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Beside Management of Hypertensive Stroke

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Abstract

Hypertension remains one of the most important preventable contributors to disease and death. Most patients with hypertension have other risk factors including lipid abnormalities, glucose intolerance and diabetes, family history of early cardiovascular events, obesity and tobacco use with or without alcohol excess. According to a review on "The Global Burden of Hypertension", the estimated prevalence of hypertension (in people aged 20 years and over) in India in the year 2000 was 20.6% among-males and 20.9% among females and is projected to increase to 22.9% and 23.6% respectively by year 2025.. If the child's blood pressure (BP) is more than the 90th percentile, the incidence of hypertension in adulthood rises 2-4 fold. Therefore, it is postulated that childhood diseases such as reflux nephropathy (RN) are the reason of hypertension in adulthood..

Based on the World Health Organization (WHO) reports, hypertension is the cause of 62% of cerebrovascular accidents (CVA) and 49% of ischemic heart diseases. For every 5 mmHg increase in diastolic BP, there is a 35% and 20% increase in the risk of CVA and coronary artery disease, respectively. In addition, hypertension is the cause of up to 50% of end stage renal diseases (ESRD) requiring dialysis and transplantation in adults.

Hypertension (HTN) is the second most common cause of death after diabetes in adults worldwide. Untreated hypertension leads to life-threatening complications that include CAD, MI, stroke, and kidney disease. So it is called "the silent killer" because symptoms generally appear only after the disease has caused damage to vital organs such as the brain, eyes, kidneys and the heart.

Introduction

Hypertension is the most important modifiable risk factor for stroke. It is estimated that 25% or more of strokes may be attributable to hypertension. This is well established that lowering BP reduces the risk of stroke. Epidemiological studies have shown that for each 10 mm Hg lower systolic blood pressure (SBP), there is a decrease in risk of stroke of approximately one third in persons aged 60 to 79 years. This association is continuous down to levels of at least 115/75 mm Hg and is consistent across sexes, regions, stroke subtypes, and even for fatal and nonfatal events. Lowering diastolic blood pressure (DBP) was once the main target to achieve stroke and other cardiovascular event reduction, but SBP has now become the target. Although hypertension in the immediate post-stroke period is frequently observed, BP tends to spontaneously fall within the first hours and days following the acute event, with the pattern of blood pressure change varying with stroke subtype. Precipitous falls in BP have, however, been associated with poor outcome and should be avoided. A 'U-shaped' association between admission BP and stroke outcome has been identified, with very high and very low BP at the onset of stroke is largely being associated with poor poststroke outcome.

The role of longer-term BP control to improve outcomes in patients with stroke is undisputed, BP management immediately after a stroke still remains controversial.

Hypertensive Hemorrhage: The Guidelines to Treat Blood Pressure

While treating the patient of acute intracerebral hemorrhage the question of controlling the blood pressure in domain of when, how and how much always remains in mind of internist. Despite of vast experience in treating such situation many times one find himself in a difficult way to deal such an emergency situation.

The argument against lowering BP in acute ICH is based on the possible existence of a perihematomal ischemic zone. Recent studies, however, indicate that low blood flow around the hematoma may be a consequence of reduced cerebral metabolism in this area rather than a primary reduction of blood flow. In addition, chronic hypertensives (due to a shift in the autoregulatory curve) and patients with increased intracranial pressure (ICP; due to lowered cerebral perfusion pressure) may develop cerebral ischemia if BP is acutely lowered but majority of the patients with intracerebral hemorrhage (ICH) often have elevated BP. Approximately one third of all patients with ICH presenting within 3 hours of symptom onset have a significant expansion of the hematoma over the next 20 hours. Initial hematoma volume and hematoma expansion are powerful predictors of mortality after ICH. Some studies have suggested an association between high BP and hematoma expansion and BP is often lowered under the assumption that high BP promotes hematoma expansion.

Treating Blood Pressure: The Question of When, How and How Much?

In absence of definitive supportive evidence, majority of experts believe that a SBP of >180 mm Hg or a mean arterial pressure (MAP) of >130 mm Hg would warrant immediate lowering. In the presence of conditions such as acute heart failure, hypertensive encephalopathy, active cardiac ischemia, and so on, lower BP targets may be appropriate.

It has been suggested that rapidly lowering MAP by approximately 15% does not lower cerebral blood flow, whereas reductions of >20% can do so. Therefore, if BP-lowering is considered, current guidelines suggest cautious lowering of BP by no more that 20% in the first 24 hours.

Therefore immediately after an ICH, it is perhaps more appropriate to tailor the target BP to each patient rather than using a generalized approach. The possibility of increased ICP and a history of chronic untreated hypertension should be considered while choosing the target. Presently, the American Heart Association/American Stroke Association (AHA/ASA) guidelines suggest maintaining a cerebral perfusion pressure of 60 to 80 mm Hg in patients with possible increased ICP and a BP of 160/90 or a MAP of 110 mm Hg in other patients.

Short and rapidly acting intravenous antihypertensive agents are preferred. Drugs like labetalol, hydralazine, esmolol, nicardipine, enalapril, nitroglycerin, and nitroprusside have been recommended. While treating the stroke patient sodium nitroprusside and nitroglycerin should be used with caution because these agents can potentially increase ICP. It is the target of BP lowering which is more important than the agent used.

Hypertensive Ischemic Stroke: The Guidelines to Treat Blood Pressure

Spontaneous elevation of blood pressure in the first 24-48 h after stroke onset with a significant spontaneous decline after a few days is established phenomenon. Several mechanisms may be responsible for the increased blood pressure including stress, pain, urinary retention, Cushing effect due to increased intra cranial pressure and the activation of the sympathetic, rennin - angiotensin and ACTH-cortisol pathways. Several arguments speak for lowering this elevated BP due to risks of hemorrhagic transformation, cerebral edema, recurrence of stroke and hypertensive encephalopathy. On the other hand, it may be important to maintain the hypertensive state due to the damaged autoregulation in the Ischemic brain and the risk of cerebral hypoperfusion exacerbated by the lowered systemic blood pressure as cerebral perfusion becomes dependent upon systemic arterial BP following stroke due to impairment of cerebral autoregulation, and therefore changes in systemic BP can directly influence cerebral perfusion. Hypertension may sustain cerebral perfusion to the ischemic penumbra, with BP having been shown to fall spontaneously in response to successful recanalization of cerebral vessels following thrombolytic treatment, perhaps suggesting the restoration of cerebral autoregulation.

Treating Blood Pressure: The Question of When, How and How Much?

In thrombolysis eligible patients the AHA/ASA guidelines recommends that before intravenous thrombolytic treatment, BP should be lowered if >185 mm Hg systolic or >110 mm Hg diastolic. During thrombolytic treatment, SBP should be kept <180 mm Hg and DBP <110 mm Hg and it

should also be maintained for first 24 hrs. Intravenous labetalol, nitropaste, nicardipine infusion, and, if BP remains elevated, sodium nitroprusside are the recommended agents. Intravenous labetolol is the preferred agent with t-PA use as it maintain cerebral and coronary blood flow. It should be given in dosage of 10-20 mg over 2 min and may even be repeated once. If BP is not reduced and maintained <180/110 mmHg do not administer t-PA so it is a golden rule that "Treat BP prior to t-PA" because clinical experiences have shown that uncontrolled BP with thrombolysis may worsen cerebral oedema and increases risk of intracerebral hemorrhage. Despite the absence of supporting evidence, these recommendations are often applied to patients receiving other forms of reperfusion therapy (e.g., intra-arterial thrombolysis, clot retrieval, and so on). Patients with other indications for BP-lowering such as acute heart failure, aortic dissection, and so on should have the BP lowered. One should be cautious about abruptly lowering BP in other patients due to the risk of worsening cerebral ischemia. In other subset of patients i.e. in non-thrombolysis patient guidelines suggest withholding antihypertensive agents in these patients unless the DBP is >120 mm Hg or the SBP is >220 mm Hg and limiting the drop in BP during the first 24 hours by approximately 15%.

Thus strategies focussing on achieving target BP with aim to maintain an adequate cerebral perfusion pressure and thus restoration of cerebral autoregulation to prevent the ongoing normal injury may result is favourable neurological outcome.

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