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ORIGINAL RESEARCH

The Contribution of Dysphagia to Stroke Morbidity and Mortality among Nigerians

Olajide OO^{1,2}, Adebayo PB^{*1,3}, Taiwo FT³, Owolabi MO^{2,4}, Ogunniyi A^{2,4}

¹Neurology Unit, Department of Medicine, Ladoke Akintola University Teaching Hospital, Ogbomoso, Nigeria

²Neurology Unit, Department of Medicine, University College Hospital, Ibadan, Nigeria

³Neurology Section, Department of Internal Medicine, Aga Khan University, Dar es Salaam, Tanzania

⁴College of Medicine, University of Ibadan, Ibadan, Nigeria

*Correspondence: Dr PB Adebayo, Neurology Section, Department of Internal Medicine, Faculty of Postgraduate Medical Education, Aga Khan University, Dar Es Salaam, Tanzania.
E-mail: philipab8@yahoo.com; ORCID - <https://orcid.org/0000-0003-0194-6968>.

Abstract

Background: The assessment of time-trend morbidity and mortality in acute stroke is critical to clinical policy decisions and resource allocation.

Objectives: To determine the prevalence of dysphagia in acute stroke and the impact of dysphagia on short term stroke outcome (30 days post-stroke).

Methods: This was a prospective longitudinal study. Bedside screening for dysphagia modified Rankin score (MRS) and Barthel Index (BI) were performed on acute stroke patients on day 1, day 7, day 14 and day 30 after stroke to determine the frequency of dysphagia. Patients with dysphagia were then compared with age- and gender-matched controls (stroke patients without dysphagia) in terms of stroke characteristics and 30-day outcome.

Results: Of the recruited 200 patients, 99 (49.5%) had dysphagia. Patients with intracerebral haemorrhagic stroke had a significantly higher prevalence of dysphagia (64% vs 36%; $p < 0.001$). At baseline, dysphagic patients had more severe (Mean NHISS score, 22.81 Vs 8.92; $p = 0.01$) and subcortical strokes (57.1% vs 42.9%, $p = 0.015$). At 30 days after stroke, the mean MRS was significantly higher in the dysphagic stroke patients (3.8 ± 1.02) compared to those without dysphagia (2.5 ± 1.3), ($p = 0.001$). Case fatality was higher among the dysphagic (79.8% vs 15.84%; $p = 0.001$) and the mean survival time was lower (12.21 days) among the dysphagic group ($p = 0.001$).

Conclusion: Severe stroke, subcortical stroke and haemorrhagic stroke types were significantly associated with dysphagia at baseline. Dysphagia adversely influenced 30-days morbidity and case fatality in this cohort of acute stroke patients.

Keywords: Acute Stroke, Aspiration pneumonia, Dysphagia, Screening, Water Swallowing Test.

Introduction

Stroke is rapidly becoming a major cause of morbidity and death, especially in low and middle-income countries (LMIC). In the African

population, the annual incidence rate is up to 316 per 100 000, with a prevalence rate of up to 315 per 100,000 and a 3-year fatality rate of up to 84%.^[1, 2] Dysphagia, after stroke, is common and its detection is an important part of acute stroke

management. Existing literature suggests that dysphagia can affect 19-81% of stroke patients. [3] 4) Dysphagia refers to difficulty in swallowing, with food stuck in the mouth and throat (food apnoea), or the presence of cough after swallowing. Other features include impaired voice, wet voice or abnormal pulse oximetry during water swallowing test. Dysphagia is considered a significant gastrointestinal complication in stroke survivors. [5]

Dysphagia, after stroke, is associated with respiratory complications, increased risk of pneumonia, nutritional compromise, and dehydration. It also reduces the quality of life. [6] The duration of hospitalization is increased in stroke patients with dysphagia, and these individuals are more likely to be discharged to nursing homes compared with stroke patients without dysphagia. [6] Fortunately, there is emerging evidence that early detection of dysphagia reduces not only pulmonary complications but also the duration of hospital stay and overall healthcare cost for acute stroke patients. [7, 8] Patients' awareness of their disabilities, including dysphagia after stroke, represents an important aspect of functional recovery. Patients with poor awareness experienced more complications of dysphagia. [6]

Africa bears a heavy burden of stroke and there is a severe scarcity of facilities and human resources for prevention, investigations, acute care and rehabilitation. Identifying and treating stroke patients at risk for dysphagia are extremely important and this could be effective in reducing morbidity and mortality in a developing world setting. This study determined the prevalence of dysphagia in acute stroke patients and determined the association between dysphagia and stroke subtypes, stroke severity, and lesion sites. The study also investigated the relationship between dysphagia in acute stroke and short-term outcome (30-day post-stroke)

such as case fatality rate and functional outcome in stroke survivors.

Methods

Study Design and Participants

This observational prospective study was conducted on consecutive acute stroke cases admitted to the medical wards and the emergency unit of the University College Hospital (UCH), Ibadan, Nigeria from February 2013 to October 2013. The University College Hospital, Ibadan, is a 1000-bed tertiary hospital in Nigeria. The hospital receives referrals from all over Nigeria, especially the south-western region. The neurology unit of the UCH attends to stroke patients presenting at the emergency department as well as those referred from other medical units. For the present study, stroke patients were recruited consecutively as soon as they presented to the hospital and each case was followed up for thirty days.

Subjects with the first-ever stroke, who were aged 18 years and above, with radiological confirmation of stroke, were included in the study. Exclusion criteria included; stroke onset >7 days before assessment, severely reduced conscious level (GCS \leq 8; the GCS was checked repeatedly within 24 hours) and history of dysphagia before stroke irrespective of aetiology. In addition, subjects who required continuous oxygen therapy and/or on mechanical ventilator, as well as those who did not give consent to participate in the study were excluded. Controls were stroke patients who did not have dysphagia on screening and met the same inclusion and exclusion criteria as cases. One hundred cases and 100 controls were recruited for the study. Figure 1 shows the participant recruitment algorithm.

Ethical approval was obtained from the Ethical Committee of UI/UCH, Ibadan, Nigeria. The patients or their relatives were educated on the

purpose of the study in the language they understood best. Thereafter, written informed consent was obtained from all participants or relatives.

Subject Assessment

Using a case report form, demographic characteristics such as age, gender, place of residence, ethnicity, marital status and socioeconomic status of the patient were obtained. The details of the recent and remote history of the patient were enquired considering other conditions that could cause dysphagia, risk factors for stroke, other co-morbidities and history of the previous stroke.

The National Institute of Health Stroke Scale (NIHSS) was used to determine the severity of stroke at admission. The Glasgow Coma Score (GCS) was checked at admission. The functional outcome was determined using the Modified Rankin Scale (MRS) while the Barthel Index (BI) was used to assess the activities of daily living post-stroke [9] at presentation, one week, two weeks and one month after stroke. The assessment of dysphagia using the Bedside Swallowing Tests was carried out at presentation, at one week, two weeks, and a month after stroke.

Bedside Swallowing Tests.

Gag Reflex: The test was clearly explained to the subjects; a sterile wooden tongue depressor was used to touch the pharynx. Those patients that retched were considered to have a positive gag reflex. No patient vomited as a result of this test. This positive response suggested that the patient could swallow.

Water Swallowing Test: The test was clearly explained to the subjects; each subject was seated upright. Three millilitres of water was measured into a teaspoon and given to the patient. For those who could not hold the teaspoon, they were helped to drink from it. [3, 10] Subjects who could swallow had the test repeated. The following

signs of swallowing difficulty were looked out for in each patient: coughing, choking after attempting to swallow, water pooling in the mouth, delayed (>2 seconds) or absent swallow, poor or absent laryngeal elevation, signs of distress or respiratory difficulty, and change in the quality of phonation. Afterwards, the test was repeated using 10mls of water, followed by 30mls of water and then by 50mls of water measured into a disposable cup. Subjects who could not hold the cup were helped to drink steadily from the container. Any subject that experienced any obvious difficulty in swallowing during any of these tests was classified as having dysphagia. These tests were omitted in any patient known to have choked on fluid that day and in those who were unconscious. Subjects who had depressed or absent gag reflex were tested initially with 3mls of water. Any subject who choked was closely monitored and the managing team was advised to place the patient on *nil per os*. Oral hygiene was well maintained. [3, 11]

Pulse Oximetry: This measurement of peripheral oxygen saturation using a pulse oximeter was carried out during the 10ml water-swallowing test. The oximeter probe was attached to the index finger of the patient's unaffected upper limb. Nail polish was removed from the finger if there was any, and the subject was instructed to keep that arm still during the study to avoid movement artefact. The pulse oximeter was allowed to equilibrate for 5 minutes. A baseline measurement of oxygen saturation was recorded. Oxygen saturation measurement was done continuously for 10 minutes from the start to allow time for the swallow assessment, any immediate or delayed aspiration, and a recovery period. The oxygen saturation measurements were noted between 0 to 5 minutes (T1) and 5 to 10 minutes (T2) The greatest fall in oxygen saturation during the two time periods from the onset of the water swallowing test (T1 and T2) were calculated as the difference between the lowest saturation and the mean baseline

saturation after excluding extreme values due to movement or other artefacts. Oxygen desaturation $\geq 2\%$ were considered to be clinically significant. [12]

Assessment for aspiration pneumonia

Aspiration pneumonia was established based on the presence of ≥ 3 of the following variables: (1) fever ($\geq 38^\circ\text{C}$), (2) abnormal respiratory examination (tachypnea [respiratory rate $\geq 22/\text{minute}$], inspiratory crackles, bronchial breathing), (3) tachycardia, (4) abnormal chest radiograph, (5) arterial hypoxaemia ($\text{PO}_2 < 70 \text{ mm Hg}$), (6) isolation of a relevant pathogen (positive Gram stain and culture) and (7) leukocytosis. Subjects who had aspiration pneumonia were treated with antibiotics based on microbial sensitivity test.

Neuroimaging studies.

Computerized tomography (CT) scan of the brain was done for all patients to ascertain the stroke type, lesion site, and lesion size. Features of early ischaemic signs (EIS) on brain CT were also searched for. These included hypodensity of brain tissue, poor grey-white matter differentiation, obscuration of lentiform nucleus (blurred basal ganglia), blurring of insular ribbon, and hyperdense middle cerebral artery (MCA). Intracerebral haemorrhage appears as a hyperdense lesion on the brain CT scan. The measurement of haematoma volume was done using the maximum length, crossed short length and slice thickness (ellipsoid equation). The lesion size of acute cerebral infarction was derived from the area of the lesion and slice thickness. The area of abnormally low attenuation was traced on each CT slice, and the area was summed for the slices showing the infarct.

Laboratory evaluation

Blood samples for full blood count, serum electrolytes, blood urea nitrogen, random blood glucose and lipid profiles were obtained from

each subject. Chest radiograph was also done for all subjects.

Operational definitions of dysphagia, stroke and stroke classification.

Dysphagia was defined as a failed 10mls water swallowing test and peripheral oxygen desaturation $\geq 2\%$. [12, 13] Computerized tomography (CT) definition of haemorrhagic stroke type includes the presence of hyper-density on non-contrast CT (NCCT) brain scan in an acute stroke patient. The presence of hypodensity or hypo-attenuation on NCCT brain scan in acute stroke patient defines ischaemic stroke. The Oxfordshire Community Stroke Project (OCSP) classification system for ischaemic stroke was used to classify ischaemic stroke. [14]

Statistical Analysis

Socio-demographic and clinical characteristic data were presented in tables and charts using summary statistics of proportions for categorical variables and means (\pm standard deviation) for normally distributed continuous variables. The non-parametric variables were presented using median (interquartile range). The Chi-Square test or its equivalent was used to compare proportions while the Student's t-test or its non-parametric equivalent where appropriate, was used to compare continuous variables. Multivariate logistic regression models were constructed to assess for independent predictors of dysphagia in subjects with acute stroke with a variable that showed significant statistical association with dysphagia both in the study and from literature fitted into the model. Repeated measures ANOVA was carried out to assess the impact of dysphagia and aspiration on the clinical outcome as measured by the Modified Rankin Scale and Barthel Index. Also, survival analysis was done to assess the impact of dysphagia and aspiration on mortality, with Kaplan-Meier curves constructed and Log-rank test performed to assess for a statistically

significant difference. Cox proportional hazard model was used to assess possible causes of death in patients with dysphagia. All statistical

analyses were performed using the SPSS version 16.0 software (SPSS, Chicago, IL, USA). The level of statistical significance was set at $p < 0.05$.

Results

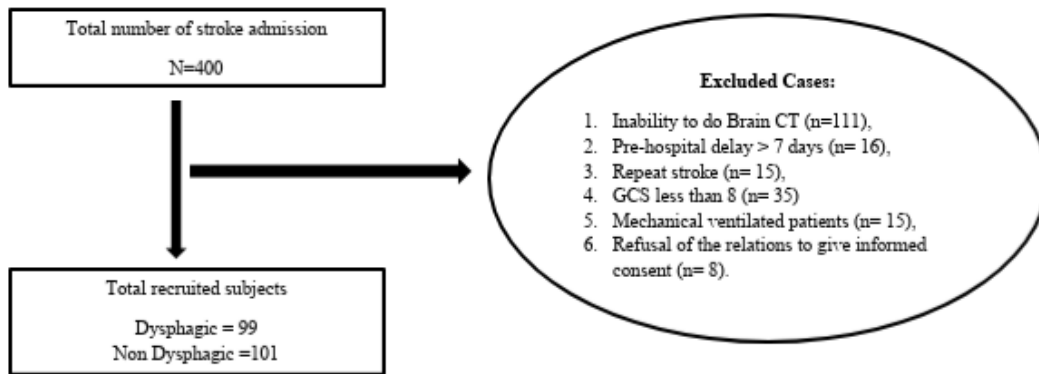


Figure 1: Flow Chart showing total stroke cases and selected subjects

Demographic and Clinical Characteristics.

The dysphagic stroke group (cases) comprised 52 (52.5%) males and 47 (47.5%) females while the non-dysphagic stroke group (controls) comprised 52 (51.5%) males and 49 (48.5%) females ($p = 0.88$). The mean ages for the non-dysphagic and dysphagic stroke groups were comparable: (59.5 ± 11.5 years *vs.* 62.1 ± 12.1 years respectively; $p = 0.11$). All the control group and 97 (97.98%) of the dysphagic stroke were right-handed ($p = 0.15$). Following the exclusion of patients with severely low GCS < 8 , the cases had a significantly higher proportion of worse GCS '9-13' at presentation (83.8% *vs.* 17.8% ; $p < 0.01$) than the non-dysphagic group as shown in Table I. Dysphagic patients had a worse mean NIHSS score at presentation (22.8 *vs.* 8.9 ; $p < 0.01$) as shown in Table I. The mean pulse rate (93.9 *vs.* 86.1 ; $p < 0.001$) and mean arterial blood pressure (136.1 *vs.* 126.2 ; $p = 0.04$) were significantly higher in the dysphagic stroke group compared to the non-dysphagic stroke group.

Stroke types, sites and vascular territories in the subjects

Table I also shows that haemorrhagic stroke was more frequent among dysphagic patients (64.6% *vs.* 35.6% ; $p < 0.001$). According to the Oxfordshire Community Stroke Project (OCSP) classification, Partial Anterior Circulation Infarct (PACI) was significantly less frequent among the dysphagic group (74.3% *vs.* 84.6% ; $p = 0.001$). No other differences were noted across the other classes of cerebral infarcts between the groups. Comparison of the occurrence of dysphagia in relation to the site of lesion shows that 51/103 (49.5%) of cases with cortical lesion were dysphagic while 52/103 (50.5%) were non-dysphagic ($p = 1.0$); 64/112 (57.1%) of the cases with subcortical lesions had dysphagia and 48/112 (42.9%) were non-dysphagic ($p = 0.015$) while 6/9 (66.7%) of those with brainstem/cerebellum had dysphagia and 3/9 (33.3%) were non-dysphagic ($p = 0.29$).

Complications and outcome of dysphagia

Table II shows the complications and outcome observed in dysphagic and non-dysphagic stroke

patients. The commonest complication observed in the dysphagic group was aspiration pneumonia. Sixty-two dysphagic (62.63%) and only one non-dysphagic (0.99%) had aspiration pneumonia ($p < 0.001$). Urinary Tract Infection (UTI) was the commonest complication observed in the non-dysphagic group. The other observed complications are highlighted in Table II.

The mean Modified Rankin Scale (MRS) of dysphagic stroke patients at week 4 post-stroke was 3.83 ± 1.029 while the MRS of non-dysphagic stroke patients at week 4 post-stroke was 2.55 ± 1.34 . This difference was statistically significant ($p < 0.001$). The difference in the mean Barthel index (BI) was statistically significantly different between the two groups (28.04 vs. 65.52; $p < 0.001$) as shown in Table III. Seventy-nine dysphagic (79.8%) and 16 non-dysphagic (15.8%) subjects died on admission, ($p < 0.001$). The average survival days was statistically significantly different between non-dysphagic (24.5 days) and dysphagic (12.2 days) groups ($p < 0.001$).

The Kaplan Meier Survival curve in Figure 2 depicts the percentage cumulative survival in the dysphagic and non-dysphagic groups. Those with dysphagia had a longer hospital stay and some of them died.

Table IV shows univariate and multivariate logistic regression analysis to assess the determinants of 30-day case fatality. On univariate analyses, the significant determinants of 30-day case fatality included dysphagia ($p < 0.001$), aspiration pneumonia ($p < 0.001$), sepsis ($p < 0.001$), severe NIHSS grade ($p < 0.001$), intracerebral haemorrhage ($p < 0.001$), and mean arterial pressure (MAP) > 145 mmHg ($p = 0.041$). Multivariate analysis shows that the most significant determinant of 30-day case fatality was dysphagia ($p = 0.005$). This was followed by aspiration pneumonia ($p = 0.024$), intracerebral haemorrhage ($p = 0.043$) and severe stroke (NIHSS > 24) ($p = 0.049$).

Table I: Demographic and baseline clinical characteristics of the groups

Profile		Dysphagic (n = 99)	Non-dysphagic (n = 101)	p-values
Mean age (years)		62.1±12.1	59.5±11.5	0.110
Sex	Male	52 (52.5)	47 (51.5)	0.880
Clinical profile				
GCS at presentation	14-15	16 (16.2)	83 (82.2)	<0.001
	9-13	83 (83.8)	18 (17.8)	
NHISS at presentation		22.8±6.2	8.9±6.4	<0.001
Stroke type	Ischaemic	35 (35.4)	65 (64.4)	<0.001
	Haemorrhagic	64 (64.6)	36 (35.6)	
Hemispheric involvement	Dominant	53 (53.5)	52 (51.5)	0.775
	Non-dominant	46 (46.5)	49 (48.5)	

GCS - Glasgow Coma Score; NHISS - National Institute of Health Stroke Scale.

Table II: Pattern of complications and case fatality among the groups

Characteristics		Dysphagic (n = 99)	Non-dysphagia (n = 101)	p-values
Stroke complications	Urinary tract infection	20 (20.2)	20 (19.8)	0.944
	Acute Kidney Injury	10 (10.1)	8 (7.9)	0.590
	Pulmonary Thromboembolism	1 (1.0)	3 (2.9)	0.675
	Deep Venous Thrombosis	2 (2.0)	3 (2.9)	0.327
	Myocardial infarction	0 (0.0)	1 (0.9)	0.321
	Aspiration pneumonitis	62 (62.6)	1 (0.9)	<0.001
Died on admission n (%)	79 (79.8)	16 (15.8)	<0.001	
Mean survival time (Days)	12.2±1.1	24.5±1.1	<0.001	

Aspiration Pneumonia

Figure 3 shows the percentage cumulative survival in the dysphagic group with aspiration pneumonia and those without aspiration pneumonia. The 30-day survival decreased drastically in those patients that had aspiration pneumonia. Table V shows the Cox proportional hazard model for patients with dysphagia while

controlling for the effect of sepsis. The hazard ratio (HR) of aspiration pneumonia among those without sepsis was 2.883 (p = 0.036) and this was statistically significant. The hazard ratio of aspiration pneumonia complicated by sepsis was 1.525 but was not statistically significant (p = 0.335).

Table III: Outcome of dysphagic and non-dysphagic stroke patients

Variables	Dysphagic (n = 99)	Non-dysphagic (n = 111)	p-value
Mean MRS on 1 st day	5.0±0.9	3.7±1.2	<0.0001
Mean MRS on 7 th day	4.4±1.2	2.9±0.9	<0.001
Mean MRS on 14 th day	4.0±1.0	2.3±1.5	<0.001
Mean MRS on 30 th day	3.8±1.0	2.5±1.3	<0.001
Mean BI on 1 st day	18.3±16.9	51.4±24.0	<0.001
Mean BI on 7 th day	28.7±20.3	60.8±23.7	<0.001
Mean BI on 14 th day	28.0±29.1	67.0±32.1	<0.001
Mean BI on 30 th day	28.0±32.1	65.5±30.3	<0.001
Died on admission n (%)	79 (79.8)	16 (15.8)	<0.001
Mean survival time (Days)	12.2±1.1	24.5±1.1	<0.001

MRS=Modified Rankin Scale; BI = Barthel Index

Table IV: Univariate and Multivariate Binary logistic regression analysis of the determinants of 30- day case fatality.

<i>Variables</i>		<i>Univariate OR (95% CI)</i>	<i>p-value</i>	<i>Multivariate OR (95%CI)</i>	<i>p-value</i>
Dysphagic	Yes	1 (reference-death)			
	No	0.20 (0.13-0.32)	<0.001	0.29 (0.08-0.63)	0.005
Aspiration pneumonitis	Yes	1 (reference-death)			
	No	0.29 (0.22-0.40)	<0.001	0.28 (0.09-0.54)	0.024
Sepsis	Yes	1 (reference-death)			
	No	0.23 (0.11-0.50)	<0.001	0.42 (0.14-1.23)	0.112
NIHSS	Severe (21-42)	1 (reference-death)			
	Mild to moderate (0-20)	0.79 (0.56-1.06)	0.112	0.36 (0.13-1.00)	0.049
Gender	Male	1 (reference-death)			
	Female	0.79 (0.56-1.06)	0.112	NC	-
Stroke subtype	Ischaemia	1 (reference-death)			
	Intracerebral haemorrhage	2.18 (1.55-3.02)	<0.001	2.34 (1.03-5.31)	0.043
Post-stroke Hyperglycaemia	RBG >140	1 (reference-death)			
	RBG<140	0.76 (0.55-1.04)	0.092	NC	-
Mean Arterial Pressure	MAP >145	1 (reference-death)			
	MAP <145	0.72 (0.54-0.97)	0.041	1.05 (0.42-2.72)	0.923

NIHSS - National Institute of Health Stroke Scale; RBG - Random Blood Glucose; MAP - Mean Arterial Pressure; NC - Not Computed; OR - Odds Ratio; CI - Confidence Interval

Table V: Cox Proportional Hazard Model for patients with dysphagia controlling for the effect of Sepsis

<i>Variables</i>	<i>Beta</i>	<i>SE</i>	<i>p-value</i>	<i>HR</i>	<i>95% CI</i>
Aspiration without sepsis	1.059	0.509	0.036	2.883	1.070-7.767
Aspiration with sepsis	0.422	0.438	0.335	1.525	0.646-3.600

HR - Hazard ratio; SE - Standard Error

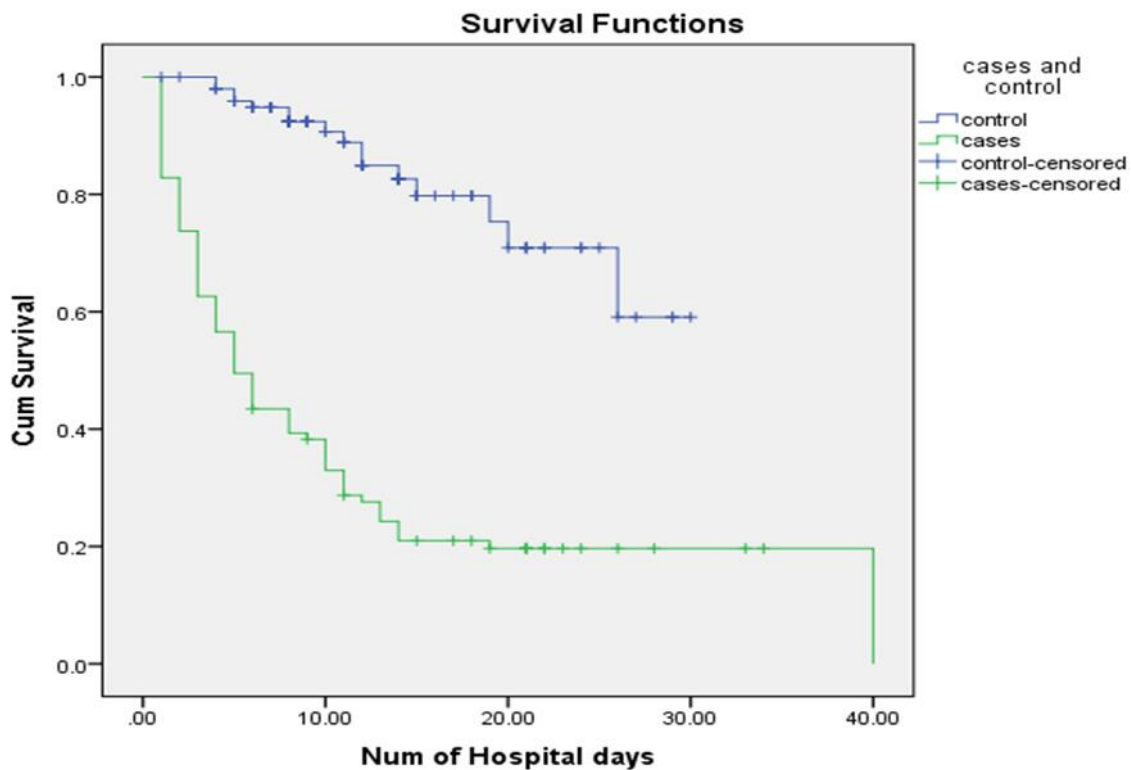
Discussion

Dysphagia following acute stroke is a common but serious problem; the findings in this study corroborate existing data from other populations. The prevalence of dysphagia in this study was 46.4% using peripheral oxygen desaturation and

48.5% using the 10mls water swallowing test. These findings are similar to reports from other studies that used similar methods. [15, 16] The prevalence of dysphagia in stroke patients ranged from 65% to 81% when videofluoroscopy and Fiberoptic Endoscopic Evaluation of Swallowing (FEES), which are the gold standards

for detecting dysphagia, were used. [17] The finding in the present study was consistent with the reports from other studies which suggested that absent gag reflex may be predictive of aspiration in stroke patients. [17, 18] However, the findings from some other studies showed that using the gag reflex alone to screen for dysphagia in stroke patients had low sensitivity and specificity. [4, 13] It was found in other studies that peripheral oxygen desaturation during swallowing of 10mls of water had higher

specificity. Studies have shown that a combination of both water swallowing test (WST) and peripheral oxygen desaturation method increased the sensitivity and specificity of detecting dysphagia in stroke patients. [11] These findings from the present study underscored the importance of checking for swallowing difficulty in all stroke patients and combining various clinical screening methods, where sophisticated equipment are not easily accessible or available.



[Num of Hospital days - Numbers of Hospital days]

Figure 2: Kaplan-Meier survival curve for dysphagic and non-dysphagic stroke patients

A significant proportion of the subjects in the present study with haemorrhagic stroke had dysphagia. This finding is similar to that of other researchers such as Sundar *et al.*, [19] and El-Sheikh, [20] who noted that dysphagia was more frequent in patients with haemorrhagic stroke. Although haemorrhagic stroke is less common than ischaemic stroke, the presence of

haemorrhagic stroke may be predictive of swallowing dysfunction. It is now increasingly recognized that unilateral hemispheric affection can cause dysphagia. [19, 21] The present study also showed that the laterality of neurological deficit did not influence the development of dysphagia. Studies by Suntrup *et al.* [22] suggest that swallowing is represented

bilaterally but asymmetrically, with no clear right or left laterality and the size of the cortical area associated with swallowing in the unaffected cortex determines the presence or absence of dysphagia. Therefore, it can be inferred that there is the possibility of unilateral hemispheric dominance which varies between individuals. Further studies requiring serial imaging and

transcranial magnetic stimulation to identify the swallow-dominant side can help determine whether there is a dominant hemisphere in each individual that, if affected, results in swallowing dysfunction. It is also plausible that the increased representation of swallowing in the unaffected hemisphere is a result of cortical reorganization and compensation. [34]

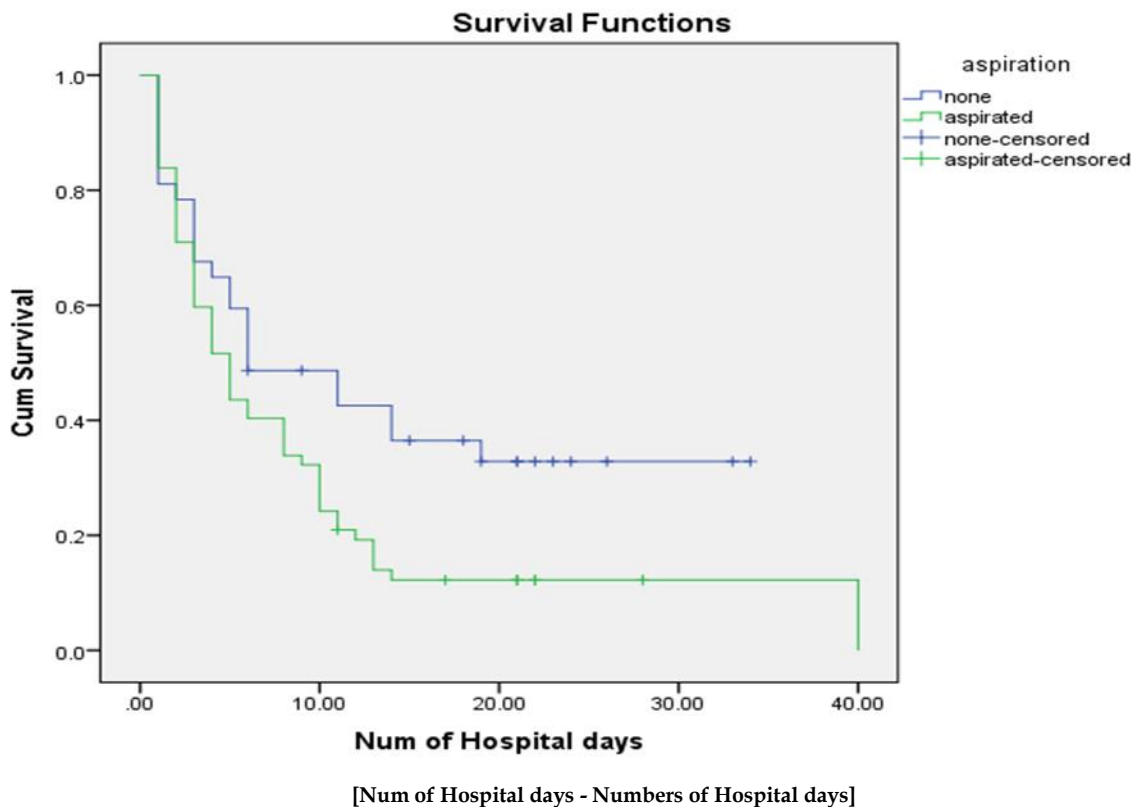


Figure 3: Kaplan-Meier survival curve for patients with dysphagia without aspiration and those with aspiration

Only six study participants had Total Anterior Circulation Infarct (TACI) based on OCSF classification of cerebral infarct. Four of the subjects with TACI had dysphagia and the other two did not have it. Although this finding lacked statistical significance probably due to the small number of subjects with TACI, Sundar *et al.* [19] in their study found that all the patients that had TACI had swallowing difficulty. The presence of dysphagia in TACI is probably due to the large area of hemispheric infarction involved. Only one out of the seven subjects in the study population that had lacunar syndrome had

dysphagia. This finding is consistent with the findings of Sundar *et al.* [19] The fact that subjects with lacunar stroke have a lower incidence of dysphagia may probably be due to smaller infarct volume and better collateral circulation through the Circle of Willis. However, the supplementary motor area, represented in the superior and middle frontal gyri, is believed to be associated with the planning of sequential movements, as occurs with swallowing. [24] Lesions in the cortical areas were not significantly associated with dysphagia in the present study as was found in some other studies. [25, 26] Severe stroke was

significantly associated with dysphagia in the present study as earlier documented that NIHSS was moderately predictive of clinically relevant dysphagia using a cut-off value of more than 9 as reported by Jevaseelan *et al.* [27] whereas a cut-off value of more than 12 reported by Okubo *et al.* [18] showed high sensitivity and specificity in detecting dysphagia.

The present study clearly showed that swallowing difficulty contributed significantly to morbidity and mortality in acute stroke patients. Stroke patients with dysphagia had worse functional outcome at the end of the 4th week based on the MRS and BI values. These findings were comparable to the results of other studies. [3, 20, 29, 30] Martino *et al.* [3] concluded that the presence of dysphagia was associated with an increased risk of death, disability, length of hospital stay, and institutional care among stroke patients. A study by Arnold *et al.* [31] revealed that stroke mortality and disability were independently associated with dysphagia.

The present study found that aspiration pneumonia contributed significantly to the morbidity and mortality of acute stroke patients. Dysphagic stroke patients that had aspiration pneumonia had worse functional outcome based on the MRS and BI at the 4th week post-stroke than those without aspiration pneumonia. The 30-day case fatality rate for dysphagic stroke cases complicated by aspiration pneumonia was significant but comparable to the findings in other studies. Feng *et al.* [32] found that pneumonia conferred a fourfold increased risk of 30-day death. Suda *et al.* [33] showed that stroke-associated pneumonia was associated with poor outcome in acute stroke patients. Teh *et al.* [34] Aspiration pneumonia was the complication with the highest attributable proportion of death in the entire stroke population within one year post-stroke. Vermeij *et al.* [35] concluded that stroke-associated infection, in particular pneumonia, was independently associated with

poor functional outcome after stroke. Confirming the above findings, the logistic regression analysis as well as the hazard ratio, after controlling for sepsis, showed that, aspiration was an independent predictor of death in acute stroke.

Study Limitations

The present study did not use the gold standards for detecting dysphagia (Videofluoroscopy and FEES) because they are unavailable. It is probable, therefore, that the frequency of dysphagia in this study was an underestimation. This limitation notwithstanding, this study showed bedside tests are valuable methods of screening for dysphagia in stroke patients, especially in resource-poor settings. Therefore, the gag reflex is still relevant in this respect as it was absent in half of the patients who were screened.

Conclusion

This study showed a high prevalence of dysphagia in acute stroke using peripheral oxygen desaturation along with impaired 10mls water swallowing test. Intracerebral haemorrhage, severe stroke, subcortical brain region and large stroke size were all associated with dysphagia. Dysphagia contributed significantly to the morbidity and mortality of acute stroke patients in this study with a 30-day case fatality rate for a patient with dysphagia alone approaching a quarter. The use of simple bedside swallowing tests is valuable screening tools for detecting dysphagia in stroke patients. Stroke patients with an identified risk factor for aspiration should be monitored for infection while early and aggressive management of pulmonary complication should be ensured to increase the chances of survival.

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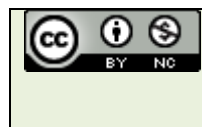
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