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Research Article

Analysis of the Impact of Muscle Mass Changes in Stroke Patients

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ABSTRACT

Stroke has become an increasingly prevalent health issue every year. Malnutrition is one of the complications that arise in post-stroke patients. Reduced physical activity, intake disturbances, and comorbidities experienced by stroke patients can trigger malnutrition. Additionally, factors such as age, sex, Body Mass Index (BMI), and stroke type accelerate the onset of malnutrition. One of the signs of malnutrition in stroke patients is a change in body composition, characterized by muscle mass loss due to inflammatory responses and catabolic reactions, which lead to increased caloric needs in stroke patients. This study aims to investigate the changes in muscle mass in stroke patients at Dr. Kariadi General Hospital, Semarang, Central Java, Indonesia. This is an observational study with a cross sectional design conducted at Dr. Kariadi General Hospital from November 2023 to April 2024. The sample size was 49 patients diagnosed with stroke, receiving medical care on the first day at Dr. Kariadi General Hospital. This study evaluates factors such as age, sex, stroke type, BMI, smoking habits, diabetes mellitus, dyslipidemia, hyperuricemia, hypertension, kidney disorders, and respiratory diseases, in relation to changes in fat-free mass, fat mass, visceral fat, and phase angle, to assess the correlation with muscle mass changes. Data analysis will be approached using Structural Equation Modeling (SEM) with Partial Least Square (PLS). The results show that various risk factors affect body composition changes, contributing to muscle mass changes by up to 84.1%. In conclusion, the decline in muscle mass in stroke patients is influenced by various factors, in addition to reductions in visceral fat and phase angle, which indicate a tendency toward malnutrition risk.

Keywords: Body Composition, Stroke, Dr. Kariadi, Muscle Mass.

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INTRODUCTION

The incidence of stroke has been increasing every year. In 2020, stroke cases reached 89.13 million, with ischemic strokes accounting for the majority at 65% of total cases, while the remaining cases were hemorrhagic strokes. The mortality rate for hemorrhagic strokes was approximately 3.7 million, which is higher compared to ischemic strokes, with around 3.48 million deaths among total sufferers.[1] In developed countries, the number of stroke cases is also quite high. In the United States, it is estimated that there are 7.6 million stroke patients, with the number doubling, especially in the elderly. The mortality rate exceeds 140,000 each year, despite advances in prevention and treatment.[2] In Indonesia, stroke has become one of the leading causes of disability and death, with mortality rates based on age and sex reaching 193 per 100,000 people, making it the highest in Southeast Asia.[3]

Various risk factors influence stroke incidence, which can be categorized into two groups: non-modifiable risks, which include age, gender, race, and a family history of vascular disease; and modifiable risks, which include a history of hypertension, diabetes mellitus, heart disease, dyslipidemia, obesity, smoking, alcohol consumption, and lack of physical activity.[4,5] These risk factors in stroke patients significantly increase complications, affecting 40-95% of hospitalized patients. The main complications often encountered are dysphagia, pneumonia, and digestive disorders.[6] Dysphagia is the most common complication in stroke patients, affecting more than half of the total stroke patients, leading to swallowing disorders and difficulties in consuming food. This increases the risk of malnutrition, which can reduce the quality of life, immunity and elevate the risk of hospital infections.[7]

Malnutrition in stroke patients is generally influenced by intake disorders, neurological deficits, cognitive impairments, and catabolic processes that lead to decreased food intake. Additionally, malnutrition is sometimes experienced by patients even before a stroke occurs, due to unhealthy lifestyles, poor socioeconomic conditions, and pre-existing chronic diseases.[8] An inflammatory response also occurs in stroke patients, marked by an increase in pro-inflammatory cytokines, which triggers oxidative stress due to an imbalance between the increased number of free radicals and the body's antioxidants.[9,10] The heightened inflammatory mechanism affects the immune system, food intake, and metabolism, thereby decreasing appetite and nutritional status. The increased need for intake, which is not balanced with adequate food consumption, further accelerates the occurrence of malnutrition.[11]

The loss of muscle mass is a major risk resulting from malnutrition. Post-stroke muscle dysfunction becomes a problem, causing muscle atrophy and weakness.[12] Most strokes occur in patients over the age of 65, and sarcopenia is common in this age group. The onset of stroke in patients with already diminished muscle mass leads to worse outcomes, as it accelerates muscle atrophy and is associated with poorer clinical results.[13] In chronic stroke patients, muscle mass loss occurs simultaneously in both paretic and non-paretic limbs. Several studies

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have shown a link between muscle mass loss and various risk factors and comorbidities suffered by the patients. Additionally, various assessments using Bioelectrical Impedance Analysis (BIA) and other modern diagnostic methods have been conducted to detect muscle mass loss.[12] However, few studies have analyzed the extent to which these risk factors contribute to muscle mass loss in stroke patients. This study aims to identify the role of various factors in muscle mass reduction in stroke patients.

DESIGN AND METHODS

This study employs an observational design with a cross-sectional approach. It was conducted at Dr. Kariadi General Hospital from November 2023 to April 2024. The study passed the ethics review on August 18, 2023 (No.1521/EC/KEPK RSDK/2023) and was approved for research at Dr. Kariadi General Hospital, Semarang, on September 14, 2023 (No.DP.04.01/I.II/9135/2023).

The sample of this study consisted of stroke patients treated at the Stroke Unit, Dr. Kariadi General Hospital, Semarang. The sample size was 49 stroke patients who met the inclusion and exclusion criteria. The inclusion criteria were patients admitted to the hospital on the first day with a stroke diagnosis, treated at the Stroke Unit, Dr. Kariadi General Hospital, without autoimmune diseases, able to receive intake either orally or enterally without GI tract disturbances, and willing to participate in the study. The exclusion criteria included patients becoming agitated or deceased, experiencing reduced intake due to clinical deterioration during the study, or having incomplete or unevaluable body composition measurements. The sample collection technique was non-random sampling using purposive sampling.

Stroke patients and their families who met the inclusion criteria were given a detailed explanation of the study and signed an informed consent. The patients and their families were interviewed to gather the patient's identity and medical history. Then, body composition was measured using Bioelectrical Impedance Analysis (BIA) seca mBCA 525 model. The measurements were taken twice: on the first day and the fifth day. The body composition measurements used in this study were fat-free mass, fat mass, visceral fat, phase angle, and muscle mass. Meanwhile, the data on patient characteristics and history included gender, age, BMI, type of stroke, smoking, diabetes mellitus, dyslipidemia, hyperuricemia, heart, lung, and kidney conditions.

The data obtained will be presented using characteristic distribution tables, and the results of the Bioelectrical Impedance Analysis (BIA) measurements will be analyzed using the Paired T-test to determine differences in measurement changes. A normality test will first be conducted to assess data distribution, followed by a homogeneity test. Subsequently, a model will be developed using Partial Least Square (PLS) to assess the influence of each variable on muscle mass reduction.

RESULTS AND DISCUSSION

The distribution of characteristics in this study is presented in Table 1, based on gender, age, BMI, type of stroke, smoking, diabetes mellitus, dyslipidemia, hyperuricemia, heart, lung, and kidney conditions. The respondents of this study were 49 stroke patients admitted on the first day at Dr. Kariadi General Hospital, Semarang, Central Java.

Based on Table 1, the gender distribution shows that there are 32 male stroke patients (65.3%) and 17 female patients (34.7%). In terms of age groups, there are 24 adults (48,9%) and 25 late elderly (51,1%). Several studies indicate that men are estimated to be up to three times more likely to experience a stroke compared to women.[14] This is attributed to generally poorer lifestyle and dietary patterns in men compared to women.[15] However, other studies suggest the opposite, showing that women are at a higher risk of stroke due to their longer life expectancy.[16] Age is also a determining factor for the high incidence

Table 1. Distribution of Study Characteristics.

Characteristic		Frequency (n)	Percentage (%)
Gender	Male	32	65,30
	Female	17	34,70
Age	Adult (19-59 years old)	24	48,9
	Elderly (≥ 60 years old)	25	51,1
BMI (Body Mass Index)	Underweight (<18,5)	2	4,08
	Normal (18,5–22,9)	11	22,45
	Overweight (23–24,99)	11	22,45
	Obesity Grade I (25–29,99)	19	38,78
	Obesity Grade II (> 30)	6	12,24
Type of Stroke	Hemorrhagic	3	6,12
	Non-Hemorrhagic	46	93,88
Smoking	Yes	4	8,2
	No	45	91,8
Diabetes Mellitus	Yes	33	67,3
	No	16	32,7
Dyslipidemia	Yes	37	75,5
	No	12	24,5
Hyperuricemia	Yes	8	16,3
	No	41	83,7
Hypertension	Yes	44	89,8
	No	5	10,2
Kidney Disorders	Yes	21	43,8
	No	27	56,3
Lung Disease	Yes	16	32,7
	No	33	67,3

of stroke in women. This is due to the higher life expectancy, while estrogen levels in pre-menopausal women act as immunomodulators, helping to prevent strokes. Therefore, the combination of longer life expectancy and decreased estrogen levels after menopause increases the risk for women as they age.[17]

In this study, Body Mass Index (BMI) measurements are categorized as follows: underweight 2 patients (4.08%), normal 11 patients (22.45%), overweight 11 patients (22.45%), obesity grade I 19 patients (38.78%), and obesity grade II 6 patients (12.24%). The majority of stroke patients in this study are obese. The increased risk of stroke in obese patients is caused by excessive inflammation of adipose tissue, leading to disrupted blood flow and an increased risk of blockage.[18,19] The inflammatory response caused by obesity results in oxidative stress and triggers lipid peroxidation, which can lead to atherosclerosis. The hydrolysis of Free Fatty Acids (FFA) occurring in the liver and endothelial cells, along with elevated levels of free fatty acids in obese patients, increases the transport of FFAs to peripheral tissues and raises total body fat.[20]

he types of stroke in this study show hemorrhagic stroke at 3 patients (6.12%) and non-hemorrhagic stroke at 46 patients (93.88%). The number of patients experiencing non-hemorrhagic stroke is higher than those with hemorrhagic stroke. This is because non-hemorrhagic stroke is mostly caused by chronic diseases such as hypertension, diabetes mellitus, dyslipidemia, and smoking habits. In contrast, hemorrhagic strokes are primarily caused by aneurysms and have a high mortality rate in the acute phase.[21,22] This accounts for the higher number of non-hemorrhagic stroke patients in the inpatient setting.

Various risk factors that can lead to stroke include smoking, diabetes mellitus, and dyslipidemia. In this study, only 4 patients (8.2%) were found to be smokers. This is due to the fact that most of the study patients are elderly and have suffered from various diseases such as diabetes

mellitus, dyslipidemia, and gout. Several studies link smoking to stroke, as cigarettes contain over 7,000 chemicals that contribute to endothelial dysfunction, inflammation, and vascular function, potentially triggering atherosclerosis and increasing the risk of cardiovascular disease.[23] The formation of atherosclerosis, thrombosis, and decreased cerebral perfusion increases the risk of non-hemorrhagic stroke; however, smoking also raises the risk of Subarachnoid Hemorrhage (SAH) from aneurysm rupture and Intracerebral Hemorrhage (ICH) due to damage to arterial wall structure.[24]

In this study, 33 patients (67.3%) with stroke had diabetes mellitus. Diabetes mellitus has a direct relationship with the risk of ischemic stroke, which can increase up to threefold in those with diabetes mellitus for more than 10 years compared to those without diabetes mellitus. The relationship between diabetes mellitus and stroke is due to an increased risk of atherosclerotic lesions and endothelial dysfunction resulting from metabolic disturbances. Additionally, diabetic patients often experience microvascular complications and vascular fibrosis, which contribute to an increased risk of stroke.[25]

In this study, 37 stroke patients (75.5%) were found to have dyslipidemia. Dyslipidemia is a major risk factor for stroke, linked to coronary artery disease and peripheral vascular disease. Health issues arising from dyslipidemia are associated with elevated LDL levels in the blood, which trigger LDL oxidation. Normally, cholesterol entering the body is processed and stored in the liver; however, excess cholesterol can enter the bloodstream as LDL. The consequences of LDL oxidation include inflammation and damage to arterial blood vessels due to the phagocytosis process by macrophages and the formation of foam cells, leading to arteriosclerosis.[26]

Only 8 stroke patients (16.3%) were found to have gout. Some studies report that hyperuricemia are associated with cardiovascular risk factors such as increased triglyceride and cholesterol concentrations, hypertension, obesity, insulin resistance, and metabolic syndrome. However, uric acid is also known to have neuroprotective effects by acting as an antioxidant. Other research presents contrasting results, indicating that elevated uric acid levels do not have a direct relationship with increased stroke risk. Despite these differing views, both agree on the pro-oxidant and antioxidant activities of uric acid. As an antioxidant (<7,0 mg/dL), it can eliminate free radicals by up to 60% in the case of singlet oxygen radicals ($O^{\bullet}$), making it an important antioxidant in plasma.[27,28]

Forty-four stroke patients (89.8%) had a history of hypertension. A history of high blood pressure is the most common risk factor for stroke, as increased tension due to arterial wall thickening can lead to elevated blood pressure, arterial wall damage, and blockages in the brain's arteries.[29] The increased tension in the brain's blood vessels can weaken the vessel walls, leading to rupture and stroke. Hypertension raises peripheral blood pressure, causing poor hemodynamic status, thickening of blood vessels, and hypertrophy of cardiac muscle. This condition can be worsened by smoking habits and consumption of high-fat and high-salt foods, leading to atherosclerotic plaque formation. Prolonged high blood pressure can cause decreased elasticity in the muscle layer of cerebral blood vessels, resulting in fixed lumen diameter that cannot dilate or constrict normally. Consequently, if systemic blood pressure rises, the perfusion pressure in the capillary walls becomes elevated, triggering bleeding in the brain.[30]

Twenty-one stroke patients (43.8%) had kidney disorders. The brain and kidneys work together to maintain normal homeostasis in extracellular fluid by regulating sodium and water balance. Chronic Kidney Disease (CKD) is a common issue among ischemic stroke patients. In a multicenter hospital-based study, approximately 35% of first-time stroke patients exhibited symptoms of CKD, such as proteinuria or low eGFR.[31] Sixteen stroke patients (32.7%) had lung disease, which is a major cause of widespread neurological disability, including respiratory abnormalities. Stroke can affect breathing patterns, airway protection, and respiratory mechanisms. At the onset of stroke, the respiratory centers can be influenced by changes in consciousness, cerebral edema, and can lead to respiratory failure.[32]

Based on Table 2, the average for each group is shown. The average values on day 1 and day 5 indicated a decline in muscle mass, fat-free mass, visceral fat, and phase angle. In contrast, the fat mass group showed an increase in average values during the periodic measurements.

In this study, stroke patients experienced a decrease in muscle mass on average, although not significantly. The relationship between muscle mass and stroke incidence is crucial. Most strokes occur in elderly patients, where sarcopenia often develops at the onset of stroke. Low muscle mass at the beginning of a stroke is associated with poor clinical outcomes and increased mortality. Sarcopenia due to stroke affects 42% of stroke patients, resulting in muscle loss in both the affected and unaffected limbs.[33]

Based on Table 3, all groups were normally distributed except for the visceral fat group ($p \leq 0.05$). In the homogeneity test, all groups were found to be homogeneous, so the difference test for the visceral fat group used the Wilcoxon test. The results showed that the p-values for the visceral fat and phase angle groups were both below 0.05. This indicates that the decrease in visceral fat and phase angle can be considered significant.

Stroke patients will experience changes characterized by the loss of a significant portion of fat-free mass. Over time, the percentage of body fat will increase, while fat-free mass and muscle will undergo atrophy. Various factors contribute to the decline in fat-free mass, such as decreased physical activity during the stroke.[34] The inflammatory process and increased catabolism associated with stroke also accelerate the loss of fat-free mass. Pro-inflammatory cytokines, including interleukin 6 (IL-6), interleukin 1β (IL-1β), and tumor necrosis factor

Table 2. Average Differences in Body Composition Values in Stroke Patients.

Group		Mean ± SD	Maximum	Minimum
Muscle Mass	Day 1	20,90 ± 6,04	34,80	7,80
	Day 5	20,51± 6,43	31,00	4,70
Fat Free Mass	Day 1	46,46 ± 9,81	64,78	27,11
	Day 5	46,50 ± 10,99	74,45	25,90
Fat Mass	Day 1	18,17 ± 7,22	31,16	0,25
	Day 5	18,18 ± 7,54	34,47	2,24
Viceral Fat	Day 1	3,07 ± 1,51	6,80	0,70
	Day 5	2,98 ± 1,57	6,50	0,40
Phase Angel	Day 1	5,53 ± 1,26	8,20	2,40
	Day 5	5,02 ± 1,26	7,50	2,00

Tabel 3. Results of Body Composition Value Comparison Tests.

Group		Normality Test	Homogeneity Test	P value
Muscle Mass	Day 1	0,200	0,886	0,762
	Day 5	0,200		
Fat Free Mass	Day 1	0,200	0,614	0,983
	Day 5	0,200		
Fat Mass	Day 1	0,200	0,816	0,998
	Day 5	0,200		
Viceral Fat	Day 1	0,009	0,728	0,021
	Day 5	0,001		
Phase Angel	Day 1	0,170	0,989	0,049
	Day 5	0,200		

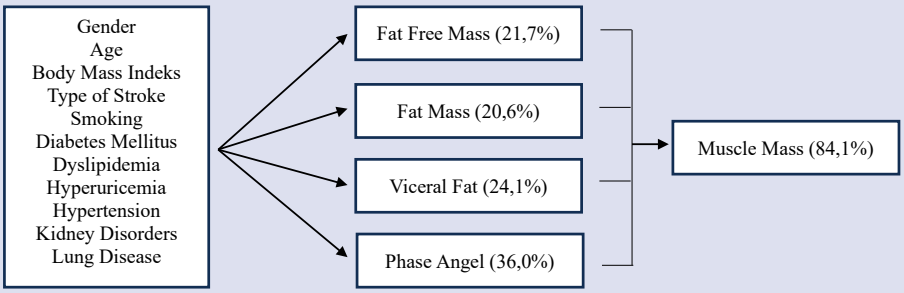

Figure 1. Structural Equation Model Analysis Results.

α (TNF-α), are released, triggering mechanisms that contribute to the pathogenesis of malnutrition. Pro-inflammatory cytokines also affect the brain's control of food intake, leading to delayed gastric emptying and increased skeletal muscle catabolism, which results in decreased food intake and weight loss.[35] Age-related factors further accelerate the decline in fat-free mass due to hormonal changes affecting muscle regeneration and protein synthesis.[36]

The increase in fat mass observed in this study was not significant. A study measuring muscle atrophy in stroke patients with weakness in the lower extremities showed declines in area, volume, and quality of muscle in the affected limbs, except for the gracilis muscle, accompanied by an increase in subcutaneous fat in the thigh. Increased intramuscular fat infiltration on the affected side is associated with insulin resistance and dyslipidemia, adversely affecting glucose homeostasis. Additionally, lack of physical activity contributes to increased fat due to systemic insulin sensitivity impairment, raising diabetes mellitus risk.[37]

In this study, stroke patients experienced a significant reduction in visceral fat. Visceral fat is the fat that surrounds abdominal organs, such as the liver, intestines, stomach, and other internal organs. Adipocytes store triglycerides in adipose tissue, which regulates lipid metabolism and glucose homeostasis.[38] Inflammatory processes in adipose tissue are characterized by the infiltration of activated M1 macrophages, leading to the production of reactive oxygen species (ROS) and the release of pro-inflammatory cytokines such as IL-6 and TNF-α.[39] Increased expression of IL-6 and leptin reduces adiponectin production and mediates the entry of macrophages into adipose tissue, triggering basal lipolysis.[40]

The significant decrease in phase angle observed in this study is noteworthy. Phase Angle is an assessment of muscle quality regarding cellular characteristics determined using Bioelectrical Impedance Analysis (BIA). A high Phase Angle indicates better cellular function, while a low Phase Angle signifies reduced cellular capability and integrity of the cell membrane. A decrease in Phase Angle typically occurs alongside diseases, inflammation, malnutrition, and physical inactivity. The Phase Angle value correlates with cellular health, muscle condition, and inflammatory responses, which can lead to a decline in the value. Additionally, loss of consciousness, cognitive impairments, and bed rest related to acute stroke can contribute to the decreased Phase Angle value.[41,42]

According to Figure 1, the distribution of characteristics affects fat-free mass by 21.7%, fat mass by 20.6%, visceral fat by 24.1%, and phase angle by 36.0%. Collectively, these factors impact muscle mass reduction by 84.1%. Stroke is primarily a disease affecting the elderly population, often accompanied by a decrease in muscle mass upon hospital admission, which tends to worsen as the disease progresses, contributing to poorer clinical outcomes.[43] Various strategies can be employed to prevent and mitigate muscle mass loss, including providing adequate nutritional intake tailored to the patient's needs and conditions at the onset of stroke. Additionally, ensuring adequate vitamin and mineral intake may involve supplementation.[44,45]

CONCLUSION

Average muscle mass reduction has occurred in stroke patients, although not significantly. However, this study demonstrates significant decreases in visceral fat and phase angle, indicating a risk of malnutrition in patients, both in terms of body composition evidenced by changes in fat mass, fat-free mass, and visceral fat, and in cellular condition evidenced by changes in phase angle. Moreover, various risk factors identified in this study have contributed to muscle mass reduction by up to 84.1%, while the remaining factors influencing this decline have not been examined in this study.

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CONFLICTS OF INTEREST

No conflicts of interest.

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REFERENCES

1. Tsao CW, Aday AW, Almarzooq ZI, Alonso A, Beaton AZ, et al. Heart Disease and Stroke Statistics-2022 Update: A Report From the American Heart Association. Circulation. 2022;145(8):e153-e639.
2. Capirossi C, Laiso A, Renieri L, Capasso F, Limbucci N. Epidemiology, organization, diagnosis and treatment of acute ischemic stroke. European Journal of Radiology Open. 2023;11:100527.
3. Venketasubramanian N, Yudiarto F, Tugasworo D. Stroke Burden and Stroke Services in Indonesia. *Cerebrovasc Dis Extra.* 2022;12 (1): 53–57.
4. Ramadhani AR, Ardhi MS, Prajitno S.Profile of characteristic, risk factor, and stroke severity on infarction stroke patients. *Malang Neurology Journal.* 2022.8:109-112.
5. Suryadinata RV, Wirjatmadi B, Adriani M, Lorensia A. Effect of age and weight on physical activity. *Journal of public health research.* 2020;9 (2):32-39
6. Yoon J, Baek S, Jang Y, Lee CH, Lee ES, et al. Malnutrition and Associated Factors in Acute and Subacute Stroke Patients with Dysphagia. Nutrients. 2023;15(17):3739.
7. Kim DY, Park HS, Park SW, Kim JH. The Impact of Dysphagia on Quality of Life in Stroke Patients. Medicine. 2020;99:e21795.

8. Ho LC, Wang HK, Chiu LT, Wang HH, Lee YC, et al. Protein energy wasting–based nutritional assessment predicts outcomes of acute ischemic stroke and solves the epidemiologic paradox. *Nutrition*. 2021;93:111431.
9. Ciancarelli I, Morone G, Iosa M, Cerasa A, Calabro RS, et al. Influence of Oxidative Stress and Inflammation on Nutritional Status and Neural Plasticity: New Perspectives on Post-Stroke Neurorehabilitative Outcome. *Nutrients*. 2022;15(1):108.
10. Suryadinata RV, Wirjatmadi B, Adriani M, Sumarmi S. The Expression Change of Mmp-8 and Collagen Type-2 Intracell in Lung Tissue Due to Electronic Smoke Exposure. *Kemas*. 2022;18(1):pp.31–40
11. Romani-Perez M, Bullich-Vilarrubias C, Lopez-Almela I, Liebana-Garcia R, Olivares M, at al. The Microbiota and the Gut–Brain Axis in Controlling Food Intake and Energy Homeostasis. *Int. J. Mol. Sci*. 2021;22:5830.
12. Landi F, Camprubi-Robles M, Bear DE, Cederholm T, Malafarina V, et al. Muscle loss: The new malnutrition challenge in clinical practice. *Clin Nutr*. 2019;38(5):2113-2120.
13. Hunnicutt JL, Gregory CM. Skeletal muscle changes following stroke: a systematic review and comparison to healthy individuals. *Top Stroke Rehabil*. 2017;24(6):463-471.
14. Hiraga A. Gender differences and stroke outcomes. *Neuroepidemiology*. 2017;48(1):61-62.
15. Abdu H, Seyoum G. Sex Differences in Stroke Risk Factors, Clinical Profiles, and In-Hospital Outcomes Among Stroke Patients Admitted to the Medical Ward of Dessie Comprehensive Specialized Hospital, Northeast Ethiopia. *Degener Neurol Neuromuscul Dis*. 2022;12:133-144.
16. Ospel J, Singh N, Ganesh A, Goyal M. Sex and Gender Differences in Stroke and Their Practical Implications in Acute Care. *Journal of Stroke*. 2023;25(1):16-25.
17. Zhong X, Sun Y, Lu Y, Xu L. Immunomodulatory role of estrogen in ischemic stroke: neuroinflammation and effect of sex. *Front. Immunol*. 2023;14:1-12.
18. Horn JW, Feng T, Morkedal B, Aune D, Strand LB, et al. Body Mass Index Measured Repeatedly over 42 Years as a Risk Factor for Ischemic Stroke: The HUNT Study. *Nutrients*. 2023;15(5):1232.
19. Kim NY, Choi YA. Obesity Impairs Functional Recovery of Older Stroke Patients with Possible Sarcopenia: A Retrospective Cohort Study. *J Clin Med*. 2023;12(11):3676.
20. Quinones-Ossa GA, Lobo C, Garcia-Ballestas E, Florez WA, Moscote-Salazar LR, et al. Obesity and Stroke: Does the Paradox Apply for Stroke?. *Neurointervention*. 2021;16(1):9-19.
21. Salvadori E, Papi G, Insalata G, Rinnoci V, Donnini I, et al. Comparison between Ischemic and Hemorrhagic Strokes in Functional Outcome at Discharge from an Intensive Rehabilitation Hospital. *Diagnostics (Basel)*. 2020;11(1):38.
22. Lee SE, Kim HJ, Ro YS. Epidemiology of stroke in emergency departments: a report from the National Emergency Department Information System (NEDIS) of Korea, 2018-2022. *Clin Exp Emerg Med*. 2023;10(S):S48-S54.
23. Suryadinata RV, Sukarno DA, Sardjono SC, Adriani M. Antioxidant activity in red mulberries on sperm development exposed by cigarette smoke. *Bali Medical Journal*. 2021;10(2):583-586
24. Luo J, Tang X, Li F, Wen H, Wang L, et al. Cigarette Smoking and Risk of Different Pathologic Types of Stroke: A Systematic Review and Dose-Response Meta-Analysis. *Front Neurol*. 2022;12:772373.
25. Mosenzon O, Cheng AY, Rabinstein AA, Sacco S. Diabetes and Stroke: What Are the Connections? J Stroke. 2023;25(1):26-38.
26. Chang Y, Eom S, Kim M, Song TJ. Medical Management of Dyslipidemia for Secondary Stroke Prevention: Narrative Review. Medicina (Kaunas). 2023;59(4):776.
27. Mehrpour M, Khuzan M, Najimi N, Motamed MR, Fereshtehnejad SM. Serum uric acid level in acute stroke patients. *Med J Islam Repub Iran*. 2012;26(2):66-72.
28. Padda J, Khalid K, Padda S, Boddeti NL, Malhi BS, et al. Hyperuricemia and Its Association With Ischemic Stroke. *Cureus*. 2021;13(9):e18172.
29. Ruksakulpiwat S, Zhou W, Phianhasin L, Benjasirisan C, Salehizadeh S, et al. Associations between diagnosis with stroke, comorbidities, and activity of daily living among older adults in the United States. *Chronic Dis Transl Med*. 2023;9(2):164-176.
30. Wajngarten M, Silva GS. Hypertension and Stroke: Update on Treatment. Eur Cardiol. 2019;14(2):111-115.
31. Zhao Q, Yan T, Chopp M, Venkat P, Chen J. Brain-kidney interaction: Renal dysfunction following ischemic stroke. *J Cereb Blood Flow Metab*. 2020;40(2):246-262.
32. Kim T, Chucalin A, Gusev E, Martynov M, Shogenova L, Panin A. The syndrome of respiratory failure in acute stroke patients. *European Respiratory Journal*. 2020;56(64):2009.
33. Honma K, Honda Y, Nagase M, Nakao Y, Harada T, et al. Impact of skeletal muscle mass on functional prognosis in acute stroke: A cohort study. *J Clin Neurosci*. 2023;112:43-47.
34. Przysada G, Czenczek-Lewandowska E, Wyszynska J, Weres A, Baran J, et al. Effect of Post-Stroke Rehabilitation on Body Mass Composition in Relation to Socio-Demographic and Clinical Factors. *Int J Environ Res Public Health*. 2020;17(14):5134.
35. Stumpf F, Keller B, Gressies C, Schuetz P. Inflammation and Nutrition: Friend or Foe?. *Nutrients*. 2023;15(5):1159.
36. Westerterp KR, Yamada Y, Sagayama H, Ainslie PN, Andersen LF, et al. Physical activity and fat-free mass during growth and in later life. *Am J Clin Nutr*. 2021;114(5):1583-1589.
37. Ryan AS, Buscemi A, Forrester L, Hafer-Macko CE, Ivey FM. Atrophy and intramuscular fat in specific muscles of the thigh: associated weakness and hyperinsulinemia in stroke survivors. *Neurorehabil Neural Repair*. 2011;25(9):865-72.
38. Chen Q, Zhang Z, Luo N, Qi Y. Elevated visceral adiposity index is associated with increased stroke prevalence and earlier age at first stroke onset: Based on a national cross-sectional study. *Front Endocrinol (Lausanne)*. 2023;13:1086936.
39. Suryadinata RV, Aditya DMN, Wiana VP, Rahman D. Brassica oleracea var. italica extract reducing free radicals and inflammation initiated by an exposure to cigarette smoke. *Healthcare in Low-Resource Settings*. 2023;11(S1):11210
40. Aparecida E, Vaseghi G, Carvalho AS, Kliemann N, Masoudkabir F, et al. Visceral Obesity and Its Shared Role in Cancer and Cardiovascular Disease: A Scoping Review of the Pathophysiology and Pharmacological Treatments. *Int J Mol Sci*. 2020;21(23):9042.
41. Sato Y, Yoshimura Y, Abe T. Phase Angle as an Indicator of Baseline Nutritional Status and Sarcopenia in Acute Stroke. *J Stroke Cerebrovasc Dis*. 2022;31(1):106220.
42. Abe T, Yoshimura Y, Imai R, Yoneoka Y, Tsubaki A, et al. Impact of Phase Angle on Physical Function in Patients with Acute Stroke. *J Stroke Cerebrovasc Dis*. 2021;30(9):105941.
43. Arsava EM, Gungor L, Sirin H, Sorgun MH, Aykac O, et al. Muscle Assessment in Stroke Study investigators. Muscle mass as a modifier of stress response in acute ischemic stroke patients. *Sci Rep*. 2024;14(1):10088.
44. Lathuiliere A, Mareschal J, Graf CE. How to Prevent Loss of Muscle Mass and Strength among Older People in Neuro-Rehabilitation?. *Nutrients*. 2019;11(4):881.
45. Manuel K, Ambroise MM, Ramdas A, Varghese RG, Vasanthi N, Ravichandran K. Clinical and diagnostic utility of platelet count and its parameters in COVID-19. *Indones J Med Sci Technol*. 2024; 6(1): 18-26.

GRAPHICAL ABSTRACT

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