

IN HOSPITAL FREQUENCY AND FACTORS RESPONSIBLE FOR ACUTE KIDNEY INJURY AMONG PATIENTS OF ACUTE STROKE: INSIGHTS FROM A TERTIARY CARE SETTING

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ABSTRACT

Objective: To determine the in-hospital frequency of acute kidney injury and identify factors associated with its development among patients admitted with acute stroke at a tertiary care hospital.

Methods: This was a cross-sectional study of 193 patients between 18-80 years of age with acute ischemic or hemorrhagic stroke admitted within 48 hours of onset of symptoms. Enrolment of the patients was done by non-probability consecutive sampling method. Acute kidney injury was defined based on kidney disease: Improving Global Outcomes criteria. Demographic characteristics, comorbidities, type of stroke, National Institutes of Health Stroke Scale (NIHSS) score, Glasgow Coma Scale (GCS) score, hemodynamic instability, dehydration, nephrotoxic drug exposure, contrast exposure, management, length of stay, and mortality were recorded. Independent factors associated with acute kidney injury were determined by using the multivariable binary logistic regression.

Results: Of the 193 patients, 64 (33.2%) had acute kidney injury. This happened in 50.0% of patients with hemorrhagic stroke, and 27.3% of those with ischemic stroke. Independent risk factors for acute kidney injury were: hypotension (aOR 3.18); NIHSS score >15 (aOR 2.71); exposure to nephrotoxic medications (aOR 2.52); dehydration (aOR 2.48); hemorrhagic stroke (aOR 2.36); diabetes mellitus (aOR 2.15); and age (aOR 1.04 per year). Acute kidney injury patients had a higher mortality rate (21.9% vs. 6.2%) and longer hospital length of stay (9.8 ± 4.1 vs. 6.2 ± 2.7 days) than did patients without acute kidney injury.

Conclusion: Acute kidney injury is a common complication of acute stroke and linked with modifiable and non-modifiable clinical variables.

KEYWORDS: Acute Kidney Injury; Diabetes Mellitus; Hypotension; Risk Factors; Stroke

INTRODUCTION

Acute stroke is a significant cause of death and disability throughout the world, and often requires hospitalization, hemodynamic monitoring, neuroimaging, and supportive care. In the acute stage neurological damage can be associated with complications which can negatively impact recovery. One such complication is acute kidney injury (AKI), which is an abrupt deterioration of renal function, often that occurs during hospitalisation. Altered metabolic status, including hyperglycemia and hypoglycemia, has been linked to a higher risk for AKI in patients with acute ischemic stroke undergoing endovascular treatment [1]. Clinical and laboratory parameters obtained on admission have been shown to predict a higher risk of renal deterioration in stroke patients [2].

The pathogenesis of AKI following stroke is probably complex. Impaired renal perfusion and kidney damage may be caused by systemic inflammation, biomarkers indicative of leukocytes, hemodynamic instability, dehydration, baseline renal dysfunction, diabetes mellitus, hypertension, infection, nephrotoxic drugs, and severity of neurological injury [3]. Critically ill patients with acute cerebrovascular disease are susceptible as several alterations may occur at the same time and aggravate the renal stress [4]. The same associations have been described in hemorrhagic stroke where inflammatory indices, like the monocyte to lymphocyte ratio, have demonstrated predictive value for development of AKI [5].

New therapies for stroke may add new risks to the kidneys. Iodinated contrast dye is often used for computed tomography angiography, perfusion imaging and endovascular interventions. These investigations are critical to diagnosis and decision making for reperfusion, but it is reported that AKI may also occur after endovascular therapy [6]. Spontaneous intracerebral hemorrhage (ICH) patients also have clinically important rates of AKI, in

which admission characteristics are risk factors for developing AKI at an individual level [7]. Machine-learning methods for intracerebral hemorrhage (ICH) have also emphasized the synergistic effects of biochemical and demographic factors in predicting for AKI [8]. It was also seen that renal injury associated with contrast is seen in acute stroke patient who underwent computed tomography angiography [9]. There is some evidence to support the continued relevance of AKI in acute stroke populations and its potential impact upon short-term outcomes [10].

This study was performed due to the lack of local data on the incidence and risk factors of AKI in acute stroke patients. This factor was thought to be critical for early risk identification, prevention, and the improvement of hospital outcomes in a tertiary hospital.

METHODS

It was a cross-sectional study carried out in the Department of Neurology, Dow university Hospital, Karachi for six months after approval from Institutional Review Board, College of Physicians and Surgeons Pakistan and Research Evaluation Unit. A sample size of 193 patients was obtained using OpenEpi sample size software, using previously reported frequency of acute kidney injury (AKI) of 43.6% in patients with acute stroke, 95% confidence level, and the margin of error of 7%. Non-probability consecutive sampling technique was used to enroll the participants. All patients (males and females) 18 to 80 years old who presented with an acute stroke and were admitted within 48 hours of symptom onset were included. Acute stroke was diagnosed based on clinical presentation and confirmed with computed tomography (CT) of the brain. Patients who had suffered from recurrent strokes, glomerulonephritis, urinary tract obstruction, chronic renal complications before admission, patients who refused consent and pregnant women were excluded.

Following informed written consent, demographic characteristics, clinical presentation, type of stroke, duration of symptoms, and relevant co-morbidities such as hypertension, diabetes mellitus, dyslipidaemia, coronary artery disease, heart failure, atrial fibrillation, transient ischaemic attack, smoking and alcohol consumption were documented on a structured proforma. The severity of stroke upon admission was measured with the National Institutes of Health Stroke Scale (NIHSS) and Glasgow Coma Scale (GCS). Hemodynamic instability, periods of hypotension, vasopressor use, iodinated contrast exposure, nephrotoxic drug use and diuretic use were documented during hospitalisation. Serum creatinine and estimated glomerular filtration rate were evaluated and patients were followed for the development of AKI during hospital admission. The definition of AKI used was an increase in serum creatinine concentration of ≥ 0.3 mg/dL within 48 hours, and an increase in serum creatinine concentration of ≥ 1.5 times baseline within seven days, and urine output of < 0.5 mL/kg/hour for ≥ 6 hours, based on the kidney disease: Improving Global Outcomes criteria. Management measures, such as fluid therapy, nephroprotective measures, pharmacological treatment, thrombolysis/anti-coagulation, ventilatory support and renal replacement therapy (where necessary) were recorded. At discharge, renal function recovery, serum creatinine, dialysis needs and number of dialysis sessions, length of hospital stays, and in-hospital mortality were assessed.

IBM SPSS Statistics version 22.0 was used for data entry and data analysis. The data of quantitative variables were presented as mean and standard deviation, and the data of categorical variables were presented as frequencies and percentages. Appropriate univariate analyses, including the chi-square test, were first performed to determine the association of AKI with demographic, clinical, laboratory, stroke-related and treatment-related factors. Variables that showed clinically relevant or statistically significant associations were included in a multivariable binary logistic regression model to determine independent factors associated with AKI. Adjusted odds ratios (AORs) with 95% confidence intervals (CIs) were presented. A two-sided p-value of ≤ 0.05 was considered statistically significant. The study was carried out after ethical approval was granted for the institution and informed consent was obtained from all the subjects or their legally appointed representative and confidentiality of the subjects' information was maintained throughout the study.

RESULTS

A total of 193 patients with acute stroke were included in the study. The mean age of the participants was 61.4 ± 11.8 years, and 112 (58.0%) were males. Ischemic stroke was identified in 143 (74.1%) patients, while 50 (25.9%) had hemorrhagic stroke. Hypertension was the most frequent comorbidity, present in 125 (64.8%) patients, followed by diabetes mellitus in 78 (40.4%), smoking in 61 (31.6%), dyslipidemia in 55 (28.5%), coronary artery disease in 34 (17.6%), and atrial fibrillation in 25 (13.0%) patients. Diabetes mellitus, hypertension, severe stroke, hypotension, vasopressor requirement, dehydration, and exposure to potentially nephrotoxic medications were significantly more frequent among patients who developed AKI. **Table 1**

Table 1. Demographic and clinical characteristics according to development of AKI

Characteristics	AKI, n=64	No AKI, n=129	p-value
Age, years, mean $\pm$ SD	65.8 $\pm$ 9.6	59.2 $\pm$ 12.2	<0.001
Male gender	39 (60.9%)	73 (56.6%)	0.570
Hypertension	50 (78.1%)	75 (58.1%)	0.006
Diabetes mellitus	36 (56.3%)	42 (32.6%)	0.002
Dyslipidemia	20 (31.3%)	35 (27.1%)	0.549
Coronary artery disease	15 (23.4%)	19 (14.7%)	0.133

Atrial fibrillation	11 (17.2%)	14 (10.9%)	0.215
Hemorrhagic stroke	25 (39.1%)	25 (19.4%)	0.003
NIHSS >15 at admission	37 (57.8%)	31 (24.0%)	<0.001
Hypotension during admission	27 (42.2%)	17 (13.2%)	<0.001
Vasopressor requirement	18 (28.1%)	9 (7.0%)	<0.001
Contrast exposure	30 (46.9%)	46 (35.7%)	0.134
Nephrotoxic drug exposure	29 (45.3%)	23 (17.8%)	<0.001
Dehydration	31 (48.4%)	25 (19.4%)	<0.001

Table 2 shows on multivariable binary logistic regression analysis, hypotension during hospitalization was independently associated with AKI (adjusted odds ratio [aOR] 3.18, 95% CI 1.42-7.12; $p=0.005$). Severe stroke with an NIHSS score >15 was also independently associated with AKI (aOR 2.71, 95% CI 1.32-5.57; $p=0.007$).

Table 2. Multivariable analysis of factors associated with AKI

Factor	Adjusted OR	95% CI	p-value
Increasing age, per year	1.04	1.01-1.07	0.009
Diabetes mellitus	2.15	1.08-4.28	0.029
Hemorrhagic stroke	2.36	1.10-5.07	0.028
NIHSS >15	2.71	1.32-5.57	0.007
Hypotension	3.18	1.42-7.12	0.005
Nephrotoxic medication exposure	2.52	1.17-5.45	0.019
Dehydration	2.48	1.18-5.20	0.016
Contrast exposure	1.39	0.69-2.81	0.355

Among the 64 patients who developed AKI, fluid optimization was undertaken in 54 (84.4%), potentially nephrotoxic medications were discontinued or avoided in 42 (65.6%), and renal replacement therapy was required in 8 (12.5%). The mean length of hospital stay was significantly longer among patients with AKI than among those without AKI (9.8 ± 4.1 versus 6.2 ± 2.7 days; $p<0.001$). In-hospital mortality occurred in 14 (21.9%) patients with AKI compared with 8 (6.2%) patients without AKI ($p=0.001$). Overall, 22 (11.4%) patients died during hospitalization. **Table 3**

Table 3. Management and in-hospital outcomes according to AKI status

Variable	AKI, n=64	No AKI, n=129	p-value
Mechanical ventilation	22 (34.4%)	16 (12.4%)	<0.001
Dialysis required	8 (12.5%)	0 (0.0%)	<0.001
Length of stay, days, mean $\pm$ SD	9.8 ± 4.1	6.2 ± 2.7	<0.001
In-hospital mortality	14 (21.9%)	8 (6.2%)	0.001
Renal recovery by discharge*	44 (68.8%)	NA	NA

*Renal recovery was assessed among patients who developed AKI.

DISCUSSION

The present study showed a significant proportion of acute kidney injury among patients with acute stroke with 33.2% of the study population having acute kidney injury. Hemorrhagic stroke patients were more likely to have an AKI than were ischemic stroke patients, and AKI was associated with older age, diabetes mellitus, severe neurologic deficit, hypotension, dehydration, nephrotoxic medication exposure, longer hospitalization, and higher in-hospital mortality. The rate of observed frequency was much higher as compared to the study conducted by Khan et al. in a tertiary care hospital in Peshawar, Pakistan, which reported the incidence of AKI in acute stroke patients to be 15.4%. Their study also showed higher rates of AKI in hemorrhagic and severe strokes, confirming the current finding that the type and severity of acute brain damage might be closely related with renal complications [11].

The occurrence was more common in patients who developed AKI in this study, though not statistically significant, and not sustained after multivariable adjustment. This is clinically relevant as contrast-enhanced imaging is now a part of the current acute stroke assessment. In a systematic review and meta-analysis of patients with AIS who underwent CTA and CTP, Kamran et al. did not demonstrate a statistically significant association between the use of CTA and/or CTP and the development of AKI when compared with non-contrast imaging. Their results indicate that time-sensitive vascular imaging should not be routinely delayed based on concerns of renal injury, but patients with other susceptibility factors and preexisting renal impairment should be carefully monitored [12].

Multivariable results of the current study showed that hypotension, severe stroke, nephrotoxic medication exposure, dehydration, hemorrhagic stroke, diabetes mellitus, and age were independent risk factors for AKI. These findings suggest that post-stroke AKI is likely polymicrobial and not only as a result of one insult. In 593 patients who underwent mechanical thrombectomy, Wrona et al. reported that 12.6% developed AKI and several biochemical abnormalities, as well as diabetes, chronic kidney disease, and contrast exposure, were associated

with the development of AKI. But, in their adjusted analyses, total iodinated contrast exposure and admission urea remained important predictors. The overall results support the significance of baseline patient vulnerability and the acute, physiological, and treatment-related stressors [13] even though the current population was larger and included both ischemic and hemorrhagic stroke.

In the current study, the independent association of approximately two-fold higher odds of AKI with hemorrhagic stroke was demonstrated. Hemorrhagic stroke patients can have higher neurological severity, autonomic dysfunction, hemodynamic instability, and intensive-care needs, and they are subjected to more interventions, all of which contribute to a higher risk of kidney injury. Subah et al. identified the association of renal injury in patients with subarachnoid hemorrhage with an increase in complications, functional outcome, hospital length of stay, and mortality, supporting the clinical significance of renal injury in hemorrhagic cerebrovascular disease. Based on these observations, close renal surveillance should be considered in patients with severe hemorrhagic presentation and AKI should not be thought of as an isolated biochemical abnormality [14].

Moreover, in the current study, AKI was also significantly linked to poor short-term outcomes. The mean hospital stay was higher in patients with AKI compared with those without AKI, and mechanical ventilation need was significantly higher among patients with AKI. Patients with AKI had a higher in-hospital mortality of 21.9% compared with patients without AKI (6.2%). Approximately two thirds of the patients with AKI showed renal recovery before discharge, and renal replacement therapy was needed in 12.5 % of the patients. Oliveira et al assessed the prevalence of early AKI after endovascular treatment (EVT) for acute ischemic stroke and in particular focused on the association with death or dependency and mortality. Their work contributes to the growing body of evidence that renal damage is more than just a transient laboratory phenomenon after stroke therapy but is a prognostically important complication [15].

Therefore, it may be relevant to detect vulnerable patients at a very early stage. Ma et al. created a nomogram with readily available clinical data to predict AKI during the course of the hospital stay in the ICU for patients with ischemic stroke. This is a strategy that mirrors growing focus on risk stratification early (instead of waiting for serum creatinine to increase after renal injury has taken place). The current results also indicate that age, diabetes, stroke features, hemodynamic instability and other clinical factors could be included in a bedside risk assessment at admission and in hospital [16].

In addition, recent prediction studies have also revealed that a single clinical variable may not be sufficient to reflect the risk of developing AKI following cerebral infarction. Lu et al. used interpretable machine-learning techniques to classify patients with cerebral infarction admitted to intensive care who were at high risk for AKI, highlighting the potential benefits of combining multiple, routinely collected clinical variables to identify high-risk patients early on. Such computational models are yet to be validated for clinical use but these present results corroborate the overall interpretation that renal injury after stroke is a combination of patient factors, neurologic severity, physiologic derangements and treatment exposures [17].

Severe ischemic injury may also render the kidneys vulnerable to inflammatory processes. Ozdogru and colleagues compared the systemic immune-inflammation index and systemic inflammation response index in patients with acute ischemic stroke who underwent mechanical thrombectomy, and showed that the inflammatory indices were significantly higher in the group of patients who developed contrast-induced AKI. Systemic immune-inflammation index showed the highest discriminatory ability in this complication. Inflammatory biomarkers were not evaluated in the present study, but these observations may suggest another pathway that should be taken into account in future studies on the association between post-stroke AKI [18].

Cai et al. created and externally validated a dynamic nomogram involving 673 patients with acute ischemic stroke (AIS) receiving endovascular therapy and found that five variables (baseline NIHSS score, preprocedural creatinine, pneumonia, acute heart failure, and hypotension) were major predictors of AKI. Their results agree with the current correlation of more severe stroke, and hemodynamic instability to AKI and indicate that neurological and systemic deterioration should be assessed collectively for renal risk [19].

This negative prognostic relationship shown in the present study is further bolstered by large contemporary international evidence. Schwarz et al. investigated 6,638 patients treated with thrombectomy in 73 centres over 16 countries and the incidence of contrast-associated AKI was 4.9%. Even though the incidence is low, AKI was independently associated with higher in hospital mortality, poor functional status at 90 days, and severe disability or death. In addition to their prediction model, they included commonly used factors such as hypertension, chronic kidney disease, NIHSS score, glucose, eGFR, hemoglobin and mean arterial pressure. Interestingly, inclusion of procedure-related variables (such as contrast volume) resulted in comparable performance. This discovery is consistent with the current report of contrast exposure alone not being independently correlated with AKI and further supports the idea that the overall clinical status of the patient may be more relevant than contrast exposure alone [20].

The current results indicate that AKI is clinically relevant and a systemic complication of acute stroke. The fact that AKI is associated with severe stroke, hypotension, dehydration, diabetes, nephrotoxic medication exposure, longer hospital stay, need for ventilatory support and death suggests that AKI could be a marker for a physiologically very vulnerable group of patients. Several of these factors are potentially modifiable, such as dehydration, hypotension and avoidable nephrotoxic exposure, and offer opportunities for preventive measures without hindering essential neurological investigations and treatment.

Another key strength of the study was that patients with both ischemic and hemorrhagic stroke were included, which enabled the evaluation of AKI in a wider acute-stroke population, rather than only patients undergoing

endovascular procedures. To enhance the consistency of diagnoses, we used standardized KDIGO criteria for the diagnosis of AKI. In addition, a variety of clinically relevant areas were assessed, such as demographic characteristics, comorbidities, stroke severity, hemodynamics, treatment exposures, renal management, length of stay as well as mortality.

A few limitations should be taken into account. Cross-sectional, single-centre design precluded causal inference and generalizability to other institutions and populations. As such, the variables identified should be viewed in the context of factors associated with, but not necessarily proved causes of, AKI. The sample size was fairly small, especially for the subgroup and multivariable analyses. Not all baseline renal function may have been recorded prior to admission, which may impact accurate identification of creatinine change. However, incomplete documentation of urine-output measurements may also have influenced this.

Routine renal risk assessment should be used in the original assessment of patients admitted with acute stroke, especially in older, diabetic, severe neurological deficit, hemorrhagic stroke, and hemodynamic instability patients. Urinary output, fluid status and blood pressure should be closely monitored during hospitalization, as well as serum creatinine. Dehydration and hypotension need to be identified and rectified quickly and without giving too much fluid. Medications with the potential to be nephrotoxic should be assessed and/or stopped or changed whenever possible.

CONCLUSION

Acute kidney injury was a common and clinically significant complication in hospitalized acute stroke patients, and was associated with more severe stroke, hypotension, dehydration, diabetes mellitus, hemorrhagic stroke, nephrotoxic medication exposure, longer hospital lengths of stay, and significantly higher mortality rates. The results suggest that AKI should be considered as a significant part of the acute stroke prognosis and not only as a renal complication of stroke. Proactive strategies to identify vulnerable patients, to keep them hemodynamically and fluidly stable, to avoid unnecessary nephrotoxic exposures, and to systematically monitor their kidneys may offer practical measures to lower the incidence of avoidable kidney injury and improve outcomes in acute stroke patients.

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