DOI: https://doi.org/10.54393/pjhs.v6i1.2286

PAKISTAN JOURNAL OF HEALTH SCIENCES
(LAHORE)
https://thejas.com.pk/index.php/pjhs
ISSN (E): 2790-9352, (P): 2790-9344
Volume 6, Issue 01 (January 2025)

Original Article

OPEN ACCESS

Relationship of Hypoalbuminemia in Colistin-Induced Acute Kidney Injury (AKI) among Adult Intensive Care Patients

Sughandh Umer Memon[1*], Saima Samad[1], Nazish Misbah[1], Saima Muzaffar[2], Sadia Ishaque[1] and Shumaila Kamran[3]

[1]Shaheed Mohtarma Benazir Bhutto Institute of Trauma, Karachi, Pakistan
[2]Dr. Ziauddin Hospital Clifton, Karachi, Pakistan
[3]Dr. Ruth K. M. Pfau Civil Hospital, Karachi, Pakistan

ARTICLE INFO

Keywords:
Acute Kidney Injury, Colistin Therapy, Intensive Care Unit, Hypoalbuminemia

How to Cite:
Memon, S. U., Samad, S., Misbah, N., Muzaffar, S., Ishaque, S., & Kamran, S. (2025). Relationship of Hypoalbuminemia in Colistin-Induced Acute Kidney Injury (AKI) among Adult Intensive Care Patients: Hypoalbuminemia in Colistin-Induced Acute Kidney Injury. Pakistan Journal of Health Sciences, 6(1), 162-167. https://doi.org/10.54393/pjhs.v6i1.2286

***Corresponding Author:**
Sughandh Umer Memon
Shaheed Mohtarma Benazir Bhutto Institute of Trauma, Karachi, Pakistan
drsughandh.memon@gmail.com

Received date: 5th October, 2024
Acceptance date: 18th January, 2025
Published date: 31st January, 2025

ABSTRACT

Acute kidney injury incidence ranges from 30-60% among critically ill patients and stands as the primary death cause within this population. A serious concern is a global rise in major drug-resistant-gram-negative organisms among hospital-acquired infections. **Objectives:** To determine the incidence of colistin-induced acute kidney injury in intensive care patients receiving colistin therapy and to investigate its relationship with albumin levels. **Methods:** It was a follow-up prospective cohort study executed at Shaheed Mohtarma Benazir Bhutto Institute of Trauma Pakistan in an adult intensive care unit over 6 months. The study end-point was an injury in intensive care injury at the end of colistin therapy. A total of 250 patients were studied. **Results:** The median age of patients was 40 (IQR=22-48) years with an age range of 18-70 years. The majority of patients were male (75.2%). Median colistin dosage was 4 (IQR=3.5-4.5) MIU. In univariate analysis, the risk of developing injury in intensive care was significantly increased with increased age, use of nephrotoxic drugs, and increasing colistin dosage whereas injury in intensive care risk was decreased with increasing albumin levels. In a multivariable model, only colistin dosage was found to be significantly associated with increasing injury in intensive care risk with increasing colistin dosage. **Conclusions:** It was concluded that the present study analyzed a higher burden of acute kidney injury incidence following colistin therapy. Albumin levels were not found to be linked to acute kidney injury incidence in the multivariable model. Acute kidney injury incidence was significantly related to increasing colistin dosage.

INTRODUCTION

Acute kidney injury (AKI) is a frequent issue in patients with deteriorating health, leading to higher morbidity and mortality rates. It also results in longer hospital stays and increased healthcare expenses [1]. In critically ill patients, AKI occurs in about 30% to 60% of cases, making it a major cause of death in this group. The Kidney Disease Improving Global Outcomes (KDIGO) guidelines define AKI as a rapid loss of kidney function, indicated by a rise in serum creatinine (SCr) of 0.3 mg/dL or more within 48 hours, or an increase to at least 1.5 times the baseline level within the past seven days [2]. The worldwide increase in antibiotic resistance presents a significant public health issue [3]. A significant concern in healthcare is the rising prevalence of multidrug resistance and the proliferation of extensively drug-resistant Gram-negative bacteria, particularly in hospital-acquired infections [4]. Colistin, a polymyxin-class antibiotic originally developed in the late 1940s and approved by the Food and Drug Administration (FDA) in 1962, has demonstrated in vitro efficacy against the majority of aerobic Gram-negative bacilli. Colistin efficacy has also been demonstrated against a broad spectrum of Gram-negative pathogens including extensively diverse and multiple-drug organisms [7, 8]. Though initially it was effective its utilization was not encouraged owing to the high risk of AKI. Subsequently, colistin was revived into medical practice in the early 2000s as a fundamental rescue therapy for countering the harmful impacts of these infections [8, 9]. In multiple studies, colistin-induced AKI

DOI: https://doi.org/10.54393/pjhs.v6i1.2286

ranges from 20% to 76% [10, 11]. In these studies, the nephrotoxicity rate was varying with different dosing regimens. Numerous determinants were reported to contribute to colistin-induced AKI such as diabetes, CKD, anemia and exposure to events which are harmful to the kidney (trauma, sepsis, use of nephrotoxic agents in critical illness) [11, 12]. Colistin-induced AKI is generally reversible and rarely results in lasting kidney damage. It is crucial to balance the potential for nephrotoxicity with the risks associated with not adequately treating a severe infection [13]. Hypoalbuminemia, commonly characterized by albumin levels below 3.5–4.0 g/dL or ≤ 3.5 mmol/L, is a notable risk factor for higher rates of morbidity and mortality. Moreover, it has been linked to an increased likelihood of developing AKI [14]. Further in the literature of the study enrolled participants in both intensive care unit (ICU) and surgical patients demonstrated that hypoalbuminemia could serve as a useful indicator to predict death in AKI patients [15-17]. However, despite these findings, the overall effect of hypoalbuminemia remains questionable due to conflicting outcomes from various studies, partly owing to diverse heterogeneity and the occurrence of different types of infection [5, 8]. Data on the prevalence of colistin-associated AKI in our local context is limited.

This study aims to assess the incidence of colistin-induced AKI among intensive care unit (ICU) patients and investigate the relationship between serum albumin levels and the development of AKI in ICU patients receiving colistin therapy.

METHODS

This prospective cohort (follow-up) study was conducted in the adult intensive care unit at the "Shaheed Mohtarma Benazir Bhutto Institute of Trauma, Pakistan", from August 2023 to December 2023. The study commenced following the approval of "Ethics Review Committee (ERC-000113/SMBBIT)". After obtaining dual linguistic consent, all patients aged 18 years who were admitted to the ICU and receiving intravenous colistin therapy for 72 hours as part of their infection treatment were included in the study. Patients with AKI injury before receiving colistin therapy, requiring renal replacement therapy before colistin initiation and those patients receiving only inhaled colistin were excluded. An informed consent was taken. WHO sample size calculator was used for a Sample size of 229 patients, estimated by taking 68.8% AKI injury among patients on colistin therapy [14] at a 95% confidence interval and 6% margin of error. Patients were enlisted into the study using non-probability consecutive sampling. In this study, the duration of colistin therapy was set at either 7 or 14 days. This study used KDIGO guidelines [2], while the primary outcomes of the study were the incidence of AKI at 48 hours and after colistin therapy (Table 1).

Table 1: KDIGO Guidelines

Stages	Serum Creatinine
Stage 1	1.5 to 1.9 Baseline OR ≥0.3 mg/dL (≥26.5 µmol/L)
Stage 2	2.0 to 2.9 Times Baseline
Stage 3	3.0 Times Baseline OR Increase in Serum Creatinine to ≥4.0 mg/dL (≥353.6 µmol/L) OR Initiation of Renal Replacement Therapy, OR inpatient <18 Years, Decrease in eGFR to <35mL/min/1.73m2

The statistical analysis was performed using SPSS version 26. Categorical data were shown as counts and percentages, whereas continuous data, due to their non-normal distribution, were displayed as medians with "interquartile ranges (IQR)". The "Shapiro-Wilk test" evaluated the normality of numerical data. "Pearson's Chi-square" or "Fisher's exact test" for categorical variables, and the "Mann-Whitney U" test for continuous data, were employed to compare patients with and without AKI. The independent sample t-test was used for inferential analysis of continuous data. A "Kaplan-Meier survival curve" assessed the likelihood of AKI recovery after therapy. A "Cox proportional hazards regression model" was used to obtain "hazard ratios (HRs)" with 95% confidence intervals. Variables with significant unadjusted hazard ratios were included in the model to compute adjusted hazard ratios. Statistical significance was set at a two-sided p-value of 0.05.

RESULTS

A total of 250 patients were studied. On Shapiro-Wilk the normality of the residual was >0.05. The median age of patients was 40 (IQR=22-48) years with an age range of 18-70 years. The majority of patients were male 200 (80%). More than three-fourths of patients were receiving concomitant antibiotics 195 (78%). Of 195 (78%) patients who were receiving concomitant antibiotics, 93 (47.7%) were on vancomycin and 72 (37%) were on meropenem. Around a quarter of them were administered with nephrotoxic drugs 204 (81.6%). Median colistin dosage was 4 (IQR=3.5-4.5) MIU (Table 2).

Table 2: Comorbidities and Diagnosis of the Patients Presented at ICU

Variables	Frequency (%)
Gender	
Male	200 (80%)
Female	50 (20%)
Hypertension	
Present	175 (70%)
Absent	75 (30%)
Diabetes	
Present	150 (60%)
Absent	100 (40%)

DOI: https://doi.org/10.54393/pjhs.v6i1.2286

Heart Disease	
Present	137 (55%)
Absent	112 (45%)
Chronic Kidney Disease	
Present	50 (20%)
Absent	200 (80%)
Respiratory Tract Infection	
Present	100 (40%)
Absent	150 (60%)
Infection in Blood	
Present	125 (50%)
Absent	125 (50%)
Infection in Urine	
Present	75 (30%)
Absent	17.5 (70%)
Infection in Sputum	
Present	25 (10%)
Absent	225 (90%)
Infection in CNS	
Present	12.5 (5%)
Absent	237.5 (95%)
Hypoalbuminemia	
Present	175 (70%)
Absent	75 (30%)
Concomitant Antibiotics	
Patients Receiving Concomitant Antibiotics	195 (78%)
Receiving Vancomycin	93 (47.7% of 78%)
Receiving Meropenem	72 (37% of 78%)
Patients Administered Nephrotoxic Drugs	204 (81.6%)
Median Colistin Dosage	4 MIU (IQR: 3.5-4.5)

The incidence rate of AKI at the end of therapy was 5.1 per 100 patients. The survival probability from AKI at the end of therapy was 2.1%. Median survival days were 14 days (95% CI: 13.3-14.7). The Kaplan-Meier survival analysis demonstrates a progressive decline in cumulative survival throughout the study, with significant drops indicating clustered event occurrences and censored data points reflecting incomplete follow-up for certain subjects. The survival analysis on the 5th, 10th, 15th and 20th days were 0.85, 0.75, 0.55, and 0.35 respectively where the curve has a visible decrease indicating more events occurring (Figure 1).

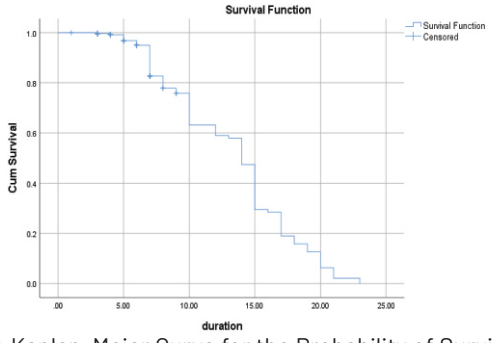

Figure 1: Kaplan-Meier Curve for the Probability of Survival from AKI During Colistin Therapy During the Study Period

At baseline and 48 hours, the median serum creatinine level was 0.5 (IQR=0.2-0.6) and 0.5 (IQR=0.5-0.3-0.9) and at the end of therapy it was 0.6 (IQR=0.3-1.4). At 48 hours, AKI was developed in 32% of patients whereas at the end of therapy, AKI incidence was seen in 42.8% which is the highest among all the stages of AKI. The serum levels at baseline and after colistin therapy and AKI incidence are displayed (Table 3).

Table 3: Serum Creatinine and AKI Incidence Following Colistin Therapy

Variables	Frequency (%)
Serum Creatinine at Baseline	0.5 (IQR=0.2-0.6)
Serum Creatinine at 48 Hours	0.5 (IQR=0.5-0.3-0.9)
Serum Creatinine at End of Therapy	0.6 (IQR=0.3-1.4)
AKI Incidence at 48 Hours	80 (32%)
AKI Staging at 48 Hours	
Stage 1	28 (35%)
Stage 2	23 (28.8%)
Stage 3	29 (36.3%)
AKI Incidence at End of Therapy	107 (42.8%)
AKI Staging at the End of Therapy	
Stage 1	37 (34.6%)
Stage 2	20 (18.7%)
Stage 3	50 (46.7%)

Patients developing AKI at the end of therapy were elderly ($p<0.001$). All co-existing diseases were significantly higher among patients developing AKI. The use of nephrotoxic drugs was significantly higher among AKI patients ($p<0.001$). Albumin levels were significantly lower in patients having AKI than those who did not have AKI at the end of therapy ($p<0.001$) (Table 4).

Table 4: Comparison of Patient's Characteristics with AKI (with and without) at the End of Colistin Therapy

Variables	Groups	AKI at the End of Therapy		p-Value
		Yes n (%)	No n (%)	
Age (in years)	-	Mean 58 (Range: 50-63)	Mean 35 (Range: 22-25)	*<0.001
Gender	Male	78 (41.5%)	110 (58.5%)	0.466
	Female	29 (46.8%)	33 (53.2%)	
Comorbid	Hypertension	36 (80%)	9 (20%)	*<0.001
	Diabetes	36 (75%)	12 (25%)	*<0.001
	CKD	12 (75%)	4 (25%)	*0.007
	Heart Diseases	16 (66.7%)	8 (33.3%)	*0.013
Concomitant Use of Antibiotics	Yes	83 (42.6%)	112 (57.4%)	0.887
	No	24 (43.6%)	31 (56.4%)	
Nephrotoxic Drugs	Yes	44 (95.7%)	2 (4.3%)	*<0.001
	No	66 (36.5%)	115 (63.5%)	
Site of Infection	Blood	83 (50.3%)	82 (49.7%)	*0.001
	Urine	14 (40%)	21 (60%)	0.718
	Sputum	9 (42.9%)	12 (57.1%)	0.996
	CNS	7 (50%)	7 (50%)	0.575

DOI: https://doi.org/10.54393/pjhs.v6i1.2286

	Respiratory tract	63 (43.2%)	83 (56.8%)	0.894
Albumin Level (Mean ± SD)		Mean 1.7 (Range: 1.3-2.3)	Mean 3.6 (Range: 3.4-4.0)	*<0.001

AKI: Acute kidney injury, CKD: Chronic kidney disease, CNS: Central nervous system, **=0.05, aIndependent Sample t-test applied, bChi Square test applied

In univariate analysis, the risk of developing AKI was significantly increasing with increased age, use of nephrotoxic drugs, and increasing colistin dosage whereas AKI risk was decreasing with increasing albumin levels. In a multivariable model, only colistin dosage was found to be significantly associated with increasing AKI risk with increasing colistin dosage (Table 5).

Table 5: Patients' Characteristics with AKI at the End of Colistin Therapy in Univariate and Multivariable Regression

Variables	HR (95% CI)	p-Value	A HR (95% CI)	p-Value
Age	1.04 (1.02-1.05)	0.176	1.01 (0.98-1.02)	0.693
Gender, Male	1.16 (0.75-1.80)	0.485	1.10 (0.90-1.35)	0.560
Hypertension	0.94 (0.63-1.40)	0.748	0.97 (0.70-1.20)	0.720
Diabetes	1.30 (0.86-1.96)	0.209	1.15 (0.85-1.45)	0.450
CKD	0.98 (0.53-1.78)	0.943	0.99 (0.65-1.52)	0.930
Heart Diseases	1.41 (0.82-2.41)	0.210	1.20 (0.95-1.60)	0.250
Concomitant Antibiotics Use	1.20 (0.76-1.89)	0.432	1.05 (0.80-1.40)	0.650
Nephrotoxic Drugs	1.48 (1-2.19)	*0.050	1.28 (0.85-1.93)	0.228
Colistin Dosage	1.08 (1.03-1.15)	*0.006	1.06 (1.01-1.14)	*0.049
Albumin	0.72 (0.53-0.98)	*0.038	0.82 (0.58-1.14)	0.232

CI: Confidence Interval at 95%, HR: Hazard ratio, *=0.05

DISCUSSION

This study of 250 patients (75.2% male, median age 40) found high exposure to nephrotoxic drugs and antibiotics, with a median colistin dose of 4 MIU. The AKI incidence rate was 5.1 per 100 patient days, and survival at therapy's end was only 2.1%, with a median survival of 14 days. Kaplan-Meier analysis showed a steady decline in survival over the treatment period, underscoring significant risks associated with colistin use. At the 48-hour mark, 32% of patients had developed AKI, which increased to 42.8% by the end of therapy, marking the highest incidence among AKI stages. Elderly patients and those with pre-existing conditions were more likely to develop AKI (p<0.001), and AKI was significantly associated with the use of nephrotoxic drugs. Lower albumin levels were noted in patients with AKI (p<0.001). Univariate analysis indicated that AKI risk increased with age, nephrotoxic drug use, and higher colistin doses, while higher albumin levels were protective. Multivariable analysis confirmed that increased colistin dosage was the only significant independent risk factor for AKI. Our study identified a rise in AKI after colistin therapy. The incidence was 11.1% at 48 hours and 25% by treatment completion, mirroring the findings of a retrospective study conducted by Deniz (12% at 48 hours, 29% by day 7) [18]. However, discrepancies exist with prior literature reporting a wider range of AKI risk (44.3%-76.1%) [19-21]. These variations likely stem from differing AKI criteria, therapy duration, infection severity, underlying conditions, and concomitant nephrotoxic medications. We observed a high prevalence of AKI stages following colistin use. At 48 hours, 35.0%, 28.8%, and 36.3% of patients had Stage I, II, and III AKI, respectively. Notably, Stage III AKI prevalence rose to 46.7% by treatment conclusion, accompanied by a decrease in Stages I (34.6%) and II (18.7%). These findings align with previous research demonstrating a link between colistin and AKI development [19]. Similar studies support this, with Alotaibi et al., reporting a distribution of AKI stages at treatment completion as 24.5% Stage I, 37.32% Stage II, and 14.5% Stage III [20]. Likewise, Moghnieh et al., documented an incidence of AKI at Stages I, II, and III of 21.2%, 28.8%, and 50.0%, respectively [21]. Univariate analysis revealed a significant correlation between increasing age and AKI occurrence, but not on multivariable analysis. This discrepancy might be due to our limited sample size. Nevertheless, our findings are consistent with prior research highlighting increasing age as a key factor in colistin-induced AKI [18, 20]. Hypoalbuminemia, frequently observed in critically ill patients, serves as an indicator of malnutrition and inflammation and is also predictive of AKI and mortality [22]. In our study, univariate analysis showed a significant association between higher albumin levels and a reduced risk of AKI, aligning with previous research that identifies hypoalbuminemia as a risk factor for colistin-induced AKI [14-15]. However, Alotaibi et al., reported no such association [20]. The link between hypoalbuminemia and colistin-induced AKI is likely multifaceted, with albumin's various physiological functions playing a significant role [14]. Shah et al., in their retrospective study, reported Kaplan-Meier survival analysis as significantly lower survival for patients with AKI-related kidney failure compared to other etiologies (log-rank test, p<0.001). Sensitivity analysis, excluding AKI patients who started dialysis with arteriovenous access and non-AKI patients who recovered renal function, yielded hazard ratios consistent with the primary model, with a marginal attenuation of roughly 10% [23]. In a study conducted by Nagata et al., Kaplan-Meier survival analysis revealed that transient AKI, persistent AKI, and AKD were all significantly linked to an increased incidence of events characterized by a reduction in estimated glomerular filtration rate (ReGFR<0.7) compared to individuals without AKI, especially within the first year. Among these groups, the rates of adverse outcomes were substantially higher in those with persistent AKI and AKD than in those with transient AKI [24]. The findings of this study carry

DOI: https://doi.org/10.54393/pjhs.v6i1.2286

important implications for clinical practice and patient management. The high incidence of AKI, particularly among elderly patients and those exposed to nephrotoxic drugs, emphasizes the need for vigilant monitoring of renal function during colistin treatment. The frequent use of concomitant antibiotics, such as vancomycin and meropenem, points to the importance of optimizing antibiotic regimens to minimize cumulative nephrotoxic effects. Additionally, the significant relationship between higher colistin dosages and increased AKI risk suggests that dose adjustments may be necessary to achieve a balance between efficacy and safety. Identifying high-risk patients, such as those with comorbidities or lower albumin levels, could inform more personalized treatment strategies to mitigate AKI risk. Overall, these results provide valuable insights for guiding future research aimed at developing safer treatment protocols and protective measures for patients undergoing colistin therapy.

CONCLUSIONS

It was concluded that this analysis identified a higher incidence of AKI following colistin therapy. However, albumin levels did not show a significant association with AKI risk in the multivariable model. Conversely, a significant correlation was observed between increasing colistin dosage and AKI incidence.

Authors Contribution

Conceptualization: SUM
Methodology: SUM, SS, NM, SK
Formal analysis: SM, SK
Writing review and editing: SI

All authors have read and agreed to the published version of the manuscript

Conflicts of Interest

The authors declare no conflict of interest.

Source of Funding

The author received no financial support for the research, authorship and/or publication of this article.

REFERENCES

[1] Hoste EA, Kellum JA, Selby NM, Zarbock A, Palevsky PM, Bagshaw SM, *et al.* Global Epidemiology and Outcomes of Acute Kidney Injury. Nature Reviews Nephrology. 2018 Oct; 14(10): 607-25. doi: 10.1038/s41581-018-0052-0.

[2] Ying J, Wang J, Ying Z, Ran X, Zeng X, Chen D, *et al.* Exploring the Relationship Between Post-Contrast Acute Kidney Injury and Different Baseline Creatinine Standards: A Retrospective Cohort Study. Frontiers in Endocrinology. 2023 Jan; 13: 1042312. doi: 10.3389/fendo.2022.1042312.

[3] Wollein Waldetoft K, Sundius S, Kuske R, Brown SP. Defining the Benefits of Antibiotic Resistance in Commensals and the Scope for Resistance Optimization. Mbio. 2023 Feb; 14(1): e01349-22. doi: 10.1128/mbio.01349-22.

[4] Morris S and Cerceo E. Trends, Epidemiology, and Management of Multi-Drug Resistant Gram-Negative Bacterial Infections in the Hospitalized Setting. Antibiotics. 2020 Apr; 9(4): 196. doi: 10.3390/antibiotics9040196.

[5] Gameiro J, Fonseca JA, Outerelo C, Lopes JA. Acute Kidney Injury: From Diagnosis to Prevention and Treatment Strategies. Journal of Clinical Medicine. 2020 Jun; 9(6): 1704. doi: 10.3390/jcm9061704.

[6] Katip W, Oberdorfer P, Taruangsri P, Nampuan T. Risk Factors for Acute Kidney Injury in Patients Receiving Intravenous Colistin. Pharmaceutical Sciences. 2024 Jan; 30(2): 244-51. doi: 10.34172/PS.2023.31.

[7] El-Sayed Ahmed MA, Zhong LL, Shen C, Yang Y, Doi Y, Tian GB. Colistin and Its Role in the Era of Antibiotic Resistance: An Extended Review (2000–2019). Emerging Microbes and Infections. 2020 Jan; 9(1): 868-85. doi: 10.1080/22221751.2020.1754133.

[8] Binsker U, Käsbohrer A, Hammerl JA. Global Colistin Use: A Review of the Emergence of Resistant Enterobacterales and the Impact On Their Genetic Basis. FEMS Microbiology Reviews. 2022 Jan; 46(1): fuab049. doi: 10.1093/femsre/fuab049.

[9] Almutairy R, Aljrarri W, Noor A, Elsamadisi P, Shamas N, Qureshi M et al. Impact of Colistin Dosing On the Incidence of Nephrotoxicity in A Tertiary Care Hospital in Saudi Arabia. Antibiotics. 2020 Aug; 9(8): 485. doi: 10.3390/antibiotics9080485.

[10] Mirjalili M, Mirzaei E, Vazin A. Pharmacological Agents for the Prevention of Colistin-Induced Nephrotoxicity. European Journal of Medical Research. 2022 May; 27(1): 64. doi: 10.1186/s40001-022-00689-w.

[11] Gai Z, Samodelov SL, Kullak-Ublick GA, Visentin M. Molecular Mechanisms of Colistin-Induced Nephrotoxicity. Molecules. 2019 Feb; 24(3): 653. doi: 10.3390/molecules24030653.

[12] Sangthawan P, Geater AF, Naorungroj S, Nikomrat P, Nwabor OF, Chusri S. Characteristics, Influencing Factors, Predictive Scoring System, and Outcomes of the Patients with Nephrotoxicity Associated with Administration of Intravenous Colistin. Antibiotics. 2021 Dec; 11(1): 2. doi: 10.3390/antibiotics11010002.

[13] Morales-Alvarez MC. Nephrotoxicity of Antimicrobials and Antibiotics. Advances in Chronic Kidney Disease. 2020 Jan; 27(1): 31-7. doi: 10.1053/j.ackd.2019.08.001.

DOI: https://doi.org/10.54393/pjhs.v6i1.2286

[14] Li N, Qiao H, Guo JF, Yang HY, Li XY, Li SL *et al.* Preoperative Hypoalbuminemia Was Associated with Acute Kidney Injury in High-Risk Patients Following Non-Cardiac Surgery: A Retrospective Cohort Study. BioMed Central Anesthesiology. 2019 Dec; 19: 1-1. doi: 10.1186/s12871-019-0842-3.

[15] Zheng LJ, Jiang W, Pan L, Pan J. Reduced Serum Albumin as A Risk Factor for Poor Prognosis in Critically Ill Patients Receiving Renal Replacement Therapy. BioMed Central Nephrology. 2021 Dec; 22: 1-1. doi: 10.1186/s12882-021-02512-w.

[16] Walther CP, Gutiérrez OM, Cushman M, Judd SE, Lang J, McClellan W et al. Serum Albumin Concentration and Risk of End-Stage Renal Disease: The Regards Study. Nephrology Dialysis Transplantation. 2018 Oct; 33(10): 1770-7. doi: 10.1093/ndt/gfx331.

[17] Chen L, Wu X, Qin H, Zhu H. The PCT to Albumin Ratio Predicts Mortality in Patients with Acute Kidney Injury Caused by Abdominal Infection-Evoked Sepsis. Frontiers in Nutrition. 2021 Jun; 8: 584461. doi: 10.3389/fnut.2021.584461.

[18] Deniz M and Alişik M. Risk Factors and Prognosis for the Development of Acute Kidney Injury in Patients Using Colistin in the Intensive Care Unit: A Retrospective Cohort Study. Medicine. 2024 Jan; 103(2): e36913. doi: 10.1097/MD.0000000000036913.

[19] Arrayasillapatorn N, Promsen P, Kritmetapak K, Anunnatsiri S, Chotmongkol W, Anutrakulchai S. Colistin-Induced Acute Kidney Injury and the Effect On Survival in Patients with Multidrug-Resistant Gram-Negative Infections: Significance of Drug Doses Adjusted to Ideal Body Weight. International Journal of Nephrology. 2021; 2021(1): 7795096. doi: 10.1155/2021/7795096.

[20] Alotaibi FM, Alshehail BM, Al Jamea ZA, Joseph R, Alanazi AH, Alhamed NA *et al.* Incidence and Risk Factors of Colistin-Induced Nephrotoxicity Associated with the International Consensus Guidelines for the Optimal Use of the Polymyxins: A Retrospective Study in A Tertiary Care Hospital, Saudi Arabia. Antibiotics. 2022 Nov; 11(11): 1569. doi: 10.3390/antibiotics11111569.

[21] Moghnieh R, Husni R, Helou M, Abdallah D, Sinno L, Jadayel M et al. The Prevalence and Risk Factors of Acute Kidney Injury during Colistin Therapy: A Retrospective Cohort Study from Lebanon. Antibiotics. 2023 Jul; 12(7): 1183. doi: 10.3390/antibiotics12071183.

[22] Sun J, Su H, Lou Y, Wang M. Association Between Serum Albumin Level and All-Cause Mortality in Patients with Chronic Kidney Disease: A Retrospective Cohort Study. The American Journal of the Medical Sciences. 2021 Apr; 361(4): 451-60. doi: 10.1016/j.amjms.2020.07.020.

[23] Shah S, Leonard AC, Harrison K, Meganathan K, Christianson AL, Thakar CV. Mortality and Recovery Associated with Kidney Failure Due to Acute Kidney Injury. Clinical Journal of the American Society of Nephrology. 2020 Jul; 15(7): 995-1006. doi: 10.2215/CJN.11200919.

[24] Nagata K, Horino T, Hatakeyama Y, Matsumoto T, Terada Y, Okuhara Y. Effects of Transient Acute Kidney Injury, Persistent Acute Kidney Injury and Acute Kidney Disease on the Long-Term Renal Prognosis After an Initial Acute Kidney Injury Event. Nephrology. 2021 Apr; 26(4): 312-8. doi: 10.1111/nep.13831.