DOI Number: 10.37506/v14/i1/2020/ijfmt/192937

Correlation of Smoking and National Institutes of Health Stroke Scale (NIHSS) in Acute Ischemic Stroke Patients

Yunike Tourisiana[1], Mohammad Saiful Islam[1], Joseph Ekowahono Rahardjo[1]

[1]Department of Neurology, Faculty of Medicine, Universitas Airlangga-Dr. Soetomo General Hospital, Surabaya 60131, Indonesia

Abstract

Background: Ischemic stroke is the most common type of stroke with 87.00% prevalence of atherosclerosis as one of the causes of cerebral vascular lumen occlusion. Smoking is known as a modifiable risk factor for stroke. The process of atherosclerosis leads to a neurological deficit resulting in impairment of the patient. One of the scales used to assess impairment is the National Institutes of Health Stroke Scale (NIHSS).

Objectives: To know the correlation between smoking status and functional degree of acute ischemic stroke patients as measured by NIHSS.

Method: The subjects were 48 patients. The study design was cross-sectional and clinical sampling consecutive admissions and an acute ischemic stroke image imaging. Smoking status data was obtained through anamnesis at the time the patient was hospitalized supported by the testimony of the immediate family. NIHSS values were obtained on the first day of hospitalization. Data analysis used chi-square test and logistic regression.

Result: Sex (p = 0.001) and smoking (p = 0.013) were variables that had significant correlations with NIHSS. There was no association between smoking and the functional degree of acute ischemic stroke patients measured by NIHSS, (p = 0.57) with Adjusted Odds Ratio 1.5 (CI 95% 0.35 - 6.9).

Conclusion: There was no significant association between smoking with functional degree of acute ischemic stroke patients as measured by NIHSS.

Keywords: *NIHSS, Acute ischemic stroke, Smoking status*

Introduction

A stroke is a focal or global neurologic deficit that suddenly occurs over 24 hours unless it dies before 24 hours or surgery is performed, in the absence of other causes other than cerebral vascular disorders [1]. The prevalence of stroke in Indonesia has increased from 8.3 each mile in 2007 to 12.1 per mil in 2013. Ischemic stroke is the most common type of stroke with the prevalence of 87.00% [2]. Ischemic stroke is classified based on the mechanism of etiopathogenesis: Large Artery Atherosclerotic Stroke (LAA), cardioembolic stroke (CE), small artery occlusion or lacunar stroke (LAC), the stroke of other determined etiology and stroke of undetermined etiology [3],

One of the risk factors that increase the incidence of stroke is smoking behavior [4]. Indonesia is ranked one in the world for the number of male smokers over the age of 15 (66.00%) [5]. Exposure to secondhand smoke triggers many pathological effects in the endothelium, such as oxidative stress, platelet activation, trigger coagulation cascades and interfere with fibrinolysis [6,7] . Smoking causes poorer functional outcomes at 3 months after the onset of ischemic stroke compared to non-smokers [8,9].

The scale to measure the functional deficit of acute ischemic stroke is NIHSS (National Institutes of Health Stroke Scale) [1]. Smoking is said to be associated with

Correspondence Author
Mohammad Saiful Islam
Email : mohammadsaifulislam2828@gmail.com

higher NIHSS scales in small vessel occlusion [10], while other studies produce different assumptions by using the same measurement tool [11]. NIHSS has a specificity of 0.90 (CI 95%, 0.86-0.94), sensitivity of 0.71 (CI 95%, 0.64-0.79) and good accuracy of 0.83 (CI 95%, 0.79-0.87) in predicting post-stroke clinical outcomes [12].

How to know someone's smoking status is with interviews that have sensitivity and specificity by 79.00% [13]. This method is known not invasive and does not require special techniques also cheap. The current studies on the relationship between smoking and functional degrees in acute ischemic stroke patients in Indonesia are not present. This study is expected to help clinicians to be more aggressive in controlling risk factors for stroke associated with the process of atherosclerosis, especially smoking, so as to reduce the degree of disability stroke patients.

Method

The subjects of the study were acute ischemic stroke patients in Dr.Soetomo General Hospital and treated in Seruni A and B rooms, from February to July 2017. The inclusion criteria of having the first attack of ischemic stroke and willing to paticipate the research. The exclusion criteria were having a history of brain tumors, brain infection, and head trauma as well as unclear or unreliable smoking status. The design used was cross-sectional study with a sample size of 24 patients using the technique of sampling consecutive admission. Patients were interviewed about smoking status and NIHSS scale measured on the first day of treatment.

Prior to the identification of the subject, the researcher conducted the ethical test (191/Panke.KKE/III/2017) in Dr.Soetomo General Hospital Surabaya, Indonesia. Primary data collection can be basic patient characteristics (age, sex, education level, smoking history, duration of smoking and number of cigarettes consumption each day) and clinical characteristics (history of brain infection, head tumor, head trauma, hypertension and diabetes mellitus). History of brain infection, brain tumor, and head trauma is evidenced by interviews, physical examinations, and radiology. Hypertension has a systolic standard of ≥ 140 mmHg and/or diastolic ≥ 90 mmHg according to the JNC 7 criterion and is measured by a sphygmomanometer. DM has a standard ≥ 200 mg/dL for laboratory examination. Measurement of functional deficits of acute ischemic stroke with NIHSS scale. Acute ischemic stroke patients were divided into 2 groups, i.e., NIHSS <5 were included in mild criteria and NIHSS ≥ 5 were included in moderate-severe criteria. The correlation between smoking status and functional grade of acute ischemic stroke patients was calculated and analyzed by chi-square statistic test and logistic regression with SPSS (SPSS, Inc., Chicago, IL).

Results

Clinical Characteristics and Basic Subject Research

The mean age of the study subjects was 58.9 ± 12.3 years, with the youngest age of 27 years and the oldest 86 years old. The minimum random blood sugar level-maximum was 85 and 915. The systolic maximum values were 90 and 240. The diastolic maximum values are 60 and 140 (Table 1).

Table 1. Clinical Characteristics of Research Subject

Variables	Mean ±SD (N=48)	Range
Age (years old)	58.9 ± 12.3	27 – 86
GDA (mg/dL)	173.6±138.4	85 - 915
Systolic (mmHg)	158.8 ± 32.1	90 - 240
Diastolic (mmHg)	92.3 ± 16.6	60 - 140

The majority of subjects were male (64.60%) with the highest number age was ≤ 60 years (68.70%). Most subject education history (52.00%) was higher education level (Senior high school and Bachelor degree). The majority of nonsmoker subjects (58.30%). In smokers, most smokers for> 20 years (90.00%) and cigarette consumption per day were 10-19 cigarettes (65.00%) (Table 2).

Table 2. Basic Characteristics of Research Subject

Variables	Total (n=48)	Percentase (%)
Sex		
Female	17	35.40
Male	31	64.60
AGE GROUPS		
≤ 60 y/o	33	68.70
> 60 y/o	18	37.50
Education Level		
Primary	23	48.00
Advanced	25	52.00
Smoking history		
Smoking	20	41.70
Non-smoking	28	58.30
Smoking Duration		
1-9 y/o	0	0.00
10-20 y/o	2	10.00
>20 y/o	18	90.00
Smoking per-day		
1-9 cigarretes	5	25.00
10-19 cigarretes	13	65.00
>20 cigarretes	2	10.00

Independent Variable Correlation and NIHSS Score

Subjects of the study were male who had moderate-weight NIHSS 90.50%, it was greater than those with mild NIHSS (44.40%). The difference was statistically significant (p = 0.001), with Crude Odds Ratio of 11.875 (CI 95%, 2.297- 61.396). In the age category, the majority of subjects aged ≤60 years, who had moderate-severe NIHSS were 57.10%, and it was smaller than those with mild NIHSS (77.8%) also were not statistically significant (p = 0.112).

Subjects with primary education (Elementary school - Junior high school) with moderate-severe NIHSS were 57.10% that greater than those with mild NIHSS (40.70%). In DM variables, those with moderate NIHSS were 28.60%, it was greater than those with mild NIHSS (22.20%). These two variables were also not statistically significant (p = 0.201) and (p = 0.431).

In hypertension variables, the study subjects who had moderate-severe NIHSS were 81.00%, smaller than those with mild NIHSS (88.90%) and were not statistically significant (p = 0.68). In smoking variables, NIHSS with moderate-weight was 62.00% greater than those with mild NIHSS (26.00%). The difference was statistically significant (p = 0.013), with Crude Odds Ratio of 4.64 (CI 95% 1.35 - 15.9) (table 3).

Table 3. Correlation of Independent Variables (Clinical and Basic) and Dependent (NIHSS)

Independent Variables	NIHSS (n=48)		p-value	OR (Cl 95%)
	Medium-Severe (%)	Mild (%)		
Sex				
Male	90.50	44.40	0.001	11.875 (2.297– 61.396)
Female	9.50	55.60		
Age				
≤ 60 y/o	57.10	77.80	0.112	0.38 (0.11 – 1.33)
> 60 y/o	42.90	22.20		
Education Level				
Primary	57.10	40.70	0.201	1.94 (0.61 – 6.16)
Advanced	42.90	59.30		
DM Statuses				
Yes	28.60	22.20	0.431	1.4 (0.38 – 5.2)
Normal	71.40	77.80		
Hyphertensi Status				
Yes	81.00	88.90	0.35	0.53 (0.11 – 2.68)
Normal	19.00	11.00		
Smoking Status				
Smoking	62.00	26.00	0.013	4.64 (1.35 – 15.9)
Non-smoking	38.00	74.00		

Multivariate Analysis

In bivariate analysis, there was a significant correlation between functional degree measured by NIHSS and sex variable also smoking status. Therefore, the analysis was continued with logistic regression. After multivariate analysis logistic regression was obtained p = 0.57 with Adjusted Odds Ratio 1.5 (CI 95% 0.35 - 6.9) meaning that there was no correlation between smoking status and NIHSS score.

Discussion

The average age was elderly (> 60 years) with the majority being male. This is consistent with studies suggesting that males have a higher risk of stroke than female and sex is one of the unmodifiable risk factors for stroke [14]. The well-documented modifiable stroke risk factors include hypertension, smoking, diabetes, dyslipidemia, atrial fibrillation, asymptomatic carotid stenosis, hormone replacement therapy, nutrition and physical inactivity [14]. Smoking was believed to cause the development of atherosclerosis by initiating endothelial lesions through the production of oxygen radicals or by direct toxic effects of the element of cigarette smoke.

Smoking was associated with higher NIHSS scores in small vessel occlusion [10]. But other studies stated that smoking was not associated with good functional outcomes after acute ischemic stroke [11]. Both studies used NIHSS as an indicator of clinical outcome.

NIHSS was a tool used to measure the functional degree of stroke. In this research, the maximum values of NIHSS were 2 and 19. The mean of NIHSS was 5.56 ± 3.8. The subjects were divided into 2 groups, those with

mild neurologic deficit were 56.30% and the group with the severe neurological deficit was 43.80%. This study compared to NIHSS scales in the smokers, diabetes, and hypertension groups. There was no association between diabetes and poor NIHSS on the first day of treatment. This was similar to other studies that there was no significant difference in the severity of stroke at baseline treated as assessed by NIHSS between patients suffering from DM and non-DM [15]. While hypertension was also not associated with NIHSS that consistent with other studies which suggest that elevated blood pressure during the onset of ischemic stroke was indicated the mild stroke [16].

There were two statistically significant variables ($p < 0.05$) that were sex and smoking. However, in the logistic regression analysis for both variables, there was no association between smoking and NIHSS. Differences in the results of the multivariate analysis with bivariate analysis results can be overcome by the addition of the number of research subjects.

Oxidative stress could cause disorders of cerebral blood vessel metabolism that resulting in vascular lesions with forms of lacunar stroke, white matter hyperintensity, and microbleeds. Oxidative stress possibly triggered by hypertension, diabetes, aging, and smoking [17]. The neurologic deficits in ischemic stroke patients who smoked in this study were mostly mild, as they may be caused by small vessel disease with clinical forms of lacunar stroke.

Although there was no significant association between smoking and the functional degree of acute ischemic stroke was assessed with NIHSS, the majority of the subjects were smoking. Although the functional degree of stroke in this study was mostly mild, smoking has been recognized as a risk factor for stroke so anti-smoking campaigns must still be aggressive for the primary prevention of ischemic stroke.

Conclusion

Based on the sex and smoking variables, it had a significant relationship with the functional degree measured by the NIHSS scale. In a multivariate analysis, there was no significant association between smoking status and functional degree of acute ischemic stroke patients as measured by NIHSS. Further research with more objective measures such as CO levels in expiratory air and blood levels of cotinine was also worth considering.

Ethical Clearance: The study has been approved by ethical comittee (191/Panke.KKE/III/2017) in Dr.Soetomo General Hospital Surabaya, Indonesia.

Conflict of Interest: The author reports no conflict of interest of this work.

Source of Funding: This study is done with individual funding

References

1. Investigators WHOMPP. The World Health Organization MONICA Project (monitoring trends and determinants in cardiovascular disease): a major international collaboration. J Clin Epidemiol. 1988;41(2):105–14.
2. RI KK. Laporan hasil riset kesehatan dasar (Riskesdas) 2013. Jakarta Kementeri Kesehat RIDinKes Jateng. 2013;
3. Adams Jr HP, Bendixen BH, Kappelle LJ, Biller J, Love BB, Gordon DL, et al. Classification of subtype of acute ischemic stroke. Definitions for use in a multicenter clinical trial. TOAST. Trial of Org 10172 in Acute Stroke Treatment. Stroke. 1993;24(1):35–41.
4. Shah RS, Cole JW. Smoking and stroke: the more you smoke the more you stroke. Expert Rev Cardiovasc Ther. 2010;8(7):917–32.
5. Eriksen M, Mackay J, Ross H. The Tobacco Atlas. Atlanta, GA: American Cancer Society; New York, NY: World Lung Foundation; 2012. 2013.
6. Burke A, FitzGerald GA. Oxidative stress and smoking-induced vascular injury. Prog Cardiovasc Dis. 2003;46(1):79–90.
7. Fowles J, Bates M, Noiton D. The chemical constituents in cigarettes and cigarette smoke: priorities for harm reduction. A Rep to New Zeal Minist Heal. 2000;1–65.
8. Kumagai N, Okuhara Y, Iiyama T, Fujimoto Y, Takekawa H, Origasa H, et al. Effects of smoking on outcomes after acute atherothrombotic stroke in Japanese men. J Neurol Sci. 2013;335(1–2):164–8.
9. Ovbiagele B, Weir CJ, Saver JL, Muir KW, Lees KR. Effect of smoking status on outcome after acute ischemic stroke. Cerebrovasc Dis. 2006;21(4):260–5.
10. Weng W-C, Huang W-Y, Chien Y-Y, Wu C-L, Su F-C, Hsu HJ, et al. The impact of smoking on

the severity of acute ischemic stroke. J Neurol Sci. 2011;308(1–2):94–7.

11. Lee J-H, Lee JY, Ahn SH, Jang MU, Oh MS, Kim C-H, et al. Smoking is not a good prognostic factor following first-ever acute ischemic stroke. J stroke. 2015;17(2):177.
12. Muir KW, Weir CJ, Murray GD, Povey C, Lees KR. Comparison of neurological scales and scoring systems for acute stroke prognosis. Stroke. 1996;27(10):1817–20.
13. Hald J, Overgaard J, Grau C. Evaluation of objective measures of smoking status a prospective clinical study in a group of head and neck cancer patients treated with radiotherapy. Acta Oncol (Madr). 2003;42(2):154–9.
14. Meschia JF, Bushnell C, Boden-Albala B, Braun LT, Bravata DM, Chaturvedi S, et al. Guidelines for the primary prevention of stroke: a statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2014;45(12):3754–832.
15. Nacu A, Thomassen L, Fromm A, Bjerkreim AT, Andreassen UW, Naess H. Impact of diabetes mellitus on 1867 acute ischemic stroke patients. a bergen norstroke study. J Res Diabetes. 2015;2015:1–12.
16. Manabe Y, Kono S, Tanaka T, Narai H, Omori N. High blood pressure in acute ischemic stroke and clinical outcome. Neurol Int. 2009;1(1).
17. De Silva TM, Miller AA. Cerebral small vessel disease: targeting oxidative stress as a novel therapeutic strategy? Front Pharmacol. 2016;7:61.