Relationship between Blood Pressure and Stroke Rate at UKI Hospital

Abstract

A complicated stroke of hypertension is dreaded in the community. Hypertension is the most common in public and causes no symptoms. Hypertension is one of the risks of stroke. Hypertension can cause injury to the veins of one of the injured blood vessels in the brain because of carelessness, deafness, and narrowing or rupture of blood vessels in the brain and can result in the brain. Nerve damage in this brain causes a stroke. The writer to know the greatest risk factors for stroke patients in hospital UKI in 2019-2021.

***Keywords**: Hypertension, Stroke*

Introduction

Hypertension is a common disease in society. Often people are not aware of suffering from hypertension because they do not have specific symptoms. If not handled properly, there is a risk of death due to complications in several vital organs. The most frightening complication is stroke because of the sudden nature of the attack, which results in death and disability. [1] According to the WHO, nearly one billion people have high blood pressure (hypertension), two-thirds of whom are in developing countries. Hypertension kills nearly 8 million people worldwide and nearly 1.5 million yearly in Southeast Asia. Approximately one-third of the adult population in Southeast Asia has high blood pressure. Data from epidemiological studies show that hypertension is found in 50% -70% of stroke patients, and the fatality rate ranges from 20% -30% in many countries. Mortality will increase (an increase of 47%) in re-stroke. [2]

Based on the 2007 Basic Health Research (Riskedas), the prevalence of hypertension in Indonesia is very high, reaching 31.7% of the total adult population, and hypertension in Indonesia reaches 31.7% of the total adult population. High blood pressure is a risk factor for stroke, heart attack, heart failure, and arterial aneurysms and is a major cause of chronic heart failure. [3] A stroke is a sudden and severe injury to the brain's blood vessels. Blockages and narrowing or rupture of blood vessels can cause injuries. A stroke may or may not show symptoms (a stroke without symptoms is called a silent stroke) depending on the location and size of the damage. Stroke causes a variety of neurologic deficits depending on the location of the lesion (in which blood vessels are blocked), the size of the area of inadequate perfusion, and the amount of blood flow. Damaged brain function cannot fully recover. (Brunner & Suddarth, 2002). [4]

According to WHO (World Health Organization), in 2012, 51% of deaths from stroke worldwide were caused by high blood pressure. In addition, it is estimated that 16% of stroke deaths are due to high blood glucose levels. [2] Based on the results of the 2013 Riskesdas, the prevalence of stroke in Indonesia increases with age. The highest cases of stroke diagnosed by health workers were those aged 75 years and over (43.1%), and the lowest were in the age group 15-24 years, namely 0.2%. The prevalence of stroke based on gender is more male (7.1%) than female (6.8%). Based on place of residence, the prevalence of stroke in urban areas is higher (8.2%) than in rural areas (5.7%). [5] Based on this, researchers are interested in knowing patients who have had a stroke caused by high blood pressure at the UKI Hospital.

Literature Review

The cerebrovascular system provides the brain with a blood flow that contains many important nutrients for brain function. Cessation of cerebral blood flow or Cerebrum Blood Flow (CBF) for just a few seconds will cause symptoms of cerebral dysfunction. If continued for several seconds, CBF deficiency causes loss of consciousness and cerebral ischemia. Normal CBF is about 50 ml/100 grams of brain tissue/minute. At rest, the brain receives one-sixth of the cardiac output; from oxygen aspiration, the brain uses 20% of the body's oxygen. [6]

Four large arteries supply the brain: the two internal carotid arteries and the two vertebral arteries (which join the basilar artery to form the vertebrobasilar system). Arterial blood to the brain comes from the aortic arch. In general, the cerebral arteries are penetrating or conducting. The conducting arteries (carotid, middle and anterior cerebral, vertebral, basilar, and posterior cerebral) and their branches form an extensive network on the brain's surface. Generally, the carotid arteries and their branches supply the bulk of the 11 cerebral hemispheres, and the vertebral arteries supply the cerebellum and cerebellum. The penetrating arteries are vessels that channel the brain at right angles and supply blood to structures beneath the cortex (thalamus, hypothalamus, internal capsule, and basal ganglia). [7]

Collateral circulation can be established slowly when there is a decrease in normal blood flow to an area. Most of the cerebral collateral circulation between the great arteries is through the circle of Willis. The effect of this collateral circulation is to ensure the distribution of blood flow to the brain. These collaterals only function when other routes are disrupted [8]. The brain's gray matter has a much higher metabolic rate than the white matter, so the number of capillaries and blood flow is also four times greater. [9]

Hypertension is when a person experiences increased blood pressure above normal, resulting in illness or morbidity and mortality or mortality. Hypertension is when a person experiences increased blood pressure above normal or chronic for a long time. [10] According to WHO, hypertension is blood pressure above 160/95 mmHg expressed as hypertension. [11] "The Sixth Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure (1997)" defines persistent hypertension as having a systolic pressure above 140 mmHg and a diastolic pressure above 90 mmHg or more or being on medication anti-hypertension. [12]

The definition of hypertension does not change with age: systolic blood pressure (TDS) > 140 mmHg and/or diastolic blood pressure (TDD) > 90 mmHg. The joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC VI) and the WHO/International Society of Hypertension guidelines subcommittees agree that TDS & both are used for the classification of hypertension. [13] Stroke is a clinical sign that develops rapidly due to focal or global disturbance of brain function, with symptoms lasting for 24 hours or more or causing death, without any other obvious cause other than vascular. [14] Stroke is an acute disorder of nerve function caused by a disturbance in the circulation of the brain, where suddenly (within a few seconds) or quickly (within a few hours) symptoms and signs appear that are appropriate to the affected area and focal area. [15] Stroke is an acute focal or global functional brain disorder, lasting more than 24 hours, originating from a

disturbance in cerebral blood flow and not caused by a transient cerebral circulation disorder, brain tumor, or secondary stroke due to trauma or infection.

In America, it is estimated that 1 in 4 adults suffers from hypertension. If this disease is not controlled, it will attack target organs and cause heart attacks, strokes, kidney disorders, and blindness. Several studies have reported that uncontrolled hypertension can lead to a seven times greater chance of having a stroke, a six times greater chance of having congestive heart failure, and a three times greater chance of having a heart attack. [16] According to WHO and the International Society of Hypertension (ISH), 600 million people with hypertension worldwide, and 3 million of them die yearly. Seven out of every ten sufferers do not get adequate treatment. [17]

In Indonesia, the problem of hypertension tends to increase. The 2001 Household Health Survey (SKRT) results showed that 8.3% of the population suffered from hypertension, which increased to 27.5% in 2004. [18] Based on data from around the world, 15 million people have a stroke each year, of which one-third would die. In the following year, and third survive with a disability, and the remaining one-third recover to normal. Stroke cases are increasing in developed countries like the United States. Based on statistical data in America, every year, there are 750,000 new stroke cases in America. The data shows that every 45 minutes, one person in America has a stroke. And in 2020, an estimated 7.6 million people will die from a stroke. [19]

In Indonesia, stroke is the number three killer. Based on Basic Health Research (Riskesdas) data in 2007, at the age of 45-54 years, the mortality rate due to stroke was 15.9% (in urban areas) and 11.5% (in rural areas). Of the total number of stroke sufferers in Indonesia, around 2.5 percent, or 250 thousand people, died, and the rest had mild or severe disabilities. [20] The increase in stroke rates in Indonesia is associated with increased incidence of stroke risk factors. The risk factors for stroke are diabetes mellitus, mental health disorders, smoking, obesity, and hypertension. Hypertension is a problem often encountered in stroke patients and persists after a stroke. A study conducted by Lamassa et al. in 4.462 stroke patients showed that hypertension was found in 48.6% of cases. Based on the WHO fact sheet in 2005, epidemiological studies show that hypertension is found in 50% -70% of stroke patients, and the fatality rate is around 20% -30% in many countries. In repeated strokes, mortality will increase to 47%. [20]

According to The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC VII), hypertension in adults can be classified into normal, prehypertension, grade I, and II hypertension groups. (table 1.) [21]

Table 1. Classification of blood pressure according to JNC VII

Classification of Blood Pressure	Systolic Blood Pressure (mmHg)	Diastolic Blood Pressure (mmHg)
Normal	<120	<80
Prehypertension	120-139	80-89
Grade I hypertension	140-159	90-99
Grade II hypertension	≥ 160	≥ 100

In Indonesia, based on the consensus produced by the First National Scientific Meeting of the Indonesian Hypertension Association on 13-14 January 2007, we have not been able to classify hypertension for Indonesians. Because national-scale research

data on hypertension in Indonesia is rare, the Indonesian Nephrology Association (Pernefri) chose the WHO/ISH classification because it has a wider distribution. [23]

Table 2. Classification of blood pressure according to WHO/ISH

Classification of Blood Pressure	Systolic Blood Pressure (mmHg)	Diastolic Blood Pressure (mmHg)
Severe hypertension	≥ 180	≥ 110
Moderate hypertension	160-179	100-109
Mild hypertension	140-159	90-99
Borderline hypertension	140-149	90-94
Borderline systolic hypertension	140-149	< 90
Isolated systolic hypertension	> 140	< 90
Normotensive	< 140	< 90
Optimal	< 120	< 80

Strokes can be classified based on several criteria. These classifications include; based on anatomical pathology and its causes (Ischemic Stroke and Hemorrhagic Stroke) based on stage or time considerations (Current Ischemic Attack, Reversible Ischemic Neurologic Deficit, Progressing Stroke, Stroke In Evolution, and Complete Stroke); and based on the vascular system (ischemic stroke and hemorrhagic stroke). [23] The risk factors for stroke are divided into two categories: risk factors that cannot be modified (age, sex, heredity, race/ethnicity) and factors that can be modified (hypertension, heart disease, diabetes mellitus, hypercholesterolemia, obesity, and smoking).

When viewed in simple terms, blood pressure is determined by two important factors: cardiac output and peripheral resistance. When a person experiences psychological stress, it will stimulate the sympathetic nerves to release NE (norepinephrine). The release of NE itself will cause a ligand-receptor reaction, in which NE as a ligand can attach to receptors in blood vessels (α1), kidneys (β1), and heart. In blood vessels (α1), a vasoconstriction reaction will occur so that the endothelium in the blood vessels closes and causes peripheral resistance to increase, and blood pressure increases automatically. It causes hypertension; if these endothelial cells continue to be exposed to continuous high blood pressure, it will cause endothelial cells to become dysfunctional; NO (nitrite oxide), which is normally produced by endothelial cells, becomes reduced so that endothelial cells cannot relax, and vasoconstriction will continue. Its permeability becomes reduced so that it can cause atherosclerosis over time.

In the heart, the presence of NE will increase the heart rate and contraction of the heart, which can increase cardiac output (COP); this COP itself will cause peripheral resistance in the blood vessels as well as when the NE event binds to receptors (α1) in the blood vessels, so it can also lead to atherosclerosis. At its receptors in the kidney, NE will cause the activation of renin secretion to increase, and we know that renin will stimulate the conversion of angiotensin to angiotensin I, angiotensin I will turn into angiotensin II which has a vasoconstrictive effect on blood vessels, in the Posterior Pituitary it will stimulate aldosterone release which acts as retention, water, and sodium. The result of water and Na retention will increase blood volume, ultimately affecting the increased venous return and CO.

The research results show the availability of NO. Another opinion states that essential hypertension is associated with changes in endothelial function and

morphology, causing an increase in cell volume so that the endothelium bulges into the lumen. The interaction between the endothelium, platelets, and monocytes in hypertensive vessels increases. Another opinion about the mechanism of NO damage is the production of oxidative stress. Oxidative stress in the form of ROS (Reactive Oxygen Species), especially the superoxide anion, can combine and destroy peroxynitrite, which produces NO, negatively affecting the structure and function of blood vessels.

Arteriosclerosis is a group of blood vessel disorders characterized by thickening and loss of elasticity of the arteries. There are three types of arteriosclerosis: a) atherosclerosis, b) Monckeberg's medial calcific sclerosis, and c) arteriosclerosis. Under normal circumstances, the endothelium blocks the penetration of large molecules such as low and very low-density lipoproteins (LDL, VLDL) into the intima, whereas higher-density lipoproteins with smaller molecules can move freely in and out of the intima. Endothelial cells also produce prostacyclin (PG12) and nitric oxide, which can prevent platelet accumulation in vessels. Increased endothelial permeability is the first complaint due to arterial injury, a non-specific response caused by viruses, toxins, immune complexes, products released by activated white blood cells or platelets, and uncontrolled physical stress. In general, this can also be caused by an increase in the concentration of lipoproteins in the blood. When lipoproteins enter the intima due to increased capillary permeability, the main protein compounds from LDL and VLDL (apolipoprotein B) are accompanied by plychosominoglycans, especially dermatan sulfate so that lipoproteins accumulate in the intima.

Blood flow to the brain (ADO) is the amount of blood going to the brain. The adult brain uses 20% of the blood pumped by the heart at rest, and blood normally fills 10% of the intracranial space. ADO strictly regulates the brain's metabolic demands, averaging ADO flow is maintained at 50 ml per 100 grams of brain tissue per minute in the adult human. Maintaining ADO within normal limits is vital because too much ADO can increase intracranial pressure and compress and damage brain tissue, whereas too little ADO will result in an inadequate blood supply. Ischemic will occur if blood flow to the brain is below 18-20 ml per 100 grams of brain tissue per minute, and brain tissue death occurs if ADO falls below 8-10 ml per 100 grams of brain tissue per minute. Within the brain tissue, there is a biochemical cascade, or what is known as the ischemic cascade, which causes the brain tissue to become ischemic, which further causes damage and death of brain cells. ADO is determined by several factors, such as blood viscosity, the ability of blood vessels to dilate, and cerebral perfusion pressure, which is determined by blood pressure and intracranial pressure. Cerebral blood vessels can change blood flow by changing the diameter of the blood vessel lumen; this process is called autoregulation when blood pressure decreases.

Solid hypertension causes pathological changes in the brain's medium and small blood vessels. Based on this, strokes caused by hypertension can be divided into groups with different pathological and clinical features. In medium blood vessels, such as a. Carotid, a. Vertebrobasilar or arteries at the base of the brain, the pathological changes are in the form of atherosclerosis, and the clinical manifestations are in addition to other factors such as diabetes mellitus, hyperlipidemia, smoking, and others. The brain's small blood vessels, namely the branches of the arteries penetrating the brain tissue, are 50-200 microns in diameter. The basis of abnormalities in vessels of this type are spasms and lipohyalinosis; Spasms occur in acute hypertension, such as malignant hypertension, and the clinical manifestations are lacunar infarctions. Lipohianilosis also

occurs in chronic hypertension, blood vessels with lipohyalinosis can experience microaneurysms that can rupture, and intracerebral hemorrhage occurs. In contrast to atherosclerosis, in lipohyalinosis, hypertension can be said to be the sole causative factor.

There are various clinical symptoms of stroke, including intracerebral hemorrhage (ICH), which can be distinguished clinically from ischemic stroke; hypertension is usually found, and an altered level of consciousness or coma is more common in hemorrhagic stroke than ischemic stroke. Often, this is due to increased intracranial pressure. Meningismus can result from the presence of blood in the ventricles. 20 Focal neurological deficit. The type of deficit depends on the area of the brain involved. If the dominant (usually left) hemisphere is involved, a syndrome consisting of right hemiparesis, right chemosensory loss, preferential gaze abandonment, truncated right visual field, and aphasia may occur. If the dominant (usually right) hemisphere is involved, a left hemiparesis syndrome, left hemisensory loss, right gaze preference, and left visual field cut. Nondominant hemisphere syndrome can also result in neglect and inattention to the left side. [24]

If the cerebellum is involved, the patient is at high risk for herniation and brainstem compression. Herniations can cause a rapid decline in consciousness, apnea, and death. Other signs of cerebellar or brainstem involvement include extremity ataxia, vertigo or tinnitus, nausea and vomiting, hemiparesis or quariparesis, hemisensory or sensory loss of all four limbs, eye movements resulting in abnormal diplopia or nystagmus, oropharyngeal weakness or dysphagia, and the face ipsilateral and contralateral to the body. [25]

An intracerebral hemorrhage starts suddenly. In about half of sufferers, attacks begin with a severe headache, often with exertion. However, in the elderly, headaches may be mild or absent. Symptoms of cerebral dysfunction describe the progression of progressively worsening bleeding. Some symptoms, such as weakness, paralysis, loss of sensation, and numbness, often affect only one side of the body. The person may be unable to speak or be confused. Vision can be disturbed or lost. The eyes may point in different directions or become paralyzed. Nausea, vomiting, convulsions, and loss of consciousness are common and can occur within seconds to minutes. [26]

Before it ruptures, an aneurysm usually causes no symptoms unless it presses on a nerve or leaks a small amount of blood, usually before a large rupture (which causes a headache), producing the following warning signs: a) Headache, which may be unusually severe suddenly sudden and severe (sometimes called lightning headache; b) pain in the eye or facial area; c) Double vision; and d) Loss of peripheral vision.

Research Method

The research conducted was a descriptive epidemiological study with a cross-sectional design. The location for data collection was carried out at the UKI General Hospital, Cawang, East Jakarta, and the time for data collection was carried out in November 2021 - January 2022. The target population of the study was stroke patients in Indonesia. The reachable population is stroke patients at UKI General Hospital, Cawang, East Jakarta. The research subjects were stroke patients treated at UKI General Hospital, Cawang, East Jakarta, who came from 2019 -2021. The research sample is the research subject according to the research criteria. The sample size in this study was determined based on the number of stroke patients treated at the UKI General Hospital, Cawang, East Jakarta, for the period 2019 – 2021. Data collection was carried out using

secondary data obtained from the medical records of stroke patients treated at the internal medicine department of the UKI General Hospital. , Cawang, East Jakarta, for 2019 - 2021, and then recorded according to the required variables. The data used is secondary data taken from medical records. Previously, researchers asked permission to access medical records. Then the researcher will look for data from medical records and write them into a worksheet. The data collected is entered and then processed using a distribution table. This study follows the rules of applicable research ethics by keeping the identity of the existing respondents confidential. Documents regarding identity and data related to the study of stroke patient profiles at the UKI General Hospital, Cawang, East Jakarta.

Result and Discussion

In this chapter, the author will present research on the medical record installation at the UKI General Hospital, Cawang, East Jakarta. Medical record data were collected from 28 November 2021 to 29 January 2022 regarding "The Relationship between Blood Pressure and Stroke Incidence Rates at UKI Hospital in 2019-2021". Data was collected by taking notes from medical records (secondary data). The method used in data collection was the Random Method, where the researcher took data from medical records that met the inclusion criteria until the desired amount of data was met. From the study results, the number of patient medical record data was 70 patients. The following research results are presented in tabular form.

Table 3. Distribution of stroke patients by age

Age group	Frequency	Percentage (%)
34-50 years	20	28,6
51-66 years	37	52,9
67-83	13	18,6
Total	70	100,0

Based on Table 3 regarding the age group, it was found that the age group 51-66 was the most compared to other age groups, namely, with 37 patients (52.9%).

Table 4. Distribution of stroke patients by Gender

Gender	Frequency	Percentage (%)
Male	43	61,4
Female	27	38,6
Total	70	100,0

Table 4 shows an overview of gender; most results were male, with 43 patients (61.4%).

Table 5. Distribution of Stroke patients based on Diagnosis

Diagnosis	Frequency	Percentage (%)
Non-Hemorrhagic Stroke	47	67,1
Hemorrhagic Stroke	15	21,4
Recurrent Non-Hemorrhagic Stroke	3	4,3
Recurrent Hemorrhagic Stroke	1	1,4
Basilar Stroke	4	5,7

Total	70	100,0

Table 5 shows that most diagnoses were non-hemorrhagic strokes, with 47 patients (67.1%) at the UKI Hospital.

Table 6. Distribution of stroke patients based on diagnosis

Past medical history	Hemorrhagic Stroke (Frequency)	(%)	Non Hemorrhagic Stroke (Frequency)	(%)
Diabetes Mellitus	1	1,4	7	10
Hypertension	9	12,8	37	52,8
Heart	0	0	2	2,8
None	2	2,8	8	11,7

Table 6 shows that the most common result in past medical history for Hemorrhagic Stroke was hypertension with nine patients (12.8%) at the UKI Hospital, and the highest result for Non-Hemorrhagic Stroke was Hypertension with 37 patients (52.8%).

Based on medical record data obtained from the UKI Hospital in 2019-2021, it was found that the ages of 51-66 years were the most affected by stroke, with 37 patients (52.9%) of the 70 patients suffering from stroke. This study follows the results of Lestari's study which found that a percentage of the age group >50 years suffers from stroke more than the age group <50 years [27]. Another related study is research which states that the risk of stroke in the age group >55 years is 3,640 times that in the age group <55 years [28]. So it can be concluded that there is a relationship between age and the incidence of stroke. The increase in stroke frequency with age is related to the aging process, in which all body organs experience a decline in function, including the brain's blood vessels. Blood vessels become inelastic, especially in the endothelial part, which is thickened in the intima, resulting in narrower lumens of blood vessels and an impact on decreasing cerebral blood flow [29].

The results of a study of 70 samples found that the incidence of stroke was more male, namely 43 patients (61.4%), compared to the female sex, namely 27 patients (38.6%). So it can be concluded that there is a relationship between gender and the incidence of stroke. This study's results align with Puspita and Putro's research which found that gender had a significant relationship with the risk of stroke, with the risk of the male gender being 4.375 times compared to females [30].

Conclusion

Knowing the risk factors for stroke is very important in preventing stroke, which is one of the efforts that have a significant role in treating stroke, in reducing the risk of stroke, namely by controlling stroke risk factors. The main factor of Stroke is Hypertension, but this factor can be modified. Thus, it is hoped that people will be more aware of the importance of living a healthy life to reduce stroke risk factors. Exercise, consuming low-sugar foods, quitting smoking, consuming low-salt foods, and controlling blood pressure regularly is one way that can be done to reduce stroke risk factors.

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