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Trends in the Prevalence and Risk Factors of Acute Kidney Injury in Hospitalized Patients with Acute Stroke in the University of Calabar Teaching Hospital, Calabar

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Abstract: Patients admitted to hospital for cerebrovascular accident are at a high risk of developing acute kidney injury (AKI) because of comorbid conditions, and other potential risk factors, such as exposure to nephrotoxic medications, dehydration or utilization of hypertonic crystalloid solutions. This study, therefore aims to determine the risk factors of acute kidney injury in patients hospitalized with acute stroke. A prospective cross-sectional study of patients hospitalized with acute stroke in the medical wards of the Department of Internal Medicine at the University of Calabar Teaching Hospital. A total of 46 patients were recruited, but only 39 patients met the inclusion criteria. Socio-demographic, clinical and biochemical data of participants were captured. There were more males 25 (64.1%) than females 14 (35.9%) with a mean age of 60.38±12.22 years for the study population. However, the mean age was 59.79±11.07 years and 60.72±13.02 years for participants with the presence and absence of AKI respectively; and AKI was significantly commoner in patients with haemorrhagic stroke (p = 0.038). The risk factors associated with AKI were haemorrhagic stroke, hypovolaemeia, malignant hypertension, diabetes mellitus and others including higher blood urea nitrogen, baseline creatinine, 48 hours creatinine and lower glomerular filtration rate (GFR). Moreso, the predictors of AKI were higher baseline creatinine (95% CI-1.500 - 2.329, p = 0.0001), lower GFR (95% CI- -1.012- (-0.00), P = 0.049) and higher baseline creatinine (95% CI-0.833 - 0.976, p = 0.011), malignant hypertension (95% CI-0.010 - 0.368, p = 0.002) in multiple linear regression and logistic regression analysis respectively.

Key words: Acute Kidney Injury · Acute Stroke · Calabar · Prevalence

INTRODUCTION

Acute kidney injury is a common occurrence in hospitalized patients, most especially in intensive care unit (ICU) patients with very poor prognosis [1]. It is associated with 5-7% of acute care hospitalizations and up to 30% of patients admitted to intensive care unit [2]. A stroke or cerebrovascular accident is defined as an abrupt onset of focal neurological deficit that is attributable to a vascular origin and is the second leading cause of mortality worldwide [3].

According to the pathophysiological mechanisms, stroke is classified as either ischaemic stroke (IS) or haemorrhagic stroke (HS) [4]. About 80-85% of all strokes are ischemic stroke while hemorrhagic stroke accounts for about 10-20% [5, 6].

According to the World Health Organization (WHO), around 15 million people, in the world over, suffer from stroke each year. Among these, 5 million are permanently disabled. Four out of five strokes occur in the low and middle-income countries; Nigeria included and these countries can least afford to manage with the consequences of this disease [7].

Stroke is a major cause of disability and poor quality of life [8]. The high rates of disability and mortality encountered in stroke are determined not only by the

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neurological deficits but also by the associated medical comorbidities such as cardiovascular disease, hypertension, diabetes and renal dysfunction [9].

Acute kidney injury (AKI), characterized by the sudden deterioration of kidney function or kidney damage occurring over hours to a few days, is a common complication encountered both in acute ischaemic stroke (AIS) and intracerebral haemorrhage (ICH) [10].

It is worth noting that, in 2012, AKI was defined according to the Kidney Disease Improving Global Outcomes (KDIGO) as any of the followings: increase in Serum Creatinine by ≥0.3 mg/dl (≥ 26.5 µmol/l) within 48 hours; or increase in Serum Creatinine to ≥1.5 times baseline, which is known or presumed to have occurred within the prior 7 days; or decrease in urine volume to <0.5 ml/kg/hour for 6 hours [11].

Patients admitted to the hospital for cerebrovascular accident are at a high risk of developing AKI because of comorbid conditions, and other potential risk factors, such as exposure to nephrotoxic medications, dehydration or utilization of hypertonic crystalloid solutions [12]. AKI has a deleterious prognosis in the outcome of various medical conditions including acute stroke and it can be one of the important determinants in defining the outcome of an acute cerebrovascular event [13]. Moreso, AKI is common and imposes a heavy burden of illness; it is amenable to prevention which ultimately warrants prompt detection and effective management [13].

The development of AKI is associated with a significantly increased risk of in-hospital and long-term mortality, longer length of stay, and increased costs of care for patients [14]. However, to the best of our knowledge, data regarding the prevalence of AKI and its risk factors among patients hospitalized with stroke is limited in our environment. This study is therefore conducted to determine the prevalence of AKI and to identify the risk factors associated with the development of AKI in acute stroke patients admitted in the medical wards of the University of Calabar Teaching Hospital.

MATERIALS AND METHODS

Design and Settings: This is a cross-sectional study performed in the medical wards of the Department of Internal Medicine at the University of Calabar Teaching Hospital. Acute stroke patients hospitalized from April to November 2021 were consecutively enrolled in the study.

Study Population: We recruited a total of 46 patients admitted with acute stroke based on clinical and brain computerized tomography findings, but only 39 met the inclusion criteria concerning having a baseline creatinine levels and were included in the study.

Inclusion Criteria:

- Patients with evidence of Ischaemic or Haemorrhagic strokes
- Patients admitted to the hospital within 48 hours of the onset of stroke
- Patient with baseline creatinine value on the day of admission

Exclusion Criteria

- Patients with a history of glomerulonephritis and urinary tract obstruction
- Patients with pre-existing chronic kidney disease
- Patients with exposure to radiocontrast agents and nephrotoxic medications

Informed consent to participate in the study was obtained from the patients or their relatives and all the researchers followed the ethical guidelines provided in WMA Helsinki declaration.

Data Collection: Baseline medical including fundoscopy examination and neurological assessments were done in all patients. The demographic data included age, sex, and history of vascular risk factors, and relevant drug history. A non-contrast CT brain scan examination was done in all patients to diagnose and classify the stroke subtype.

Serum creatinine was measured (using kinetic colorimetric assay based on the Jaffe method) during the first day of admission for all patients and repeated after 48 hours. The KDIGO criteria were applied to define and categorize patients who developed AKI into stages using the Acute Kidney Injury Network (AKIN) [11]. Accordingly, patients who had a creatinine of 0.3 mg/dl (26.5 µmol/l) or more within 48 hours or 1.5 fold increment or more within 7 days from first reading was considered to have AKI. Subsequently, AKI is categorized into stages as follows:

Stage 1 - Serum creatinine 1.5-1.9 times baseline or ≥ 26.5 µmol/l increase OR Urine output < 0.5 ml/kg/hr for 6-12 hours.

Table 1: Demographic and clinical characteristics of study participants

Variable	Total (N=39), n (%)	Ischaemic Stroke (N=25), n (%)	Haemorrhagic Stroke (N=14), n (%)	Statistics Value	p-value
Gender					
Female	14(35.9)	9(36.0)	5(35.7)	0	0.986
Male	25(64.1)	16(64.0)	9(63.3)	(LR)	
Age Category (years)					
18–30	1(2.6)	1(4.0)	0(0.0)		
31–50	7(17.9)	3(12.0)	4(28.6)	4.807	0.186
51–70	24(61.5)	18(72.0)	6(42.9)	(χ^2)	
Above 70	7(17.9)	3(12.0)	4(28.6)		
Place of Residence					
Rural	8(20.5)	4(16.0)	4(28.6)	0.844	0.358
Urban	31(79.5)	21(84.0)	10(71.4)	(LR)	
Level of Sensorium					
Conscious	20(51.3)	17(68.0)	3(21.4)	8.148	0.004*
Unconscious	19(48.7)	8(32.0)	11(78.6)	(LR)	
Family History of Stroke					
Yes	3(7.7)	2(8.0)	1(7.1)	0	0.923
No	36(92.3)	23(92.0)	13(92.9)	(LR)	
Hypertension					
Present	34(87.2)	21(84.0)	13(92.9)	0.682	0.409
Absent	5(12.8)	4(16.0)	1(7.1)	(LR)	
Risk of AKI					
Present	28(71.8)	15(64.0)	12(85.7)	2.246	0.134
Absent	11(29.2)	9(36.0)	2(14.3)	LR	
Comorbidities					
Present	35(89.7)	21(84.0)	14(100.0)	3.81	0.051
Absent	4(10.3)	4(16.0)	0(0.0)	(LR)	
AKI					
Present	14(35.9)	6(24.0)	8(57.1)	4.284	0.038*
Absent	25(64.1)	19(76.0)	6(42.9)	(×2)	
Stage of AKI	**(N=14),n (%)**	**(N=6), n (%)**	**(N=8), n (%)**		
Stage 1	6(42.9)	5(83.3)	1(12.5)		
Stage 2	5(35.7)	1(16.7)	4(50.0)	8.711	0.013*
Stage 3	3(21.4)	0(0.0)	3(37.5)	(LR)	

AKI - Acute kidney injury, χ^2-Chi-square, LR-Likelihood ratio; *Significant p-value

Stage 2 - Serum creatinine 2-2.9 times baseline OR Urine output < 0.5 ml/kg/hr for 12 hours.

Stage 3 - Serum creatinine 3.0 times baseline or increase in serum creatinine to ≥ 353.6 µmol/l OR Initiation of dialysis OR Urine output < 0.3 ml/kg/hr for >24 hours OR anuria for >12hours.

Laboratory Evaluation: Laboratory measurements included serum creatinine (SCR), electrolytes, blood sugar, and routine examination of urine. CrCl (ml/min) was calculated using the Cockroft and Gault formula [15]: CrCl = (140-age) × weight (kg)/ (serum creatinine × 72 (×0.85 for women)).

Data Analysis: The data generated in the study was analyzed using Statistical Package for Social Sciences (SPSS) version 20.0. The appropriate statistical methods were applied in determining the prevalence and risk factors for AKI. Categorical variables were expressed as percentages and continuous variables as means ±SD. The chi-square test was used to determine significant associations between categorical variables while the student's t-test was used to assess the difference between two means. Pearson's correlation coefficient (r) was used to determine the association between and across means of the variables. Logistic regression analysis was used to determine the predictors of the dependent variables. Results are considered significant at $p < 0.05$.

RESULTS

Table 1 showed that the majority of the study participants were males (64.1%) and most presented with ischaemic stroke (64.1%). In terms of their sensorium, most participants with ischaemic stroke were conscious (68.0%) while those with haemorrhagic stroke were

Table 2: Univariate analysis to determine the risk factors for AKI

Variable	Odds Ratio (OR)	95% CI	p-value
Male Gender	1.667	0.407-6.818	0.475
Level of Sensorium (Unconscious)	0.589	0.157-2.207	0.431
Haemorrhagic Stroke	0.237	0.058-0.961	0.038*
Hypertension	2.476	0.249-24.646	0.409
Sepsis	1.333	0.350-5.076	0.673
Hypovolaemia	9.600	0.951-96.922	0.030*
Nephrotoxic Medications	1.583	1.242-2.019	0.342
Malignant Hypertension	9.778	1.965-48.665	0.003*
Diabetes Mellitus	4.629	1.142-18.752	0.027*

*Significant p-value

Table 3: Student's T-Test to determine the risk factors for AKI

Variable	Total (N=39), Mean ± SD	AKI Present (N=14), Mean ± SD	AKI Absent (N=25), Mean ± SD	p-value
Age (years)	60.38±12.22	59.79±11.07	60.72±13.02	0.814
Glasgow coma score	11.92±3.68	11.29±3.93	12.28±3.57	0.441
Systolic BP	167.18±42.30	187.86±48.70	155.60±34.04	0.040*
Diastolic BP	95.64±21.13	101.43±21.79	92.40±20.47	0.216
Sodium (mmol/L)	137.45±5.24	138.03±5.91	137.12±4.93	0.631
BUN (mmol/L)	10.41±3.19	13.56±6.05	8.64±3.70	0.012*
Baseline Creatinine (µmol/L)	111.42±44.65	148.42±55.56	90.70±15.42	0.002*
48 hours Creatinine (µmol/L)	150.57±109.89	258.00±123.87	90.40±17.49	0.0001*
eGFR (mLs/min)	71.40±35.76	36.70±22.79	90.83±25.44	0.0001*

BP-Blood pressure, BUN-Blood urea nitrogen, eGFR-Estimated glomerular filtration rate; *Significant p-value

Table 4: Multiple regression analysis to determine the predictors of AKI

Variable	Beta	t	95% CI	p-value
Age (years)	-0.081	-1.707	-1.600-0.142	0.098
Glasgow coma score	-0.074	-0.451	-12.128-7.709	0.654
Systolic BP	0.045	0.568	-0.303-0.536	0.574
Diastolic BP	-0.016	-0.216	-0.897-0.726	0.831
Sodium (mmol/L)	-0.025	-0.534	-2.506-1.467	0.591
BUN (mmol/L)	0.069	0.934	-1.732-4.661	0.357
Baseline Creatinine (µmol/L)	0.780	9.541	1.500-2.329	0.0001*
eGFR (mLs/min)	-0.165	-2.044	-1.012-(-0.00)	0.049*

BP-Blood pressure, BUN-Blood urea nitrogen, eGFR-Estimated glomerular filtration rate; *Significant p-value

Table 5: Logistic regression analysis to determine the predictors of AKI

Variable	Exp(B)	95% CI	p-value
Age (years)	1.020	0.960-1.084	0.524
Glasgow coma score	1.057	0.848-1.317	0.624
Systolic BP	0.970	0.939-1.094	0.076
Diastolic BP	1.021	0.963-1.094	0.552
Sodium (mmol/L)	0.921	0.758-1.118	0.404
BUN (mmol/L)	1.165	0.883-1.537	0.280
Baseline Creatinine (µmol/L)	0.901	0.833-0.976	0.011*
48 hours Creatinine (µmol/L)	0.809	0.629-1.040	0.099
eGFR (mLs/min)	1.286	0.626-2.641	0.493
Gender	0.565	0.116-2.756	0.480
Level of Sensorium	0.842	0.148-4.792	0.846
Type of Stroke	4.565	0.839-24.830	0.079
Hypertension	0.584	0.044-7.825	0.684
Sepsis	0.735	0.176-3.066	0.673
Hypovolaemia	0.104	0.010-1.051	0.055
Malignant Hypertension	0.061	0.010-0.368	0.002*
Diabetes Mellitus	0.279	0.067-1.161	0.079

BP - Blood pressure, BUN - Blood urea nitrogen, eGFR - Estimated glomerular filtration rate; *Significant p-value

unconscious (78.6%) and this is statistically significant ($p<0.05$). AKI was present in 35.9% of the patients and there is a significantly higher incidence in those with haemorrhagic stroke as compared to those with ischaemic stroke (57.1% vs 24.0%, $p=0.038$) and a significant proportion with ischaemic stroke are in stage 1 AKI (83.3%), $p<0.05$).

In Table 2, the univariate analysis showed that the risk factors associated with the development of AKI are patients with haemorrhagic stroke, diabetes mellitus, and the presence of hypovolaemia and malignant phase hypertension.

The independent sample T-Test in Table 3 showed that the risk factors for AKI were higher systolic BP, higher levels of BUN, baseline creatinine, 48 hours creatinine and lower eGFR.

In the multiple regression analysis in Table 4, the predictors of AKI were higher baseline creatinine level and lower eGFR.

In Table 5, further analysis for the predictors of AKI using logistic regression showed that higher baseline creatinine level and malignant phase hypertension were predictors of AKI.

DISCUSSION

This study was conducted to determine the prevalence and risk factors associated with the development of AKI in patients admitted with acute stroke. In this study, 64.1% of the study population had ischaemic stroke while 35.9% had haemorrhagic stroke, and this has a similar proportion to the global distribution of stroke type; but has varied actual percentages (80-85%) for ischaemic stroke and 10-20% for haemorrhagic stroke [5, 6]. The difference in the stroke type population could be attributed to the patients that were excluded in the study, as they all had ischaemic stroke. Also, our study had more males (64.1%) with higher prevalence in both ischaemic (64.0%) and haemorrhagic stroke (63.3%), and this is similar to reports in other studies [13, 16-18].

In this study, AKI is shown to be a common complication as 35.9% of the study population developed AKI. This finding varies from the prevalence in a Nigerian study and other studies outside Nigeria. The study in Nigeria reported a prevalence of 24% for renal dysfunction in acute stroke, mainly acute renal dysfunction [19]. On the other hand, some studies outside Nigeria reported prevalence of AKI in acute stroke ranging from 13-18%, but a study in Mexico reported a higher prevalence of 54% [13, 16-18, 20]. The variation in the prevalence of AKI in acute stroke in the various studies enumerated may be possibly explained in the differences in the sample sizes and the criteria used in defining AKI in these studies. In addition, the pre-admission creatinine level was not available in our study; as such the exact baseline level of kidney function is unknown.

Moreso, the incidence of AKI in patients with HS is significantly higher than those with IS (57.1% vs 24.0%), and this finding is in keeping with reports from earlier mentioned studies [13, 17, 18]. It has been shown that the significant difference in AKI in the stroke type could be attributed to the use of nephrotoxic medications such as mannitol which is more frequently administered in patients with HS [18]. In this study, the vast majority of patients with AKI were in AKIN stage 1 and most of them were patients with HS; this is keeping with report by Khatri *et al.* [18].

The risk factors for AKI in this study using univariate analysis were HS, hypovolaemia, malignant HTN and the presence of DM. However, analysis with the independent T-Test revealed elevated SBP invariably HTN, elevated BUN, higher baseline SCR, higher 48 hours SCR and lower GFR as risk factors of AKI. Further analysis showed that the predictors of AKI were higher baseline SCR, lower GFR and higher baseline SCR in multiple regression and logistic regression analysis respectively. Several studies reported similar findings as enumerated in our study, while some observed significant risk factors for AKI; others reported predictors of AKI in regression analysis. There are studies in keeping with the observations described in our study regarding the risk factors of AKI in acute stroke in the univariate and independent T-Test analysis, but HS was not statistically significant in the study by Khatri *et al.* [12, 16-19, 21]. Moreso, hypovolaemia in our study could be due to the use of osmotic diuretics such as mannitol especially in HS patients which is observed in other studies [17, 21]. In as much as our study revealed a significant association of HTN and AKI in stroke, like in other studies, but we went ahead to classify the HTN as malignant HTN, which was not done in the other studies [12, 16]. The presence of DM was shown to be a risk factor for AKI in this study, corroborated with some studies, but a study found elevated blood sugar as risk factor rather than DM [16, 17, 21]. Regarding higher 48 hours creatinine in our study, a study in Iraq however reported a consecutive rise in SCR from baseline reading from the first day to second day and the seventh day of admission of patients [16]. The studies by Covic et al and Khatri et al reported higher baseline SCR and lower GFR as independent predictors of AKI in acute stroke patients which is in tandem with our observations as earlier mentioned in the regression analysis [17, 18].

CONCLUSION

The study has shown that HS, hypovolaemia, malignant HTN, DM, higher BUN, higher baseline SCR, higher 48 hours SCR and lower GFR were significant risk factors for the development of AKI in acute stroke patients. However, the independent predictors of AKI were higher baseline SCR, lower GFR and malignant HTN.

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REFERENCES

1. Koza, Y., 2016. Acute kidney injury: Current concepts and new insights. Journal of Injury and Violence Research, 8: 58-62.

2. Waikar, S.S. and J.V. Bonventre, 2018. Acute kidney injury. Harrison's Principles of Internal Medicine, Eds., In: Jameson, J.L., A.S.Fauci, D.L. Kasper, *et al*. New York: McGraw-Hill Education, 2099-2111.
3. Smith, W.S., S.C. Johnston and J.C. Hemphill, 2018. Cerebrovascular disease. Harrison's principles of internal medicine, Eds., In: Jameson, J.L., A.S. Fauci, D.L. Kasper, *et al*. New York: McGraw-Hill Education, 3068.
4. Koh, S.H. and H.H. Park, 2017. Neurogenesis in stroke recovery. Translational Stroke Research, 8: 3-13.
5. Boursin, P., S. Paternotte, B. Dercy, C. Sabben and B. Maïer, 2018. Semantics, epidemiology and semiology of stroke. Soins; La Revue De Reference Infirmiere, 63: 24-27.
6. Nicholas, L., A. Matthew, M.B. Martin, G. Joan and S. Robert, 2016. Stroke and Cerebrovascular Diseases. Neurology A Queen Square Textbook, Eds., In: Charles C, et al. Wiley Blackwell, London, pp: 133-135.
7. Thrift, A.G., D.A. Cadilhac, T. Thayabaranathan, G. Howard, V.J. Howard, P.M. Rothwell and G.A. Donnan, 2014. Global stroke statistics. International Journal of Stroke, 9: 6-18.
8. De haan, R.J., M. Limburg, J.H. Vander Meulen and H.M. Jacobs, 1995. Quality of life after stroke. Impact of stroke type and lesion location. Stroke, 26: 402-408.
9. Theofanidis, D., 2015. Management of acute stroke: a debate paper on clinical priorities. a literature review. International Journal of Caring Science, 8: 201-210.
10. Ansaritoroghi, M., S.P. Nagaraju, R.P. Nair, V. Kumar, L.I. Kongwad, R.P. Attur and A. Hegde, 2019. Study on acute kidney injury in patients with spontaneous Intracerebral hemorrhage: an overview from a tertiary Care Hospital in South India. World Neurosurgery, 123: 740-746.
11. Kellum, J., N. Lameire, P. Aspelin, R. Barsoum, E. Burdmann, S.L. Goldstein and C.A. Herzog, 2012. Kidney disease: improving global outcomes (KDIGO) acute kidney injury work group. KDIGO clinical practice guideline for acute kidney injury. Kidney International, Suppl(2): 1-138.
12. Nadkarni, G.N., A.A. Patel, I. Konstantinidis, A. Mahajan, S.K. Agarwal, S. Kamat, N. Annapureddy, A. Benjo and C.V. Thakar, 2015. Dialysis Requiring Acute Kidney Injury in Acute Cerebrovascular Accident Hospitalizations. Stroke, 46: 3226-3231.
13. Bhattacharjee, K., N. Paul and S. Roy, 2020. Acute kidney injury in acute stroke in relation to immediate outcome - a hospital based study. Journal of Evidence Based Medicine and Healthcare, 7: 475-479.
14. Waikar, S.S. and J.V. Bonventre, 2013. Acute kidney injury. Harrison's Nephrology and acid-Base disorders, Eds., In:Jameson, J.L, *et al*. New York:, McGraw Hill-Hill Education, pp: 104-115.
15. Cockroft, D.W. and M.H. Gault, 1976. Prediction of creatinine clearance from serum creatinine. Nephron, 16: 31-41.
16. Zaid, W.A., A.A. Ali and A.A. Hasan, 2019. Acute kidney injury in patients with acute stroke. Iraqi Postgraduate Medical Journal, 18: 234-241.
17. Covic, A., A. Schiller, N.G. Mardare, L. Petrica, M. Petrica, A. Mihaescu and N. Posta, 2008. The impact of acute kidney injury on short-term survival in an Eastern European population with stroke. Nephrology Dialysis Transplantation, 23: 2228-2234.
18. Khatri, M., J. Himmelfarb, D. Adams, K. Becker, W.T. Longstreth and D.L. Tirschwell, 2014. Acute kidney injury is associated with increased hospital mortality after stroke. Journal of Stroke and Cerebrovascular Disease, 23: 25-30.
19. Eze, C.O., 2023. Prevalence of Renal Dysfunction in Acute Stroke Patients At Abakaliki Nigeria: An Observational Study. West African Journal of Medicine, 40: S20.
20. Olguin, L., F. Góngora-Rivera, H. Martinez and J. Celis-Jasso, 2019. Acute kidney injury increases mortality in acute stroke. A study in Hispanic population. Journal of the Neurological Sciences, 405S: 105258.
21. Lin, S.Y., S.C. Tang, L.K. Tsai, S.J. Yeh, L.J. Shen, F.L. Wu and J.S. Jeng, 2015. Incidence and Risk Factors for Acute Kidney Injury Following Mannitol Infusion in Patients with Acute Stroke: A Retrospective Cohort Study. Medicine (Baltimore), 94: e2032.