

Socio-Clinical Demographics of Acute Stroke Cases Could Determine Onset of Stroke

DR OSARENKHOE OSARETIN JOHN

Medicine department, Igbinedion university/teaching hospital, Nigeria

Abstract- Stroke or cerebrovascular Accident according to WHO is focal or global neurological deficit of vascular origin lasting more than twenty-four hours or resulting in death before twenty-four hours. Stroke is a health condition with global impact, causing significant morbidity and mortality worldwide. Many economies of the world commit to combating this problem. Worldwide and especially in subsaharan Africa, the victims are mainly in the productive age ranges, leading to huge economic loss. Stroke is currently the second leading cause of death worldwide after Ischaemic heart disease. Most strokes are preceded by modifiable risk factors. Controlling these risk factors would reduce the occurrence, morbidity and mortality of stroke. And trend of socio-clinical demographics in stroke patients is not elucidated in the literatures hence this study comes in handy

I. INTRODUCTION

Stroke according to World Health Organization (WHO) is an acute neurological deficit of cerebrovascular origin that persists beyond twenty-four hours or is interrupted by death within twenty-four hours. It has been projected that stroke could soon be the most common cause of death worldwide as it is currently the second leading cause of death in the world, ranking after heart disease.¹⁻⁹

Globally there is an increasing trend in the burden of non-communicable diseases especially cardiovascular and cerebrovascular diseases particularly in developing countries. Across Africa and in Nigeria the prevalence of stroke is also increasing. This transition imposes more constraints in dealing with the double burden of communicable and non-communicable diseases in a poor economy characterized by inadequate health systems.

This has been attributed to more people now living up to and beyond middle age because of improvement in sanitation and reduction in prevalence rate of infectious diseases coupled with increasing use of tobacco, westernized lifestyle and urbanization with reduced physical activity, increased caloric consumption and psychosocial stress also been implicated.

These acts synergistically to cause increased cardiovascular and cerebrovascular risk via weight gain, hypertension, dyslipidaemia, dysglycaemia and hyperuricaemia. By year two thousand and twenty it is predicted that non-communicable diseases will cause seven out of every ten deaths in developing countries compared with less than half that is obtained today.²⁻⁹ About eight hundred thousand people in the United States, have stroke each year, one hundred and thirty thousand of them die each year. One American dies from stroke every four minutes on average. Stroke cost the United States, an estimated \$36.5 billion each year. Worldwide, stroke is the second leading cause of death after ischaemic heart disease, and is followed by lower respiratory tract infections, chronic obstructive lung disease, diarrhea and HIV/AIDS, as the leading six killers worldwide as at 2013.^{10,11}

The incidence of stroke increases exponentially from thirty years of age, and the etiology varies with age. Advanced age is one of the most significant stroke risk factors. Ninety five percent of stroke occurs in people aged forty five and above, two-thirds of stroke occurs in those over the age of sixty five.^{3,12}

Disability affects seventy five percent of stroke survivors enough to decrease their employability and stroke can affect patients physically, mentally, emotionally, or its combination. The result of stroke varies widely depending on size and location of lesion. Thirty to fifty percent of stroke survivors suffer post stroke depression, which is characterized by lethargy,

irritability, sleep disturbance, lowered self-esteem and withdrawal while up to ten percent of all stroke patients develop seizures most commonly in the weeks subsequent to the stroke event and the severity of the stroke increases the likelihood of a seizure.^{8, 13-15}

Stroke can be classified into ischaemic stroke and haemorrhagic stroke²

Ischaemic stroke occurs as a result of an obstruction within a blood vessel supplying blood to the brain. It accounts for about eighty seven percent of all stroke cases. The underlying condition for this type of obstruction is the development of fatty deposits lining the vessel wall. This condition is called atherosclerosis. These fatty deposits can cause obstruction mainly as shown below;

Types of Ischaemic Stroke

(a)Cerebral thrombosis. Refers to a thrombus that develops at the clogged part of the vessel. (b) Cerebral embolism. Refers generally to a blood clot in the cerebrovascular system from another location in the circulatory system, usually the heart and large arteries of upper chest and neck, these tend to be associated with atrial fibrillation and other heart diseases. (c)Systemic Hypo-perfusion. This is a general decrease in blood supply for example in shock. (d)Venous thrombosis. This leads to stroke due to locally increased venous pressures which exceeds the pressure generated by the arteries. These infarcts are more likely to undergo haemorrhagic transformation (leaking of blood into the damaged area) than other types of ischaemic stroke. (e) Cryptogenic stroke. This is stroke of unknown origin. Constitutes thirty to forty percent of all ischaemic stroke.^{9, 16-19}

Less frequently used though stroke can also be classified based on the Oxford classification into 4 types;Total Anterior Circulation Infarct (TACI). (b) Partial Anterior Circulation Infarct (PACI). (c) Lacunar infarct (LAC). (d) Posterior Circulation Infarct (POCI). These four entities predict the extent of the stroke, the area of the brain affected, the underlying cause and the prognosis.^{20, 21}

Haemorrhagic stroke arises from bleeding within the brain parenchyma or intra ventricular spaces. They constitute about fifteen percent of stroke. They result

in tissue injury by causing compression of tissue from expanding haematoma or haematomas. This can distort and injure tissues. In addition, the pressure may lead to a loss of blood supply to the affected tissues with resulting infarction, and the blood released by brain haemorrhage appears to have direct toxic effects on brain tissue and vasculature. Inflammation also contributes to the secondary brain injury after haemorrhage^{19, 22, 23}

- Aims and objectives: To determine the trend of socio clinical demographics in stroke cases

II. METHODOLOGY

Data from 120 admitted acute (within twenty-four hours) stroke patients in University of Benin teaching hospital, Nigeria was reviewed. Data was analyzed using SPSS 21 package.

Study area/design: This study was carried in the University of Benin Teaching Hospital (UBTH) which is one of the six first generation hospitals in Nigeria that offers secondary and tertiary care to patients in Edo and neighboring states. This was a descriptive study that assessed the difference of electrocardiographic abnormalities between diabetic and non-diabetic cases.

Sampling method: A simple non-randomized sampling method was used in selecting patients recruited for this study. One hundred and twenty patients presenting for the first time with clinical features and imaging findings of stroke (CT brain scan was performed in all cases) and were admitted into the UBTH medical wards. They had a detailed history and physical examination finding entered into the data acquisition sheet. ECG was performed on the stroke patients within the first twenty-four hours of presentation.

Inclusion criteria:

- A. Patients that have first ever occurrence of stroke.
- B. Patients that are eighteen (18) years old and above.
- C. The patients that remain alive for at least 7 days post presentation.

Exclusion criteria:

- A. Patients that have two or more occurrence of stroke (recurrent stroke).
- B. Patients less than eighteen (18) years of age.
- C. Stroke resolved within twenty-four (24) hours, as evidenced by resolution of presenting complaints.
- D. Patients that died within 7 days of presentation.
- E. HIV positive patient.
- F. Patients with malignancies.
- G. Patients on immunosuppressive therapy.
- H. Patients with electrolyte abnormalities.

Data analysis: Anthropometric measurement and data collected using the preformat were collated and analyzed using the International Business Machines Statistical Product and Service Solutions (IBM- SPSS) version 22. Data were presented using tables and charts. Frequencies and percentages were used to present categorical data while continuous data were

expressed as mean (Standard Deviation). Frequencies were compared using the Pearson’s Chi-square test while means were compared using the independent t-test. Where the data was skewed, continuous data were expressed as mean (inter-quartile range) and compared using the Mann Whitney U test. Significant chi-square comparisons were further tested using a binomial logistic regression where applicable. A p value less than 0.05 were considered significant for all statistical comparisons.

Ethical clearance: Ethical clearance was obtained from the Research and Ethics Committee of the University of Benin Teaching Hospital, Benin City, Edo State. Informed consent was obtained from patients before participation in the study. Autonomy: Respect for respondents and confidentiality was maintained throughout the process of extracting the data.

III. RESULTS

	Day 1 n (%)	Day 3 n (%)	Day 7 n (%)	DAY 1/7 p value
Age (years)	58.5	58.5	58.5	
Sex				
Male	56(46.7)	56(46.7)	56(46.7)	
Female	64(53.3)	64(53.3)	64(53.3)	
Respiratory distress	100(83.3)	8(6.7)	4(3.3)	<0.001*
Facial palsy	68(56.7)	68(56.7)	68(56.7)	
Impaired speech	64(53.3)	64(53.3)	64(53.3)	
Headache	32(23.3)	20(16.7)	16(13.3)	0.01*
Loss of consciousness	28(23.3)	20(16.7)	20(16.7)	0.197
Vomitting	28(23.3)	26(21.7)	20(16.7)	0.197
Fever	12(10.0)	20(16.7)	4(3.3)	0.07
Palour	12(10.0)	12(10.0)	12(10.0)	
Seizures	4(3.3)	4(3.3)	8(6.7)	0.374
Hypertension	92(76.7.)	92(76.7)	92(76.7)	

Diabetes mellitus	28(23.3)	28(23.3)	28(23.3)
Obesity	12(10.0)	12(10.0)	12(10.0)

* $p < 0.05$ for day 1 vs. day 7 comparisons.

The mean age of the stroke cases was 58.47 +/- 12.80 years. The stroke cases were sixty-four females and fifty-six males. One hundred (83.33%) of the stroke cases were married, twelve (10.00%) were single and eight (6.67%) were widowed. All the stroke cases were Christians. Among the cases forty eight (40.00%) were Binis, twenty four (20.00%) were urhobos, sixteen (13.33%) were igbos and eight (6.67%) were esan..

One hundred (83.33%), eight (6.67%) and four (3.23%) were in respiratory distress on day 1, 3 and 7 respectively. This was significant with $p < 0.001$. Sixty-eight (56.67%) of the cases had facial nerve paralysis and number was same on days 1, 3 and 7. Sixty-four (53.33%), of the cases had impaired speech on day 1, 3 and 7 respectively. This was not statistically significant between day 1 vs. day 7,. Thirty-two (26.67%), twenty (16.67%) and sixteen (13.33%) of cases had headache on day 1, 3 and 7 respectively. There was statistically significantly difference between day 1 vs day 7 $p = 0.01$.

Loss of consciousness was in twenty-eight (23.33%), twenty (16.67%) and twenty (16.67%) of the cases on day 1, 3 and 7 respectively. There was no statistically significant difference in day 1 vs day 7 $p = 0.197$. Among the stroke group sixteen (40.00%) of the forty haemorrhagic stroke patients and twelve (15.00%) of the eighty ischaemic cases presented with loss of consciousness. This difference was statistically significant, $p = 0.002$ OR = 3.78, CI 1.57 – 9.12. Twenty-eight (23.33%) of the cases presented with vomiting, twenty six (21.67%) and twenty (16.67%) were still vomiting on day 3 and 7 respectively. There was no statistically significant difference on day 1 vs day 7 $p = 0.197$

Twelve (10.00%), twenty (16.67%) and four (3.33%) cases had fever on day 1, 3 and 7 respectively. The difference between day 1 and day 7 was not statistically significant, $p = 0.07$. Twelve (10.00%) of the cases were pale on presentation and on day 7. There was no statistical significance. Four (3.33%),

four (3.33%) and eight (6.67%) had seizures on day 1, 3 and 7. There was no statistically significant difference between day 1 vs day 7 $p = 0.374$.

Ninety-two (76.67%) of the cases were known hypertensive. Twenty-eight (23.33%) of the cases were diabetics and twelve (10.0%) were obese. Ninety-six (80.00%) of the cases were ill looking as at presentation. There was no statistical significance.

IV. DISCUSSION

Majority of the stroke cases were in their middle age of life (The mean age of stroke cases was 58.47 years). Stroke risk increases with age while the chances of developing a stroke after the age of forty five years double every ten years. Furthermore, there is increase in life expectancy with women having a higher life expectancy compared to males supporting existing evidence that stroke occurrence tends to increase with longevity probably accounting for the higher female preponderance in this study and other studies.²⁴⁻²⁷

Previous findings as that in this study show that stroke is commoner in married cases than single as marital strain, job stress and depression have been noted to contribute to the development of heart disease and stroke.²⁸⁻³⁰ Impaired speech, headache, vomiting and loss of consciousness respectively are the most common presenting symptoms of stroke cases. These and other symptoms would improve within seven days but headache would have significantly resolved by this time.

Hypertension, diabetes mellitus and obesity respectively are the most common risk factors for stroke. Hypertension is three-fold and seven-fold more common than diabetes mellitus and obesity respectively among stroke cases.

These findings in this study were further corroborated and buttressed by existing knowledge in the literature that show diabetes is an independent risk factor for stroke same as hypertension and obesity. Obese

patient are more susceptible to death or disability from stroke. Excess fat tissue has been shown to have a significant correlation with stroke and TIA. An increase in body mass index increases the risk of atherosclerosis and ischaemic stroke. Coexisting obesity and diabetes increase the risk of left ventricular hypertrophy.³¹⁻³⁵

Hypertension is modifiable; therefore, controlling hypertension would prevent eighty percent of strokes and lowering diastolic blood pressure by 10 mmhg decrease risk of stroke by fifty percent.^{36, 37}

Respiratory distress and facial palsy are the most common examination findings in stroke cases. However, respiratory distress and headache would have resolved by day three post event while facial palsy would persist. From this study, one can safely infer that absence of respiratory distress and or headache in a presenting stroke case should mean that that stroke is more than forty eight hours old in onset.

CONCLUSION

Headache and respiratory distress significantly resolve within three days post event. While facial palsy would persist, thus could be used to time the stroke onset

- Recommendation: There is need to do similar studies using multicenter, larger number of patients and for longer duration to look at socio clinical demographics in stroke cases to see if any and or some of these features could be used to determine onset of the stroke event
- Limitations of this study: This is a single center study thus the sample size though adequate can be improved upon. A larger sample size would involve a large multicenter study which will take more time and resources beyond that available for this research.

REFERENCES

[1] World Health Organization. Cerebrovascular Disorder Geneva: World Health Organization.1978.24-6

[2] Boutayeb A and Boutayeb S. The burden of non-communicable disease in developing countries. *Int. J. Equity Health.* 2005; 4: 2-6.

[3] Ellekjaer H, Holmen J, Indredavik B, et al. Epidemiology of stroke in Innherred, Norway, 1994 to 1996: Incidence and a 30 – Day case fatality rate. *Stroke.* 1997; 28: 2180 -2184.

[4] Kocan M J. Cerebrovascular effects of acute stroke. *Prog Cardiovascular Nurs.*1999; 1:61—7.

[5] Goldstein D S. The electrocardiogram in stroke: relationship to pathophysiological type and comparism with prior tracings. *Stroke.* 1979; 10: 253-9.

[6] FAMILONI O.B. The pattern and prognostic features of QT intervals and dispersion in patients with acute ischaemic stroke. *J Natl Med. Assoc.* 2006; 98: 1758-62.

[7] Tokgozoglu S.L, Batur M.K, Topcuoglu M.A, et al. Effects of Stroke localization on Cardiac Autonomic Balance and Sudden Death. *Stroke.* 1999; 30:1307-11.

[8] World Health Organization. The World Health Report 2004. Annex Table 2: Deaths by cause, sex and mortality stratum in WHO regions, estimates 2002. Geneva. World Health Organization.2004.

[9] Donnan G A, Fisher M, Macleod M, et al. *Stroke.* Lancet. 2008; 371: 1612 -15.

[10] Go A.S, Mozaffarian D, Roger V .L et al. Heart and stroke statistics 2013update report from the American Heart Association. *Circulation.* 2013; 2: 241-6.

[11] WHO. The top 10 causes of death. Geneva. World Health Organization. 2017. Available from www.who.int/en/news-room/factsheets/detail/the-top-10-causes-of-death (Accessed on 16th May 2018).

[12] Senelick R, Rossi C, Peter W, et al. Living with stroke: A Guide for facilities. Chicago. Contemporary books. 1994.10-6

[13] Coffey C, Edward C. Jeffery L, et al. *Stroke .The American psychiatric press textbook of Geriatric Neuropsychiatry .* Washington DC: American Psychiatric press.2000.2 edition. 601 – 617.

- [14] Lisa D. Sandra E. Fuqiang G et al. Correlating lesion size and location to deficits after ischaemic stroke the influence of accounting for altered peri-necrotic tissue and incidental silent infarcts. *Behav Brain Funct.* 2010; 6: 6-10.
Available at <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2823642> Accessed 12th September, 2019
- [15] Reith J, Jorgensen H. S, Hakayama H. et al. Seizures in acute stroke: predictors and prognostic significance. *The Copenhagen stroke study.*1997; 28: 1585-9.
- [16] Ocarroll C.B, and Barrette K.M. Cardioembolic Stroke. *Continuum Lifelong Learning in Neurology.*2017; 23:111-132.
- [17] Shuaib A, Hachinski V. C, "Mechanisms and management of stroke in the elderly". *CMAJ.* 1991; 145: 433 - 43.
- [18] Stam J. "Thrombosis of the cerebral veins and sinus." *The New England Journal of Medicine.* 2005; 352:1791 – 8.
- [19] National Institute of Neurological Disorders and Stroke (NINDS). 'Stroke Hope Through Research'. National Institute of Health.1999.112-5
- [20] Bamford J. Classification and natural history of clinically identifiable subtypes of cerebral infarction. *Lancet.*1991; 337:1521 – 6.
- [21] Bamford J. M. 'The role of the clinical examination in the subclassification of stroke'. *Cerebrovas. Dis.*2000; 10:2 – 4
- [22] Wang J. 'Preclinical and clinical research on inflammation after intracerebral haemorrhage'. *Prog. Neurobiol.* 2010; 92: 463–77.
- [23] Adeloye D. An estimate of the prevalence of hypertension in Nigeria: a systematic review and meta-analysis. *J. Hypertension.* 2015; 2: 260 – 262.
- [24] Ezenwa C, and Gutierrez J. Secondary stroke prevention: challenges and solutions. *Vasc. Health Risk Manag.* 2015; 11: 437-450.
- [25] Kelly-Hayes M. Influence of Age and Health Behaviours on Stroke Risk: Lessons from Longitudinal Studies. *J Am Geriatr Soc.* 2010; 58: 325-8.
- [26] Glader E.L, Stegmayr B, Norrving B, et al. Sex differences in management and outcome after stroke: A Swedish national perspective. *Stroke.* 2003; 34:1970-5.
- [27] Ghandehari K and Izadi Z. The Khorasan Stroke Registry: Results of a five-year hospital-based study. *Cerebrovasc Dis.*2007; 23:132-9.
- [28] Eaker ED, Sullivan LM, Kelly-Hayes M et al. Marital status marital strain, and risk of coronary heart disease or total mortality: The Framingham Offspring study. *Psychosom Med.* 2007; 69: 509-513.
- [29] Whooley MA, de JP, Vittinghoff E, et al. Depressive symptoms, health behaviours, and risk of cardiovascular events in patients with coronary heart disease. *JAMA.* 2008; 300: 2379-2388.
- [30] Hackett ML, Yapa C, Parag V, et al. Frequency of Depression after stroke: A systematic review of observational studies. *Stroke* 2005; 36: 1330-40.
- [31] Kurt T, Gaziano J M, Berger K, et al. Body mass index and risk of stroke in men. *Arch Intern Med.* 2002; 32: 2575-2579.
- [32] Rost N S, Wolf P A, Kase C S, et al. Plasma concentration of C-reactive protein and risk of ischaemic stroke and transient ischaemic attack: The Framingham Study. *Stroke.* 2001; 32: 2575-2579.
- [33] Roger V L, Go A S, Lloyd-Jones D M et al. American Heart Association. *Heart Disease and Stroke Statistics.* *Circulation.*2011;1:123-4
- [34] Winter Y, Rohrmann S, Linseisen J, et al. Contribution of obesity and abdominal fat to risk of transient ischaemic attacks. *Stroke* 2008; 39: 3145-3151.
- [35] Lauer RM, Clarke WR. Childhood risk factors for high adult blood pressure: the Muscatine study. *Paediatrics* 1989; 84: 633-641.
- [36] Balami J.S, Chen R.L, Grunwald I.Q, et al. Neurological complication of acute ischaemic stroke. *Lancet Neurol.* 2011; 10:357-371.
- [37] Ozturk S, Ege F. and Ekmeko H. Language Disorders due to Posterior System Strokes: An Ignored Dysfunction. *Noro Psikiyatrs Ars.* 2014 ;51: 313-17.