidities is also highlighted by another study in Iran [23, 24]. Some laboratory data including AST, LDH and BUN in both survival and non-survival groups were significantly out of range ($p < .05$). This result is also supported by some researchers which identified lower range of lymphocyte counts, Hb, platelet, and albumin levels as well as higher range of inflammatory biomarkers and cytokines in non-survival patients [16]. other studies also found that there are some changes in hematological, biochemical, and inflammatory biomarkers in severe or non-survived patients which is in line with our results indicating that our data can be compared with other populations [25, 26].

As shown in Table 3, multivariate Cox's proportional-hazard model was done in order to find variables which are independently predicted mortality. This test showed that higher rate of death is significantly related to the older age, LDH, AST and BUN. It is in good agreement with another study which revealed that moderate anemia, increased level of LDH and creatine kinase are independent risk factors for mortality [18, 27].

Conclusion

In this study we found higher rate of death is significantly related to the older age, LDH, AST and BUN. Evaluation of the risk factors of this disease can be beneficial for clinicians to find the risk of disease progression, to perform proper intervention earlier to get the best therapeutic goal, although we feel that it can be useful, in conjunction with other prognostic markers regarding clinical management decisions.

Declarations

Ethics approval and consent to participate

This study was approved by Ethical Committee of Kashan University of Medical Sciences (IR.KAUMS.MEDNT.REC.1401.022), All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and national research committee and with the 1964 Helsinki Declaration and its later amendments. Written informed consent was obtained from all participants or if participants are under 16, from a parent and/or legal guardian.

Consent for publication

All study process was presented to patients and they were reassured about confidentiality of their records, they were requested to present their written consent of participation in the study.

Availability of data and material

The primary data for this study is available from the authors on direct request.

Competing interests

The authors declare no conflict of interest.

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Authors' contributions

ESH, MJA and HHK were responsible for the study conception and design and MS, LV, and ESH performed data collection and AP, HN and HHK preparing the first draft of the manuscript. MJA did the data analysis, HHK made critical revisions to the paper for important intellectual content and supervised the study. All authors read and approved the final manuscript.

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Figures

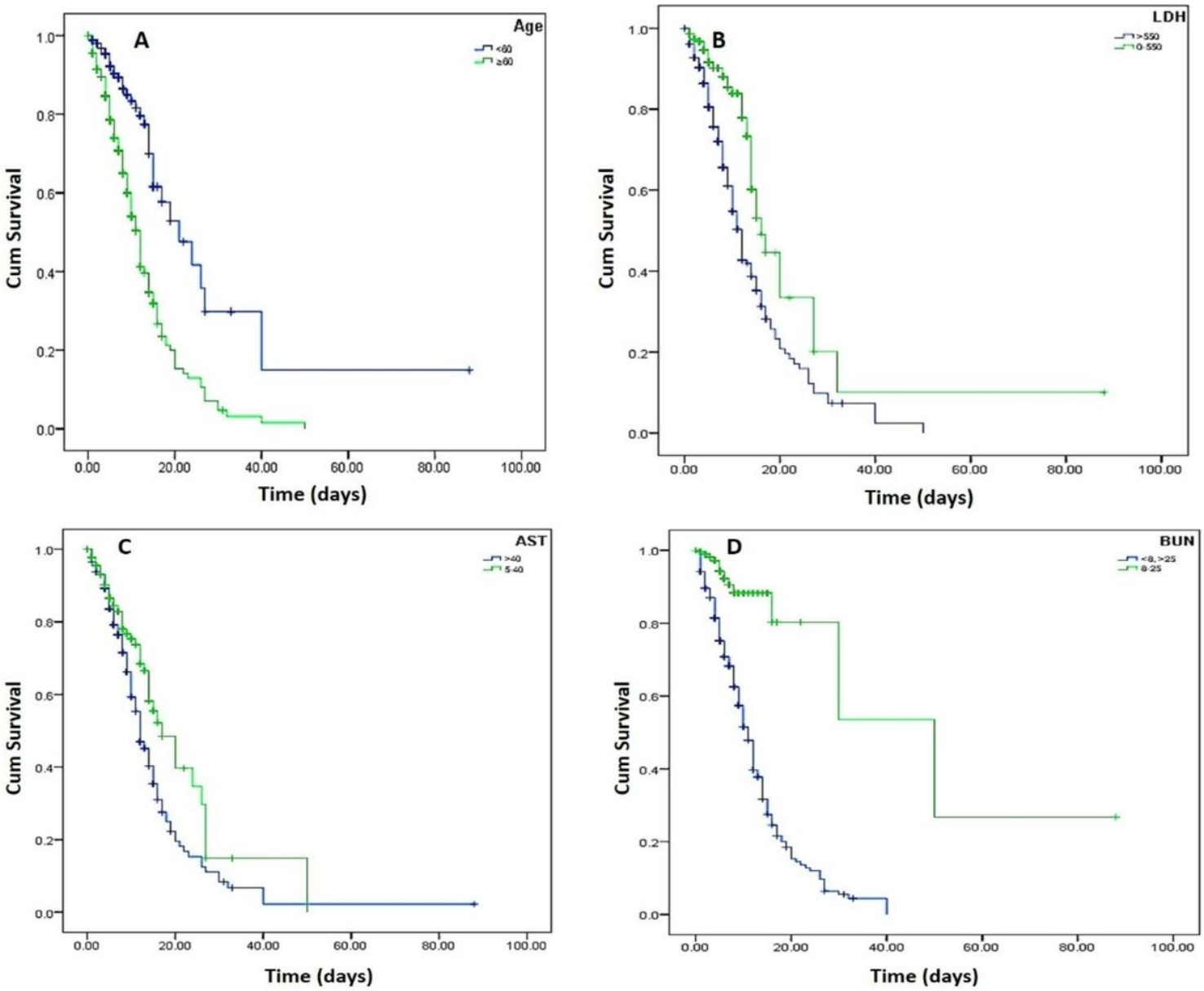

Figure 1

Kaplan-Meier survival plots based on risk factors. (A) Age, (B) LDH, (C) AST and (D) BUN.