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Research Article

Clinical and Laboratory Features of COVID-19 in Ulin Referral Hospital of South Kalimantan: Predictors of Clinical Outcome

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ABSTRACT

Corona Virus Disease (COVID-19) is becoming a global pandemic. Indonesia, especially South Kalimantan had recorded increasing cases with a high fatality rate of 3.7%. Information about factors related to outcomes based on clinical and laboratory features in Indonesia is still limited. Identification of the risk is crucial to determine optimal management and reducing mortality. This retrospective study enrolled 455 adults COVID-19 patients and data were extracted from medical records of Ulin General Hospital Banjarmasin. The latter is COVID-19 referral hospital in South Kalimantan between March-November 2020. Demographic data, comorbidities, and laboratory were all collected. Data were compared between survivors and non-survivors. Fisher's exact test and chi-square were used to compare categorical variables. The Mann-Whitney U test was used to compare continuous variables. Analysis was continued by multivariate logistic regression then receiver operating characteristic (ROC) curve to determine cut-off value. The multivariate analysis showed that number of comorbidities [odds ratio (OR) 1,339 (95% confidence interval (CI): 1,064-1,685, P = 0,013) was significant risk factor to the outcome. In laboratory, lactate dehydrogenase (LDH) [OR: 1.001, 95% CI: 1,000-1.002, P = 0.001], Ferritin (OR 1.000, CI: 1,000-1.001, P = 0.013), APTT (OR: 1.045, CI: 1.010-1.082, P = 0.012), and D-dimer (OR: 1.188, CI: 1.064 - 1.327, P = 0.002) were significant predictor factors but only LDH, ferritin and D-dimer were obtained good AUC 0.731, 0.715, and 0.705, respectively. The cut of the value of LDH was 656.5 U/L, ferritin was 672.18 ng/ml, and D-dimer was 2.28 mg/L. Sensitivity and specificity were 66.7% and 68,0% for LDH, 83,2% and 56,3% for ferritin, and 62,8 and 70,8% for D-dimer. From this research, we revealed that the number of comorbidities was a risk factor for death. Elevated LDH, ferritin, and D-dimer could be good predictive factors for poor outcomes, thereby considering the accelerating management of COVID-19 patients.

Keywords: COVID-19, Comorbidities, D-dimer, Ferritin, LDH, Outcome

Introduction

COVID-19 was first reported in Wuhan, China, as pneumonia caused by new viral infection 2019 novel coronavirus (2019-nCOV). It was known as severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2). World Health Organization (WHO) declared COVID-19 a pandemic on March 11, 2020, because since the discovery of the virus, the number of cases outside China has increased by 13 times within two weeks. The number of affected countries also increased threefold. In early April 2020, WHO reported a more than tenfold increase in less than a month with more than 1 million confirmed cases of COVID-19 worldwide [1, 2].

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This virus affected 222 countries with 184 of these countries being local transmission areas. Since it was first announced in Indonesia, the number of COVID-19 cases has continued to increase, so it needs attention. As of January 20, 2021, Indonesia recorded 939,948 confirmed cases of COVID-19 and 26,857 died with a case fatality rate (CFR) of 2.9%, still higher than the global CFR of 2.2%. Meanwhile, South Kalimantan contributed 16,837 cases with 618 deaths, including a high CFR of 3.7% [3]. South Kalimantan has so far recorded a significant increase in the number of cases.

Early epidemiological studies showed that the main symptoms of COVID-19 infection were fever, dry cough, shortness of breath, and headache, with progression to pneumonia. Many patients also reported other symptoms such as fatigue, myalgia, diarrhea, nausea/vomiting, neurological symptoms such as anosmia/hyposmia and dysgeusia even non-specific dermatological manifestations such as urticaria. Haemoptysis is a rare clinical manifestation [4]. Rapid transmission with a wide spectrum of manifestations makes this new virus more difficult to control. Preventive measures include using face masks, hand hygiene practices (regularly wash hands or at least 60% alcohol if soap is not available), avoidance of public contact, and avoidance of contact with people infected. Case detection, contact tracing, and quarantines have been discussed as ways to reduce transmission. This protocol must be implemented to reduce the spread of COVID-19 [5].

Unfortunately, this was still a difficulty where everyone must do protocol compliance to control the disease. The number of patients that continues to increase causes an increase in health care costs. The fluctuating mortality rates due to COVID-19 virus need to be considered. Several measures that may slow down the transmission were the isolation of patients with COVID-19 cases, identification and follow-up contacts, environmental disinfection, and personal protective equipment. Governments also carried out various efforts to prevent the spread of the COVID-19 virus in countries worldwide to break the chain of spreading the virus [3, 5].

Comorbidity in COVID-19 patients was one factor that affects the degree of disease, more complex management, and increased health care costs and affects poor outcomes. This was a matter of concern in patient care [6]. COVID-19 patients also show various laboratory tests before admission. The laboratory tests were complete blood count, blood chemistry analysis, coagulation factors, C-reactive protein (CRP), liver and kidney function that showed various abnormalities. These laboratory values were said to describe the infectious conditions in patients [7]. These laboratory values continue to focus on whether they were significant for the severity of disease or the possibility related to patient prognosis.

Clinical and laboratory information on COVID-19 patients in Indonesia was limited. More importantly, significant differences had been identified in the clinical and demographic features of COVID-19 patients in different regions of the world. Therefore, we aimed to study clinical features and laboratory characteristics that affect patient outcomes at Ulin General Hospital Banjarmasin as a COVID-19 referral hospital in South Kalimantan. The latest information regarding the new coronavirus is essential and is expected to describe disease trends that will guide medical personnel in finding optimal approaches and priorities for COVID-19 patients. This can help determine which interventions are given to the patient at a suitable time to provide optimal management and prevent poor outcomes.

This study hopes that clinical and laboratory features could be an objective assessment that helps doctors and medical personnel to determine the alarm about the poor outcome in patients. This is an early marker in determining when treatment should be more aggressive. Also, it helps to provide the best education to the patient's family about prognostic and the care being performed.

Material and Methods

Study design

The research design used was a retrospective study. Patient and laboratory data were taken from the medical records of the Ulin General Hospital in Banjarmasin. Data were used as predictors of clinical outcome. This research was approved by the Ethical Committee of Ulin General Hospital, Banjarmasin No.83/V-Reg Riset/RSUDU/20.

Subjects

The research subjects were all data on COVID-19 patients at Ulin General Hospital between March-November 2020. The COVID-19 patient was someone who tested positive for the COVID-19 virus, as evidenced by reverse trans-

criptase-polymerase chain reaction (RT-PCR) laboratory tests. As many as 455 subjects were included in this study and divided into 2 groups, 96 patients were non-survivors and 359 were survivors. Non-survivors are those who died during hospitalization. Survivors are discharged from the hospital, living with a negative evalua-tion of RT-PCR or self-isolation. Inclusion criteria for this study were as follows: all patients who have the required demographic data variables such as age, sex, comorbidities, number of comorbidi-ties, and clinical manifestations. The comorbidities include a history of smoking, smoking, asthma, chronic obstructive pulmonary disease (COPD), pulmonary tuberculosis, diabetes mellitus, hypertension, cardiovascular disease, neurological disease, hepatitis B, cancer, chronic kidney disease, and obesity. Laboratory data included haemoglobin, leukocytes, platelets, eosinophils, neutrophils, Neutrophil Lymphocyte Ratio (NLR), Absolute Lymphocyte Count (ALC), Lactate Dehydrogenase (LDH), Aspartate Aminotransferase (AST), Alanine Aminotransferase (ALT), Urea, Creatinine, Sodium, Potassium, Chloride, C-reactive protein (CRP), Ferritin, Partial Thromboplastin Time (PTT), activated Partial Thromboplastin Time (APTT), International Normalized Ratio (INR) and D-dimer. Patients who did not have data according to the specified variables were excluded from this study. The laboratory tests that were listed were carried out at the time of admission. All baseline values were taken before the severity classification of the patients was determined. Initial values at admission were used to construct predictive models and pick the efficacy of predicting poor outcomes in COVID-19 patients.

Statistical method

Before analysis, the data were tested for normality and homogeneity. Hypothesis testing was performed to compare continuous and categorical variables using the Mann-Whitney U test and Fisher's exact test. The correlation between comorbidities and survivor was used chi-square test. Relationship with patient outcomes logistic regression was used to explore the independent early predictors and risk factors associated with the outcome. The predictive efficacy of each laboratory predictor was measured by the receiver operating characteristic (ROC) curve. All significance levels were computed for two-tailed testing and the cut-off of significance was set at $P < 0.05$. A good area under the curve (AUC) is obtained if it is above 0.7. Data were analyzed with a confidence interval 95% using software SPSS for Windows version 25.0.

Results and Discussions

Clinical features

Based on the baseline patient characteristics, age and gender had significant differences. In the non-survivor group, it was found that the age was older than the survivor group. Older age tends to have an unfavorable outcome such as death. It was known that morbidity and mortality rates were higher in old age. In another study, it was stated that in Asia, Europe, and the United States of America (USA), 80% of deaths due to COVID-19 were aged > 65 years [8].

Male patients were more significantly different in the non-survivors group. In the other study, males had different morbidity and mortality rates in that they were more at risk of developing a severe infection, three times more likely to need intensive care than women and were 50% more likely to cause death. However, there may be some bias in existing gender studies [9, 10].

Meanwhile, comorbidities factors such as smoking, asthma, pulmonary tuberculosis, diabetes mellitus, hypertension, cardiovascular disease, cerebrovascular disease such as stroke, hepatitis B, cancer and obesity showed no significant difference in the two groups with p value > 0.05 (Table 1). This could be due to the comorbidity of the patients in admission have heterogeneous conditions and severity and statistically, in this study, the differences were not considered significant between survivors and non-survivors groups.

The results of the chi-square test between the survivors and the comorbidities obtained $p = 0.032$. It means the number of comorbidities was a statistically significant difference. Patients with two or more comorbidities tended to have a higher percentage of poor outcomes than no and single comorbidities. In the multivariate analysis, the number of comorbidities was still a significant predictor factor in the outcome, increasing the chance of death by OR 1.339 (95% CI: 1.064-1.685) in Table 2. The other studies found that comorbidities contribute to increasing severity and ICU admission, but only cardiovascular or neurological disease together with COVID-19 in-

Table 1. Demographic and baseline characteristic of non-survivors and survivors groups

Variables	Non-survivors (n = 96)		Survivors (n = 359)		*P*
	n	%	N	%	
Age, median (IQR)	54	16	50	18	0.001
Male sex	67	69.8	184	51.2	0.001
Comorbidities:					
History of smoke	4	4.2	16	4.5	1.000
Smoking	9	9.4	22	6.1	0.259
Asthma	3	3.1	10	2.8	0.742
COPD	1	1.0	1	0.3	0.378
Pulmonary Tuberculosis	1	1.0	7	1.9	1.000
Diabetes Mellitus	31	32.3	87	24.2	0.117
Hypertension	43	44.8	127	35.4	0.097
Cardiovascular Disease	7	7.3	20	5.6	0.476
Neurological Disease	4	4.2	14	3.9	1.000
Hepatitis B	2	2.1	4	1.1	0.611
Cancer	6	6.3	12	3.3	0.234
Chronic Kidney Disease	13	13.5	42	11.7	0.600
Obesity	42	43.7	130	36.2	0.193
Number of comorbidities					0.032
0	14	14.6	87	24.2	
1	31	32.3	135	37.6	
2	25	26.0	77	21.4	
≥3	26	27.1	60	16.7	
Clinical Manifestation					
Fever/history of fever	81	84.4	261	72.7	0.023
Shortness of breath	83	86.5	205	57.1	< 0.001
Cough	73	76.0	237	66.0	0.065
Sore throat	13	13.5	49	13.6	1.000
Cold	2	2.1	10	2.8	1.000
Haemoptysis	2	2.1	4	1.1	0.611
Anosmia	3	3.1	32	8.9	0.082
Nausea/vomiting	25	26.0	113	31.5	0.320
Diarrhea	16	16.7	59	16.4	1.000
Myalgia	0	0.0	7	1.9	0.354
Fatigue	24	25.0	73	20.3	0.328
Cephalgia	2	2.1	20	5.6	0.190

IQR: Interquartile Range

fection carries a higher risk of mortality [6].

In clinical manifestations, it was found that shortness of breath and fever/history of fever were statistically significant differences in the two groups. Shortness of breath was the most complained about by a non-survivor group. Shortness of breath was the subjective experience of respiratory discomfort, has been reported to affect less than 50% of patients infected with SARS-CoV-2 and is more common in patients who were about to die than those who will recover. Shortness of breath was a marker for warning symptoms of pneumonia COVID-19. Of course, this complaint was the main complaint of patients with acute respiratory distress syndrome (ARDS), which often occurs in patients with critical degrees of COVID-19. Perhaps that is why most patients with poor outcomes complained of shortness of breath the most. Patients with shortness of breath showed a more severe phase of infection than patients with mild symptoms in the absence of pneumonia [11].

Whereas, history of fever is the most common finding observed among patients 84.4% in the non-survivors group and 72.7% in survivors group with p-value 0.023. Fever indicates a host's response to a substance that changes the temperature regulation center. Fever may be a beneficial signal from immunity response but also increase

Table 2. Factors associated with COVID-19 death using logistic regression analysis

Variables	OR	95% CI	*P*
Age	1.014	0.991-1.039	0.238
Male Sex	1.483	0.807-2.725	0.205
Number of comorbidities	1.339	1.064-1.685	0.013
Leukocytes × 10^9/L,	0.972	0.912-1.036	0.377
Eosinophils %	1.067	0.853-1.335	0.570
Neutrophils %	1.065	0.984-1.152	0.117
Lymphocyte %	1.002	0.907-1.108	0.962
NLR	0.994	0.960-1.029	0.731
ALC	1.000	1.000-1.001	0.459
LDH	1.001	1.000-1.002	0.001
AST	0.998	0.995-1.002	0.391
Urea	1.000	0.995-1.005	0.921
Creatinine	0.993	0.957-1.030	0.700
CRP	1.003	0.999-1.006	0.105
Ferritin	1.000	1.000-1.001	0.013
PTT	1.054	0.906-1.226	0.493
APTT	1.045	1.010-1.082	0.012
INR	0.822	0.343-1.969	0.660
D-dimer	1.188	1.064-1.327	0.002

OR: Odds Ratio, CI: Confidence Interval

Table 2. Comparison of laboratory parameters between non-survivors and survivors groups

Variables	Normal Range	Non-survivors N = 96		Survivors N = 359		*P*
		Median	IQR	Median	IQR	
Haemoglobin, g/dL	12.0-18.0	13.4	3.38	13.30	2.80	0.181
Leukocytes, × 10^9/L	4.0-10.5	9.5	6.36	7.61	4.40	0.003
Platelet count, × 10^9/L	150-450	241.0	168.50	252.0	137.00	0.207
Eosinophil, %	1.0-3.0	0.0	1.00	0.3	1.50	0.002
Neutrophil, %	50-81	82.55	11.43	73.4	16.70	<0.001
Lymphocyte, %	20-40	10.6	9.95	16.5	13.50	<0.001
NLR		7.79	7.77	4.29	4.92	<0.001
ALC		1037.5	756.50	1300.0	796.00	0.001
LDH, U/L	122-220	839.0	482.25	488.0	474.00	<0.001
AST, U/L	5-34	53.0	60.25	40.0	33.00	<0.001
ALT, U/L	0-55	37.0	40.75	36.0	39.00	0.153
Urea, mg/dl	0-50	47.755	58.00	26.0	29.00	<0.001
Creatinine, mg/dl	0.77-1.25	1.3	1.31	0.97	0.66	<0.001
Sodium, Meq/L	136-145	136.0	6.00	136.0	5.00	0.320
Potassium, Meq/L	3.5-5.1	4.155	0.90	3.9	0.70	0.077
Chloride, Meq/L	98-107	106.0	7.00	106.0	6.00	0.286
CRP, mg/dl	< 6	64.65	105.03	32.0	79.10	<0.001
Ferritin, ng/ml	4.63-274.66	1680.89	1215.25	532.53	1208.93	<0.001
PTT, s	9.9-13.5	12.3	2.08	11.3	1.80	<0.001
APTT, s	22.2-37.0	30.35	7.05	28.0	5.50	<0.001
INR,		1.14	0.21	1.04	0.19	<0.001
D-dimer, mg/L	<0.22	3.445	4.30	1.19	2.34	<0.001

IQR: Interquartile Range

the need for metabolism energy. This increase in metabolic energy requirements can be a factor that needs to be considered in patient care [4].

Laboratory study

From the results of the comparison laboratory study (Table 3), it was found that laboratory findings showed significant differences in the levels of leukocytes, eosinophils, neutrophils, lymphocytes, NLR, ALC, LDH, AST, Urea, Creatinine, CRP, PTT, APTT, INR, D-dimer, and ferritin. Leukocyte levels were significantly higher in the non-survivors group. Mild neutrophilia often occurs in COVID-19 patients who died. Lymphocytopenia was common in patients and lower in the group who died. Increased NLR and decreased ALC also tended to be found in the group who died. Elevated serum LDH in the non-survivors group was significantly higher two times than the survivors group. The elevated liver enzyme as AST was higher in the non-survivors group. High levels of urea and creatinine were also found in died patient. CRP levels were significantly two times higher and ferritin three times higher in those who died. Meanwhile, PT, APTT, INR and D-dimer also tended to be significantly prolonged and higher in the non-survivors group.

To analyze the risk of fatal outcomes, logistic regression was used (Table 2). Factors that have significant differences between the non-survivors and survivors groups then entered into the regression model. After multivariate logistic regression analysis, it was found that only LDH, Ferritin, APTT and D-dimers played a significant role in predictors of poor outcome. Meanwhile, other factors not significant enough to be a factor in determining the prognostic value of COVID-19 patients.

The analysis showed that LDH [odds ratio (OR): 1.001, 95% confidence interval (CI): 1,000-1.002, P = 0.001], Ferritin (OR 1.000, CI: 1.000-1.001, P = 0.013), APTT (OR: 1.045, CI: 1.010-1.082, P = 0.012), and D-dimer (OR: 1.188, CI: 1.064 - 1.327, P = 0.002) were significant predictive factors for poor clinical outcomes of COVID-19 patients. Furthermore, they were analyzed using the ROC Curve to predict efficacy of varia-ble (Table 4). An AUC above 0.7 was obtained in LDH (AUC = 0.731), ferritin (AUC=0.715) and D-dimer (AUC=0.705) with a cut of value of LDH 656.5, ferritin 672.18 and D-dimer 2.28.

Lactate dehydrogenase

In this study LDH predicted poor outcome even with an OR of 1.001. According ROC Curve analysis results for LDH (Table 4), the cut of value was 656.5 and the area under the curve (AUC) was 0.731 (95% CI: 0.678-0.785). Sensitivity values were 66.7% and specificity values were 68%. Comparable to other studies have shown that elevated LDH was significantly different in the group of patients with severity. Meanwhile, survivor and non-survivor patients also had a significant difference [12]. In another study LDH was demonstrated to have high accuracy with an AUC value of 0.949 with high sensitivity and specificity for determining patient severity and mortality [13]. In this study, LDH also had the highest AUC value compared to other variables, although the difference was not too big.

The elevated in LDH indicates the presence of inflammation and non-specific tissue injury that occurs in COVID-19 patients. This marked increase was attributed to severe cases of COVID-19 and reflected wider tissue damage. LDH was most active in the liver, kidneys, lungs, brain and red cells (erythrocytes). If there was cell damage, lactate dehydrogenase released, so that the amount of concentration in the blood increases. Since LDH was present in lung tissue, it was expected that patients with COVID-19 infection in the form of interstitial pneumonia or severe infections that can release LDH into the circulation and manifesting as acute respiratory distress syndrome (ARDS). Extremely high serum LDH activity related with a negative prognosis in these patients. It was noted that the inflammatory response to COVID-19 may be more extensive than common viral infections, even causing cytokine storms that can lead to disease complications and dysfunction multi-organ. High levels of LDH indicates a biomarker of disease extent [14, 15].

Ferritin

In this study, it was shown that high ferritin levels have an effect on poor outcomes with OR 1.000 (95% CI: 1.000-1.001). With the median ferritin value in the non-survivors group was 1680.89 ng/ml, this was three times higher than 532.52 ng/ml in the survivor group (Table 2). Ferritin be predictive value with AUC 0.715 (95% CI: 0.660-0.769) with sensitivity and specificity are 83.2% and 56.3% (Table 4). It is comparable with other studies that ferritin can be predictive of the

Table 4. Predictive efficacy* of the poor outcome COVID-19 risk model.

Variables	AUC	P Value	95% CI	Cut of Value	Sensitivity	Specificity
LDH	0.731	< 0.001	0.678-0.785	656.5	66.7%	68.0%
Ferritin	0.715	< 0.001	0.660-0.769	672.18	83.2%	56.3%
APTT	0.656	< 0.001	0.594-0.719	28.35	71.9%	53.8%
D-dimer	0.705	< 0.001	0.648-0.761	2.28	62.8%	70.8%

AUC: Area Under the Curve
*The predictive efficacy was evaluated by receiver operating characteristic (ROC) curve

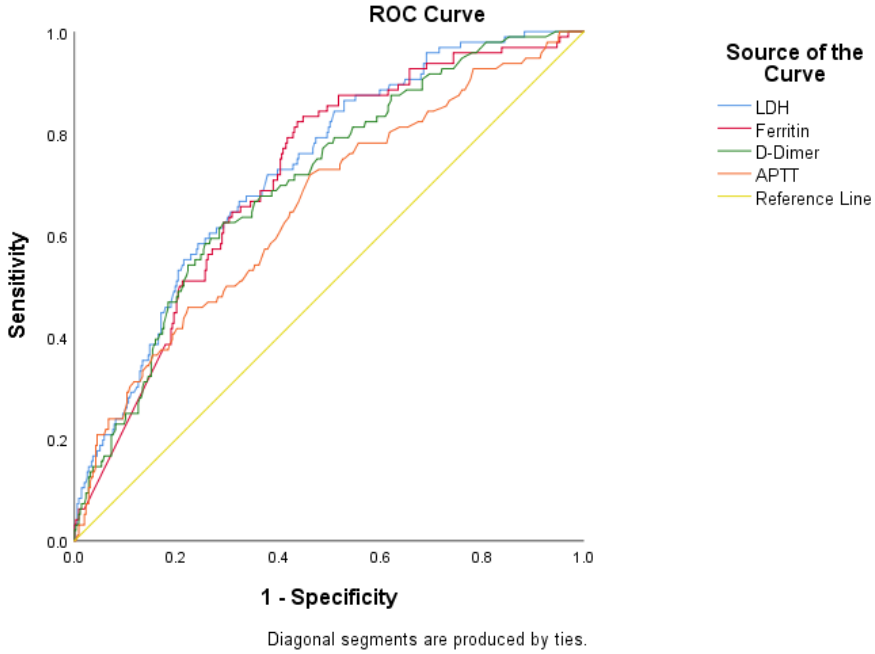

Figure 1. ROC Curve of LDH, ferritin, APTT and D-dimer

mortality with AUC 0.762 (95% CI: 0.690-0.835) [16]. Although, other studies mention ferritin was not accurate predictor for outcomes [17].

Other research comparing ferritin levels states that ferritin levels tend to be higher in the severe patients than mild patients [18]. Patients who need to be treated in the ICU show ferritin 5.8 times higher than those with mild COVID-19 [19]. Serum ferritin secretion is associated with macrophages that produce cytokines and are responsible for most of the immune reactions in the pulmonary parenchyma. Ferritin synthesis can also be induced inflammatory stimulation of interlukin-6. Interestingly, high IL-6 concentrations are known to be positively correlated with disease degree [20, 21].

Apart from being induced by cytokines, ferritin was also reported to have a feedback mechanism that can induce pro and anti-inflam-matory formation as well. It was known that ferritin consists of 2 different subunits, namely H and L. Expression of H is driven by inflammatory stimuli and can work as an immunomodulatory molecule. It is interesting to explore in future how ferritin is expressed in COVID-19 patients. Ferritin is a significant marker of macrophage activation. Individuals with hyperferritinemia show a characteristic pattern of reticuloendothelial system activation and multiple organ dysfunction. Therefore, hyperferritinemia has been associated with high mortality regardless of the underlying pathology [22].

Coagulopathic parameters

Abnormalities of coagulopathic parameters such as APTT and D-dimers were found to be correlated with patient outcome. In contrast to several studies where the APTT value did not significantly different in outcome [23, 24]. In this study, APTT

actually showed that significant different and correlated a poor outcome with odds 1,045. Although on the ROC curve, the predictor assessment was not significant with an AUC is 0.656. The results of the APTT study tended to be more varied than the D-dimers which remain in relation to the outcome in several studies with different backgrounds [24, 25].

The increase in D-dimers showed a positive correlation that 1.188-fold increased odds the risk of poor outcomes (OR: 1.188 CI 95%: 1.064-1.327). This odds ratio was higher than three others factor that significant role in poor outcome. According to ROC Curve, an AUC of 0.705 was obtained with a cut of value of D-dimer was 2.28 mg/L with a sensitivity and specificity were 62.8% and 70.8%, respectively. It was not much different from studies elsewhere that the optimum cut off of D-dimer was 2.0 mg/L (fourfold increase) but with high sensitivity and specificity in that study. In other study, they found cut off was 2.14 mg/L effectively predicts mortality [26, 27].

D-dimers were predictors of the tendency of blood to develop hypercoagulability. This coagulation mechanism was related to the cytokine storm mentioned earlier. Excessive systemic inflammatory response can lead to systemic endotheliopathy and a hypercoagulable state that leading to increased activation of the coagulation cascade and excessive thrombin production. This increases the risk of systemic macrothrombosis and microthrombosis. Patients with critical COVID-19 showed alveolar and microvascular changes in the lung suspected to have platelet-rich strings of inflamed endothelium and intra-alveolar deposition of fibrin to form localized or diffuse microthrombus which plays a role in the incidence of ARDS and multiorgan failure. In addition, vasoconstriction and decreased blood flow due to severe hypoxemia also increase the microthrombotic vasoocclusion process [28, 29].

Regular D-dimer evaluated was a crucial need for COVID-19 patients with critical severity to determine therapeutic management that focuses on hemostasis. Although the use of thromboprophylaxis using low molecular-weight heparin (LMWH) or unfractionated heparin (UFH) has been given, the thrombotic incidence rate was still high. Aggressive doses of LMWH or UFH were recommended in patients with multiple risk factor for thromboembolism [28].

However, we had some limitations, we only studied patients who were hospitalized in Ulin Regional Hospital Banjarmasin and cannot report if there are differences in characteristics and outcomes for patients who are being quarantined at home or another hospital in South Kalimantan. We suggest large prospective studies are needed to further build on our data and provide insights into this ongoing pandemic in the Indonesia.

Conclusion

In summary, this study investigates clinical and laboratory features in admission that can predict high risk of poor outcome of COVID-19. Current research revealed that number of comor-bidities were risk factor for death. The laboratory data such as elevated of LDH (656.5 U/L), ferritin (672.18 ng/ml) and D-dimer (2.28 mg/L) could be a good predictive factor for poor outcomes. This increase of these markers was believed to be the result of a cytokine storm that common occurs in severe patients. Also, from this study may risk poor outcomes. Thereby, we suggest to consider accelerating management of COVID-19 patients who at high risk.

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