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Nephrotoxic Medication Exposure and Clinical Outcomes Following Acute Kidney Injury.

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ABSTRACT

Acute kidney injury (AKI) is a common comorbidity in patients admitted to hospitals and can also be affected by exposure to potentially nephrotoxic drugs. Medication-related and clinical predictors of adverse outcomes can be identified, which is significant to enhance risk assessment and management. An analytical study, secondary-data study was carried out on a subset of 1,000 cases of hospitalized adults with AKI based on ELAIA-2 dataset that was cleaned. ACEi/ARB, NSAIDs and PPIs exposure were evaluated in relation to AKI progression, dialysis, mortality and composite adverse outcomes. Descriptive statistics, chi-square tests, independent samples t-tests, correlation and regression were used. Crude associations between PPI exposure and AKI progression, dialysis, death, and composite outcome were significant. There were lower rates of selected outcomes with ACEi/ARB and NSAID exposure in the unadjusted analysis. Nonetheless, the composite outcome was not adjusted and independently related to individual medication classes. The overall outcome, AKI progression, death and dialysis were always predicted by the higher SOFA score. Predictors of 14-day mortality were age, and predictors of dialysis were baseline creatinine. Clinical severity and baseline renal status seemed to be more critical predictors of short-term outcomes of AKI compared to medication exposure alone. Close review of medications should thus be coupled with evaluation of severity of systemic illness and renal function.

Keywords: Acute kidney injury; nephrotoxic medications; NSAIDs; proton pump inhibitors; clinical outcomes

1. Introduction

Acute kidney injury (AKI) is a frequent and clinically significant complication in hospitalized patients and manifests itself with a sudden deterioration of kidney function. It is linked to morbidity, mortality, need of dialysis, long hospitalization and risk of chronic kidney disease in the future. Patients who have been exposed to nephrotoxic drugs are also at risk and may be especially susceptible with kidney reserve already compromised, or other comorbid conditions. Kidney injury in relation to medications can thus play a significant role in the overall weight of AKI along with their long-term effects [1]. The intensity of exposure also can play a role in risk because increased vancomycin levels have been linked to increased AKI occurrence [2]. Potentially nephrotoxic drugs are still very common in routine treatment of chronic kidney disease patients, which highlights the importance of closely evaluating medications [3]. Exposure to medication is a potentially modifiable risk factor of AKI. Patients admitted to the hospital and those who are critically ill often receive numerous agents that can impair renal perfusion, lead to direct tubular toxicity, result in interstitial nephritis, or cause immune-mediated kidney injury. Combination of broad-spectrum antibiotics have been linked to nephrotoxicity among critically ill groups [4], other antibiotics have reported significant AKI markers during pharmacovigilance and clinical trials [5]. Recent classes of therapeutics, such as immune checkpoint inhibitors, have also been associated with renal side effects [6]. Since AKI can result in chronic renal failure and other chronic complications, it is worthwhile to detect the risk of medications to prevent the pathology not only in the short term but in the long term to protect the kidney [7].

Drug AKI occurs in a variety of ways and its clinical presentation can vary depending on the drug, dose, length of treatment, renal function, and disease severity [8]. Treatment intervention strategies to detect and minimize nephrotoxic exposure have shown that avoidable medication-induced kidney injury could be minimized by systematic surveillance and medication

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scrutiny [9]. Exposure to nephrotoxins has also been linked to AKI risk and is also a consideration in the hospital practice in adults [10]. Moreover, survivors of AKI might face repeated kidney damage and chronic kidney disease, cardiovascular complications, and high mortality, with the significance of early detection of risk factors that can be modified [11]. A number of classes of drugs have been linked with kidney damage. Drug accumulation and tubular precipitation by high-dose methotrexate can cause AKI [12], whereas nephrotoxicity in general can be caused by a change in glomerular hemodynamics, tubular injury, crystal deposition, or inflammatory processes [13]. This risk can be exaggerated in case of co-administration of several nephrotoxic drugs [14]. Antimicrobials are considered to be one of the best-known causes of medication-related nephrotoxicity [15], and the risk is also increased in critically ill patients by hemodynamic instability, infection, and other organ dysfunction [16].

Non-steroidal anti-inflammatory medications are some of the frequently used medications that have a kidney impact, and may decrease renal perfusion by inhibiting the prostaglandin-mediated vasodilation, and may also alter the glomerular hemodynamics in the conditions of low renal perfusion. Kidney damage through proton pump inhibitors has also been linked to kidney damage especially using interstitial nephritis. The AKI caused by drugs thus necessitates an analysis of the exposure to the drug and the renal and clinical status of the patient [17]. The exposure to multiple potentially nephrotoxic drugs simultaneously could also alter the AKI risk and clinical outcome trends [18]. The clinical outcomes of AKI are progressive renal damage, dialysis, acute mortality, inpatient mortality, readmission, and delayed recovery. The age, sex, and baseline kidney functioning, chronic kidney disease, diabetes, hypertension, heart failure, comorbidity burden, and overall illness severity also contribute to these results. Hence, crude comparisons and adjusted analyses should be used to evaluate medication-outcome relationships. The current research sought to assess the relationship between the exposure to potentially nephrotoxic drugs and the clinical outcomes of hospitalized patients with AKI.

Objectives of the study

1. To assess associations between nephrotoxic medication exposure and major AKI outcomes.
2. To identify independent predictors of adverse outcomes following acute kidney injury.

2. Methodology

2.1 Study Design

An analytical secondary-data study was performed to assess the relationship between possibly nephrotoxic medication exposure and clinical outcome of hospitalized patients with acute kidney injury. The evaluation was based on medication exposure during randomization, and the renal and clinical outcomes. Descriptive and inferential statistical tests were used to analyze patient-level data. The study directly evaluated the individual classes of medication, cumulative cumulative medication exposure, AKI severity, renal function, comorbidities, and the adverse outcome such as AKI progression, dialysis, mortality and the composite clinical outcome.

2.2 Data Source

The data were obtained from the publicly available Zenodo repository containing the ELAIA-2 dataset titled Automated, Medication-Targeted Alerts for Acute Kidney Injury – A Randomized Trial [19]. The initial dataset consisted of 5,060 hospitalized adults with AKI who had an active order of at least one medication of interest: NSAID, renin-angiotensin-aldosterone system inhibitor or proton pump inhibitor. In the current research, 1000 patients with 62 complete variables (cleaned analytical subset) were used.

2.3 Study Population and Sample Selection

The population of the study was a group of adult patients with acute kidney injury, hospitalized, and who were taking one of the chosen classes of medications. Records containing full information of the retained demographic, the clinical, renal, medication-exposure, and outcome variables were eligible to be analyzed. A proportional stratified random sample was drawn based on medication count and status on composite outcome. The analytical sample was 1,000 patients. There were no duplicate patient identifiers or missing values within the final cleaned dataset and no statistical imputation was done.

2.4 Data Cleaning and Preparation

Analysis of the original data set included checking its completeness, duplicate records, relevance of variables, and consistency. With large structural or conditional missingness, variables were not imputed, but dropped. Only records that had all the data on the variables that were retained were identified. Duplicate rows and duplicate patient identifiers were verified and done away with where necessary. The original coding of binary variables was preserved, whereas continuous variables were maintained as numbers. The resulting clean data consisted of 1,000 full patient records and 62 variables that were available to be used in statistical analyses.

2.5 Medication Exposure Assessment

Three binary variables of ACEi/ARB, NSAID, and PPI use at the time of randomization were used to measure medication exposure. Patients were categorized as either being exposed to a specific type of medication or not. The number of classes of medication that each patient received was also evaluated. Patients were grouped into one, two or three classes of medication. These variables were employed to assess how individual medication exposures and escalating medication burden related to a variance in AKI progression, dialysis, mortality, and composite adverse clinical outcomes.

2.6 Clinical and Renal Variables and Outcomes

The clinical variables were age, sex, AKI stage, SOFA score, vital signs, comorbidities, and Elixhauser comorbidity score. Renal parameters were the baseline creatinine, admission creatinine, creatinine at randomization, blood urea nitrogen and admission eGFR. The major events were AKI progression, dialysis, death in 14 days, and the composite adverse outcome. Some of the secondary outcomes were inpatient mortality, inpatient dialysis, stage progression, 30-day readmission, renal consultation, duration of AKI, and length of stay in the hospital.

2.7 Statistical Analysis

Demographic, clinical, renal, and medication-exposure

and outcome variables were summarized using descriptive statistics. Continuous variables were in the form of mean and SD whereas the categorical variables were in the form of frequencies and percentages. Chi-square tests were used to evaluate the relationships between exposure to medication and categorical outcomes. Continuous variables in two outcome groups were compared using independent-samples t-tests. Correlation was used to study the associations between continuous renal and clinical variables. Adjusted odds ratios with 95% confidence intervals of major outcomes were estimated using regression. The p-value was set to $p < 0.05$.

3. Results

3.1 Baseline Demographic and Clinical Characteristics

One thousand patients with acute kidney injury were used in the analysis. The average age was 67.90 +/- 15.90 years and the distribution in both sex groups was almost equal. The majority of patients were diagnosed with stage 1 AKI (90.7%), with stage 2 and stage 3 representing 7.3% and 2.0, respectively. The most prevalent comorbidity was hypertension (68.2%), then there was diabetes mellitus (39.3%), and congestive heart failure (33.2%). Table 1 shows baseline renal and clinical features.

Table 1. Baseline Demographic and Clinical Characteristics of the Study Population

Category	Characteristic	Value
Demographic characteristics	Age, years	67.90 ± 15.90
	Male	519 (51.9%)
	Female	481 (48.1%)
AKI severity	AKI stage 1	907 (90.7%)
	AKI stage 2	73 (7.3%)
	AKI stage 3	20 (2.0%)
Comorbidities	Chronic kidney disease	269 (26.9%)
	Congestive heart failure	332 (33.2%)
	Pulmonary disease	296 (29.6%)
	Diabetes mellitus	393 (39.3%)
	Hypertension	682 (68.2%)
	Malignancy	213 (21.3%)
	Liver disease	129 (12.9%)
Comorbidity burden	Elixhauser comorbidity score	5.91 ± 3.87
Renal function parameters	Baseline creatinine	1.13 ± 0.65
	Creatinine at randomization	1.64 ± 0.83
	Blood urea nitrogen	33.37 ± 20.12
	Admission eGFR	64.48 ± 30.54
Clinical severity	SOFA score	2.78 ± 2.11

3.2 Pattern of Medication Exposure

The most common medication exposure was PPI exposure, which happened in 673 patients (67.3%), then ACEi/ARB exposure in 503 patients (50.3%), and NSAID exposure in 312 patients (31.2%). When it comes to the overall number of medication classes, 578 (57.8) patients were exposed to one class, 356 (35.6) to two classes, and 66 (6.6) to all three classes.

Figure 1 demonstrates the distribution of the categories of medication exposure among patients with acute kidney injury.

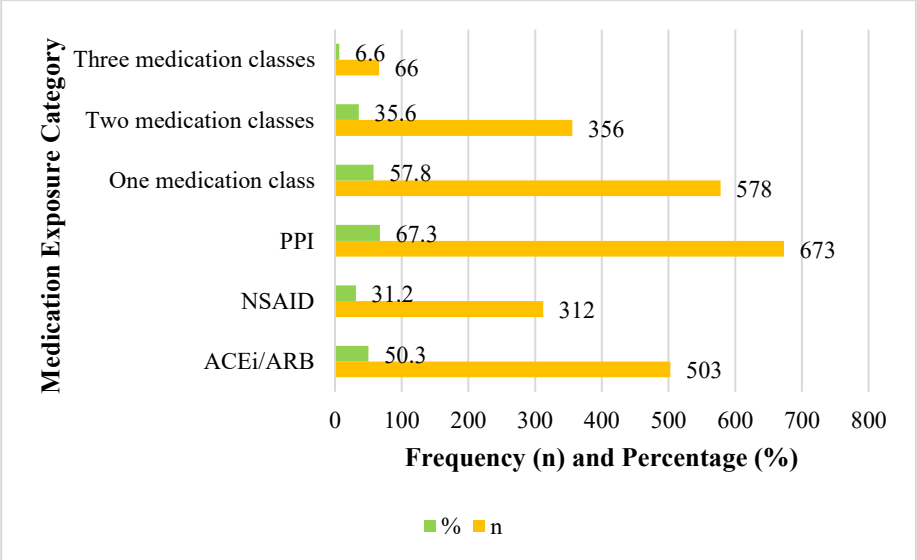

Figure 1. Medication Exposure Pattern

As illustrated in Figure 1, the exposure to medication was not uniform among the study population with PPI and ACEi/ARB being the most common individual categories of medications. The patients were mostly subjects of one medication class and comparatively few to multiple classes.

3.3 Association Between Medication Exposure and Major Outcomes

Notable variations in the key clinical outcomes were found based on personal exposure to medication. The proportions of dialysis, death, and composite adverse outcome were also lower in case of ACEi/ARB exposure than non-exposure. Dialysis was also significantly linked to NSAID exposure as opposed to AKI progression, death, and the composite outcome. Conversely, exposure to PPI was linked to increased AKI progression, dialysis, death, and composite adverse outcome. Table 2 shows that the most significant crude association was found between PPI exposure and dialysis ($p < 0.001$).

Table 2. Association of Individual Medication Exposure with Major Clinical Outcomes

Medication	Outcome	Not exposed n (%)	Exposed n (%)	χ^2	p-value
ACEi/ARB	AKI progression	105 (21.1)	89 (17.7)	1.671	0.196
ACEi/ARB	Dialysis	31 (6.2)	14 (2.8)	6.160	0.013
ACEi/ARB	Death	65 (13.1)	36 (7.2)	9.013	0.003
ACEi/ARB	Composite outcome	134 (27.0)	105 (20.9)	4.764	0.029
NSAID	AKI progression	137 (19.9)	57 (18.3)	0.273	0.601
NSAID	Dialysis	40 (5.8)	5 (1.6)	7.906	0.005
NSAID	Death	73 (10.6)	28 (9.0)	0.466	0.495
NSAID	Composite outcome	171 (24.9)	68 (21.8)	0.943	0.332
PPI	AKI progression	50 (15.3)	144 (21.4)	4.865	0.027
PPI	Dialysis	3 (0.9)	42 (6.2)	13.299	<0.001
PPI	Death	21 (6.4)	80 (11.9)	6.650	0.010
PPI	Composite outcome	61 (18.7)	178 (26.4)	6.929	0.009

3.4 Clinical Outcomes Following Acute Kidney Injury

The AKI progression in 14 days was observed in 194 patients (19.4%), and 45 patients (4.5%) underwent dialysis and 101 patients (10.1) died in 14 days. The composite adverse outcome was found in 239 patients (23.9%). Inpatient mortality and inpatient dialysis rates were 14.6% and 5.4, respectively. Advancement to stage 2 and stage 3 AKI were found in 10.0% and 9.4% of the patients, respectively. In 13.4 per cent of the study population, 30-day readmission was observed.

The distribution of major clinical outcomes among patients with acute kidney injury is presented in Figure 2.

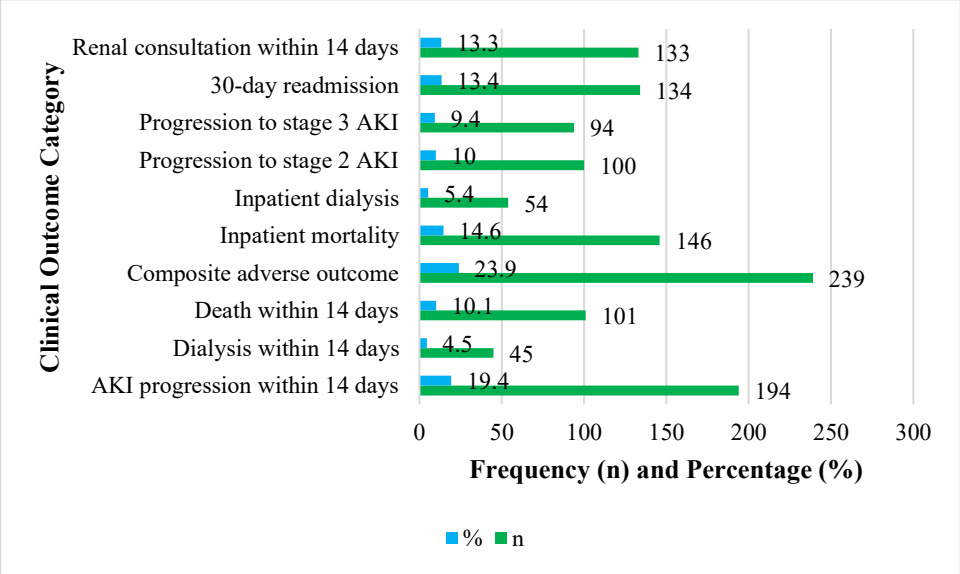

Figure 2. Clinical Outcomes Following AKI

Figure 2 shows the general trend of adverse outcomes following acute kidney injury. The composite adverse outcome and AKI progression were one of the more common occurrences whereas dialysis-related outcomes were relatively less common among the study population.

3.5 Multivariable Predictors of Adverse Clinical Outcomes

ACEi/ARB, NSAID and PPI exposure showed no independent relationship with the composite adverse outcome after the covariates of demographic, renal, comorbidity and clinical-severity were taken into consideration. SOFA score remained a significant predictor of the composite outcome (Aor = 1.39, $p < 0.001$), AKI progression (Aor = 1.37, $p < 0.001$), mortality (Aor = 1.42, $p < 0.001$), and dialysis (Aor = 1.43, $p < 0.001$). Mortality was predicted by age, and dialysis predicted by baseline creatinine. Notable adjusted results are summarized in Table 3.

Table 3. Adjusted Logistic Regression for Major Clinical Outcomes

Predictor	Outcome	Adjusted OR	95% CI	p-value
ACEi/ARB exposure	Composite outcome	0.93	0.65–1.33	0.672
NSAID exposure		0.89	0.61–1.29	0.532
PPI exposure		1.12	0.76–1.66	0.567
SOFA score		1.39	1.29–1.51	<0.001
Elixhauser score		0.91	0.86–0.96	0.001
SOFA score	AKI progression	1.37	1.26–1.49	<0.001
Elixhauser score		0.92	0.87–0.98	0.009
Age	14-day mortality	1.03	1.01–1.05	0.001
SOFA score		1.42	1.27–1.59	<0.001
NSAID exposure	Dialysis	0.32	0.11–0.91	0.032
SOFA score		1.43	1.24–1.66	<0.001
Baseline creatinine		1.79	1.06–3.05	0.031

3.6 Clinical Outcomes According to Number of Medication Classes

There was no significant difference in clinical outcomes based on the number of medication classes used. Composite adverse outcome had been experienced in 24.4, 23.6, and 21.2 percent of patients who were exposed to one, two, and three classes of medications respectively. On the same note, the AKI development was experienced in 19.0, 20.2 and 18.2 percent of these groups, respectively.

Figure 3 demonstrates the distribution of the key clinical outcomes in relation to the number of the medication classes.

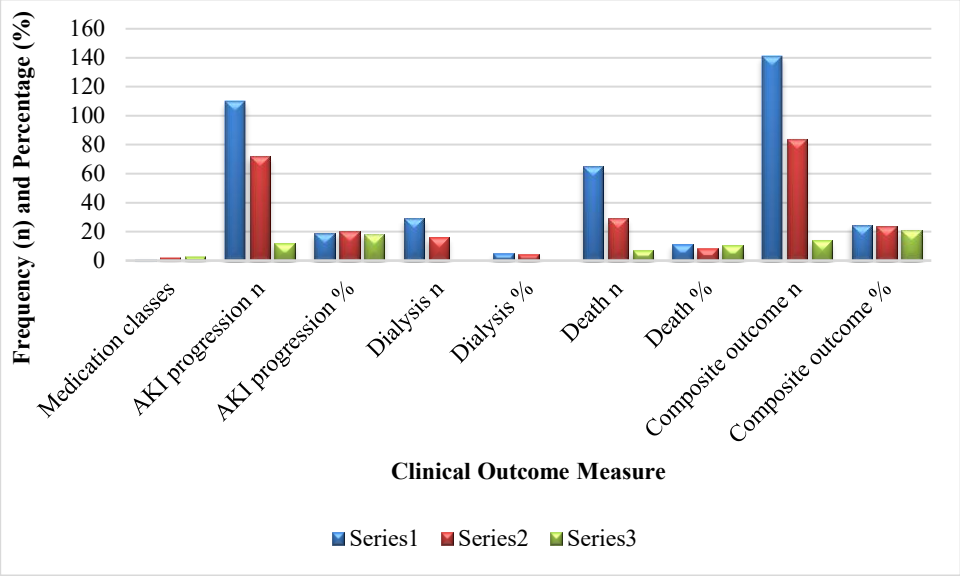

Figure 3. Clinical Outcomes by Medication Class Count

Figure 3 compares key clinical outcomes of patients who were exposed to one, two, or three medication classes. There were generally comparable patterns across exposure groups and no evident rise in negative outcomes with the number of medication classes.

4. Discussion

The current research assessed the clinical outcomes and the relationship between potentially nephrotoxic drug exposure and clinical outcomes in hospitalized patients with acute kidney injury. Almost a quarter of patients had the composite adverse outcome and AKI progression, mortality, and dialysis were also clinically significant. Crude comparisons indicated that PPI exposure was linked with increased rates of AKI progression, dialysis, death and the composite outcome, but ACEi/ARB exposure was linked with reduced rates of multiple adverse events. But upon multivariate adjustment, none of the three classes of medication predicted the composite outcome, suggesting that patient severity and baseline clinical factors might have played a significant role in the crude relationships. Various mechanisms affect drug-associated nephrotoxicity, such as the change in renal hemodynamics, tubular toxicity, interstitial inflammation, and individual susceptibility. Sales and Foresto termed drug induced nephrotoxicity as a multifactorial process that is dependent on both patient and medication related factors [20]. This helps the current observation that the exposure to medications was not adequate to describe the occurrence of adverse outcomes after adjusting. The findings thus imply that nephrotoxic drug exposures must be viewed in the broader clinical context instead of being viewed as a solitary determinant of AKI prognosis.

The current results are also significant since nephrotoxic exposure can still affect the renal health in the aftermath of the acute event. According to Schreier et al., the exposure to nephrotoxin within the years after the discharge of a hospitalized patient was linked with the onset or progressive deterioration of chronic kidney

disease in survivors of AKI [21]. In the present study, the composite adverse outcome patient group demonstrated enhanced creatinine, blood urea nitrogen, illness severity, prolonged AKI period and enhanced hospitalization. On the same note, Searns et al. revealed that AKI and nephrotoxic exposure were both linked to longer length of stay in the hospital [22]. Other vulnerable populations have also provided evidence of a relationship between nephrotoxic medication exposure and AKI. According to Steflik et al. the early AKI was linked to neonatal nephrotoxic drug exposure, with a greater role in clinically frail patients [23]. The existing evidence also suggests that exposure to medications should be taken into account together with the renal status at the baseline and the severity of the systemic illness. It aligns with the general idea that nephrotoxic AKI is a frequent condition in patients with multiple risk factors interacting to cause the disease, such as hemodynamic instability, comorbid illness, concomitant medications, and low renal reserve [24].

One of the significant results of this research was that SOFA score was the most reliable independent predictor of adverse outcomes. The composite outcome, AKI progression, mortality, and dialysis were significantly related to higher SOFA scores, which indicated that the severity of systemic illness was more likely to be a significant predictor of prognosis than medication exposure alone. The original ELAIA-2 randomized trial showed that medication-targeted alerts increased discontinuation of medications of interest, but did not produce a statistically significant improvement in the overall composite outcome [25]. This fact is very much consistent with the current analysis where the exposure to medication demonstrated a number of raw associations but few independent effects when adjusted. The independent predictors of 14-day mortality were age, and the baseline creatinine was predictive of dialysis, meaning that patient vulnerability and pre-existing kidney function continued to be significant predictors of prognosis. Yousef et al. also found that clinical features, the severity of renal damage, and underlying disease, but not drug exposure, affected the outcomes in drug-induced AKI [26]. The negative correlation found between the Elixhauser score in certain adjusted models ought to be viewed with some caution since the residual confounding or correlation between covariates with each other might have affected this finding.

The clinical implications of the findings include the need to be cautious in reviewing medications in hospitalized patients, especially those with AKI and higher illness severity or with a compromised baseline kidney function. The study was constrained by the fact that it was a secondary-data study, it was limited to three medication classes, and did not include medication-discontinuation variables due to structural missingness, and a cleaned 1,000-patient subset. Causal associations could not hence be determined. Subsequent investigations are needed to assess dose, time of medication usage, and time of withdrawal, exposure to nephotoxins with other drugs, and long-term renal outcome in larger cohorts.

5. Conclusion

This was a study to assess the relationship between the possible exposure to nephrotoxic drugs and patient clinical outcomes in hospitalized patients with acute kidney injury. The results indicated that the unfavorable outcomes such as AKI progression, dialysis, mortality, and the composite outcome were clinically relevant in the study population. PPI exposure showed a number of important crude relationships with worse outcomes and ACEi/ARB and NSAID exposure displayed lower rates on a number of outcomes. Nevertheless, these associations with medication were significant, but after the multivariate adjustment, they were highly diluted, which suggests that the effects of medication exposure alone did not significantly explain the overall risk of adverse clinical outcomes. The severity of illness turned out to be a significant prognosis factor. The composite adverse outcome, AKI progression, mortality, and dialysis had a consistent predictor of higher SOFA scores. There was an independent age-related relationship with 14-day mortality and a greater baseline creatinine was a predictor of dialysis. These results indicate that a clinical severity and baseline renal status might play a greater role than personal exposure to medication in short-term outcomes after AKI. On the whole, the findings confirm the vigilance of reviewing medications in hospitalized patients with AKI, but stress that risk assessment must also involve systemic illness severity, renal function, and comorbidities of the patients. Further studies are needed on the dose, duration, combinations, and discontinuation time, as well as the long-term renal outcomes in larger prospective cohort studies.

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