Stroke and Hypertension

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Stroke is defined by WHO (World Health Organization) as rapidly developing clinical symptoms and signs of focal, and at times global, loss of cerebral function lasting more than 24 hours or leading to death, with no apparent cause other than that of vascular origin.[1] This definition includes patients presenting with clinical signs and symptoms suggestive of subarachnoid hemorrhage, intracerebral hemorrhage, and cerebral ischemic or embolic infarction. However, with new imaging technology, it is realized that some lacunar infarction can fully recover within less than 24 hours.

Incidence and prevalence of stroke

Stroke is the third most common cause of death in most developed countries.[1] It is a worldwide problem. Data show that the incidence of stroke varies according to age, sex and race.[2] Stroke can occur at any age, but half of all strokes occur in the elderly people over 70 years old.[1] Several studies obtained the same evidence that men had stroke more than women. In the USA, black people have risk of stroke more than white people.[3] Furthermore, stroke is a high incident health problem among Chinese people.[4]

WHO MONICA Project, in 9 European countries and 2 Asian countries, revealed that age-standardized stroke incidence rates per 100,000 varied from 101 to 285 in men and from 47 to 198 in women.[5]

The community survey in Singapore 2005 found a crude stroke prevalence rate of 4.05% (95% CI, 3.75 to 4.38) and WHO world population age-gender-standardized rate of 3.65% (95% CI, 3.36 to 3.96).[6] The prevalence rates rose with age ($P<0.001$ for trend) and were higher among men compared to women, 4.53% (95% CI, 4.05 to 5.07) versus 2.91% (95% CI, 2.57 to 3.29). Crude stroke prevalence and annual incidence rates in South America from a systematic review of community studies 2003 were 1.74 to 6.51 per 1,000 and 0.35 to 1.83 per 1,000, respectively.[7] In Taiwan, stroke crude prevalence from the 1994 National Health Interview Survey was 5.95 per 1,000.[8]

Risk factors of stroke

Risk factors of stroke are different among stroke subtypes, i.e. subarachnoid hemorrhage, intracerebral hemorrhage, and cerebral ischemic or embolic infarction. However, hypertension is a common major risk factor to all stroke subtypes.

Atherogenic host factors of stroke include hypertension, diabetes mellitus, cardiac disorders (atrial fibrillation, congestive heart failure, coronary heart disease, left ventricular hypertrophy, and echocardiographic abnormalities), dyslipidemia, obesity, family history of stroke, fibrinogen and other clotting factors disturbance, homocysteine levels, race, complicated migraine and migraine with aura, cigarette smoking, oral contraception, less physical activity, and unhealthy diet.

In addition to the risk of infarction, cigarette smoking is clearly linked to intracerebral hemorrhage and subarachnoid hemorrhage.[9,14]

Hypertension and the risk of stroke

From Framingham study, the incidence of stroke, particularly atherothrombotic brain infarction, was approximately three times greater in people with systolic blood pressure e" 160 mmHg, and 50% higher among people with systolic blood pressure 140-159 mmHg, than in people with systolic blood pressure less than 139 mmHg.[9] This finding was the same in all age groups and in both sexes. Moreover, the incidence of stroke rises with increasing level of blood pressure. Systolic blood pressure level is clearly directly related to the risk of stroke, particularly after the age 65. However, evidence of the relation of diastolic blood pressure and stroke is still lacking. In addition to blood pressure level, duration of hypertension is significantly related to stroke.

High blood pressure management in acute stroke

Any type of acute stroke is often accompanied by a transient high blood pressure, both in individuals with or without long-standing hypertension. There is much debate regarding whether it is advisable to intervene with drugs against this transient high blood pressure. The Cochrane Review of randomized controlled trials found that clinical outcome of acute stroke is the same regardless of whether blood pressure is acutely lowered or left to settle spontaneously.[10] This elevated blood pressure has been viewed as a beneficial physiologic response to acute cerebral ischemia. It is attributed to several factors such as stress inducing catecholamine secretion, other neuroendocrine factors, topographic presentation of cerebral ischemia. As cerebral autoregulation is transient deficit from acute stroke, lowering transient mild to moderate high blood pressure tends to increase stroke volume, particularly the penumbra zone. This transient high blood pressure usually spontaneously declines within a week or so. Reducing transient high blood pressure during deficient cerebral autoregulation period may lead to severely reduction of cerebral perfusion pressure. However, some exceptions to this recommendation are acute congestive heart failure due to severe hypertension, hypertensive encephalopathy, subarachnoid hemorrhage, immediate or progressing hypertensive or anticoagulant-induced intracranial hemorrhage, hyperperfusion syndrome

after carotid endarterectomy, myocardial ischemia, dissection of the thoracic or abdominal aorta, and the use of thrombolytic therapy. As for severe hypertension without urgency-emergency conditions, it is recommended to treat when blood pressure is higher than 220 mmHg systolic or 130 mmHg diastolic.[11]

Antihypertensive drugs of choice should have rapid onset and short duration intravenous agents such as nicardipine, nitroglycerine, or sodium nitroprusside, etc. The use of oral nifedipine, particularly crushing and swallowing, is very harmful since there is a possibility of severe steal from ischemic regions and prolonged uncontrolled blood pressure drop. Therefore, oral nifedipine is contraindicated for emergency antihypertensive treatment! From animal studies, angiotensin-converting enzyme inhibitors (ACEI) and angiotensin II receptor blockers (ARB) may shift the lower and upper limit of autoregulation of cerebral blood flow towards lower blood pressure.[12] Recently, there were several studies investigating the role of ACEI and ARB in treating high blood pressure in acute stroke.[14] Some studies found favourable outcomes, however, some did not. More studies or meta-analysis are needed for definite conclusion on the benefit of these two antihypertensive groups in acute stroke.

Hypertension and secondary prevention of stroke

Since hypertension is the common independent strong risk factor to any type of stroke, i.e., ischemic stroke, hemorrhagic stroke, and subarachnoid hemorrhage. Controlling hypertension, therefore, has long been emphasized to prevent secondary stroke and there are rich evidence on this issue. A systematic review (Rashid P et al., 2003) confirmed that hypertension treatment had been associated with significant stroke recurrence reduction.[13] Meta-analyses of randomized controlled trials found an approximate 30%-40% stroke risk reduction with blood pressure reduction.[14]

Data on the relative benefits of specific antihypertensive regimens for secondary stroke prevention are inadequate to draw conclusion. A meta-analysis (Rashid P et al., 2003) proved a significant stroke recurrence reduction with diuretics alone or with diuretics plus ACEI, but not with beta-blockers or ACEI alone.[13]

The American Heart Association has provided comprehensive and timely evidence-based recommendation on the prevention of ischemic stroke in the year 2006 regarding hypertension as:[14]

1. Antihypertensive treatment is recommended for both prevention of recurrent stroke and prevention of other vascular events in persons who have had an ischemic stroke or TIA and are beyond the hyper-acute period. Because this benefit extends to persons with and without a history of hypertension, this recommendation should be considered for all ischemic stroke and TIA patients. An absolute target blood pressure level and reduction are uncertain and should be individualized, but its benefit has been associated with an average reduction of 10/5 mmHg, and normal blood presure levels have been defined as < 120/80 mmHg by JNC-7.

2. Several lifestyle modifications have been associated with blood pressure reductions and should be included as part of a comprehensive antihypertensive therapy. The optimal drug regimen remains uncertain; however, the available data support the use of diuretics and the combination of diuretics and an ACEI. The choice of specific drugs and targets should be individualized on the basis of reviewed data and consideration of specific patient characteristics (e.g., extracranial cerebrovascular occlusive disease, renal impairment, cardiac disease, and diabetes).

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