Original Article

Predictors of mortality among acute stroke patients with kidney failure in University of Port Harcourt Teaching Hospital, Port Harcourt, Rivers State

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Abstract

Background: Stroke is considered a leading cause of morbidity and mortality globally. Renal disease is also increasing globally with a consequent increase in morbidity and mortality. The cross-link between the brain and the kidney leading to the complex relationship between stroke and renal disease which may have adverse effects on mortality has been studied.

Aim: The study sought to determine the predictors of mortality among acute stroke patients admitted into the University of Port Harcourt Teaching Hospital (UPTH), Port Harcourt.

Methods: This was a hospital-based, prospective cohort study conducted among 150 acute stroke patients presenting at the UPTH. Selected stroke patients with kidney failure and those without kidney failure were assessed and followed up for 6 weeks for mortality. A multivariate logistic regression analysis was carried out to demonstrate the predictors of mortality.

Results: Of the one hundred and fifty participants studied, there were 93(62.0%) male and 57(38.0%) female patients, the mean age was 54.6±10.6 years. There were 95 stroke patients with Kidney Failure (KF) giving a prevalence of 63.3%. The predictors of mortality among respondents included age 70 – 79years (aOR=51.14; p – 0.005), severe Glasgow Coma Score (GCS) (aOR=7.58; p – 0.001),haemorrhagic stroke (aOR=3.99; p- 0.001), admitting hyperglycaemia (aOR=2.53; p – 0.042) and CKD (aOR=2.81; p – 0.00).

Conclusion: The predictors of mortality included age group 70 – 79years, severe GCS, haemorrhagic stroke, admitting hyperglycaemia and CKD. Therefore, early detection and prompt intervention will reduce morbidity and mortality in these patients.

Keywords: Stroke, predictors, mortality

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INTRODUCTION

Stroke is one of the major non-communicable diseases contributing to Global Disease Burden (GDB) due to its high mortality and morbidity.[1] Deaths from stroke accounted for 11.9% of all global deaths in 2015.[2] In Africa, there were over 483,000 new cases of stroke in 2009.[3] This high incidence was attributed to population growth and a rise in many modifiable vascular disease risk factors, including smoking, excessive use of alcohol,

physical inactivity, increased prevalence of hypertension, diabetes and obesity.[3] Furthermore, the incidence of stroke is seen to be declining in developed countries due to the vigorous efforts at lowering blood pressure and reducing smoking.[4]

In Nigeria, there is an increase in the prevalence of stroke and other cardiovascular diseases due to the epidemiological transition from a traditional socio-cultural setting to a Western, sophisticated life in the city.[5]

Chronic kidney disease as a cause of death worldwide has risen by 31.7% between 2005 and 2015, with an increase in the ranking of total Years of Life Lost (YLL) from the 21st to the 17th position over the same period.[2] The prevalence of impaired kidney function has been estimated to be between 10% and 20% of the adult population in most countries worldwide.[6] A moderate-to-severe decrease in eGFR was also associated with a high incidence of first-ever stroke and all-cause mortality in an ethnic Chinese population-based cohort.[7]

Acute kidney injury is characterised by abrupt deterioration in renal function and has a deleterious prognosis in the final outcome of various medical conditions, notably stroke. However, it is a common comorbid condition in the community and may be associated with other cardiovascular diseases, diabetes mellitus, hypertension and cerebrovascular events. It complicates 5-7% of acute care hospitalisations and around 30% of those in intensive care units.[8]

Kidney failure has been associated with a high prevalence of cardiovascular disease, and patients with reduced renal function are at high risk for the subsequent development of cerebrovascular diseases, including stroke. Renal function among patients with stroke has been studied in the developed countries in hospital-based studies.[9-10] Almost all types of vascular disease, including stroke are associated with varying degrees of kidney failure, and the severity of stroke could be an indication of the degree of injury in small renal vessels.[11] However, mortality among stroke patients with kidney failure has not been well investigated in Africa, particularly in Nigeria, despite the rising incidence of stroke and kidney failure following the event. This study is aimed at determining the predictors of mortality among acute stroke patients with kidney failure in UPTH.

METHODOLOGY

Study area

The study was conducted in the Accident and Emergency (A&E) department, the intensive care unit and the Department of Internal Medicine, UPTH. The University of Port Harcourt Teaching Hospital is a 740-bed hospital and referral centre for Rivers State and neighbouring states (Bayelsa, Abia, Cross Rivers, Edo, Delta and Imo states). It is located in Alakahia in the Obio-Akpor Local Government Area of Rivers State. Rivers State has a heterogeneous population consisting of several local tribes as well as foreigners involved in various activities in the area mainly of the oil and gas sector.

The hospital is made up of a 130-bed space medical ward and a 30-bed space accident and emergency. A total of 212 cases of stroke were admitted via the A & E of the hospital from January 1st, 2021 to December 31st, 2021 (Data from the hospitals' A & E records).

Study design

The study was a hospital-based prospective cohort study.

Study population

This consisted of all consecutive adults (≥18 years) admitted for stroke.

Inclusion criteria

Respondents eligible for the study should be adults aged 18 years and above admitted to the hospital within 7 days of onset of stroke, patients with neuroimaging evidence of stroke and stroke patients who grant informed consent personally or from their caregivers.

Exclusion criteria

Respondents with stroke mimics, for example, patients with intracranial space-occupying lesions, and hypoglycaemia, who died within

24 hours of admission, who has repeat stroke and had kidney transplant were excluded from the study.

Sampling

Patients who satisfied the eligibility criteria and had none of the exclusion criteria were recruited consecutively until a sample size of 150 was obtained.

Sample size determination

The minimum sample size required for this study was calculated from the method of Kish[12]

$n = z^2pq/ d^2$

Where:

n = sample size when the population is infinite

z = the standard normal deviation, usually set at 1.96 which corresponds to a 95% confidence interval

p = Prevalence of renal dysfunction among stroke patients estimated at 9.3%[13]

q = 1-p

d = degree of accuracy desired, 0.05%

n = (1.96)2 x 0.093 x 0.907/ (0.05)2

n =129.6

10% attrition gives 12.9 (approximated to 13)

Therefore, the minimum sample size would be 129.6 +13 =142.6

However, a sample size of 150 stroke patients was recruited, for the study.

Study proforma

A structured questionnaire was used as the survey instrument to collect information about the subjects' socio-demographic characteristics, risk factors for stroke, assessment of kidney disease, assessment of stroke, anthropometry, laboratory investigations, radiological features of the kidneys and interim outcomes.

Two research assistants were trained to administer the questionnaire and extract data. They also helped in ensuring adequate follow-up of the participants.

Study protocol/procedure

A structured questionnaire was used as an instrument to collect information about the respondent's socio-demographic data, medical history of renal disease in the patient and family, diabetes, hypertension, alcohol consumption, smoking, medication use, trauma, history of TIA/stroke, cardiovascular disease, etc. Information about the risk factors of stroke and stroke assessment was also collected through the questionnaire, which was administered by the researcher after obtaining informed consent. Subsequently, a baseline physical and neurological examination was performed.

Selected stroke patients were categorised as having AKI, pre-existing CKD, acute-on-chronic CKD and no kidney failure after assessment of their renal function using electrolyte, urea and creatinine at presentation, 48 hours, 7th day and 6 weeks post-stroke. In addition, urinalysis and measurement of urine output were performed to assess renal function while renal imaging was performed to assess kidney size. Patients were then followed up for 6 weeks for mortality outcomes and the predictors of mortality determined.

Measurements

Anthropometric measurements were carried out with the participants' privacy duly respected and with a chaperone when required.

Participants' Waist Circumference was measured at the midpoint between the inferior margin of the last palpable rib and the top of the iliac crest, while the Hip Circumference was measured at the largest posterior extension of the buttocks. Waist and Hip Circumferences were measured to the nearest 0.1cm. Their Waist-to-Hip Ratio (WHR) was calculated and metabolic risks noted as WHR $\geq$ 0.90cm for men and $\geq$ 0.85cm for women.[14]

The blood pressure (BP) of the participants was measured with a mercury sphygmomanometer and stethoscope to obtain both systolic blood pressure (SBP) and diastolic blood pressure (DBP). Two BP

readings were taken for each arm, and the average BP was used in the study.

Blood pressure measurement was done according to recommendations of the American Heart Association Council on Hypertension.[15]

Sample collection

A spot urine sample was obtained in a clean, dry universal bottle provided for each participant. to assess for proteinuria, which is measured by dipstick using combi 9 (meditest combi 9 – Duren Germany). Furthermore, urine was measured over a 24-hour period by the researcher to estimate the 24-hour urine output.

For biochemical investigations, serum creatinine was measured using the modified Jaffe reaction method. Serum creatinine, urea & electrolytes were measured at presentation and repeated after 48hours, on the 7th day of admission and 6th-week post-stroke.

A fluoride oxalate bottle using standard techniques was used to estimate fasting plasma glucose, while a lithium heparin bottle was also used to collect the sample for the estimation of the following: Total Cholesterol (TC), High-Density Lipoprotein Cholesterol (HDL-C), and Triglycerides (TG).

Using the Friedwald equation[16] $LDL=TC-HDL-\frac{TG^*}{5}$.

These samples were analysed in the chemical pathology laboratory of the hospital.

The eGFR was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation[17] as follows:

$$eGFR = 141 \times min\ (SCr/\kappa, 1)\ \alpha \times max\ (SCr/\kappa, 1) - 1.209 \times 0.993Age \times 1.018\ [if\ female] \times 1.159\ [if\ Black]$$

Where, eGFR = ml/ min/1.73m^2

SCr (standard serum creatinine) = mg/dl

K= 0.7 (females) or 0.9 (males)

α= -0.329 (female) or -0.411 (male)

Min= indicates the minimum of SCr/κ or 1

Max= indicates the maximum of SCr/κ or 1

Age= in years

The severity of CKD was assessed by the National Kidney Foundation Kidney Disease Outcomes Quality Initiative Improving Global Outcome (NKF KDOQI)[18] criteria to stratify CKD, while the severity of AKI was stratified according to KDIGO, as shown in Table 1.

Table 1: Staging of AKI according to KDIGO

Stage	Serum creatinine	Urine output
1	1.5–1.9 times baseline within 7 days OR ≥0.3 mg/dl (≥26.5 µmol/l) increase within 48 hours	< 0.5 ml/kg/h for 6–12 hours
2	2.0–2.9 times baseline	< 0.5 ml/kg/h for ≥12 hours
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 In patients <18 years, decrease in eGFR to <35 ml/min per 1.73 m2	≤0.3 ml/kg/h for ≥24 hours OR Anuria for ≥12 hours

Renal USS was used to assess the features of CKD, such as reduced bipolar length and transverse diameter, increased echogenicity, and loss of cortico-medullary differentiation. This was done by the researcher in the renal unit under a radiologist's guidance, while a

mobile ultrasound machine was used for patients who could not be moved to the renal unit.

Participants were categorised into 4 groups:

1. Stroke patients with no kidney failure which were considered as stroke patients who did not have any renal compromise: defined as serum creatinine of 52-106 µmol/l, an eGFR of > 90mL/min/1.73 m^2 according to the guidelines of the National Kidney Foundation.[17]
2. Stroke patients that develop AKI: defined as either an increase in serum creatinine by ≥26.5µmol/l or 0.3mg/dl within 48hours or an increase in serum creatinine to >1.5 times baseline, which is known or presumed to have occurred within the prior 7 days with urine volume of <0.5ml/kg/hour for 6 hours.[18]
3. Stroke patients with pre-existing CKD: For this study, patients were considered to have a pre-existing CKD if they had a hospital record of CKD or if patients or their relatives gave a history of CKD or had previous evidence of serum creatinine elevation or had evidence of elevated serum creatinine (>1.5 mg/dl) or have structural evidence kidney disease.[19]
4. Stroke patients with Acute-on-Chronic renal impairment: defined as stroke patients who develop AKI with evidence of background pre-existing CKD.

Respondents' level of consciousness was assessed by the Glasgow Coma Scale (GCS) and rated as mild (GCS >13), moderate (GCS 8–13), or severe (GCS ≤7).

The severity of the stroke was graded according to the National Institute of Health Stroke Scale[20] (NIHSS) score, as shown in Table 2. The NIHSS questionnaire was administered by the researcher within 24 hours of presentation and on the 7th day of admission. The maximum score obtainable for the NIHSS was 42, and the minimum score was 0.[21] The poor outcome was defined as less than an 8-point improvement in the NIHSS score by the 7th day of admission or death on or before the 7th day.[22] The outcome of interest was death within 6 weeks.

Table 2: National Institute of Health stroke score and stroke severity

Stroke severity	Score
No stroke symptoms	0
Mild stroke	1-4
Moderate stroke	5-15
Moderate to severe stroke	16-20
Severe stroke	21-42

Data management

Data entry was done using a Microsoft Excel sheet and exported into a Statistical Package for Social Sciences (SPSS 23). (IBM Corp, Armonk, New York, USA). The SPSS package was used for data cleaning and analysis. Before data entry was done into Microsoft Excel, the study questionnaire was thoroughly checked after the data collection exercise to ensure the correctness and completeness of each for all participants in the study.

Data analysis

Data analysis was done at the univariate and multivariate analysis levels. At the univariate analysis level, categorical variables like sex, educational level, ethnicity, prevalence of kidney failure, pattern of kidney failure, type of stroke etc were summarized as frequencies and percentages while continuous variables like age, urine output, serum creatinine, serum potassium, eGFR were summarized using median and interquartile range since the data were not normally distributed (skewed).

Bivariate analysis was carried out to identify the characteristics associated with mortality. All stroke patients with kidney failure were identified, and the factors associated with mortality were assessed by comparing the occurrence of these factors in stroke patients with kidney failure and stroke patients without kidney failure using the Chi-square test. The factors associated with mortality compared included age group 70–79years, diabetes mellitus, hypertension/diabetes mellitus, severe stroke, severe GCS score, eGFR stage 2 – 5 at presentation, proteinuria, haemorrhagic

stroke, mannitol use and admitting hyperglycaemia., A multivariate logistic regression analysis was carried out to further explore the predictors of mortality. The level of significance was set at p-value < 0.05.

ETHICAL CONSIDERATION

Ethical clearance for the study was obtained from the Ethical Review Committee of the University of Port Harcourt Teaching Hospital, Port Harcourt, with reference number UPTH/ADM/90/S.11/VOLXI/1318.

Written informed consent was obtained from the subjects and the caregivers gave consent for the unconscious patients. The participants were informed that participation was voluntary and that they would not suffer any consequences if they chose not to participate.

All information gathered was kept confidential, as access to this information was limited to the investigator and the managing team when appropriate. All participants were identified using only serial numbers.

All laboratory investigations were done at no cost to the participants, and results were used to aid the management of the participants. In addition, participants were counselled on the findings of their clinical data and the results of their laboratory investigations.

RESULTS

Sociodemographic characteristics, anthropometric measurements and fasting lipid profile of the patients

The study consisted of 150 participants, of which 93 were males, and 57 were females with a male- to-female ratio of 1.6:1. The age range of patients was between 30 and 75 years with a mean age of 54.6 ±10.6years. About half of the respondents had tertiary education, almost 90% are Christians by religion and over 50% of the respondents are high income earners.

Table 3 shows the sociodemographic characteristics of the study participants.

Table 4 shows the anthropometric measurements and fasting lipid profile, of the study participants. The mean Waist Hip Ratio of the participants was 0.92 ± 0.44cm and the mean of the lipid components was described.

Fifty-eight patients had high metabolic risk based on the anthropometric measurements while sixty-nine of patients were seen to have dyslipidaemia. (Table 5)

Table 5 shows the proportion of patients with high metabolic risk based on anthropometry and lipid profile among the study participants.

Clinical characteristics of stroke patients in the study

It was noted that 96.7% of the respondents were hypertensive, eighteen (12%) patients were current smokers while majority of the patients (77.3%) live a sedentary life. Family history of hypertension was the most prominent family history of medical condition as found in 92 (61.3%) patients. Seventy-one (47.3%) patients and eighty (53.3%) patients had a history of changes in urination and changes in urine appearance respectively.

Medical and clinical characteristics of patients in the study are shown in Table 6.

Severity of stroke among study participants

Figure 1 shows the severity of stroke assessed by the NIHSS score. The median score for the stroke patients at presentation was 20 (IQR:18 – 24); on the 7th day post-stroke it was 10 (IQR: 8 – 14) while on the 6th week post-stroke it was 4 (IQR: 2 – 6).

Characteristics associated with mortality among stroke patients in the study

Relationship between sociodemographic characteristics/medical history and mortality outcomes.

Table 7 showed the relationship between sociodemographic characteristics and mortality demonstrating that of the 150 respondents, 95(63,3%) died and 55(36.7%) survived. Only age group 70 – 79 years (χ^2 = 19.32, p – 0.006) had statistically significant relationship with the occurrence of mortality in the study. The presence of diabetes (χ^2 =13.86, p – 0.001) and presence of diabetes/hypertension (χ^2 =15.70, p – 0.004) were also significant medical histories associated with the occurrence of mortality in

these patients. The gender of participants did not show significant relationship with mortality. (χ^2 =1.02, p – 0.312).

Relationship between stroke severity/clinical features and mortality outcome.

Table 8 showed that severe stroke symptoms depicted as high NIHSS score at presentation (χ^2 =30.02, p – 0.001), severe LOC as determined by the GCS (χ^2 =26.30, p – 0.001), eGFR stages 3 - 5 (p – 0.002, 0.001, 0.001 respectively) at presentation, presence of proteinuria (χ^2 =5.86, p – 0.017), haemorrhagic stroke (χ^2 =4.55, p - 0.001), mannitol use (χ^2 =2.65, p – 0.005) admitting hyperglyceamia (χ^2 = 3.55, p – 0.001) and presence of CKD (χ^2 =12.19, p – 0.0004) were significantly related to the occurrence of mortality in the study.

Table 9 demonstrated that age group 70 - 79years (aOR=51.14,95% CI: 3.32 – 788.66, p – 0.005), severe GCS (aOR=7.58, 95% CI:3.28 – 17.51, P – 0.001), hemorrhagic stroke (aOR=3.99,95% CI: 1.66 – 9.56, p – 0.002), admitting hyperglycemia (aOR=2.53,95% CI:1.03 – 6.19, p – 0.042) and presence of CKD (aOR= 2.81, 95% CI: 1.47 – 9.54 , p – 0.001) are the predictors of mortality among the participants in the study.

Table 3: Sociodemographic characteristics of stroke patients in the study

Characteristics	Frequency N = 150	Percent (%)
Sex		
Male	93	62.0
Female	57	38.0
Age group		
30 - 39 years	14	9.3
40 - 49 years	39	26.0
50 - 59 years	46	30.7
60 - 69 years	43	28.7
≥ 70 years	8	5.3
Age in years – Mean ± SD	**54.6 ± 10.6**	
Education		
No formal Education	18	12.0
Primary Education	8	5.3
Secondary Education	55	36.7
Tertiary Education	69	46.0
Ethnicity		
Ijaw	99	66.0
Igbo	42	28.0
Hausa	5	3.3
Yoruba	4	2.7
Religion		
Christian	134	89.3
Islam	10	6.7
Others	6	4.0
Income		
High-income Earner	88	58.7
Middle-income Earner	35	23.3
Low-income Earner	27	18.0

Table 4: Anthropometric and biochemical findings among stroke patients in UPTH, Port Harcourt

Parameter	Mean ± SD
Anthropometric measurement (in cm)	
Waist Circumference	93.2 ± 9.8
Hip Circumference	99.6 ± 12.4
Waist Hip Ratio	0.92 ± 0.44
Lipid profile (in mmol/l)	
Total Cholesterol	5.2 ± 0.5
Triglycerides	1.5 ± 3.0
LDL Cholesterol	3.3 ± 0.3
HDL Cholesterol	1.5 ± 0.8

LDL –Low Density Lipoprotein, HDL – High Density Lipoprotein

Table 5: Proportion of patients with high metabolic risk and dyslipidaemia among participants in the study

Variables	Frequency N = 150	Percent (%)
Metabolic risk		
Normal	92	61.3
High metabolic risk	58	38.7
Lipid profile		
Normal lipid profile	81	54.0
Dyslipidemia	69	46.0

Table 6: Medical history and clinical characteristics of patients in the study

Characteristics	Frequency	Percent (%)
Comorbidity		
Diabetes mellitus	58	38.7
Hypertension	145	96.7
No HTN/no DM	16	10.7
DM only	4	2.7
HTN only	88	58.7
Both HTN/DM	42	28.0
Social history		
Tobacco Use	18	12.0
Exercise		
No Exercise	116	77.3
Some exercise	34	22.7
Family history of medical conditions		
Family history of Stroke	12	8.0
Family history of hypertension	92	61.3
Family history of Diabetes	28	18.7
Family history of CKD	27	18.0

History of symptoms		
History of Body Swelling	32	21.3
History of Reduced Urine Output	47	31.3
History of Change in Urination	71	47.3
History of Frequency	17	11.3
History of change in urine appearance	80	53.3
History of Frothy Urine	56	37.3
History of bloody Urine	5	3.3
History of dark colored Urine	33	22.0
History of Obstructive Urinary Symptoms	17	11.3
Use of Herbal Concoction	36	24.0

HTN – Hypertension, DM – Diabetes

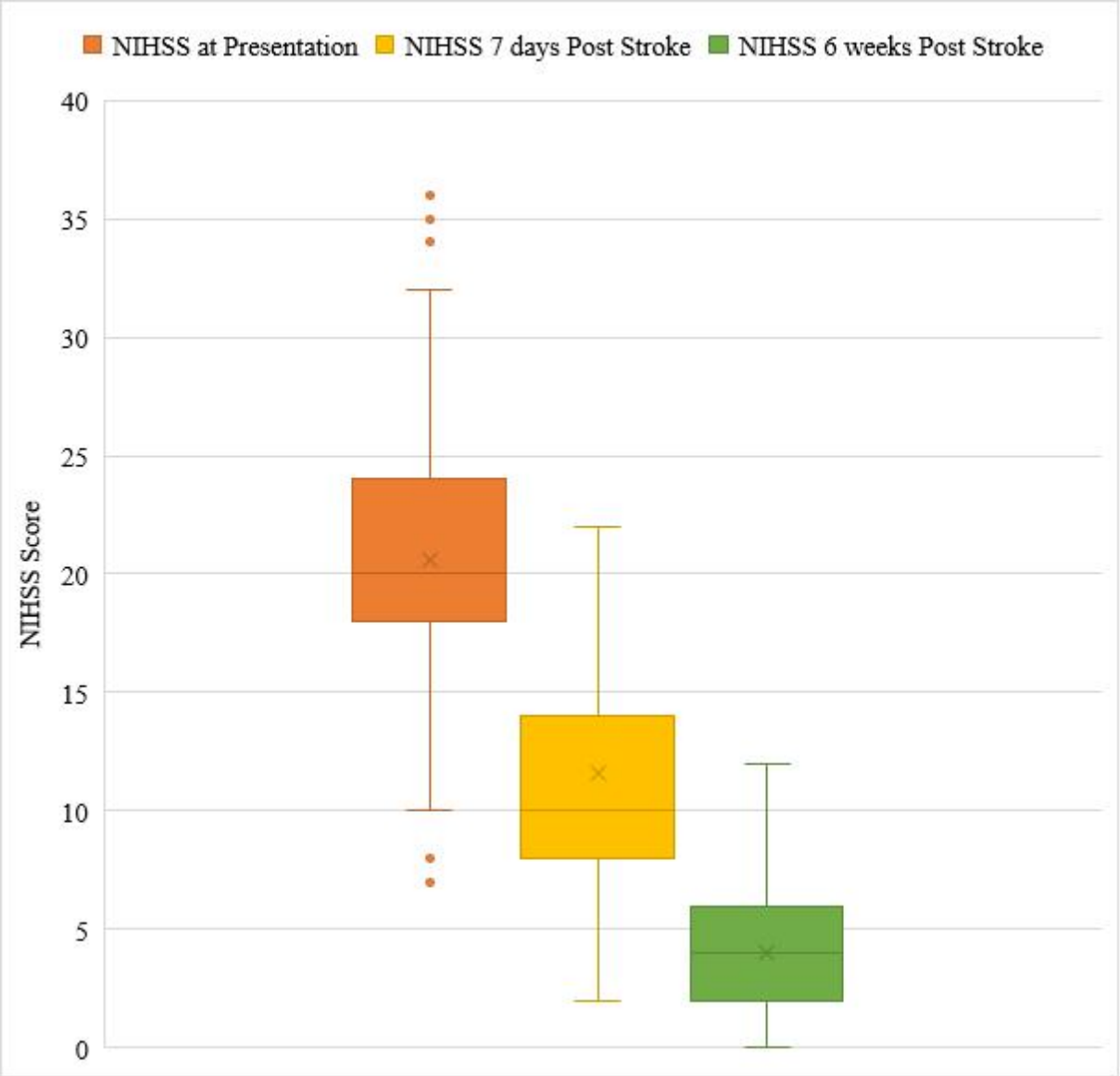

Figure 1: Box and whisker chart showing the NIHSS scores in patients at presentation, 7th day and 6th-week post-stroke among study participants

Table 7: Association between sociodemographic characteristics/ clinical characteristics and mortality outcome among stroke patients in UPTH, Rivers state.

Characteristics	Total N = 150	Outcome		χ^2 (pValue)	Crude Odd Ratio (95% CI)	pValue
		Died N = 55 (%)	Survived N = 95 (%)			
Sex						
Male	93	37 (39.8)	56 (60.2)	1.02 (0.311)	1.43 (0.71 – 2.87)	0.312
Female	57	18 (31.6)	39 (68.4)		1	
Age Group						
30 - 39 years	14	1 (7.1)	13 (92.9)	19.32 (0.001*)	1	
40 - 49 years	39	10 (25.6)	29 (74.4)		4.48 (0.52 – 38.76)	0.173
50 - 59 years	46	25 (54.3)	21 (45.7)		15.48 (1.87 – 128.30)	0.11
60 - 69 years	43	13 (30.2)	30 (69.8)		5.63 (0.67 – 47.67)	0.113
70 - 79 years	8	6 (75.0)	2 (25.0)		39.00 (2.93 – 518.84)	0.006*
Use of Herbal Concoction						
Yes	41	17 (41.5)	24 (58.5)	0.56 (0.455)	1.32 (0.63 – 2.76)	0.455
No	109	38 (34.9)	71 (65.1)		1	
Metabolic risk						
Normal	92	33 (35.9)	59 (64.1)	0.06 (0.799)	1	
High metabolic risk	58	22 (37.9)	36 (62.1)		1.09 (0.55 – 2.16)	0.799
Hypertension						
Yes	130	51 (39.2)	79 (60.8)	2.76 (0.097)	2.58 (0.82 – 8.16)	0.106
No	20	4 (20.0)	16 (80.0)		1	
Diabetes mellitus						
Yes	46	27 (58.7)	19 (41.3)	13.86 (0.001*)	3.86 (1.86 – 8.00)	0.001*
No	104	28 (26.9)	76 (73.1)		1	
Hypertension and Diabetes Mellitus						
No HTN/DM	16	2 (12.5)	14 (87.5)	15.70 (0.001*)	1	
HTN only	88	26 (29.5)	62 (70.5)		2.93 (0.62 – 13.84)	0.174
DM only	4	2 (50.0)	2 (50.0)		7.00 (0.60 – 81.68)	0.121
HTN+DM	42	25 (59.5)	17 (40.5)		10.29 (2.07 – 51.22)	0.004*

HTN – Hypertension, DM – Diabetes Mellitus

Table 8. Relationship between stroke severity/clinical features and mortality outcome.

Characteristics	Total N = 150	Outcome		χ^2 (pValue)	Crude Odd Ratio (95% CI)	PValue
		Died N = 55 (%)	Survived N = 95 (%)			
NIHSS Score at Presentation						
Moderate Stroke Symptom	22	1 (4.5)	21 (95.5)	30.02 (0.001*)	1	
Moderate to Severe Stroke Symptom	61	14 (23.0)	47 (77.0)		6.25 (0.77 – 50.72)	0.086
Severe Stroke Symptom	67	40 (59.7)	27 (40.3)		31.11 (3.95 – 245.23)	0.001*
Glasgow Coma Scale						
Moderate LOC	82	15 (18.3)	67 (81.7)	26.30 (0.001*)	1	
Severe LOC	68	40 (58.8)	28 (41.2)		6.38 (3.05 – 13.37)	0.001*
eGFR at Presentation						
Stage 1	37	4 (10.8)	33 (89.2)	24.16 (0.001*)	1	
Stage 2	22	4 (18.2)	18 (81.8)		1.83 (0.41 – 8.22)	0.428
Stage 3	33	15 (45.5)	18 (54.5)		6.88 (1.98 – 23.84)	0.002*
Stage 4	28	14 (50.0)	14 (50.0)		8.25 (2.31 – 29.52)	0.001*
Stage 5	30	18 (60.0)	12 (40.0)		12.38 (3.48 – 44.02)	0.001*
Proteinuria						
Proteinuria	90	40 (44.4)	50 (55.6)	5.86 (0.015*)	2.40 (1.17 – 4.92)	0.017*
No Proteinuria	60	15 (25.0)	45 (75.0)		1	
Stroke Type						
Ischaemia	98	24 (24.5)	74 (75.5)	18.05 (0.001*)	1	
Haemorrhagic	52	31 (59.6)	21 (40.4)		4.55 (2.21 – 9.35)	0.001*
Mannitol Use						
Yes	70	34 (48.6)	36 (51.4)	8.01 (0.005*)	2.65 (1.34 – 5.26)	0.005*
No	80	21 (26.3)	59 (73.8)		1	
FBS						
Normoglycaemia	111	32 (28.8)	79 (71.2)	11.29 (0.001*)	1	
Hyperglycaemia	39	23 (59.0)	16 (41.0)		3.55 (1.66 – 7.58)	0.001*
CKD						
Yes	36	22(40.0)	14(14.7)	12.19 (0.0004*)	3.81 (1.74 – 8.54)	0.001*
No	114	33(60.0)	81(85.3)		1	
AKI						
Yes	45	9 (20)	36 (80)	2.04 (0.15)	1	0.015
No	95	30(49.5)	65(50.5)		0.54(0.23-1.27)	

Predictors of mortality among stroke patients in the study

Table 9: Predictors of mortality among stroke patients in UPTH, Port Harcourt

Characteristics	B-coefficient	Adjusted OR (95% CI)	pValue
Age Group			
30 - 39 years		1	
40 - 49 years	1.28	3.60 (0.37 – 34.53)	0.267
50 - 59 years	2.51	12.33 (1.34 – 113.43)	0.027
60 - 69 years	1.22	3.39 (0.36 – 31.80)	0285
70 - 79 years	3.93	51.14 (3.32 – 788.66)	0.005*
Glasgow Coma Scale			
Moderate loss of consciousness		1	
Severe loss of consciousness	2.03	7.58 (3.28 – 17.51)	0.001*
Stroke Type			
Ischaemia		1	
Haemorrhagic	1.38	3.99 (1.66 – 9.56)	0.002*
Fasting Blood Sugar			
Normoglycaemia		1	
Hyperglycaemia	0.93	2.53 (1.03 – 6.19)	0.042*
eGFR @ Presentation			
Stage 1		1	
Stage 2	0.69	2.00 (0.27 – 15.05)	0.500
Stage 3	0.97	2.64 (0.48 – 14.56)	0.265
Stage 4	1.38	3.98 (0.62 – 25.56)	0.146
Stage 5	1.20	3.32 (0.59 – 18.71)	0.173
Hypertension + Diabetes mellitus			
No HTN/DM	-0.87	0.42 (0.03 – 5.76)	0.514
HTN	-0.10	0.90 (0.10 – 8.37)	0.929
HTN+DM	1.21	3.34 (0.30 – 37.49)	0.328
DM only		1	
NIHSS at Presentation			
Moderate Stroke Symptom		1	
Moderate to Severe Stroke Symptom	0.76	2.13 (0.20 – 22.59)	0.530
Severe Stroke Symptom	1.65	5.20 (0.36 – 74.72)	0.225
Mannitol Use			
Yes	-1.03	0.36 (0.10 – 1.25)	0.107
No		1	
Proteinuria			
Proteinuria	-0.34	0.71 (0.19 – 2.62)	0.611
No Proteinuria		1	
CKD			
Yes	1.34	2.81 (1.47 – 9.54)	0.001*
No		1	

DISCUSSION

This study observed that more males than females were found among the stroke population. This was similar to the finding by Shittu[23] in Ogbomosho, Medhat et al[24] in Egypt, and Pereg et al[25] in Israel who reported more males among acute stroke patients in their studies. The similarity in the findings from these studies may be because of the improvement of more women from stroke than men in some countries due to the sensitivity of women to health information, health-seeking behaviours and early access to primary

prevention of stroke[26] as well as increased neurovascular risk, factors such as current cigarette smoking, use of illicit drugs and significant alcohol consumption in men.

The mean age of the study population was 54.6 ±10.6 years. This is comparable to the mean age of 53.9 ± 18.1 years reported in the study by Sulaiman et al[27] in Maiduguri, Nigeria. The preponderance of young and middle-aged acute stroke patients in these studies may be attributed to the increasing prevalence of stroke in the young globally. However, these findings are in contrast to those reported by Okaka et al[28] in Benin and Vijay et al[29] in India, with higher mean ages of 63.28 ±15.22 years and 60.36 ±10.7years respectively. The disparity may be attributed to the small sample sizes used in their studies.

This study revealed that advanced age (70 – 79 years) was a predictor of mortality among acute stroke patients (aOR = 51.14, p – 0.005). This finding is similar to that reported by Nedeltchev et al[30] in Switzerland and kamil et al[31] in Poland. In Egypt, Madhat et al[24] demonstrated that advanced age is a predictor of mortality in their study to evaluate the prevalence of renal dysfunction among acute ischemic stroke patients and its role on the early overall mortality. Here in Nigeria, Busari et al[32] in their study to evaluate renal dysfunction and 30 – day mortality risk in patients with acute stroke in a tertiary centre in Lagos also reported a comparable result among the study participants.

The aging of the general population worldwide is viewed as one of the critical health challenges in the twenty-first century. With advancing age, patients are prone to more co - morbidities, having at least three chronic disorders. For instance, about 60% of the population greater 70 years have dementia which will lead to non- compliance with medications increasing the risk of repeat stroke leading to increased morbidity and mortality in these patients.

The study participants' severe Glascow Coma Score of was demonstrated to be a predictor of mortality in the study (aOR =7.58, p –0.001). This findings constrast that done by Sharanabasappa Nandyal and Nanda Nandyal[35] in India which showed that moderate GCS was a predictor of mortality among the study participants. The differences in the findings from both studies may be explained by the average age of the study populations. The average age of the participants in this study is 54.6 ± 10.6 years, with most patients in the age range of 50 – 59years compared to the work by Sharanabasappa Nandyal and Nanda Nandyal[35] which had most study participants with age > 65years. Busari et al[32] also revealed that severe GCS score was not a predictor of mortality. This finding is at variance with that shown in this study and the disparity in the results from both studies may be due to the small sample size and the shorter follow-up period of only 30 days in the study by Busari et al.[32]

A decreased level of GCS has a strong association with adult stroke patients' mortality. This is because the stroke may lead to injury to the brain stem which houses the respiratory and circulatory centres, so they may develop respiratory and circulatory compromise.[34]

Furthermore, decreased GCS following stroke worsens the stroke-induced autonomic hyperactivity which brings about sympathetic activity and systemic inflammation disrupting renal homestasis[35] leading to AKI. Patients with severe stroke at presentation are often at increased risk of dehydration as they have a reduced level of consciousness, are physically dependent, unable to communicate, have difficulties in swallowing and decreased oral intake which may predispose them to acute kidney injury. AKI which is a risk factor for CKD and accelerates the progression of CKD to ESRD in stroke patients who had background CKD, CKD is also a risk factor for AKI[9,36]. This vicious circle may also contribute to the increased mortality in stroke patients presenting with severe GCS.

The presentation with haemorrhagic stroke was noted as a predictor of mortality in this study. (aOR =3.99, p – 0.002). This corroborates with the findings of Busari et al,[32] and Colombo et al.[39] Jover-Saenz et al[40] and

Mbala et al[39] reported similar findings. The poorer prognosis in haemorrhagic than ischemic stroke is probably due to the lower GCS at presentation, more rapid deterioration, and the greater frequency of complete paralysis which may have accounted for the higher mortality in patients with haemorrhagic stroke. Also, the higher frequency of systemic hypertension and higher average SBP and DBP in patients with haemorrhagic stroke may also contribute to the increased mortality in these patients.

In contrast, Kallol et al[40] in a hospital-based study to evaluate AKI in acute stroke in relation to immediate outcomes and Sharanabasappa Nandyal[33] showed that haemorrhagic stroke is not a predictor of mortality in their study participants. This disparity in the findings may be due to the fact that Kallol et al recruited only stroke patients with AKI while Sharanabasappa considered only stroke patients with CKD. Recall that the participants in this study include those with AKI and CKD.

Admitting hyperglycaemia among stroke patients with renal impairment has been reported to be a predictor of mortality in these patients (aOR =2.53, p – 0.044). This finding is coherent with Busari et al.[32]

The mechanisms that could explain presenting hyperglycaemia as a predictor of mortality among stroke patients with renal impairment are as follows: First, hyperglycaemia may be directly toxic to the ischaemic brain probably due to accumulation of lactate and intracellular acidosis in the ischemic brain through anaerobic cerebral glucose metabolism[41] resulting in a greater infarct volume thereby promoting the recruitment of potentially salvageable neuron into the infarction. Second, Patients with stress- hyperglycaemia are more likely to have undiagnosed diabetes which predisposes them to sustain more ischaemic damage at the time of the infarction.[42] Third, reactive hyperglycemia may disrupt the blood-brain barrier and promote hemorrhagic infarct conversion.[43]

Furthermore, admitting hyperglycaemia may lead to renal hypoxia, oxidative stress, altered renal vasoreactivity and volume depletion caused by osmotic diuresis thereby increasing the risk and progression of AKI with consequent increase in morbidity and mortality.

The finding from this study is at variance with that done by Columbo et al[37] which showed that admitting hyperglycaemia is not a predictor of mortality in their study. The difference in the findings from this study and those conducted by Columbo et al may be attributed to the definition of random blood glucose using different cut-off values.

The presence of CKD was also demonstrated as a predictor of mortality among stroke patients in this study (aOR =2.81, p – 0.001). This finding is similar to that reported by Busari et al[32] in Lagos, Seifu et al[46] in Ethiopia, Tsagalis et al[9] and Hao et al[45] in Europe.

CKD serves as a predictor of mortality among acute stroke because of the higher prevalence of both traditional and non-traditional cardiovascular risk factors[46-47] thereby making it serve as an independent risk factor for cardiovascular events or death.[9]

CONCLUSION

The predictors of mortality among acute stroke patients demonstrated in this study include; Advanced age, Admitting hyperglycaemia, severe Glasgow coma score, haemorrhagic stroke and CKD.

Limitations of the study

Due to the lack of an adequate renal database, some patients with pre-existing CKD may have been missed.

GFR was assessed using prediction equations rather than by gold standard methods of measurement such as radioisotope methods and measurement using Iothalamate and Ioxohol.

Recommendations

Random blood glucose should be done for all stroke patients on presentation at the emergency department irrespective of their diabetic status and Patients with severe Glasgow coma score should be managed in an

intensive care unit for better care in order to reduce mortality.

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Conflict of interest:

There are no conflicts of interest.

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