Brain-Stroke – Role of Blood Pressure

Kulwinder Kaur[1], Dheeraj Jamwal[2], Dr Chandan Kumar Jha[3]
[1] *M.Sc., Department of Human Genetics, Punjabi University Patiala*
[2,3] *Ph.D.; Department of Human Genetics, Punjabi University Patiala*

***Abstract*- Stroke is a very diverse disorder that affects people of all ages. Adult ischemic stroke is primarily a condition of comorbidities. The increasing knowledge on stroke with the understanding of genetic and environmental factors and daily lifestyles help in managing patients with an acute ischemic stroke. Higher baseline blood pressure has repeatedly been shown to increase the risk of stroke. In comparison to average systolic blood pressure, higher systolic blood pressure variability (SBPV) has been found to be a more accurate predictor of all-cause and cardiovascular mortality. This paper represents a thorough study to understand the role of blood pressure, whether chronic or a prolonged one in inducing a brain stroke.**

Keywords : **Stroke, Blood pressure and Hypertension.**

INTRODUCTION

The most prevalent chronic medical condition that can be found in a range of groups is high blood pressure (BP)[1]. Higher baseline blood pressure has repeatedly been shown to increase the risk of end-stage renal disease (ESRD), cardiovascular morbidity and death, and all-cause mortality, whether it is untreated or treated[2-8]. Distinctively, a significant meta-analysis from the Prospective Study Group showed that an elevated risk of mortality from coronary heart disease (CHD), stroke, and other vascular causes was substantially and linearly associated to the mean or "usual" BP, corrected for regression dilution[9,15]. Studies conducted by epidemiologists have linked high blood pressure to an increased risk of cerebral vascular disease, including both cerebral hemorrhage and cerebral infarction[10]. Additionally, in individuals with severe hypertension, lowering blood pressure through pharmacological intervention reduces the risk of future strokes in both those without cerebrovascular symptoms[11] and those who have already had a stroke[12]. This strongly implies that blood vessel damage and strokes are directly related to elevated blood pressure[13]. Pathologists have been looking for an underlying pathological mechanism that can explain the increased susceptibility of cerebral arteries to rupture and occlusion for a long time. It is commonly accepted that as compared to patients with normotension, the walls of the major cerebral arteries from chronic hypertension patients exhibit greater thickness and hyalinization of the muscular coat. Additionally, in big arteries, atheroma is more severe and widespread.[14,15] In comparison to average systolic blood pressure, higher systolic blood pressure variability (SBPV) has been found to be a more accurate predictor of all-cause and cardiovascular mortality[16-18], stroke[19-20], and cardiac illness[21-24]. Comorbidity is a defining feature of stroke that worsen prognosis while also raising the incidence of stroke. The most significant modifiable risk factor for stroke is hypertension, which affects a large portion of the stroke population. Increased shear stress, endothelial dysfunction, and stiffness of the major arteries, which carry pulsatile flow to the cerebral microcirculation, are all factors that contribute to hypertensive diseases' promotion of stroke. Through a number of factors, including hypoperfusion, reduced autoregulatory function, and locally increased blood-brain barrier permeability, hypertension also encourages cerebral small vessel disease.[25]

Stroke is a very diverse disorder that affects people of all ages. Adult ischemic stroke is primarily a condition of comorbidities (such as hypertension, diabetes mellitus, hyperlipidemia), and/or altered coagulation states, as opposed to pediatric stroke (e.g. pregnancy and preeclampsia). Stroke is currently the fifth greatest cause of death in the United States and the top cause of long-term disability.[26-29] The intensive treatment of hypertension and dyslipidemia, both of which are prevalent in the stroke population, can be partly blamed for the decrease in stroke mortality over the past ten years.[28,30] Actually, the most significant modifiable risk factor for all forms of stroke is

hypertension. Systolic and diastolic blood pressure both increase the incidence of stroke proportionally, which raises the relative risk 3.1 times for men and 2.9 times for women.[31,32] Through inexplicable reasons, blood pressure also elevates during the acute phase of a stroke, adding fuel to the clinical debate about whether and how much treatment is necessary. Stroke outcomes are also worsened by hypertension. Although decreased blood pressure is sometimes harmful in stroke, patients with pre-existing hypertension had less salvageable tissue (penumbra) and greater infarctions than normotensive patients. Additionally significant is the risk of stroke in the presence of hypertension. The main cause of cerebral small vessel disease (CSVD), which results in cognitive impairment and lacunar stroke, is hypertension. Additionally, recent research has revealed that the long-term risk of stroke is 4-5 times higher in women who have already experienced preeclampsia, a common hypertension disease of pregnancy. Therefore, the effect of hypertension on cerebral circulation is crucial to the field of stroke research.[25]

This paper represents a thorough study to understand the role of blood pressure, whether chronic or a prolonged one in inducing a brain stroke.

PATHOPHYSIOLOGY OF STROKE

Atherosclerosis is a degenerative disease and is the underlying process responsible for stroke primarily affecting medium and large sized arteries[33] (Crowther, 2005). Atherosclerosis can be described as a vascular inflammatory disease caused by disturbed vascular homeostasis resulting in the formation of atherosclerotic lesions[34] (Schafer and Bauersachs, 2008). There are three main theories hypothesized to be the triggerer for stroke. These are - the response to injury theory, the response to retention theory and the oxidation theory. Under the first theory the process is initiated in response to the endothelium becoming damaged, for example by diabetes, smoking, raised blood pressure, injury or infection resulting in an inflammatory response[35,36] (Mallika *et al.*, 2007, Pongnimitprasert 2009). The second theory is that the atherosclerosis process arises as a response to accumulation of extracellular matrix molecules and lipoproteins[36] (Pongnimitprasert, 2009). Finally, the oxidative theory proposes that the process begins with the oxidation of low-density lipoprotein (LDL) through free radicals; the oxidation results in the attachment of monocytes to the vessel wall and also damages to the endothelium, resulting in endothelial dysfunction[35,36] (Mallika *et al.*, 2007, Pongnimitprasert 2009).

PREVALENCE

Over 60% of patients presenting with stroke to the ED had high blood pressure in a nationally representative big data set of the US.[37] In 1972, a 92 percent participation rate was used to examine a representative sample of the population aged 35 to 59 in two counties of Eastern Finland. Standardized epidemiological measurements on individuals (both sexes) were taken for height, weight, serum lipids, blood pressure, and questionnaire data on smoking. Through the use of national hospital discharge and death certificate registers, the cohort was monitored for seven years. 77 men and 65 women experienced a brain stroke during the follow-up. Age, smoking, blood pressure, history of a previous stroke, and diabetes were found to be independent predictors of cerebral infarction and other strokes in males based on multiple logistic risk function analysis. Men with no history of stroke had a 1.9-fold (90% CI = 1.1-3.6) increased risk of cerebral infarction, while women with no history of stroke had a 2.5-fold (90% CI = 1.1-5.6) increased risk when their diastolic blood pressure was 100 mm Hg or above. In men, systolic blood pressure of 150 mm Hg or higher was responsible for 37% (90% CI = 21-53%) and diastolic blood pressure of 95 mm Hg or higher for 27% (90% CI = 11-42%) of all cerebral strokes. [38] In low- and middle-income nations, including India, the incidence of stroke increased by more than 100% between 1970-1979 and 2000-2008.Throughout the country over the past ten years, the cumulative incidence of stroke varied from 105 to 152/100,000 people annually, and the gross prevalence of stroke varied from 44.29 to 559/100,000 people.[39]

COMPARATIVE STUDY

A poor outcome is associated with arterial hypertension, which affects 80% of patients with acute stroke[40,41]. The pathophysiology is extremely complicated, and a number of underlying mechanisms might be in play, including pre-existing hypertension

[40,42], activation of the renin-angiotensin-aldosterone axis[39], and psychological stress induced by being in the emergency room and being admitted to the hospital[42]. Autonomic dysfunction may also be involved. Urinary catecholamine levels and blood pressure changes in AIS were found to be correlated in one study[43]. In acute stroke patients, BRS had a negative correlation with acute hypertension at admission, hypertensive episodes, and blood pressure variability (BPV) within the first 72 hours[44,45].

Three main factors, including vascular structure, vascular function, and blood pressure, influence arterial stiffness. The cyclic strain on the arterial wall, primarily the cyclical variation in blood pressure, determines arterial stiffness. When blood pressure is low, elastin regulates composite behaviour, and the vessel wall is still somewhat extensible. However, when blood pressure is high, collagen's stiffer properties become more significant, and the vessel wall eventually becomes in-extensible.[46,47] As a result, even without structural change, arterial stiffness rises at increased blood pressure which is a cause observed in several cases of stroke and heart attack.[48]

Two thirds to three quarters of patients with acute ischemic stroke experience elevated blood pressure.[49,50] Early hypertension that develops after an ischemic stroke frequently reflects both a neuroendocrine response to physiological stress and undiagnosed or undertreated hypertension. [51] In the first 24 hours following the onset of a stroke, blood pressure drops on its own in a lot of individuals.[52] In low- and middle-income nations, 70% of strokes and 87% of stroke-related deaths and disability-adjusted life years take place globally.[53-55] In low- and middle-income nations, the incidence of stroke has more than doubled during the previous 40 years. In high-income nations during the past four decades, stroke incidence has decreased by 42%.[53]

High blood pressure (BP) is associated with worse clinical outcomes in the setting of acute ischemic stroke, but the optimal blood pressure target is still a matter of debate.[56]

Although it is well known that hypertension is the primary risk factor for stroke, most people are unaware of the intricacy of the cerebrovascular issues linked to hypertension. There are numerous ways in which hypertension can lead to stroke.

Intracerebral arteries' endothelium and smooth muscle function will be significantly altered by a high intraluminal pressure. Increased endothelial stress can lead to localized or multi-focal brain oedema as well as increased permeability across the blood-brain barrier. Localized thrombi development and ischemic lesions can be brought on by endothelial injury and altered blood cell-endothelium interactions. Focal stenosis and occlusions brought on by fibrinoid necrosis can result in lacunar infarcts. Intracerebral haemorrhages are predisposed by deteriorating endothelium and smooth muscle cells. Additionally, as hypertension quickens the arteriosclerotic process, there is a higher chance that cerebral lesions caused by stenosis and embolism from the aortic arch, the heart, or other big extracranial veins would develop. Although adaptive structural changes in resistance vessels reduce artery wall tension, they also raise peripheral vascular resistance, which could limit collateral circulation and increase the risk for ischemic events in conjunction with hypotensive episodes or distal to a stenosis.[57]

CONCLUSION

Initiated in 1948 with the goal of determining probable and treatable causes of cardiovascular illnesses, the Framingham Heart Study is the longest-running prospective epidemiology project. After six decades of research, the project, which was initially intended to examine the causes of coronary heart disease in men under the age of 60, has produced important insights into the impact of many factors on the risk of stroke and cognitive impairment. According to the first cohort of subject results, depending on age and gender, hypertension individuals (with BP > 160/95 mm Hg) had a five to 30 times higher incidence of stroke than normotensive people (140/90 mm Hg). As-called "borderline hypertensives" were likewise at an increased risk.[58] However, when blood pressure is high, collagen's stiffer properties become more significant, and the vessel wall eventually becomes in-extensible. As a result, even without structural change, arterial stiffness rises at increased blood pressure which is a cause observed in several cases of stroke and heart attack.

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