Predictors and prognoses of new onset post-stroke anxiety at one year in black Africans

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Background: There is relatively limited information on the risk factors and outcome of new onset Poststroke Anxiety (PSA) in Low- and Middle-Income Countries. We estimated incidence, cumulative incidence, risk factors and outcome of new onset anxiety in the first year of stroke among African stroke survivors. *Methods:* We analyzed the dataset of a completed clinical trial comprising patients enrolled to test an intervention designed to improve one-year blood pressure control among recent ($\leq$ one month) stroke survivors in Nigeria. Anxiety was measured using the Hospital Anxiety and Depression Scale. Outcomes were assessed using the modified Rankin Scale (mRS), Community screening instrument for dementia (CSID) and Health Related Quality of Life in Stroke Patients (HRQOLISP-26). *Results:* Among 322 stroke survivors who were free of anxiety at baseline, we found a one-year cumulative incidence of 34% (95% CI = 28.6−39.3). Rates were 36.2% (95% CI =29.6−42.7) for men and 29.2% (95% CI =19.9−38.3) for women. In multivariate Cox regression analyses, haemorrhagic stroke type was associated with higher risk of new onset PSA (Hazard Ratio=1.52, 95% CI =1.01−2.29). New onset PSA was independently associated with cognitive [(mean difference (MD) in CSID scores=1.1, 95% C.I=0.2, 1.9)] and motor decline (MD in mRS scores= −0.2, 95% C.I= −0.4, −0.02), as well as poorer quality of life overtime (MD in total HRQOLISP-26 scores=3.6, 95% C.I=1.0, 6.2). *Conclusion:* One in 3 stroke survivors in Nigeria had PSA at one year. Clinicians in SSA should pay special attention to survivors of haemorrhagic stroke as they are at higher risk of incident anxiety and therefore its consequences.
Keywords: Mental health complications—Stroke outcomes—Poststroke emotional disturbances—Stroke burden—cohort studies

Introduction

Anxiety rates in stroke survivors[1] are higher than rates reported in the general population.[2] While poststroke depression is also highly prevalent,[3] stroke survivors worry about recurrence of stroke, their ability to return to work, stigma and falls.[4] Poststroke anxiety (PSA) is associated with more chronicity than depression,[5] and in this way, PSA may compromise rehabilitation efforts and result in functional dependency,[6] poor quality of life[7] and many other unwanted outcomes of stroke.[5] Fortunately, PSA is both pharmacologically and psychosocially treatable.[8, 9]

An important step towards early intervention is the identification of stroke survivors who are likely to be at increased risk of new onset anxiety. Evidence,[7, 10] derived mostly from high income countries (HICs), appears to suggest that new onset PSA may be associated with

unique sets of risk factors and outcomes. Information on incidence, risk factors and outcome of new onset PSA is currently unavailable in Low- and Middle-Income Countries (LMICs).

Low- and Middle-Income countries endure more than two thirds of the worldwide burden of stroke.[11] Of seven studies[1, 12, 13] on PSA conducted across six LMICs in the previous eleven years, only one implemented a prospective observation of anxiety after stroke. However, by focusing their aim on the longitudinal trajectory of severity, Sutter et al.[12] did not quantify incidence, risk factors and outcome of new onset PSA in Colombian survivors. Such information is desirable as it could inform the design and implementation of context appropriate interventions for the huge numbers of stroke survivors in LMICs.

In the present study, which is based on a Nigerian sample of stroke survivors, we aimed to estimate incidence, cumulative incidence, risk factors and outcome of new onset anxiety in the first year of stroke.

Methods

Sites

We evaluated a dataset comprising information collected as part of an intervention to improve one-year blood pressure control among recent (<1 month) stroke survivors who were discharged from four referral hospitals serving the Nigerian and West African population. These hospitals were the University College Hospital (UCH) Ibadan, Blossom Neurorehabilitation Hospital Ibadan, Federal Medical Center (FMC) Abeokuta, and the Sacred Heart Hospital Ibadan.

Ethical approval

Ethical approval was obtained from the institutional review boards of the University of Ibadan/UCH joint ethics committees (which cover the WFNR-Blossom), FMC Abeokuta, and Sacred Heart Hospital. Participants provided written, informed consent before interviews were conducted.

Subjects

The subjects comprised consecutive adult ischaemic or haemorrhagic stroke survivors. The diagnosis of stroke was confirmed based on neuro-imaging and clinical examination criteria.[14]

Patients were informed about the study, and the procedure was explained to them in their home language. We excluded patients with severe communication difficulties (N=34) or aphasia (N=42), and those with severe conditions that could limit participation in follow-up assessments (N=94). These included those with severe cognitive impairments or dementia [(Modified Community Screening Instruments for Dementia (CSID) ≤ 20)], global disability [(Modified Rankin Scale (MRS) ≥ 3)], as well as those with significant comorbid medical illnesses (e.g., chronic kidney disease).[15]

Stroke survivors meeting study criteria underwent baseline assessments within the first month of stroke. This was followed by a prospective observation of the same cohort in five time points at 1, 3, 6, 9 and 12 months.

Measures

Ascertainment of anxiety

Anxiety was ascertained using the Anxiety subscale of the Hospital Anxiety and Depression Scale (HADS-A).[16] The HADS was developed for the assessment of emotional disturbances in non-psychiatric patients within a hospital setting. It includes a total of 14 items, each with a score of between 0 and 3. One half of the items are related to anxiety while the other half is specific for depression. The developers of the scale recommend a cut off ≥11 for the ascertainment of 'probable' cases in research. This score is also often used in the determination of clinically significant anxiety. Lower scores of between 8 and 10 are recommended for less severe anxiety symptoms. The HADS has been previously validated in Nigeria[17] where the HADS-A was found to have a sensitivity ranging 85.0−92.9% and a specificity of 86.5−90.0%.

Outcomes

Cognitive functioning

Cognitive functioning was assessed using the CSID.[18] The instrument includes a global cognitive test and informant account of patient's ability to cope with Activities of Daily Life (ADL). The CSID has been validated in Nigeria.[19] The Nigerian version of the CSID is widely used to screen for cognitive functioning in the general and older adults populations, including stroke survivors.[20]

Motor functioning

Motor functioning was assessed using the modified Rankin Scale (mRS).[21] The mRS is a clinician-reported measure of global disability that has been widely applied for evaluating recovery from stroke. Although it does not provide a comprehensive assessment of ADL, it correlates strongly with the Bartel index and other measures of physical functioning.[22] The mRS includes five operationally defined degrees of disability ranging from slight to severe disability, with higher scores corresponding to more severe disability.

Quality of life

Quality of life was assessed using the 26-items version of the Health Related Quality of Life in Stroke Patients (HRQOLISP-26).[23] The HRQOLISP-26 is a stroke-specific

measure developed from a large cross-cultural, transnational, patient-controlled sample.[24] The measure is flexible and valid for regular assessment of all domains of health-related quality of life. The HRQOLISP-26 has been tested and validated for use in Nigeria[23] and is applicable multiculturally. The instrument is comprised of four therapeutically relevant domains: physical, psycho-emotional, cognitive, and eco-social. Apart from the cognitive domain with 5 items, the other domains are composed of 7 items. Higher scores on the HRQOLISP-26 represents better quality of life.

Other data collection

The following information was obtained from all participants using a standardized questionnaire: demographic data, personal history of smoking and alcohol consumption, medical history of systemic hypertension, information on dietary patterns (obtained using the food frequency questionnaire). The severity of stroke was ascertained using the Stroke Levity Scale.[25] The average of two blood pressure (B.P) measurements was recorded. Each B.P measurement was obtained using an Omron HEM-907 XL 26 blood pressure monitor and the readings recorded according to standardized protocol provided by the manufacturers.

Records of other relevant risk factors for stroke were additionally collated. Stroke subtypes were determined based on neuroimaging evidence of lesion characteristics. Depression status was assessed using the depression sub-scale of the HADS.

Data analysis

We estimated the rates of anxiety after stroke and its 95% confidence interval (95% CI) at baseline and at 1, 3, 6, 9, and 12-months follow-up. A patient was classified as having anxiety if HADS-A score was ≥ 11 (clinically significant anxiety). New onset anxiety at each follow-up time point was the number of stroke survivors having clinically significant anxiety for the first time during the corresponding wave of follow-up. These were determined after censoring cases with clinically significant anxiety in the preceding waves. Socio-demographic characteristics of participants in the new onset anxiety cohort who could not be followed up at 12 months were compared with those who were followed up using chi-square test.

Incidence was calculated as the ratio of new cases of anxiety (incident cases) to the number at risk (excluding the prevalent cases and losses to follow-up). We also estimated the cumulative incidence of new onset PSA (with 95% CI) according to demographic characteristics using the Kaplan−Meier method. To achieve this, we calculated person time for those who developed new onset PSA or were censored using the interval from the date of first enrolment into the study to the last wave in which the person was followed up.

To investigate the predictors of new onset PSA over the 12-month period, we fitted Cox proportional hazards regression models and estimated unadjusted hazards ratios (with 95% CI) as measures of effect. Socio-demographic characteristics, vascular risk factors, life style (smoking, alcohol use and exercise) and diet (intake of fruits, fish and vegetables) were included as time invariant factors. Variables with p-value<0.1 were entered in a multivariable model to obtain adjusted hazards ratios.

We next investigated the relationship between new onset PSA and stroke outcomes (CSID, mRS and HRQOLISP-26 scores) at 12 months follow up. For this objective, we compared the mean values for the listed outcomes between those with and without incident PSA using independent samples t-test. For the stroke outcome measures, we reported unadjusted mean differences (MD) with 95% CI between the two groups. Subsequently, linear regression models were fitted to adjust the MD for age, education, stroke type and stroke severity. For all analyses, p-values<0.05 were considered statistically significant. Stata MP Version 14 was used for statistical analyses.

Results

Study sample

A total of 400 stroke survivors were eligible, gave consent and participated in the study. Of these, 322 (81%) were free of anxiety at baseline and constituted the new onset anxiety cohort that was subsequently followed up at five time points over twelve months Fig. 1. The mean age of the new onset anxiety cohort was 57.6 ($\pm$ 11.7) years. Compared with those who were lost to follow-up or had died before the last follow-up date, survivors who were successfully followed up were more likely to have stroke of mild severity (Supplementary Table 1).

Incidence and risk factors for new onset anxiety in the first year of stroke

In all, the anxiety scores on the HADS increased steadily in the first year of stroke (Table 1). Apart from records in the first month poststroke, HADS anxiety and depression scores demonstrated non-significant correlations. We identified 29, 27, 24, 17 and 7 new cases of anxiety at 1, 3, 6, 9 and 12 months, respectively. These cases produced a cumulative incidence rate of 34% (95% CI = 28.6−39.3) for new onset anxiety in the first year of surviving a stroke. The rates for men was 36.2% (95% CI =29.6−42.7) and for women was 29.2% (95% CI =19.9−38.3). The highest rate of new onset anxiety, 46.8% (95% CI =35.7−57.9), was recorded in survivors of haemorrhagic stroke (Supplementary Table 2).

Supplementary Table 3 shows the risk factors for incident poststroke anxiety investigated in the study. In multivariate cox regression analyses, haemorrhagic stroke (HR =1.52, 95% CI =1.01−2.29) was the main predictor of time (in months) to new onset anxiety in the first year of stroke (Table 2).
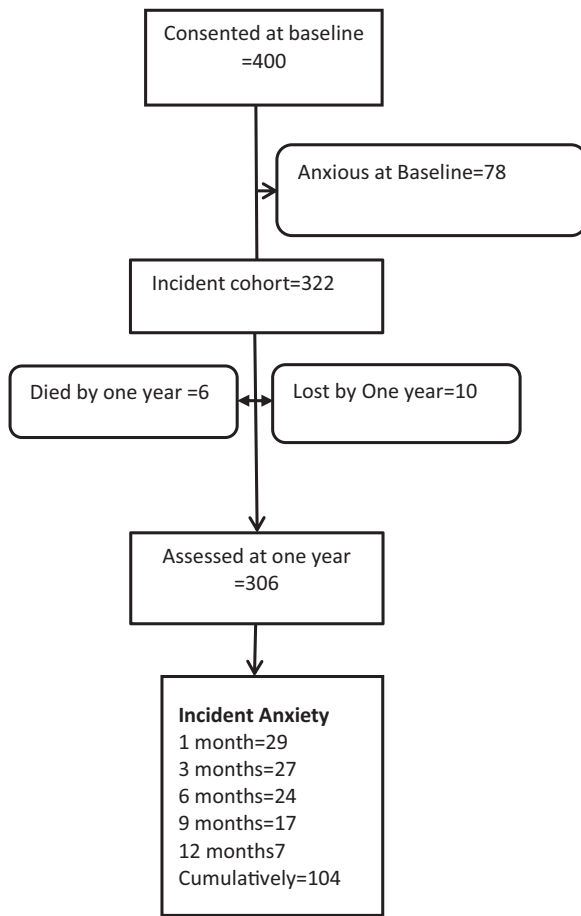
Fig. 1. *Flow chart of the incident poststroke anxiety study.*

Outcomes of new onset anxiety

In linear regression analyses adjusting for the effect of age, gender, stroke sub-types and stroke severity, new onset PSA resulted in poorer quality of life overall (MD=3.6, 95% CI =1.0, 6.2), as well as in most domains of the HRQOLISP-26 (Table 3). In the same table, stroke survivors with new onset anxiety showed significantly greater decline in cognitive (MD in CSID scores=1.1, 95% CI =0.2, 1.9) as well as motor functioning overtime (MD in mRS scores= −0.2, 95% C.I= −0.4, −0.02).

Discussion

We found that over one-third of Nigerian patients will develop new onset anxiety in the first year of surviving a stroke. A majority of survivors in this population developed the emotional disorder in the first four months of stroke. Haemorrhagic stroke survivors were at significantly increased risk. New onset anxiety in the first year of stroke was associated with decline in cognitive and physical functioning as well as poorer quality of life.

The 34% one-year cumulative incidence of new onset PSA observed in the present study is about three times

Table 1. *Incidence of new onset poststroke anxiety (PSA) in the first year of stroke.*

Follow-up time	HADS-anxiety scores Mean (SD)	HADS-depression scores Mean (SD)	Correlation of HADS anxiety and depression scores, r (p-values)	Incident cases, n	Incidence % (95% C.I)	Cumulative incidence, n	Kaplan−Meier estimate of cumulative PSA incidence (95% C.I)
1 month	8.4 (2.0)	11.7 (2.2)	0.14 (0.007)	29	9.5 (6.2 − 12.8)	29	9.4 (6.7 − 13.2)
3 months	8.7 (2.0)	11.7 (1.9)	0.05 (0.322)	27	9.8 (6.3 − 13.3)	56	18.2 (14.4 − 22.9)
6 months	8.7 (2.0)	11.8 (1.7)	−0.02 (0.695)	24	9.3 (5.7 − 12.8)	80	25.9 (21.4 − 31.1)
9 months	8.8 (1.8)	11.8 (1.5)	−0.02 (0.768)	17	7.6 (4.1 − 11.1)	97	31.7 (26.9 − 37.2)
12 months	8.7 (1.6)	11.8 (1.5	0.04 (0.401)	7	3.4 (0.9 − 5.9)	104	34.0 (29.1 − 39.5)

HADS= Hospital Anxiety and Depression Scale.

Table 2. *Results of multivariate Cox regression analyses showing haemorrhagic stroke as the main predictor of incident post-stroke anxiety in the first year of stroke.*

	12-month cumulative incidence			
Characteristics	*Absent: n=202 (%)*	*Present: n=104 (%)*	*Unadjusted HR (95% CI)*	*Adjusted HR (95% CI)*
Groups				
Control	Reference	Reference	1.00	1.00
Intervention	93 (64.1)	52 (35.9)	1.13 (0.77−1.65)	1.21 (0.82−1.78)
Age group, years				
45	Reference	Reference	1.00	1.00
45−65	123 (65.4)	65 (34.6)	1.09 (0.64−1.85)	1.03 (0.60−1.77)
> 65	46 (67.7)	22 (32.4)	1.02 (0.55−1.92)	0.91 (0.48−1.74)
Stroke type				
Ischemic	Reference	Reference	1.00	1.00
Haemorrhagic	42 (53.2)	37 (46.8)	1.76 (1.19−2.61)*	1.52 (1.01−2.29)*
Stroke severity				
Mild	Reference	Reference	1.00	1.00
Moderate/severe	29 (58.0)	21 (42.0)	1.43 (0.82−2.47)	1.00 (0.56−1.81)
Cognitive impairment				
No	Reference	Reference	1.00	1.00
Yes	18 (54.6)	15 (45.4)	2.30 (0.92−5.55)	2.22 (0.91−5.42)
Low vegetable				
No	Reference	Reference	1.00	1.00
Yes	118 (70.7)	49 (29.3)	1.13 (0.99−1.28)	1.12 (0.98−1.27)
Low fruits				
No	147 (64.8)	80 (35.2)	1.28 (0.98−1.67)	1.24 (0.95−1.62)
Yes				
Alcohol use				
Never	Reference	Reference	1.00	1.00
Formerly	83 (61.0)	53 (39.0)	1.80 (0.91−3.56)	1.18 (0.79−1.76)
Currently	19 (73.1)	7 (26.9)	1.02 (0.35−2.97)	1.09 (0.38−3.10)

Notes: Only risk factors with unadjusted alpha values of $p<0.1$ are included in the table.
*$p<0.05$.

Table 3. *Motor, cognitive, and quality of life outcomes of new onset anxiety in stroke survivors.*

	Mean (SD) scores			
Outcome measures at 12 months	Anxiety free (n=202)	Anxiety present (n=104)	Unadjusted Mean difference (95% C.I)	*Adjusted Mean diff (95% C.I)*[a]
CSID score	27.6 (2.7)	26.6 (4.9)	1.1 (0.2, 1.9)*	1.1 (0.2, 1.9)*
MRS score	2.2 (1.1)	2.4 (1.1)	−0.2 (−0.5, 0.01)	−0.2 (−0.4, −0.02)*
HRQOLISP-26 scores				
Overall	79.1 (11.6)	75.6 (12.7)	3.5 (0.7, 6.4)*	3.6 (1.0, 6.2)*
Domains				
Physical	82.9 (14.3)	77.5 (15.5)	5.4 (1.9, 8.9)*	4.5 (1.6, 7.4)*
Psychological	74.1 (16.0)	69.8 (16.8)	4.3 (0.4, 8.2)*	4.3 (0.8, 7.9)*
Cognitive	80.0 (14.4)	76.3 (16.6)	3.7 (0.1, 7.3)*	4.0 (0.5, 7.7)*
Social	79.8 (12.3)	77.1 (14.1)	2.8 (−0.3, 5.8)	2.8 (−0.03, 5.6)
Soul	79.3 (12.3)	77.0 (13.3)	2.3 (−0.7, 5.3)	2.6 (−0.3, 5.4)
Spiritual	80.8 (10.2)	77.0 (12.2)	3.8 (1.2, 6.4)*	4.1 (1.6, 6.7)*
Spiritual interaction	76.8 (13.3)	74.2 (14.6)	2.6 (−0.7, 5.9)	3.0 (−0.2, 6.1)

[a] Adjusted for stroke severity, stroke type, age and education.
*$p<0.05$, CSID= Community Screening Instrument for Dementia, MRS= Modified Rankin Scale, HRQOLISP-26= 26-items version of the Health Related Quality of Life in Stroke Patients.

higher than the pooled estimate derived from a systematic review of similar studies conducted in the general adult population.[26] It is also nearly two times higher than the one-year cumulative incidence of PSA estimated from the much-cited South London stroke registry.[7] Our study adds to the contributions made by prior studies in the global literature on PSA in two major ways.

First, most previous studies[6, 7, 27] of incidence, cumulative incidence, risk factors and outcome of PSA have relied on samples comprising stroke diagnosed without recourse to standard neuroimaging criteria.[14] Without protocol inclusion of neuroimaging in the definition of stroke, surgically treatable stroke mimics such as brain tumors or subdural haematoma may not be completely eliminated. Secondly, and to the best of our knowledge, there are no previous studies of incidence, risk factors and outcome of new onset PSA in LMICs.

We think that the incidence of new onset PSA found in the present study is likely to be an underestimation of the extent of its burden in LMICs, especially those in sub-Saharan Africa (SSA). This is partly because a suggestion of mental illness continues to be associated with self-perceived stigma in many parts of SSA.[28] It is thus likely that some of the respondents in the present study may have been too embarrassed to admit to some of the anxiety related symptoms in the HADS.

Another reason for a possible underestimation of the burden of new onset PSA in the present study is our reliance on a more stringent diagnostic cut-off score of HADS-A $\geq$ 11 in defining clinically significant anxiety. Some prior longitudinal studies of PSA in the global literature have used a HADS-A diagnostic cut-off score of 11[7] while others have relied on scores of 7 or 8.[6, 27] However, evidence from the developers of the instrument[16] suggests that a cut-off score of 11 may have better construct validity for anxiety requiring specific clinical intervention. In addition to the stringent definition of anxiety, a closer look at our data suggests that the majority of survivors included in the present study were those with stroke of mild severity. It is reasonable to expect that stroke of greater severity may be associated with more physical, as well as mental health morbidities.

We observed that while most survivors in the present study developed new onset PSA within the first four months, the severity scores on the HADS-A increased steadily in the first year of stroke. This is in contrast to a previous report from Colombia[12] where the severity scores on the HADS-A decreased over the first year of follow-up. In the same study, which was based on a small sample of 50 stroke survivors, the severity of stress increased over the same time period.[12] Notably, stress is the physical component of anxiety.[29] A recently updated systematic review of 53 studies of PSA in the global literature[30] also found that the pooled prevalence of anxiety increased steadily in the first year of stroke, and did not show much decline in the first 24 months.

We found that haemorrhagic stroke survivors were at increased risk of new onset anxiety compared with other subtypes. This finding appears to be in keeping with some observations that haemorrhagic stroke subtypes may be associated with greater stroke severity, poststroke morbidities and mortality.[31, 32] Even though a higher proportion of survivors with moderate or severe stroke could be classified as having haemorrhagic subtypes in the present study, we could not establish a statistical association between stroke types and severity.

Our finding that gender was not associated with new onset PSA was not surprising as the link between gender and anxiety has been inconsistently reported in stroke[33] and general population literature.[34] An important pointer to this inconsistency is that while there was no statistical association between female gender and anxiety in the present study, our prior report[1] from the same population found significant cross-sectional association between the two variables.

Similar to findings in the present study, two previous studies in the global literature found that new onset PSA was associated with poorer motor functioning at 3 months[6] and greater decline in quality of life scores at one year.[7] In addition to these previously reported outcomes of incident PSA, we found in the present study that new onset PSA was associated with poorer cognitive functioning at one year.

Our study has several limitations. First, the present report is based on a sample comprising participants in a clinical trial. Even though intervention conditions did not significantly affect the risk of PSA, findings in the present study are still unlikely to be generalizable to the entire spectrum of stroke survivors. Secondly, patients with aphasia were excluded. Presumably, these patients may have had more severe stroke and, consequently, they may have higher levels of anxiety. Even though the HADS-A is widely used with good validity in stroke populations across cultures, it is unable to make substantive diagnoses of anxiety disorders according to DSM V criteria. As could be expected in cohort studies of this length, we recorded attritions. Those who did not complete follow-up were more likely to have severe stroke.

In conclusion, we have found that within the first year of stroke, over one-third of survivors living in LMICs of SSA will develop new onset anxiety requiring specific interventions. New onset PSA results in decline in motor and cognitive functioning, as well as in poorer quality of life. In this population, haemorrhagic stroke was the main predictor of onset of anxiety in the first year of stroke. Clinicians seeing stroke survivors in SSA should therefore pay special attention to survivors of haemorrhagic stroke

as they are at a significantly higher risk of anxiety and therefore its consequences.

Declaration of Competing Interest

The authors declare that there is no conflict of interest.

Supplementary materials

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.jstrokecere brovasdis.2020.105082.

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