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ORIGINAL ARTICLE

Impact of Prognostic Nutritional Index (PNI) and Inflammatory Index in Critically Ill Patients with Acute Kidney Injury

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ABSTRACT

Background: Prognostic nutritional index (PNI) could be used to evaluate the immuno-nutritional status. There is a possible association between nutritional and inflammatory indices and acute kidney injury (AKI) among critically ill patients. This research aimed to investigate the relationship between PNI and all-cause mortality among critically ill patients with AKI.**Methods:**Sixty-nine patients with AKI who were at the intensive care units of Zagazig University hospitals in this prospective observational study were classified regarding outcome into (nonsurvivors): 25 patients who died (36.2%) and (Survivors): 44 (63.8%) patients who recovered. The CBC, CRP, Albumin, Serum Creatinine and Pelvi-abdominal ultrasound were assessed in all patients in addition to the calculation of PNI.**Results:** Statistically significant positive correlations were revealed between PNI and total protein, serum calcium, C reactive protein and potassium ($p<0.001$, <0.001, 0.026, and 0.004). Among factors significantly related to mortality in univariate analysis, increasing white blood cell count and high APACHE II score independently increase mortality risk by 1.211 and 4.763 folds, respectively.Total protein (unstandardized $\beta=2.111$, $p=0.001$), calcium (unstandardized $\beta=1.158$, $p=0.006$), and CRP (unstandardized $\beta=0.009$, $p=0.038$), significantly independently associated with PNI.The phosphorus level (unstandardized $\beta=1017.1$, $p=0.002$) is significantly independently associated with the inflammatory index.**Conclusion:** PNI and SII were comparable between died and survivors among Critically Ill Patients with AKI however, Neutrophil/lymphocyte ratio was significantly higher among died patients compared to survivors.PNI and SII still could be significant risk factors associated with increased mortality.

Keywords: Acute Kidney Injury, Critically Ill, Prognostic Nutritional Index, Systemic Inflammatory Index.

INTRODUCTION

Early detection of Acute kidney injury is mandatory for the initiation of proper treatment. The most commonly utilized markers for glomerular filtration rate (GFR) were plasma creatinine (PCR) and urine output; nevertheless, GFR gradually diminishes as damage progresses. Unfortunately, creatinine has limited diagnostic use due to its sensitivity to a wide range of renal and extrarenal variables. When high creatinine levels are detected, the diagnosis is often delayed, restricting treatment options [1].

Patients with AKI often suffer from malnutrition; several pathogenic mechanisms contribute to low plasma albumin levels, such as impaired synthesis, accelerated degradation and redistribution of albumin. Hypoal-buminemia is thought to result from an increase in albumin catabolism in hemodialysis patients caused by inflammation [2].

Because of its high concentration of reduced sulfhydryl groups, albumin is an antioxidant, yet too much of this protein can harm the kidneys. People with low albumin levels are more likely to develop acute kidney injury (AKI) and chronic kidney disease (CKD), which means their kidney function will not recover quickly and puts them at a higher risk of mortality [3].

The pathophysiology and development of acute kidney injury (AKI) are primarily influenced by inflammation, although non-inflammatory processes are sometimes present. It has been increasingly acknowledged in recent years that CRP plays a role in the development and progression of AKI by worsening local inflammation, reducing the development of damaged tubular epithelial cells and urging fibrosis of injured renal tissue [4].

Significant healthcare resources are consumed when acute kidney injury develops during sepsis since it increases patient morbidity, predicts higher mortality, significantly affects numerous organ functions, is associated with an increased period of stay in the intensive care unit, and so on. The pathogenesis of sepsis-associatedAKI is different from that of non-septic AKI, necessitating a different therapy. The pathogenesis, diagnostic methods and suitable treatment options for sepsis remain controversial despite remarkable progress in other areas of medicine [5].

Patients with malignancy and renal disease are more likely to have poor nutritional status, which is associated with higher rates of illness, death, hospitalization duration and worse quality of life. One can compute a prognostic nutritional index (PNI) utilizing serum albumin levels and total lymphocytes to assess immune-nutritional status. Our working hypothesis is that undernutrition is a contributing factor to the poor clinical outcomes seen in patients suffering from sepsis-induced acute kidney injury [6].

To our knowledge, this could be the first study in Zagazig University Hospitals to evaluate the association between nutritional and inflammatory indices and acute kidney injury in critically ill patients and improve the outcome of critically ill patients with AKI.

METHODS

Between June 2023 and December 2023, this prospective observational studywas carried out in the Intensive Care Unit, Internal Medicine Department, Zagazig University Hospitals,on 69AKI patients.Verbal and

written informed consentwas obtained from all participants after explaining the procedure and medical research. 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) (#10722/21-5-2023).

Cases with the following criteria were included:those aged 18 or older withAKI based on Kidney Disease Improving Global Outcomes [7] as long as they agreed to participate.Cases with the following characteristics were excluded: cases who were younger than 18 years, Patients whohad a history of renal transplants, who were on chronic dialysis, cases who had a nephrectomy and patients with a length of ICU stay less than 48 hours and patients with missed data during the study. All patients were subjected to Full history taking involving age, name, sex, history of medical diseases, and family history of end-stage renal disease. Complete clinical examination to exclude any hidden medical condition that may interfere with the results and diagnosis. Two groups of patients were classified regarding outcome into (nonsurvivors): 25 patients who died (36.2%) and (Survivors): 44 (63.8%) patients who recovered.

Laboratory investigations: Every patient had their peripheral blood samples taken the day they checked into the hospital. Albumin was assessed using spectrophotometry (Beckman Coulter AU 2700), C-reactive protein (CRP) was measured using an immunonephelometric technique, and the complete blood count (CBC) was performed (NFL BN-II). An enzyme-coupled assay was used to determine serum creatinine levels.The Chronic Kidney Disease Epidemiology Collaboration's (CKD) calculation was used to estimate the glomerular filtration rate. [GFR=141*min (Serum creatinine/kappa,1) ^{alpha}*max (Serum creatinine/kappa, 1) $^{-1.209}$ ∗ $0.993^{Age * Sex * Race}$] Pelvic and abdominal ultrasounds were done to exclude chronic renal failure. All patients referred for renal ultrasonography whose creatinine levels were measured on the same day of the procedure were considered.Ultrasound of the kidneys was conducted utilizing a 3.5–5 MHz sector curved array transducer For APACHE II score calculation, data for the acute physiology score (temperature, mean arterial pressure, pH, heart rate, respiratory rate, sodium, potassium, creatinine, hematocrit, white blood cell count, GCS, fraction of inspired oxygen (FiO2) with age according to appendix and the conventional B Mode grey scale ultrasonography (Knaus et al. 1985).The SII was defined as the number of platelets multiplied by neutrophils (10^9/L) and divided by the number of lymphocytes (10^9/L) (Jomrich et al., 2020).

Calculation of PNI: The following method was used to calculate the prognostic nutritional index (PNI), which was used to evaluate the nutritional state of patients before surgery. Ten times the serum albumin plus 0.005 times the total lymphocyte count. Modified Glasgow prognostic score: derived from measuring C-reactive protein and serum albumin. A high blood CRP concentration (>10 mg/L) was assigned one point to the mGPS, while a decreased serum albumin level (<35 g/L) was given 1 point to individuals with elevated CRP levels. A scale

from 0 to 3 was used to categorize patients' risks: low, middle, and high (2 points).

The endpoint of the study: For patients suffering from AKI, renal replacement therapy or death during one week of hospital stay were estimated.

Statistical analysis: Data analysis was performed using the software SPSS (Statistical Package for the Social Sciences) version 26. Categorical variables were described using their absolute frequencies and were compared using the chi-square test. Kolmogorov–Smirnov test was used to verify assumptions for parametric tests. Quantitative variables were described using their means and standard deviations or median and interquartile range according to the data type. The independent sample t-test (for normally distributed data) and Mann-Whitney test (for not normally distributed data) were used to compare quantitative data between the two groups. Binary backward Wald logistic regression was used to identify independent risk factors associated with specific health problems. Pearson and Spearman, rank correlation coefficient, was used to assess the strength and direction of correlation between two variables according to the type of data distribution. Linear stepwise regression analysis was performed to measure associated independent factors for the dependent variable, where all significant factors in correlation analysis were included. The level of statistical significance was set at $P<0.05$. A highly significant difference was present if $p\leq0.001$.

RESULTS

Table 1 show that he ages of the 69 patients involved in this study ranged from 23 to 58 years, with a mean age of 58.32 years. Females represented 49.3% of patients.

Table 2 show that older age was significantly associated with mortality ($p<0.001$) and there were statistically significant relations between mortality and white blood cells, neutrophil count, and neutrophil/lymphocyte ratio (significantly higher among non-survivors than survivors) ($p=0.009$, 0.016 respectively). There was a statistically significant relation between mortality and APACHE II (higher APACHE II was significantly associated with mortality) ($p<0.001$) .

Table 3: In univariate analysis, among factors significantly related to mortality, increasing white blood cell count and high APACHE II score independently increased the risk of mortality by 1.211 and 4.763 folds, respectively.

Table 4 show that there were statistically significant positive correlations between PNI and total protein, serum calcium, C reactive protein and potassium ($p<0.001$, <0.001, 0.026, and 0.004).

Table 5 show factors linked to PNI in a linear stepwise regression analysis: Total protein (unstandardized $\beta=2.111$, $p=0.001$), calcium (unstandardized $\beta=1.158$, $p=0.006$), and CRP (unstandardized $\beta=0.009$, $p=0.038$), significantly independently associated with PNI.

Table 6 show that there was a tatistically significant positive correlationsbetween the inflammatory index and serum creatinine, urea, potassium, and phosphorus ($p=0.005$, 0.004, 0.024, and 0.018).

Table 7: In the Linear stepwise regression analysis of the factors associated with the inflammatory index, Phosphorus level (unstandardized $\beta=1017.1$, $p=0.002$) was

significantly independently associated with the inflammatory index.

Supplementary Table 1 show that PNI was lower among the older group compared to younger ones in both survivors and died Moreover, PNI was also lower among died in both younger and older patients.

Table (1) Baseline data, Laboratory and clinical data, Nutrition scores of studied patients

	N=69	%
Gender:		
Female	34	49.3%
Male	35	53.7%
Comorbidities		
Diabetes	32	46.4%
Hypertension	44	63.8%
Hepatic	20	29%
UTI	42	60.9%
Outcome		
Died	25	36.2%
Survived	44	63.8%
	Mean ± SD	Range
Age (year)	58.32 ± 14.4	23 – 85
	Mean ± SD	Range
Hemoglobin (g/dl)	10.13 ± 3.06	6.2 – 22
WBCs ($10^3/mm^3$)	13.2(12 – 18) ¥	5 – 35
Neutrophils ($10^3/mm^3$)	7.1(5.96 – 10.25) ¥	2.7 – 21
Lymphocytes ($10^3/mm^3$)	2.01(1.3 – 3.7) ¥	0.1 – 4.8
	N=69	%
Neutrophil/lymphocyte ratio	3.38(1.76 – 8.94) ¥	0.56 – 39
Platelet ($10^3/mm^3$)	174(140 – 250) ¥	30 – 450
Total protein (g/dl)	6.08 ± 0.64	4 – 7.3

	N=69	%
Albumin (g/dl)	2.56 ± 0.35	1.7 – 3.1
Creatinine (mg/dl)	4.2(2.5 – 5.9) ¥	1.6 – 16
BUN (mg/dl)	85(59.5 – 113.5) ¥	42 – 230
Sodium	133.25 ± 5.56	125 – 135
Calcium	7.43 ± 0.88	6 – 12
Potassium	4.29 ± 0.82	2.5 – 6.2
Phosphorus	3.8 ± 0.86	2.5 – 6.5
CRP	132(80 – 180) ¥	50 – 467

Table (2) Relation between outcome and baseline data of studied patients and relation between outcome prognostic nutrition index and inflammatory index:

	Survivors N=44 (%)	Died N=25 (%)	χ^2	p
Gender: Female Male	 23 (67.6%) 21 (60%)	 11 (32.4%) 14 (40%)	 0.437	 0.509
Comorbidities Diabetes Hypertension Cardiac Hepatic UTI	 21 (65.6%) 26 (59.1%) 18 (58.1%) 12 (60%) 24 (57.1%)	 11 (34.4%) 18 (40.9%) 13 (41.9%) 8 (40%) 18 (42.9%)	 0.089 1.15 0.793 0.173 2.039	 0.765 0.284 0.373 0.677 0.153
	Mean ± SD	Mean ± SD	t	p
Age (year)	52.91 ± 13.83	67.84 ± 9.82	-4.753	<0.001**
	Recovery (n=44)	Died (n=25)	t	p
	Mean ± SD	Mean ± SD		
Hemoglobin (g/dl)	9.97 ± 3.02	10.4 ± 3.15	-0.567	0.573
WBCs (10^3/mm^3)	12.75(11.93 – 15) ¥	16(12.55 – 21.5) ¥	-2.6	0.009*
Neutrophil (10^3/mm^3)	6.9(5.68 – 8.78) ¥	9(6.7 – 12.95) ¥	-2.405	0.016*
Lymphocyte (10^3/mm^3)	2.06(1.43 – 4.05) ¥	1.8(0.91 – 2.75) ¥	-1.531	0.121
Neutrophil/lymphocyte ratio	3.17(1.64 – 6.53) ¥	6.43(2.7 – 11.34) ¥	-2.254	0.024*
Platelet (10^3/mm^3)	171.5(141.75 – 250) ¥	174(114 – 302) ¥	-0.194	0.846
Total protein (g/dl)	6.13 ± 0.72	6.01 ± 0.46	0.804	0.424
Albumin (g/dl)	2.6 ± 0.35	2.5 ± 0.35	1.064	0.291
Creatinine (mg/dl)	4(2.5 – 6.2) ¥	4.5(3 – 5.9) ¥	-0.65	0.516
Urea (mg/dl)	85(58.25–116.75) ¥	85(63 – 110) ¥	-0.432	0.666
Sodium	132.75 ± 3.96	134.12 ± 7.64	-0.835	0.41
Potassium	4.27 ± 0.77	4.32 ± 0.92	0.248	0.805

	Survivors N=44 (%)	Died N=25 (%)	χ^2	p
Calcium	7.46 ± 0.63	7.39 ± 1.21	0.309	0.758
Phosphorus	3.76 ± 0.74	3.88 ± 1.05	-0.551	0.584
CRP	126(80–195)¥	140(82.5 – 176) ¥	-0.125	0.9
	Recovery (n=44)	Died (n=25)	t	p
	Mean ± SD	Mean ± SD		
APACHE II	18.43 ± 2.82	24.32 ± 2.1	-9.111	<0.001**
	Survived (n=44)	Died (n=25)	t	p
	Mean ± SD	Mean ± SD		
PNI	25.97 ± 3.48	25.04 ± 3.47	1.068	0.289
	Median (IQR)	Median (IQR)	Z	p
Inflammatory index	509.11(260.29 – 1176.07)	1059.84(372.01 – 3090.9)	-1.76	0.078

UTI: Urinary tract infection, WBCS: White blood cells, BUN: Blood urea nitrogen, CRP: C reactive Protein, PNI: prognostic nutrition indext independent sample t test *p0.05 is statistically significant ¥data is represented as median and interquartile range and compared using Mann Whitney test

Table (3) Binary regression analysis of factors associated with mortality among AKI patients:

	β	p.	AOR	95% C.I.	
				Lower	Upper
WBCs	0.191	0.074	1.211	0.982	1.493
APACHE II score	1.561	0.001**	4.763	1.947	11.647

WBCS: White blood cells .AOR adjusted odds ratio CI Confidence interval **p≤0.001 is statistically highly significan

Table (4) Correlation between PNI and studied parameters:

	r	p
Age (year)	-0.12	0.324
Hemoglobin (g/dl)	-0.062	0.612
WBCs (10^3/mm^3)	-0.091¥	0.455
Neutrophil (10^3/mm^3)	-0.184¥	0.129
Lymphocyte (10^3/mm^3)	-0.023¥	0.853
Neutrophil/lymphocyte ratio	-0.038¥	0.757
Platelet (10^3/mm^3)	0.149	0.222
Total protein (g/dl)	0.49	<0.001**
Creatinine (mg/dl)	0.178¥	0.143

	r	p
Urea (mg/dl)	0.191¥	0.116
Sodium	-0.097	0.427
Potassium	0.343	0.004*
Calcium	0.436	<0.001**
Phosphorus	0.212	0.08
CRP	0.267¥	0.026*
APACHE II	-0.098¥	0.424
Inflammatory index	0.029¥	0.815

WBCS: White blood cells, BUN: Blood urea nitrogen, CRP: C reactive Protein

r Pearson correlation coefficient ¥Spearman rank correlation coefficient *p<0.05 is statistically significant **p≤0.001 is statistically highly significan

Table (5) Linear stepwise regression analysis of factors associated with PNI:

	Unstandardized Coefficients		Standardized Coefficients	t	p	95.0% Confidence Interval	
	β	Std. Error	Beta			Lower	Upper
(Constant)	2.945	3.837		0.767	0.446	-4.718	10.607
Total protein	2.111	0.562	0.387	3.757	0.001**	0.989	3.233
Calcium	1.158	0.411	0.292	2.816	0.006*	0.337	1.979
CRP	0.009	0.004	0.210	2.119	0.038*	0.000	0.017

CRP: C reactive Protein

*p<0.05 is statistically significant **p≤0.001 is statistically highly significant

Table (6) Correlation between Inflammatory index and studied parameters:

	r	p
Age (year)	0.076	0.537
Hemoglobin (g/dl)	0.205	0.091
WBCs (10^3/mm^3)	0.075	0.54
Total protein (g/dl)	0.112	0.361
Albumin (g/dl)	0.132	0.28

	r	p
Creatinine (mg/dl)	0.333	0.005*
Urea (mg/dl)	0.34	0.004*
Sodium	-0.044	0.718
Potassium	0.271	0.024*
Calcium	0.107	0.382
Phosphorus	0.284	0.018*
CRP	-0.039	0.748
APACHE II	0.212	0.08

WBCS: White blood cells, BUN: Blood urea nitrogen, CRP: C reactive Protein, PNI: prognostic nutrition index .r Spearman rank correlation coefficient *p<0.05 is statistically significant

Table (7) Linear stepwise regression analysis of factors associated with inflammatory index:

	Unstandardized Coefficients		Standardized Coefficients			95.0% Confidence Interval	
	β	Std. Error	Beta	t	p	Lower	Upper
(Constant)	-2329.88	1255.17		-1.856	0.068	-4835.22	175.45
Phosphorus	1017.1	321.996	0.36	3.159	0.002*	374.391	1659.802

*p<0.05 is statistically significant **p≤0.001 is statistically highly significant

DISCUSSION

Patients with AKI often suffer from malnutrition, which increases the risk of hospital death, complications and medical expenses and influences the incidence and development of AKI apart from non-nutritional variables [8].One accurate and thorough way to evaluate a patient's nutritional and inflammatory condition is by using the PNI. This systemic immune-nutrition indexconsiders serum albumin levels and peripheral lymphocyte counts [9].Xie et al. [10] reported that Patients with decompensated liver cirrhosis were discovered to have PNI as a predictive biomarker for all-cause death. Moreover, Zhang et al. [11] reported that Patients with type 2 diabetes have PNI as a solid predictive indicator for death from different causes.

This study aimed to determine whether there was a correlation between PNI and the overall mortality rate in critically sick patients with acute kidney injury.The present study found that older age was significantly associated with mortality. Also,Yu et al. [12] reported thatamong the 30-day mortality group and survival groups, the age of the patients was significantly different between both groups.In agreement with our findings,Gao and Yu. [7] documented that There was a low risk of 30-day mortality in individuals with high PNI who were 65 years old and older. Among patients with AKI, 51.87 percent were 65 years old or older and the average age was 64.61 years.

The present study revealed that TLC, neutrophil count and neutrophil/lymphocyte ratio were significantly higher among non-

survivors. Moreover, Higher APACHE II is significantly associated with mortality, similar to the findings of Gao and Yu. [6] and Yu et al. [12]. Another study found that NLR was a separate risk factor for RRT use and all-cause mortality in 1168 AKI hospitalized patients. After accounting for several possible confounding factors, patients with an NLR of 5.51 or above had a 1.7-fold increased chance of RRT necessity and a 2.5-fold increased risk of death compared to patients with a low NLR [13].

Patients with AKI had a considerably greater neutrophil-to-lymphocyte ratio, according to multiple studies (NLR). This meant that the lymphocyte count was lower in AKI patients compared to non-AKI patients [14,15]In addition, the present study results showed that serum albumin levels were lower in the non-survivor group compared to the survivor group, although this difference was not statistically significant. Yu et al. [16] reported that A high incidence of acute kidney injury (AKI) was seen in hospitalized patients with plasma albumin levels below 35 g/L. The risk of contrast-associated AKI (CA-AKI) was 1.59 times higher in patients with hypoalbuminemia, according to a meta-analysis by Liu et al. [17]. The risk of mortality was 94% higher in patients with insufficient albumin, according to research by Yu et al. [12]. A comparable finding was reported by Suzuki et al. [18] in hemodialysis patients; these patients had a declining trend in albumin levels three years before death. This is thought to be caused by a decrease in albumin synthesis due to liver malfunction.

Moreover, Gao and Yu. [7] reported that a reduced risk of 30-days death was revealed in patients with mild AKI who had a Sequential Organ Failure Assessment (SOFA) score of 6 or lower or a Simplified Acute Physiology Score (SAPS) II (SAPS-II) score of 43 or higher and had a high PNI. Patients with AKI often suffer from malnutrition, which is associated with more extended hospital stays and death from any cause. This could be explained by the long-term complications in patients in our study, which reflected higher PNI among critically ill patients.

The present study findings revealed that increasing TLC with low Lymphocyte count and high APACHE II score is independently associated with a higher rate of mortality.Also, Yu et al. [12] discovered that people whose lymphocyte counts were at the lowest 25% were 21% more likely to die than those whose counts were at the highest 25%. A reduced lymphocyte count is a sign of imminent death in individuals with renal failure, according to Troya et al. [19].

The present study revealed that the Inflammatory Index was higher among non-survivors than among survivors but without a statistically significant difference. Jia et al. [20] showed that there was an increased risk of death from any cause related to both lower and higher SII levels. Consequently, SII can potentially be a valuable novel biomarker for AKI patient prognosis prediction.

In the present study, PNL was lower among non-survivors than survivors but without a statistically significant difference. Besides, total protein and CRP are significantly independently associated with PNI.

High PNI levels were linked to a decreased risk of 30-day death, according to Gao and Yu [7]. Because lymphocyte count—a component of the PNI score—reflects age rather than nutritional status, it is not a suitable nutritional marker for older patients. This could be because the pathophysiological mechanisms underlying the association between high PNI and 30-day mortality in AKI are still not fully understood.Similarly, Miyasato et al. [21] showed that hemodialysis patients with higher PNI had a lower risk of death. However, when they separated the patients by age 65, they found a significant difference in PNI between the younger and

older groups, which is consistent with our results.

Shimoyama et al. [22] demonstrated in a pilot investigation that PNI preceded the prognosis of septic-associated AKI. The PNI was proposed by Hu et al. [23] as a potential tool for identifying coronary care unit patients at high risk of AKI and mortality. In addition, Sertdemir et al. [24] showed that PNI is a distinct risk factor for Contrast-induced Acute Kidney Injury (CI-AKI). In ST-elevation myocardial infarction, Kurtul et al. [25] found that PNI was substantially and inversely related to CI-AKI (STEMI) development.

According to Fanetti et al. [26], who used limited cubic splines with a critical value of 50.5 to analyze patients with renal failure, there is an L-shaped association between the PNI and all-cause mortality. Their 14-year study period and median follow-up of 5 years may account for this discrepancy since they more accurately represent the mortality risk of kidney failure patients.

After adjusting the possible confounding factors, Yu et al. [12] showed that decreased PNI was still linked to an increased risk of all-cause death in individuals with renal failure. According to their findings, those with a low PNI (<47.5) had a substantially lower overall survival rate compared to those with a high PNI (≥47.5).

Some studies have verified the significance of lymphocytes in the human immune system, and they are another component of the PNI score. The prognosis of critically sick patients is frequently dictated by the degree of inflammation, with a negative correlation between the number of lymphocytes and the advancement of inflammation. Patients' immunological condition and the severity of their sickness are reflected in PNI, which is why it is an independent risk factor for the prognosis of critically ill patients[27].

There is a statistically significant association between overall mortality and PNI &Glasgow prognostic score (GPS), according to a prior observational study by Kato et al. [28] that examined the relationship between six inflammation-based prognostic scores and mortality in hemodialysis patients. These scores include PNI, modified GPS, prognostic index, NLR, and platelet-to-lymphocyte ratio.

It is still not known how a low PNI score could cause critically sick patients to have a bad prognosis. According to some research, one possible explanation for the correlation between PNI and prognosisis that low serum albumin levels suggest the patient is underweight[29].

A patient's age, comorbidities, SOFA score and length of hospital stay before intensive care unit admission are among the many factors NUTRIC takes into account. A clear positive correlation exists between patients with more outstanding NUTRIC scores and higher six-month mortality [30].

In the present study, correlation analysis failed to identify any correlation between PNI and SII similar to Waley et al. [30] findings. Two objective and commonly performed laboratory tests serum albumin and lymphocyte count—form the basis of the PNI, which helps to remove human error among assessors. By quickly evaluating the patient's state, clinicians can implement clinical measures to lower the chance of fatality. Although there is some debate regarding whether or not critically ill patients' prognoses are improved by exogenous albumin infusion, it is widely acknowledged that inflammation control can enhance prognoses. Thus, reducing inflammation and raising the PNI score could be a useful therapeutic strategy [32].

Limitations: Since this study is a small-scale, center-prospective cohort, there is bound to be some selection bias in the results. Adjusting for all potential confounders, such as the unknown intervention outside of the intensive care unit, is challenging. Second,

our findings only apply to the Egyptian population. Differences in baseline serum albumin and TLC levels may have resulted from the fact that the condition in Egypt is distinct from other countries in terms of its etiology, comorbidities and severity.

CONCLUSION

Our study concluded that PNI and SII were comparable between died and survivors among Critically Ill Patients with AKI however, Neutrophil/lymphocyte ratio was significantly higher among died patients compared to survivors. Furthermore, PNI and SII still could be significant risk factors associated with increased mortality. Further well-designed, multi-center, large-scale studies are needed to reach a better understanding of the relation between PNI and AKI among critically ill patients to improve patient survival.

No potential conflict of interest was reported by the authors.

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Supplementary Table 1: Relation between age group and prognostic nutrition index and inflammatory index of studied patients:

	Recovery (n=44)	Died (n=25)	t	p
	Mean ± SD	Mean ± SD		
PNI				
Young	26.05 ± 3.62	25.26 ± 4.3	0.532	0.598
Older	25.68 ± 3.04	24.93 ± 3.15	0.583	0.566
t	0.273	0.216		
p	0.786	0.831		
	Median (IQR)	Median (IQR)	Z	p
Inflammatory index				
Young	540.95(261.54 – 1321.64)	640.48(276.69 – 3909.96)	-0.406	0.685
Older	446.67(213.46 – 781.43)	1200(492.08 – 3090.9)	-2.075	0.038*
Z	-0.975	-0.699		
p	0.33	0.485		

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