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A Review of Muscle Tone Alteration on Post Stroke Patient

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ABSTRACT
Stroke is the second leading cause of death and the third disability in the world. Basic health research in Indonesia in the last 12 years shows that the prevalence of stroke is still high. A stroke occurs when a blood vessel in the brain is blocked or bursts, so that part of the brain does not get a blood supply and leads to cell death. A patient can avoid death but can still have the risk of experiencing a permanent or temporary disability that affects their productivity. One of the signs of disability in stroke patients is a change in muscle tone. The socioeconomic impact of stroke on a country is so enormous that it needs serious attention both in prevention and treatment. This review aimed to find out and review the development of research related to stroke, especially regarding risk factors, clinical manifestations, and muscle spasticity in post-stroke patients. Article writing was done by summarizing and analyzing the muscle tone of post-stroke patients. The sources of literature were from national and international journal providers by using the keywords "stroke risk factor, "stroke clinical feature", and "muscle tone related to post-stroke spasticity". This review concluded that there is an increase in muscle tone in post-stroke patients that causes muscle spasticity. This increased muscle tone can also be a sign of suboptimal therapy in stroke patients. It is hoped that there will be a wearable device developed to detect muscle tone that is concise and easy to use by the therapist or the patient's family.

Keywords: *Muscle Tone, Post Stroke, Patient, Spasticity*

1. INTRODUCTION

The first recorded use of "stroke", which was related to the Greek word "aplopexia", was in 1599 [1]. Stroke is characterized by a neurological disorder attributed to an acute injury of the central nervous system. "Stroke" is broadly used to include all the following conditions: CNS infarction, silent CNS infarction, intracerebral haemorrhage, silent cerebral haemorrhage, subarachnoid haemorrhage, cerebral venous thrombosis [2].

Stroke still becomes a health problem in both developed and developing countries [3]. There are currently more than 80 million people worldwide who have suffered a stroke, and there are more than 13.7 million new strokes each year [4,5]. Five and a half million people die from stroke each year, and each year more than 116 million years of healthy life are lost to deaths related to stroke and disability [4]. Basic health research in Indonesia in the last 12 years shows that the prevalence of stroke is still high [3].

The impact of stroke is extraordinary for the country. Stroke is the third leading cause of death in Indonesia, followed by diabetes mellitus and hypertension, with a mortality rate of 138,268 people or 9.7% of the total deaths. A patient can avoid death by stroke, but they still have the risk of experiencing a permanent or temporary disability that affects their productivity. Aside from mortality, stroke disability represents an economic burden on families and weighs on emotional and mental burdens that hinder the productivity of other family members. This creates a double burden in overcoming health problems in Indonesia. This is also related to the slow decline in the incidence of communicable diseases and tends to persist and the incidence of non-communicable diseases that progress quite rapidly [3,6]. The socioeconomic

impact of stroke on a country is so enormous that it needs serious attention both in prevention and treatment.

The risk factors of stroke are so many, as well as the mortality and disability caused by stroke. The duration, cost, and quality of treatment are considered to be the main contributing factors to the high mortality and disability caused by stroke. With higher stroke treatment costs than Malaysia, Indonesia can only reduce the mortality prevalence ratio to 0.16 and the stroke prevalence ratio to 2.80. This shows the need to improve the stroke management system in Indonesia [7]. One method to improve the treatment quality for spasticity is by measuring the muscle tone of post-stroke patients. But before we develop the tool, we need to dig into the stroke risk factor, stroke clinical feature, and especially muscle tone related to post-stroke spasticity.

2. METHOD

The purpose of this article is to find out and review the development of research related to stroke, especially regarding risk factors, clinical manifestations, and muscle spasticity in post-stroke patients. This paper also laid the groundwork for further research by the author, creating wearable technology for detecting muscle tone that is both concise and simple for the therapist and the patient's family to use. Article writing was done by summarizing and analyzing the articles that were related to stroke. The sources of literature were from national and international journal providers at Thieme, ScienceDirect, Pubmed, etc., by using the keywords "stroke risk factor," "stroke clinical feature," and " muscle tone related to post-stroke spasticity". The articles that fit the subject of the review were then reviewed by paraphrasing the core and common thread of the research. The details of the articles that have been revised and paraphrased were described in Table 1.

Table 1. Details of the search method results for articles with specific keywords

No.	Keyword	Total	Reference
1	Stroke risk factor	8	[5], [8], [9], [10], [11], [12], [13], [14]
2	Stroke clinical feature	2	[15], [16]
3	Muscle tone related to post-stroke spasticity	5	[17], [18], [19], [20], [21], [22]

3. RESULTS AND DISCUSSION

3.1. Stroke Risk Factor

Identifying risk factors for stroke is made difficult by the fact that stroke comes in many varieties. A stroke occurs when a blood vessel in the brain is blocked (ischemic) or bursts (hemorrhagic). The majority of strokes are ischemic, although the relative burden of hemorrhagic versus ischemic stroke varies between different population groups. Although there are some similarities in the risk factors for hemorrhagic and ischemic strokes, there are some significant differences. In general, stroke risk factors are divided into two groups, namely modifiable factors, genetic factors, and nonmodifiable factors. Genetic risk factors are increasingly recognized as potentially changeable, either directly or through alteration of gene-environment interactions. Therefore, genetics is put in an overlapping area between modifiable and nonmodifiable to highlight this reality [8].

Age, sex, and race-ethnicity are nonmodifiable risk variables (also known as risk indicators) for stroke. Stroke is, in general, an aging illness. The risk of stroke rises with age, with the risk doubling every decade beyond 55, but new data shows that the risk of ischemic stroke is growing in the 20 to 54-year-old age range. Women have the same or higher risk of stroke as men at early ages; however, the proportional risk is somewhat higher for males as they get older. The increased risk of stroke in younger women is most likely due to hazards associated with pregnancy and the postpartum period and other hormonal variables such as the use of hormonal contraceptives. Race, compared to their white counterparts, blacks have double the risk of incident stroke and have a greater death rate associated with stroke. In some cohorts, Hispanic/Latino Americans have an elevated risk of stroke [8].

Modifiable risk factors of stroke consist of several clusters. Physiological risk factors include hypertension, obesity, diabetes mellitus, dyslipidemia, and kidneys disfunction. The most important risk of the physiological factor for all stroke subtypes was hypertension, which was a more potent risk factor for intracerebral haemorrhagic stroke than ischemic stroke and was

more critical in those aged 45 and younger. Behaviour risk factors, including a diet high in sodium and red meat, a diet low in fruit and vegetable, alcohol consumption, tobacco smoking, and low physical activity. Environmental risk factors, including air pollutants ($PM_{2,5}$, $PM_{10,}$ O_3, NO_2, CO, and SO_2), metal pollutants (lead, cadmium, mercury), and second-hand smoke. The last one is psychosocial risk factors; however, the magnitude of the effect and whether it would be deemed a clinically meaningful impact remain unknown [5,8–14].

3.2. Stroke Clinical Feature

The neurological effects of a stroke vary depending on which part of the brain is damaged. Therefore, knowledge of cerebral vascular anatomy and the locations of different functions inside the brain are required for localization. The localization is divided into four clusters: anterior cerebral artery, middle cerebral artery, vertebrobasilar, and small-vessel occlusion [15,16]. The stroke clinical feature of each localization is described in Table 2.

Table 2. Stroke clinical feature on each localization

Localization	Clinical Feature
Anterior cerebral artery	Leg more than arm involvement with hand sparing Urinary incontinence Gait apraxia Akinetic mutism
Middle cerebral artery	Homonymous hemianopia/quadrantanopia (involvement of inferior division) Face-arm- leg involvement Aphasia (Broca's = superior division; Wernicke's = inferior division) Inattention Gaze paralysis (usually indicates a large area of frontal damage)
Vertebrobasilar	Occipital lobe - homonymous hemianopia, cortical blindness, other cortical visual deficits Cerebellum - ataxia, nystagmus Brainstem cranial nerve palsies - diplopia, facial numbness/ weakness, vertigo, dysphagia, dysphonia Spinal tracts - hemiparesis and hemisensory loss
Small Vessel Occlusion	Pure motor hemiparesis Pure sensory stroke Sensorimotor stroke Ataxic hemiparesis

3.3. Post-Stroke Spasticity

Stroke has many complications, including musculoskeletal, neurological, psychological, skin, etc. Spasticity is one of many neurological complications after stroke [17]. It is defined by a velocity-dependent increase in resistance during passive stretch as a result of the stretch reflex's hyperexcitability. Spasticity frequently causes immobility of a joint due to decreased muscle length, resulting in contracture [18].

Spasticity is common after stroke. The researcher estimates post-stroke spasticity prevalence was highly variable, ranging from 4% to 42.6%, with the prevalence of disabling spasticity ranging from 2% to 13% [19]. The onset of spasticity in the post-stroke era is very varied, with studies showing that spasticity develops and peaks at 1-3 months following a stroke. Although the neural components of spasticity peak three months after a stroke, the muscular components of spasticity may grow over time, leading to an increase in spasticity at six months post-stroke [20].

One of the upper motor neuron disorders that cause hypertonia is spasticity. Muscle tone abnormalities can be caused by any lesion or injury along the pyramidal tract or extrapyramidal fibers. The local activation of muscle spindles causes spasticity, but it requires the participation of the central nervous system to propagate and manifest. Damage to the upper motor neurons causes a breakdown in communication between the brain

and the spinal cord, resulting in net disinhibition of the spinal reflexes. When a patient's muscles are stretched passively, sensory input from muscle spindles is sent to the spinal cord via primary group Ia afferent fibers, and alpha-motoneurons are activated, resulting in a loss of supra-spinal inhibitory control and excessive muscular activation [20].

At a seven-year follow-up, one-third of ischemic stroke patients under 70 had increased muscular tone. Classic spasticity was also present in half of them. Increased muscle tone post-stroke was predicted by age, arm paresis, aphasia, and facial palsy at the time of the index stroke. This may indicate sub-optimal treatment, related to use, but also to intensity, duration, and frequency, of interventions such as physiotherapy, surgery, and pharmacological treatments [21].

Spasticity places a significant strain on patients and caregivers [22]. In addition, spasticity has a big impact on the patient's activities of daily living and health-related quality of life [23]. The good news is that spasticity can be measured in clinical and quantitative evaluation methods [24]. This assessment can be beneficial in monitoring post-stroke patients' muscle tone to avoid stiffness and assessing therapy efficacy.

4. CONCLUSION

Stroke is a neurological illness caused by central nervous system damage that occurs suddenly. There are both controllable and nonmodifiable risk factors for stroke. The clinical signs of a stroke are determined by where the central nervous system lesion occurs. Muscular spasticity, characterized by an increase in muscle tone, is one of the clinical signs that might develop. This rise in tone might potentially suggest that your treatment is not working. Based on the unpredictability of muscle spasticity's development and the potential of life-threatening, debilitating, and costly repercussions for patients and their families, it's critical to prevent and treat muscle spasticity as soon as feasible. Measuring the muscular tone of the stroke patient by the family or therapist is one approach to detecting spasticity. This article is expected to be the basis for developing a wearable tool to measure the patient's muscle tone.

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