JURNAL KOMPLIKASI ANESTESI
VOLUME 12 NOMOR 3, August 2025

ARTICLE

Proteinuria as a Predictor of Acute Kidney Injury in Intensive Care Unit Patients at Dr. Sardjito Hospital

Ismail Ihsan, Calcarina Fitriani Retno Wisudarti, Ahmad Yun Jufan
Department of Anesthesiology and Intensive Care, Faculty of Medicine, Public Health, and Nursing, Gadjah Mada University, Yogyakarta, Indonesia
*Coresponden author: Adisty Chandra, DDepartment of Anesthesiology and Intensive Care, Faculty of Medicine, Public Health, and Nursing, Gadjah Mada University, Yogyakarta, Indonesia (fauzi_agungn@yahoo.co.id)

How to cite: Ihsan I, et al, Proteinuria as a Predictor of Acute Kidney Injury in Intensive Care Unit Patients At Dr. Sardjito Hospital. Jurnal Komplikasi Anestesi 12(3)-2025.

Receive: October 28, 2025
Accepted: December 16, 2025

ABSTRACT

Background: Acute kidney injury (AKI) accounts for 32–77% of mortality in intensive care unit (ICU) patients in Indonesia and increases the risk of death up to eightfold at Dr. Sardjito General Hospital. Patient outcomes are greatly influenced by early detection, but diagnosis still relies on serum creatinine, which only increases after severe kidney damage. Proteinuria has the potential to be an early predictor of AKI with simple, inexpensive, and rarely studied tests.

Objective: To assess proteinuria as a predictor of AKI in ICU patients at Dr. Sardjito General Hospital, Yogyakarta.

Subject and Methods: This prospective cohort study involved ICU patients who met the inclusion and exclusion criteria and were grouped based on proteinuria status. Data analysis was performed univariately (descriptive tables), bivariately (Chi-square/Fisher's Exact and Mann-Whitney tests), and multivariate (logistic regression) for variables with $p < 0.25$ at the bivariate stage. Model validation was performed using receiver operating characteristic (ROC) curve analysis to assess discriminatory ability and Hosmer–Lemeshow testing for model calibration.

Result: Of the 187 patients screened, 151 patients met the criteria (median age 52 years; 47.7% male). Proteinuria was found in 51.7% of patients, with a AKI incidence of 10.6%. Bivariate analysis showed a significant association between proteinuria and AKI (19.2% vs. 1.4%; RR 14.04; p<0.001). In addition, male gender (18.1% vs. 3.8%; RR 4.76; p=0.004) and a history of heart disease (33.3% vs. 8.1%; RR 4.12; p=0.011) were also associated with the occurrence of AKI. Conversely, an APACHE II score ≥17 only showed a trend toward an increased incidence of AKI (19.2% vs. 8.8%; p=0.155), but this was not statistically significant. Age, hypertension, and diabetes mellitus were not statistically significant. Multivariate analysis confirmed proteinuria as an independent predictor of AKI (OR 20.02; 95% CI: 1.32–304.52; p=0.031). The ROC curve yielded an AUC of 0.782 (p=0.001) with a sensitivity of 93.8% and specificity of 53.3%, PPV of 19.2% and NPV of 98.6%. The Hosmer–Lemeshow test indicated good model calibration (p=0.225). In addition, patients with proteinuria more often required vasopressors (34.6% vs. 12.3%; p=0.001), while diuretic use was more common in the group without proteinuria (19.2% vs. 5.1%; p=0.008).

Conclusion: Proteinuria is a moderate predictor of acute kidney injury (AKI) in ICU patients at Dr. Sardjito General Hospital. Absence of proteinuria may serve as a strong indicator for ruling out the risk of AKI

Keywords: Acute kidney injury, intensive care unit, proteinuria, predictor

INTRODUCTION

Acute kidney injury (AKI) is a global health problem that affects more than 10 million people worldwide each year. This condition is defined as a significant decline in kidney function within less than seven days, with criteria established by KDIGO (Kidney Disease: Improving Global Outcomes) including an increase in serum creatinine ≥0.3 mg/dL within 48 hours, an increase of ≥1.5 times the baseline value within seven days, or a decrease in urine output <0.5 ml/kg/hour for six hours.[1] The incidence of AKI in intensive care units (ICUs) is reported to reach 30–60%, with mortality ranging from 50–80%, and contributes to increased ICU length of stay.[2,3] In Indonesia, a study at Dr. Sardjito General Hospital showed that AKI is an important predictor of ICU patient mortality with a relative risk of 8.0 (p<0.05).[2,3]

Early detection of AKI is crucial in improving patient clinical outcomes. AKI diagnosis currently relies on serum creatinine levels, but this test is less sensitive because it is influenced by various non-renal factors (age, muscle mass, hydration status, dietary intake) and only increases after significant kidney damage has occurred.[4,5] Meanwhile, in subclinical AKI, structural damage may occur even though serum creatinine levels remain within normal limits due to the presence of tubular functional reserve (TFR).[6]

Proteinuria is one parameter that reflects the integrity of the glomerular filtration barrier. Under normal conditions, urinary protein excretion is <150 mg/24 hours, equivalent to a negative or 1+ dipstick urine test result. Several studies have shown that proteinuria is associated with an increased risk of CKD and the need for renal replacement therapy.[7-9] Preoperative proteinuria is associated with postoperative CKD (OR 1.74; 95% CI 1.45–2.09), the need for renal replacement therapy (OR 1.70; 95% CI 1.25–2.32), and severe AKI (OR 3.24; 95% CI 1.42–7.38).[7] Similarly, dipstick proteinuria ≥30 g/100 mL was found to increase the risk of AKI requiring dialysis (aOR 2.79).[8]

Other parameters such as urine output and various renal biomarkers (e.g., NGAL, IL-18, KIM-1, cystatin C) have also been studied, but their sensitivity and specificity have not been consistent, in addition to requiring high costs and complex laboratory methods.[5] In contrast, proteinuria testing is relatively simple, inexpensive, and easy to implement in ICU facilities, including in Indonesia.[10,11]

To date, several studies at Dr. Sardjito General Hospital have evaluated mortality in patients with acute kidney injury in the ICU, but no studies have specifically assessed proteinuria as a predictor of acute kidney injury in this population. Therefore, this study was conducted to assess proteinuria as a predictor of acute kidney injury in ICU patients at Dr. Sardjito General Hospital, Yogyakarta.

METHODS

This study is a prospective cohort study based on primary data conducted at the ICU of Dr. Sardjito General Hospital, Yogyakarta. Recruitment was carried out using consecutive sampling in 2025 after obtaining ethical approval from the Research Ethics Committee of the Faculty of Medicine, Public Health, and Nursing, Gadjah Mada University, as well as research permission from the Education and Training Unit of Dr. Sardjito General Hospital. The study population consisted of all ICU patients, while the accessible population included those who met the inclusion criteria: aged 18–75 years and without exclusion criteria such as a history of autoimmune disease, pregnancy, or palliative status.[12] Patients who died within 48 hours, had anuria at ICU admission, or presented with serum creatinine levels >1.2 mg/dL upon admission were excluded.

The minimum sample size was calculated using Fleiss formula based on the proportion of acute kidney injury (AKI) in patients with and without proteinuria.[13,14] The calculation showed a minimum requirement of 112 patients, with an additional 20% dropout rate, resulting in a total minimum sample size of 135 patients. All patients underwent urine protein testing upon admission to the ICU using photometric urinalysis with a fully automated urine chemistry analyzer.[15] Proteinuria was determined if the urinalysis results showed ≥1+ protein. Serum creatinine levels were checked at 48 hours and

7 days after admission, or when the patient was discharged from the ICU if the length of stay was <7 days.[16]

The main variables in this study were proteinuria as an independent variable and AKI occurrence as a dependent variable, defined as an increase in serum creatinine levels >1.2 mg/dL within 48 hours–7 days or an increase >0.3 mg/dL compared to baseline values.[17] Confounding variables analysed included age, hypertension, diabetes mellitus, and heart disease. Urine collection was performed using an aseptic non-touch technique,[18,19] while venous blood collection for creatinine testing was performed according to laboratory standards.[20]

Data were analyzed using SPSS version 26. Categorical variables were presented as frequencies and percentages, while numerical data were expressed as median (minimum–maximum). The association between proteinuria and AKI was analyzed using the Chi-square test and relative risk (RR) calculation, while Fisher's Exact test and Mann-Whitney test were applied as appropriate according to data distribution and type. Variables with $p < 0.25$ in the bivariate analysis were included in logistic regression models to control for confounders, and results were reported as odds ratios (OR) with 95% confidence intervals (CI). Statistical significance was defined as $p < 0.05$. Model validation was conducted using receiver operating characteristic (ROC) curve analysis to assess discriminative ability and the Hosmer–Lemeshow test for calibration.

RESULTS

This study was conducted among patients admitted to the Intensive Care Unit (ICU) of Dr. Sardjito General Hospital during the study period. Of the 187 patients screened, 36 were excluded for not meeting the study criteria, due to age ineligibility, incomplete laboratory data, a history of autoimmune disease, or death within 48 hours of ICU admission. Thus, 151 patients were included in the final analysis. The baseline characteristics of the study population showed a median age of 52 years (range 17–75), with 47.7% males and 52.3% females. The median body mass index (BMI) was 22.2 kg/m² (range 12.7–35.16), which fell within the normal category. Most patients were classified as ASA II (46.9%) and ASA III (46.9%), reflecting moderate to severe clinical status. The most common comorbidities were hypertension (28.5%), diabetes mellitus (12.6%), and heart disease (9.9%). Based on the APACHE II score, the majority of patients (82.8%) had a score <17 (low risk), while 17.2% were categorized as high risk (≥17). Nearly all patients received mechanical ventilation (96%), approximately one-fourth required vasopressors (23.8%), and 11.9% received diuretics. ICU mortality was 9.9%. Urinalysis revealed proteinuria in 51.7% of patients, distributed as 1+ (31.1%), 2+ (17.9%), and 3+ (2.6%). Acute kidney injury (AKI) was identified in 10.6% of patients. These findings indicate that proteinuria was relatively common among critically ill patients and may be associated with worse clinical outcomes.

Bivariate analysis showed that patients with proteinuria had a significantly higher risk of developing AKI compared with those without proteinuria (19.2% vs 1.4%; $p < 0.001$). The relative risk (RR) was 14.04 (95% CI: 1.90–103.62), confirming that proteinuria increased the risk of AKI by approximately 14-fold.

Other clinical variables were also analysed for potential associations with AKI. Male sex was significantly associated with AKI (18.1% vs 3.8% in females; $p = 0.004$; RR = 4.76). A history of heart disease was also associated with higher AKI incidence (33.3% vs 8.1%; $p = 0.011$; RR = 4.12). Meanwhile, APACHE II score ≥17 showed a trend toward increased AKI occurrence (19.2% vs 8.8%), but the association was not statistically significant ($p = 0.155$). Other variables, such as age, hypertension, and diabetes mellitus, showed no significant association with AKI.

Multivariate logistic regression analysis demonstrated that proteinuria remained an independent predictor of AKI (OR 20.02; 95% CI: 1.32–304.52; $p = 0.031$), even after controlling for other variables. In contrast, male sex, history of heart disease, APACHE II score ≥17, and age were not significant predictors.

Validation of the prediction model demonstrated that proteinuria had a reasonably good discriminatory ability in predicting AKI.

Table 1. Characteristics of Subjects

		Median (min-max)	n (%)
Age		52 (17-75)	
Gender	Male		72 (47.7%)
	Female		79 (52.3%)
Weight		56 (9-105)	
Height		160 (140-187)	
BMI		22.2 (12.7-35.16)	
ASA status	1		3 (2.3%)
	2		61 (46.9%)
	3		61 (46.9%)
	4		5 (3.8%)
Hypertension	Yes		43 (28.5%)
	No		108 (71.5%)
Heart Disease	Yes		15 (9.9%)
	No		136 (90.1%)
Diabetes	Yes		19 (12.6%)
	No		132 (87.4%)
Total Apache II Score	≥17		26 (17.2%)
	<17		125 (82.8%)
Ventilator	Yes		145 (96%)
	No		6 (4%)
Vasopressor	Yes		36 (23.8%)
	No		115 (76.2%)
Diuretics	Yes		18 (11.9%)
	No		133 (88.1%)
Dyspnea	Yes		5 (3.3%)
	No		146 (96.7%)
Proteinuria	Negative		73 (48.3%)
	1+		47 (31.1%)
	2+		27 (17.9%)
	3+		4 (2.6%)
AKI	Yes		16 (10.6%)
	No		135 (89.4%)

Table 2. Bivariate Analysis of Proteinuria with AKI

		AKI	No AKI	P	RR	CI 95%
Proteinuri	Yes	15 (19.2%)	63 (80.8%)	<0.001*	14.04	1.90-103.62
	No	1 (1.4%)	72 (98.6%)			

Table 3. Bivariate Analysis of Confounding Variables with AKI

		AKI	NO AKI	*p*	RR	CI 95%
Age		56.5 (23-75)	52 (17-74)	0.147		
Gender	Male	13 (18.1%)	59 (81.9%)	0.004*	4.76	1.41-16.01
	Female	3 (3.8%)	76 (96.2%)			
Hypertension	Yes	6 (14%)	37 (86%)	0.393$	1.51	0.58-3.89
	No	10 (9.3%)	98 (90.7%)			
Heart Disease	Yes	5 (33.3%)	10 (66.7%)	0.011*$	4.12	1.65-10.27
	No	11 (8.1%)	125 (91.9%)			
Diabetes	Yes	2 (10.5%)	17 (89.5%)	1.000$	0.99	0.24-4.03
	No	14 (10.6%)	118 (89.4%)			
Total APACHE II Score	≥17	5 (19.2%)	21 (80.8%)	0.155$	2.47	0.78-7.83
	<17	11 (8.8%)	114 (91.2%)			

Table 4. Logistic Regression Test

			95% C.I	
	P	OR	Lower	Upper
Proteinuria	.031*	20.015	1.316	304.518
Age	.787	.992	.935	1.053
Male Gender	.181	4.772	.484	47.081
Heart Disease	.083	7.084	.776	64.673
Total APACHE II score (≥17)	.170	5.281	.490	56.866

ROC curve analysis yielded an AUC of 0.782 (95% CI: 0.68–0.88; p=0.001), indicating moderate discriminative ability. The optimal cutoff was determined at proteinuria ≥1+, with a sensitivity of 93.8% and a specificity of 53.3%. Furthermore, predictive value analysis showed a negative predictive value (NPV) of 98.63%, suggesting that the absence of proteinuria can reliably exclude the risk of AKI in ICU patients. In contrast, the positive predictive value (PPV) was only 19.23%, indicating that not all patients with proteinuria ≥1+ progressed to

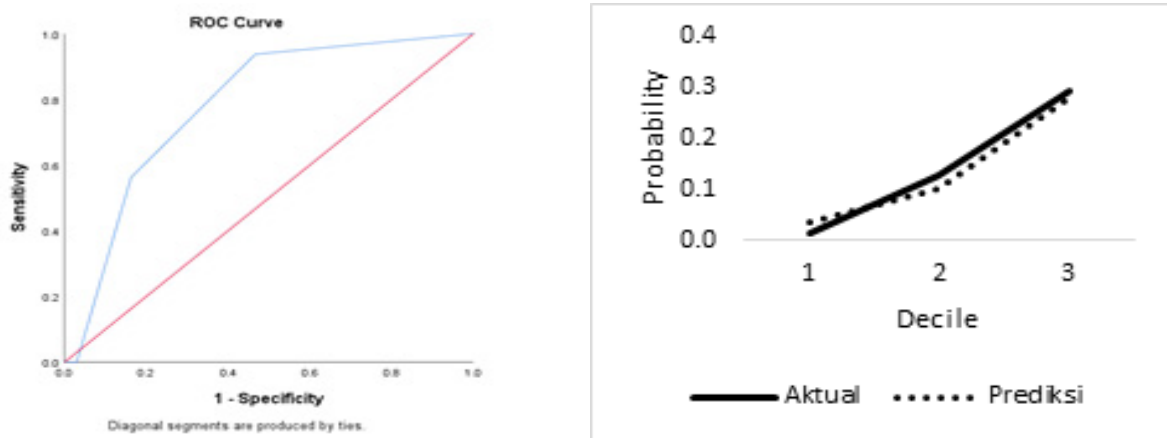

Figure 1. ROC Curve and Hosmer-Lemeshow Calibration Test

Table 5. Youden Index Table

Proteinuria	Sensitivity	1 - Specificity	Youden index
0	1.000	1.000	0.000
1	0.938	0.467	0.471
2	0.563	0.163	0.400
3	0.000	0.030	-0.030

Tabel 6. Other Outcome

	Ventilator	Vasopressor	Diuretic	Dyspnea	Death
Proteinuria	76 (97.4%)	27 (34.6%)	4 (5.1%)	5 (6.4%)	11 (14.1%)
Negative	69 (94.5%)	9 (12.3%)	14 (19.2%)	0 (0%)	4 (5.5%)
P	0.430$	0.001*	0.008*	0.059$	0.077

AKI. Calibration testing using the Hosmer–Lemeshow test produced a p-value of 0.225, demonstrating good agreement between the predicted probability of AKI and the actual observed events in the study population.

Additional outcome analysis showed that vasopressor use was more frequent in patients with proteinuria compared to those without proteinuria (34.6% vs 12.3%; p = 0.001). Conversely, diuretic use was higher among patients without proteinuria (19.2% vs 5.1%; p = 0.008). Mortality tended to be higher in the proteinuria group (14.1% vs 5.5%), although this difference did not reach statistical significance (p = 0.077).

DISCUSSION

Acute kidney injury (AKI) is defined as a rapid decline in renal function over hours to days, characterized by elevated serum creatinine levels, reduced glomerular filtration rate (GFR), or decreased urine output. In ICU patients, AKI is associated with high morbidity and mortality, as well as prolonged hospital stays and increased healthcare costs.[2,3] Currently, AKI diagnosis still largely depends on serum creatinine, which typically rises only after approximately 50% of renal function is lost.[4,5] This delay hampers timely diagnosis and management. In this context, proteinuria may serve as a useful predictive biomarker.[7,8,12]

The study confirmed that proteinuria is an independent predictor of AKI in ICU patients. Logistic regression analysis demonstrated that patients with proteinuria had nearly a 20-fold higher likelihood of developing AKI compared to those without proteinuria. Furthermore, ROC curve analysis showed an AUC of 0.782, indicating moderate discriminative ability. With high sensitivity (93.8%) and a strong negative predictive value (NPV) of 98.63%, the absence of proteinuria serves as a reliable indicator to rule out AKI risk in ICU patients. This has important clinical implications, as this simple test may help exclude low-risk patients, allowing interventions to be more effectively focused on those at higher risk.

However, the specificity (53.3%) and positive predictive value (PPV) (19.23%) were relatively low, indicating that the presence of proteinuria alone is not sufficient to confirm AKI. This explains why the majority of patients with proteinuria in this study did not develop AKI (80.8%). This phenomenon is consistent with the concept of subclinical AKI, a condition in which structural kidney injury occurs even though creatinine levels and GFR remain normal due to tubular functional reserve. Recent studies have reported that 15–20% of patients who do not meet AKI criteria based on serum creatinine still experience acute tubular injury, which is associated with poor clinical outcomes. Furthermore, proteinuria in ICU patients is heterogeneous; in some cases, it may be transient due to non-renal systemic stress, and the intensity of proteinuria also plays a critical

role, with moderate–severe proteinuria (≥2+) being more strongly associated with AKI than mild proteinuria.[6,10,11,12, 21]

These findings are consistent with previous studies reporting a significant association between proteinuria and AKI. Preoperative proteinuria has been shown to increase the risk of postoperative AKI in both cardiovascular and non-cardiovascular surgeries.[7] A similar association was reported in hospitalized COVID-19 patients.[17] In patients with severe burns, proteinuria was demonstrated to be a significant risk factor for AKI.[12] Furthermore, proteinuria and hematuria were associated with both AKI and mortality in critically ill patients.[13] Pathophysiologically, proteinuria in critically ill patients reflects glomerular endothelial damage, systemic inflammatory activation, and renal microcirculatory dysfunction. These mechanisms precede the decline in GFR or rise in serum creatinine, explaining why proteinuria emerges earlier as a predictive biomarker.[8,9]

The incidence of AKI in this study was 10.6%, with mortality being higher in the proteinuria group, although not statistically significant. These findings are consistent with a study at Cipto Mangunkusumo Hospital (RSCM), which reported an AKI prevalence of 12.25% with 32.59% mortality,[22] and a study at Hasan Sadikin General Hospital, Bandung, which found an AKI prevalence of 41.2% with 77% mortality among septic ICU patients.[23] A multicenter study in the United States also reported similar rates, with hospital-acquired AKI at 13.6% and community-acquired AKI at 9.2%.[24]

In this study, male sex and a history of heart disease were significantly associated with AKI in bivariate analysis, but these associations were lost in multivariate analysis. This suggests that proteinuria was a more robust predictor than other clinical variables. Nevertheless, biological plausibility supports sex differences in renal vulnerability, with estrogen exerting protective effects, while heart disease may worsen renal perfusion through cardiorenal interactions.[25]

Proteinuria was also associated with other clinical outcomes. Patients with proteinuria required vasopressors more frequently, suggesting greater hemodynamic instability, whereas diuretic use was higher in the non-proteinuria group. Mortality was higher among patients with proteinuria, although not statistically significant. These results align with previous literature indicating that proteinuria is associated with increased need for intensive interventions and poorer prognosis in critically ill patients.[26] This study has some limitations, proteinuria assessment was semi-quantitative using dipstick urinalysis, which may affect accuracy. Future studies should employ quantitative methods to enhance measurement precision.

CONCLUSION

Proteinuria is a moderate predictor of acute kidney injury (AKI) in ICU patients at Dr. Sardjito General Hospital. Absence of proteinuria may serve as a strong indicator for ruling out the risk of AKI.

RECOMMENDATION

Proteinuria testing is simple and easy to perform; therefore, it should be considered as part of routine screening in the ICU for early detection of AKI risk. Future studies are encouraged to employ quantitative measurement methods.

REFERENCES

1. KDIGO Clinical Practice Guideline for Acute Kidney Injury. Kidney Int Suppl. 2012;2(1):1–138.
2. Ardiansyah F, Widyastuti Y, Jufan AY. Identifikasi faktor risiko kematian di ICU RSUP Dr. Sardjito. J Komplikasi Anestesi. 2021;9(2):35–42. Doi:10.22146/jka.v9i2.8345.
3. Baskoro R, Fitriani C. Acute kidney injury sebagai faktor prediktor kematian pasien di ICU RSUP Dr. Sardjito. UGM Repository. 2014. Available from: https://doi.org/10.22146/jka.v2i2.7206
4. Li SY, Chuang CL, Yang WC, Lin SJ. Proteinuria predicts postcardiotomy acute kidney injury in patients with preserved glomerular filtration rate. J Thorac Cardiovasc Surg. 2015;149(3):894–9. Doi: 10.1016/j.jtcvs.2014.10.054

5. Parikh CR, Lu JC, Coca SG, Devarajan P. Tubular proteinuria in acute kidney injury: a critical evaluation of current status and future promise. Annals of Clinical Biochemistry: Ann Clin Biochem. 2010;47(4):301–12. Doi: 10.1258/acb.2010.010076
6. Zou C, Wang C, Lu L. Advances in the study of subclinical AKI biomarkers. Front Physiol. 2022;13:1–15. Doi: https://doi.org/10.3389/fphys.2022.960059.
7. Chang CY, Chien YJ, Kao MC, Lin HY, Chen YL, Wu MY. Pre-operative proteinuria, postoperative acute kidney injury and mortality. European J Anaesthesiol. 2021;38(7):702–14. Doi: 10.1097/EJA.0000000000001542.
8. Grams ME, Astor BC, Bash LD, Matsushita K, Wang Y, Coresh J. Albuminuria and Estimated Glomerular Filtration Rate Independently Associated with Acute Kidney Injury. J Am Soc Nephrol. 2010;21(10):1757–64. Doi: https://doi.org/10.1681/asn.2010010128.
9. Sharma A, Gupta N, Srivastava D. Carotid intima-media thickness, flow-mediated dilatation and proteinuria in patients of human immunodeficiency virus-positive patients: A case–control study. J Fam Med Prim Care. 2018;7(2):362–67. Doi: 10.4103/jfmpc.jfmpc_34_17.
10. Haider MZ, Aslam A. Proteinuria . StatPearls. [internet]. 2023. Available from: https://www.ncbi.nlm.nih.gov/books/NBK564390/.
11. Wahed A, Quesada AE, Dasgupta A. Monoclonal gammopathy and its detection. In: Hematology and coagulation. 2015:117–132. Doi:10.1016/B978-0-12-800241-4.00007-3.
12. Hu JY, Meng XC, Han J, Xiang F, Fang YD, Wu J, et al. Relation between proteinuria and acute kidney injury in patients with severe burns. Crit Care. 2012;16(R172):1–9. Doi: https://doi.org/10.1186/cc11649.
13. Han SS, Ahn SY, Ryu J, Baek SH, Chin HJ, Na KY, et al. Proteinuria and hematuria are associated with acute kidney injury and mortality in critically ill patients: a retrospective observational study. BMC Nephrol. 2014;15(93):1–8. Doi: https://doi.org/10.1186/1471-2369-15-93
14. Charan J, Biswas T. How to Calculate Sample Size for Different Study Designs in Medical Research?. Indian J Psychol Med. 2013;35(2):121–6. Doi: 10.4103/0253-7176.116232
15. Sysmex Corporation. Fully automated urine chemistry analyzer: UC-3500 instructions for use. Kobe: Sysmex; 2016. Available from: https://www.sysmex-ap.com/product/uc-3500/
16. Yeter HH, Yildirim T, Eyupoglu ND, Pasayev T, Aslan AT, Cetik S, et al. Mild to moderate proteinuria is a heralding sign for acute kidney injury and mortality for intensive care unit patients. Turk J Med Sci. 2019;49(2):543–50. Doi: http://doi.org/10.3906/sag-1802-183
17. Chaudhri I, Moffitt R, Taub E, Annadi RR, Hoai M, Bolotova O, et al. Association of proteinuria and hematuria with acute kidney injury and mortality in hospitalized patients with COVID-19. Kidney & Blood Pressure Research. 2020;45(6):1018–32. Doi: https://doi.org/10.1159/000511946.
18. Herawati S. Proper sampling and analysis of urine. Departemen Patologi Klinis, Fakultas Kedokteran Universitas Udayana/RSUP Prof Dr. IGNG Ngoerah. Denpasar. 2023.
19. Shepherd E. Specimen collection 2: obtaining a catheter specimen of urine. Nursing Times. 2017: 29–31. Available from: https://s3-eu-west-1.amazonaws.com/emap-moon-prod/wp-content/uploads/sites/3/2017/07/170712-Specimen-collection-2-obtaining-a-catheter-specimen-of-urine_V2.pdf.
20. UPTD Laboratorium Kesehatan Daerah Pasuruan. Pedoman prosedur laboratorium klinik. Pasuruan: UPTD; 2017. Available from: https://labkesda.pasuruankab.go.id/halaman/standar-pelayanan-klinik
21. Park S, Woo J, Leem S, Heo NH, Cho NJ, Gil H, et al. Transiently observed trace albuminuria on urine dipstick test is associated with All-Cause death, cardiovascular death, and incident chronic kidney disease: a National Health Insurance Service-National Sample cohort in Korea. Frontiers Cardiovasc Med. [Internet]. 2022;9:882599:1–10. Available from: https://doi.org/10.3389/

fcvm.2022.882599.
22. Lydia A, Rebecca RV, Sedono R, Arif Mansjoer. FFactors associated with mortality of intensive care unit patients with acute kidney injury at Cipto Mangunkusumo national central general hospital. Acta Med Indones. 2019;51(4):324–30. Available from: https://pubmed.ncbi.nlm.nih.gov/32041916/
23. Hidayat H, Pradian E, Kestriani ND. Angka Kejadian, Lama Rawat, dan Mortalitas Pasien Acute Kidney Injury di ICU RSUP Dr. Hasan Sadikin Bandung. J Anestesi Perioperatif. 2020;8(2):108–18. Doi: https://doi.org/10.15851/jap.v8n2.2054
24. Golestaneh L, Basalely A, Linkermann A, El-Achkar TM, Kim RS, Neugarten J. Sex, Acute kidney injury, and Age: A Prospective Cohort Study. Am J Kidney Dis. 2024;85(3):329–38. e1. Doi: 10.1053/j.ajkd.2024.10.003
25. Ronco C, Bellasi A, Lullo LD. Cardiorenal Syndrome: An Overview. Adv Chronic Kidney Dis. 2018;25(5):382–90. Doi: 10.1053/j.ackd.2018.08.004
26. AbdaAllah EE. Proteinuria and outcome of acute kidney injury in critically III patients. Jordan J Applied Sci Nat Sci Series. 202; 15:1-5. Available from: https://jas.asu.edu.jo/index.php/jjoas-n/article/download/2181/959/18017