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Prognostic Nutritional Index (PNI) at Admission Predicts In-Hospital Mortality of COVID-19-Infected Patients

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Abstract

Corona Virus Disease-2019 (COVID-19) was declared a pandemic in March 2020 and caused considerable deaths in all parts of the world. Mortality is influenced by the immune system status and increased vulnerability to infection, both related to nutritional status. The Prognostic Nutritional Index (PNI), calculated using lymphocyte count and albumin levels, may have the ability to more accurately characterize the nutritional and inflammatory conditions of COVID-19 patients. This retrospective study analyzed 967 COVID-19 patients at Ulin Hospital Banjarmasin, Indonesia, by examining demographic data, laboratory results, and PNI in relation to survival outcomes. The study revealed that factors such as age, sex, comorbidities (including hypertension, diabetes mellitus/DM, obesity, etc.), number of comorbidities, and disease severity correlated with mortality. Leucocyte count, lymphocyte count, albumin levels, and PNI all showed significant correlations with survival ($p < 0.001$), suggesting that these factors may serve as useful prognostic indicators for COVID-19 patient's survival. The PNI was associated with an increased risk of mortality, with a univariate odds ratio (OR) of 0.923. Receiver operating characteristic (ROC) analysis demonstrated that a PNI cut-off value of <41.9 had a sensitivity of 44.9% and a specificity of 82.4%, with an area under the curve (AUC) of 0.666 ($p < 0.001$). Hence, PNI at admission, which reflects patients' immune system and nutritional status upon hospital admission, can be used as a simple, cost-effective, and reliable predictor of mortality in COVID-19 patients.

Keywords: COVID-19, leucocyte, mortality, prognostic nutritional index, prognosis

Introduction

The World Health Organization (WHO) declared Coronavirus Disease 2019 (COVID-19) a pandemic on March 11, 2020, resulting in extensive global mortality. By the end of 2022, Indonesia's case fatality rate (CFR) was 2.4%, higher than the global CFR of 1%, while South Kalimantan, one of the provinces in Indonesia, had an even higher CFR of 2.9%. COVID-19 severity ranges from mild to critical.[1] The World Health Organization (WHO) declared Coronavirus Disease 2019 (COVID-19) a pandemic on March 11, 2020, resulting in extensive global mortality. By the end of 2022, Indonesia's case fatality rate (CFR) was 2.4%, higher than the global CFR of 1%, while South Kalimantan, one of the provinces in Indonesia, had an even higher CFR of 2.9%. COVID-19 severity ranges from mild to critical.

Previous studies raised concerns about patients with severe pneumonia, who experienced protein loss and impaired immune defenses..[2] Patients with COVID-19 also exhibited protein loss symptoms, including decreased albumin levels and compromised organ function, emphasizing the significance of nutrition risk assessment and its prognostic value for COVID-19 patients. Accumulating evidence suggests that patients infected with COVID-19 have an inferior prognosis if they have significant systemic inflammation and a deficient nutritional status.[3]

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Nutritional screening should ideally be simple and non-invasive. The Prognostic Nutritional Index (PNI), calculated using serum albumin and lymphocyte counts, is a widely used indicator of nutritional, immunological, and inflammatory status. It has demonstrated prognostic value in various conditions, including cardiovascular disease, infections, and cancer.[4,5] Malnutrition weakens the immune system and increases infection vulnerability. Hypoalbuminemia mostly reflects nutritional status, but it has also been linked to the severity of COVID-19. The declining albumin level could indicate a severe cytokine storm and organ damage. As evidenced by albumin levels, the poor nutritional state of COVID-19 patients is unfavorable to tissue repair and recovery. Lymphocyte count, a critical component of PNI, was found much lower in non-survivors. Nevertheless, lymphopenia has been confirmed as an independent risk factor for mortality among COVID-19 patients. The lymphocyte drop could be linked to diminished immunological activity and a substantial cytokine increase. The PNI could more precisely characterize COVID-19 patients' nutritional and inflammatory conditions.[6] Low PNI may suggest severe conditions and a poor prognosis for patients. Therefore, this study aims to determine whether the nutritional status at hospital admission, as assessed by PNI, can predict in-hospital mortality among COVID-19 patients, particularly in resource-limited settings like Indonesia.

Methods

This study used a retrospective design based on medical records of COVID-19 patients treated at Ulin Regional Hospital, the primary referral hospital for COVID-19 in South Kalimantan, Indonesia, between March 2020 and December 2021.

Data were taken using purposive sampling with the inclusion criteria of all medical record data for COVID-19 patients aged 18 years who were treated at Ulin Regional Hospital, Banjarmasin with complete research variables in admission. Variables based on clinical and laboratory data with all adult patients had either hospital discharge or mortality as their definitive clinical outcome. Demographic factors include age, gender, co-morbidities, number of co-morbidities, disease severity, and body mass index (BMI). The patients' blood test parameters, such as lymphocyte and albumin on the first day of admission, were compared to survival. The Prognostic Nutritional Index (PNI) was calculated on the first day of admission using the following formula:

[10 x serum albumin (g/dL)] + [0.005 x lymphocyte count (per mm^3)] 11, on the first day of hospitalization.

A total of 976 patients met the inclusion criteria and were included in the final analysis. Patients were divided into two groups: survivors (those discharged from the hospital) and non-survivors (those who died during hospitalization).

Statistical analysis was performed using SPSS software version 26.1. The Kolmogorov-Smirnov test was used to assess the normality of data distribution. The Mann-Whitney U test was used to compare continuous variables between groups. The Chi-square test was applied for examining independent qualitative data, while Fisher's exact test was utilized when the conditions for the Chi-square test were not met. Univariate logistic regression was performed to estimate the risk factors for mortality. T Receiver operating characteristic (ROC) curve analysis was used to evaluate the diagnostic value and optimal cut-off point of the PNI. A p-value of <0.05 was considered statistically significant. This study received ethical approval from the Ethics Committee of Ulin Regional Hospital under protocol number 25/KSM.Paru/Litbang/RSUDU/II/2023.

Results

This study included 976 hospitalized COVID-19 patients, consisting of 772 survivors and 204 non-survivors. Table 1 summarizes the patients' demographic, clinical, and laboratory characteristics. Non-survivors were significantly older than survivors ($p<0.001$), and male sex was also associated with increased mortality ($p=0.011$)Comorbidities such as hypertension ($p=0.004$), diabetes mellitus ($p=0.001$), and obesity ($p=0.036$) were more prevalent in non-survivors. Additionally, the number of comorbid conditions was significantly associated with mortality ($p<0.001$). Disease severity at admission was also linked to survival outcome ($p<0.001$). Laboratory results indicated that non-survivors had higher leukocyte counts (8700; 6555-11300), lymphocytopenia (10.5%; 7.1–15.5), lower albumin levels (3.2; 2.9-3.5), and lower PNI ratios (33; 37.1–41.1), all with p-values <0.001 (Table 1).

Table 1 Baseline Demographic, Clinical, and Laboratory Characteristics of COVID-19 Patients on Admission by Survival Outcome

Variable	All patients (n=976)	Survivors (n=772)	Non-Survivors (n=204)	p-value
Age, median (range), year	54 (44–62)	52 (43–60)	58 (49–65)	<0.001*
Male	54 (44–62)	53 (43–61)	60 (49–66)	<0.001*
Female	53 (43–61)	52 (42–60)	57 (49–64)	0.001*
Sex, no (%)				
Male	540 (55.3)	411 (42.1)	129 (13.2)	0.011α
Female	436 (44.7)	361 (37.0)	75 (7.7)	
Co-morbidities, no (%)				
Hypertension	443 (45.4)	332 (34.0)	111 (11.4)	0.004α
Diabetes Mellitus	322 (33.0)	234 (24.0)	88 (9.0)	0.001α
Chronic Kidney Disease	101 (10.3)	74 (7.6)	27 (2.8)	0.128α
Cardiovascular disease	80 (8.2)	60 (6.1)	20 (2.1)	0.347α
Stroke	39 (4.0)	30 (3.1)	9 (0.9)	0.733α
Malignancy	7 (0.7)	5 (0.5)	2 (0.2)	0.641β
Hepatitis B	11 (1.1)	6 (0.6)	5 (0.5)	0.059β
Lung tuberculosis	33 (3.4)	24 (2.5)	9 (0.9)	0.360α
COPD	4 (0.4)	3 (0.3)	1 (0.1)	1.000β
Asthma	27 (2.8)	21 (2.2)	6 (0.6)	0.864α
Obesity	477 (48.9)	364 (37.3)	113 (11.6)	0.036α
Number of Co-morbidities, no (%)				
0	171 (17.5)	158 (16.2)	13 (1.3)	
1	330 (33.8)	262 (26.8)	68 (7.0)	<0.001α
2	260 (26.6)	202 (20.7)	58 (5.9)	
≥3	215 (22)	150 (15.4)	65 (6.7)	
The severity of illness, no (%)				
Mild	94 (9.6)	92 (9.4)	2 (0.2)	
Moderate	240 (24.6)	234 (24)	6 (0.6)	<0.001α
Severe	160 (16.4)	146 (15)	14 (1.4)	
Critically ill	482 (49.4)	300 (30.7)	182 (18.6)	
Body Mass Index, no (%)				
Underweight (<18.5)	21 (2.2)	18 (1.8)	3 (0.3)	
Normal (18.5–22.9)	267 (27.4)	214 (21.9)	53 (5.4)	0.148α
Overweight (23–24.9)	211 (21.6)	176 (18)	35 (3.6)	
Obesity (≥25)	477 (48.9)	364 (37.3)	113 (11.6)	
Leucocyte, median (range)	7600 (5700–9800)	7300 (5502–9300)	8700 (6555–11300)	<0.001*
Lymphocyte, median (range)	15.5 (9.72–23.3)	17 (11.2–25)	10.5 (7.1–15.5)	<0.001*
Albumin, median (range)	3.4(3–3.8)	3.5 (3.1–3.9)	3.2 (2.9–3.5)	<0.001*
PNI, median (range)	40.0 (35.6–45.1)	41.0 (36.0–46.2)	33 (37.1–41.1)	<0.001*

α=Pearson Chi square, β=Fisher exact test *Mann-Whitney test

Table 2 Univariate Logistic Regression Analysis of Laboratory Parameters Associated With COVID-19 Mortality

Variables	Univariate		
	Odds Ratio (OR)	95% CI	p-value
Leucocyte	1.000	1.000-1.000	<0.001
Lymphocyte	0.914	0.894-0.934	<0.001
Albumin	0.472	0.375-0.624	<0.001
Prognostic Nutritional Index	0.923	0.901-0.945	<0.001

Univariate logistic regression analysis (Table 2) showed that leukocyte count (OR 1.000, 95% CI 1.000–1.000), lymphocyte percentage (OR 0.914, 95% CI 0.894–0.934), albumin level (OR 0.472, 95% CI 0.375–0.624), and PNI (OR 0.923, 95% CI 0.901–0.945) were significantly associated with mortality (all p<0.001). Receiver operating characteristic (ROC) analysis of PNI (Figure 1) identified a cut-off value of ≤41.9, with a sensitivity of 44.9%, specificity of 82.4%, and area under the curve (AUC) of 0.666 (p<0.001), indicating moderate discriminatory ability for mortality prediction.

Following the cut-off result of PNI (<41.9), we further analyzed the data to identify any significant correlations (Table 3). Interestingly, we found that age and gender were strongly correlated with PNI (p<0.001). Moreover, our analysis also revealed that disease severity and the number of co-morbidities were significantly correlated with PNI (p<0.001 and p=0.032, respectively), with co-morbidities such as diabetes mellitus (p=0.016) and asthma (p=0.034) demonstrating a particularly strong correlation.

Discussion

Older adultstend to be hospitalized, require intensive care, or die from COVID-19. Immunosenescence occurs with aging. Aging increases inflammation. Inflammaging may also cause cytokine release syndrome, a hyperinflammatory response that can damage organs and cause death.[7]

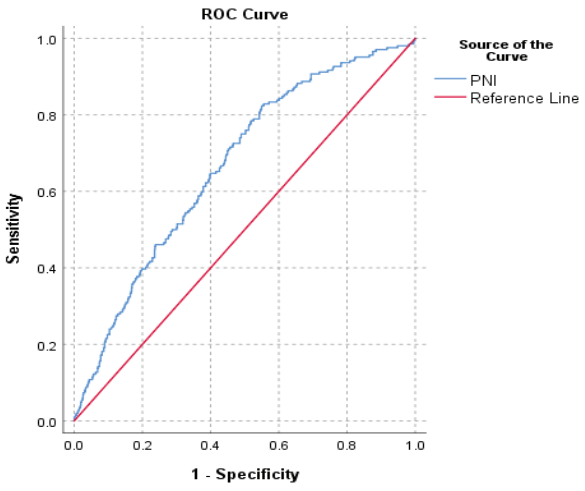

Figure 1 Receiver Operating Curve (ROC) for PNI
AUC = 0.666; cut-off ≤41.9; sensitivity 44.9%; specificity 82.4%; p<0.001

Table 3 Clinical Characteristics According to PNI

Variable	PNI≤41.9 (n=590)	PNI>41.9 (n=386)	p-value
Age, median (IQR), year	56 (48–64)	49 (37–58)	<0.001*
Male	57 (48–64)	49 (38–58)	<0.001*
Female	55 (48–62)	49 (36–58)	<0.001*
Sex, no (%)	590 (60.5)	386 (39.5)	<0.001α
Male	354 (36.3)	186 (19.1)	
Female	236 (24.2)	200 (20.5)	
Co-morbidities, no (%)			
Hypertension	277 (28.4)	166 (17.0)	0.226 α
Diabetes Mellitus	212 (21.7)	110 (11.3)	0.016 α
Chronic Kidney Disease	70 (7.2)	31 (3.2)	0.055 α
Cardiovascular disease	55 (5.6)	25 (2.6)	0.113 α
Stroke	24 (2.5)	15 (1.5)	0.887 α
Malignancy	6 (0.6)	1 (0.1)	0.255 β
Hepatitis B	8 (0.8)	3 (0.3)	0.541β
Lung tuberculosis	23 (2.4)	10 (1.0)	0.269α
COPD	3 (0.3)	1 (0.1)	1.000β
Asthma	11 (1.1)	16 (1.6)	0.034α
Obesity	280 (28.7)	197(20.2)	0.274 α
Number of Co-morbidities, no (%)			0.032α
0	88 (9.0)	83 (8.5)	
1	197 (20.2)	133 (13.6)	
2	169 (17.3)	91 (9.3)	
≥3	136 (13.9)	79 (8.1)	
The severity of illness, no (%)	590 (60.5)	386 (39.5)	<0.001α
Mild	24 (2.5)	70 (7.2)	
Moderate	84 (8.6)	156 (16.0)	
Severe	112 (11.5)	48 (4.9)	
Critically ill	370 (37.9)	112 (11.5)	
Body Mass Index			0.319α
Underweight (< 18.5)	16 (1.6)	5 (0.5)	
Normal (18.5–22.9)	168 (17.2)	99 (10.1)	
Overweight (23–24.9)	126 (12.9)	85 (8.7)	
Obesity (≥25)	280 (28.7)	197 (20.2)	
Leucocyte	8000 (6000–10.200)	7000 (5500–9100)	<0.001*
Lymphocyte	12 (8–17)	23 (15–31)	<0.001*
Albumin	3.1 (2.9–3.4)	3.9 (3.7–4.2)	<0.001*

α=Pearson Chi square, β=Fisher exact test, *=Mann -Whitney test

The present study observed that male patients exhibited a higher mortality rate, which aligns with prior research. Multiple factors may contribute to the increased mortality among COVID-19-positive males. It also may be related to the protective effects of estradiol, which has been demonstrated to boost immune responses to viral infections.[8] Lastly, behavioral factors, such as higher smoking rates and reduced mask-wearing and social distancing, may contribute to the higher mortality rate in males with COVID-19.

This study found that pre-existing conditions like hypertension, diabetes and obesity may need more intensive treatment to survive. As with previous study, this comorbidity has been identified as a significant risk factor for COVID-19-related mortality. [9] There are several possible mechanisms between diabetes mellitus and mortality. Uncontrolled hyperglycemia increases pro-inflammatory cytokines and decreases anti-inflammatory cytokines, which can enhance COVID-19-related inflammation. In regard to hypertension, the pathophysiology underlying the relationship between hypertension and COVID-19 mortality is not fully understood. Nevertheless, the renin-angiotensin-aldosterone system (RAAS) may affect COVID-19 severity. SARS-CoV-2, which produces COVID-19, penetrates cells through the angiotensin-converting enzyme 2 (ACE2) receptor, widely expressed in the lungs, heart, and kidneys. ACE2 controls the RAAS, which controls blood pressure. SARS-CoV-2 may overactivate the RAAS, causing hypertension.[10]

In obesity, adipose tissue's participation in inflammation and immunological response underlies the obesity-COVID-19 mortality. Inflammatory cytokines and adipokines, such as leptin, are produced by adipose tissue and decrease immunological function. Insulin resistance and obesity can cause persistent inflammation and immunological dysfunction. Obesity also reduces lung capacity and respiratory muscle strength, making breathing and recovering from respiratory illnesses difficult.[11] COVID-19 mortality is also strongly correlated with the number of co-morbidities. United Kingdom studies found COVID-19 individuals with two or more co-morbidities were found more likely to die.[12] In terms of disease severity, we also found it correlates with mortality. Many studies have linked COVID-19 illness severity to death. In a comprehensive review and meta-analysis of 46 studies, Chaudhry et al. observed that severe COVID-19 had a greater mortality rate than severity and mortality.[13]

In contrast, body mass index (BMI) did not significantly predict mortality in this study. This finding reflects inconsistencies in the literature, with some studies reporting associations between elevated BMI and mortality, while others do not. A meta-analysis by Klanget al. [14] involving over 149,000 patients, similarly found no strong relationship between BMI and mortality. This suggests thatBMI may not significantly predict mortality in COVID-19 patients. However, it is essential to note that BMI is just one aspect of body composition and does not necessarily reflect overall health status. Further research is needed to understand the potential association between BMI and other COVID-19 outcomes, such as disease severity and hospitalization rates.[14]

Leucocyte, lymphocyte, albumin, and PNI were measured. In all analyses, survivors exhibited greater median ranges than non-survivors ($p<0.001$). In the univariate study, leucocyte, lymphocyte, albumin, and PNI had significant relationships with survival ($p<0.001$), suggesting they may be good COVID-19 prognostic markers. Leucocyte, lymphocyte, and albumin demonstrated mortality correlation ($p<0.001$). PNI also correlated with death, suggesting that these parameters may be valuable prognostic indicators for COVID-19 patients, with similar results in univariate analysis. An Italian study found that COVID-19 patients who died had greater leukocyte counts than survivors. Leukocytosis in COVID-19 may signify immunological activation and inflammation. In severe COVID-19 cases, immune activation may cause cytokine storms that damage tissues and organs. Leukocytosis also increases the risk of thrombotic events, which can cause death in severe COVID-19 patients.[15]

Lymphocytes also have a role in COVID-19 immunity, and their numbers correlate with disease severity and mortality. According to several studies, prevalence and impact of lymphopenia, or low lymphocyte counts is frequent in severe COVID-19 patients and associated with poor clinical outcomes. In Wuhan, China, COVID-19 non-survivors had higher lymphopenia than survivors. Severe cases had much lower lymphocyte counts than moderate cases.[16] It is believed that the depletion of lymphocytes in COVID-19 may be due to the virus's ability to directly infect and destroy these cells, as well as the cytokine storm that can occur in severe cases, leading to immune dysregulation and lymphocyte apoptosis.[17]

Low plasma albumin levels are linked to

COVID-19 mortality. Albumin governs cell entry, maintains intravascular fluid balance, and binds medicines and other substances. It is a practical laboratory measure used to assess malnutrition. Inflammatory cytokines inhibit albumin production, a negative acute phase reactant. Inflammation lowers albumin. The reasons behind low albumin levels in COVID-19 are not yet fully understood. The liver produces albumin and circulates for 21 days. However, previous and current studies have found that severe cases of COVID-19 are more likely to have low levels of albumin compared to mild cases. This cannot be solely explained by liver dysfunction due to damaged liver cells, as the onset of hypoalbuminemia is much quicker than it takes for albumin to break down in the body. Therefore, it is unlikely that severe COVID-19 leads to decreased albumin production.[18]

As a computed inflammatory index of hypo albumin and lymphopenia conditions in patients, this study conducted an analysis of the PNI value as a predictor of COVID-19 mortality with a cut-off value of PNI<41.9. From the characteristic analysis of the cut-off value of PNI, disease severity and co-morbidities were found to be significantly correlated with PNI. The co-morbidities related to PNI are diabetes mellitus and asthma. Notably, no correlation was found between BMI and PNI, emphasizing the importance of considering multiple factors in assessing an individual's overall health status. In addition, our analysis revealed that several biomarkers, including leucocytes, lymphocytes, and albumin, were strongly correlated with PNI, providing further evidence of the potential clinical utility of these markers in assessing an individual's nutritional and immunological status. These findings have important implications for managing patients with various co-morbidities, highlighting the need for a comprehensive approach that considers a wide range of factors.

This study also found that PNI correlates with mortality. Fever, respiratory muscular exertion, and endocrinological disturbances that increase gluconeogenesis, protein breakdown, and lipid oxidation contribute to COVID-19-related malnutrition.[19] A recent study found that PNI significantly predicted ICU admission and mortality in COVID-19 patients.[20] In this study, a PNI cut-off value of <41.9 was significantly associated with mortality, comorbidities (notably diabetes and asthma), and disease severity. These results are in line with prior research. Studies by Aciksari et al.,[21] Cakirca et al.,[22] and Kosovali et al.[23] have each demonstrated similar PNI thresholds correlating with increased mortality in COVID-19 patients. In addition, Doganci et al. found that patients with a higher PNI (>44.7) were in the survivor group.[24]

The study also examines the relationship between PNI and several other variables, including age, gender, diabetes mellitus, asthma, number of co-morbidities, and disease severity. Hung et al.'s systematic review and meta-analysis supported the use of PNI as a predictor of mortality with an aggregated sensitivity of 0.76 and specificity of 0.71 (AUC of ROC: 0.79) but also revealed a negative correlation between PNI and disease severity in COVID-19 patients. In hospitalized COVID-19 patients, a low PNI was associated with a sevenfold increase in mortality risk, as demonstrated by their findings. The last one is the correlation between PNI and disease severity with several research findings indicating that PNI correlates with severity and can be used as a predictor.[25,26]

This study has several limitations. First, it is a single-center study, which means the findings may not be generalizable to all COVID-19 patients, as the cases included were likely severe or critical. Second, selection bias may have influenced the results, as only hospitalized patients were included in the study. Additionally, this retrospective study relied on data available from the patients' medical records during the first 48 hours of admission, including anthropometric measurements (weight and height). The vaccination status of the patients was not included in the study.

In conclusion, the prognostic nutritional index (PNI) is a simple and inexpensive method that may be quickly computed using common laboratory values. The results suggest that the PNI, which reflects the patients' immune system and nutritional status upon hospital admission, can be a reliable predictor of in-hospital mortality COVID-19 patients.

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