



MANAGEMENT OF BLOOD PRESSURE IN ACUTE STROKE: GUIDELINES AND PRACTICAL TIPS

Ansh Chaudhary¹, Sheetal Sharma¹, Sharique Rahman¹ and Bhupendra Chaudhary²

¹Department of Medicine Bharati Vidyapeeth Hospital & Research Centre, Pune

²Deptt. of Neurosciences, Jaswant Rai Speciality Hospital, Meerut (U.P.)

ARTICLE INFO

Article History:

Received 13th May, 2022

Received in revised form 11th

June, 2022

Accepted 8th July, 2022

Published online 28th August, 2022

Keywords:

Hypertension

ABSTRACT

Hypertension is the most important modifiable risk factor in stroke. It is well established that lowering the blood pressure definitely reduces the risk of stroke as each 10mmHg lowering of systolic blood pressure reduces the risk of stroke in approximately one third in the person between 60-79 years of age group, however lowering diastolic blood pressure was upon thought to be the main target to reduce stroke and related cardiovascular events but now systolic blood pressure has become the target. A U Shaped well established association exists between level of blood pressure at the time of admission and the stroke outcome is established with poor post stroke outcome at very high and very low blood pressure at onset of stroke. The judicious role of Anti-Hypertensive agents in stroke unit is at times the determining factor for the ultimate stroke outcome in any situation as systolic blood pressure of more than 180mmHg or a mean arterial blood pressure of more than 130mmHg should get immediate attention. Short and rapidly acting intravenous Anti-Hypertensive agents like Labetalol, Hydralazine, Esmolol, Enalapril, & Nitroprusside are the preferred agents in Acute Stroke setting. In the end is the target of BP lowering which is more important than the agent used.

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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. Because many patients with stroke have mild hypertension or prehypertension, we have shifted our focus and now think of stroke on a continuum of risk based on blood pressure (BP) level rather than on a threshold effect. Because high BP may not exist in isolation, a wider definition of hypertension has been proposed that also takes into account the absolute risk of cardiovascular events and associated metabolic factors or early disease markers.

Several observations have demonstrated spontaneous elevation of blood pressure in the first 24-48 h after stroke onset with a significant spontaneous decline after a few days. 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, renin-angiotensin and ACTH-cortisol pathways. Despite of increased prevalence of hypertension following stroke, optimal management has not been yet established. Several arguments speak for lowering the elevated BP: 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.

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 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. As recently shown, even the elderly with sustained SBP elevation may gain from BP reduction in relation to less fatal or nonfatal stroke, death, and heart failure.

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

Blood Pressure Management after Intracerebral Hemorrhage

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

*Corresponding author: **Ansh Chaudhary**

Department of Medicine Bharati Vidyapeeth Hospital & Research Centre, Pune

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.

Nonetheless, there may be other reasons to lower BP. Hypertensive patients with ICH may have heart failure or elevated cardiac troponin in which lowering BP might be helpful.

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.

Dilemma about Blood Pressure Management after Acute Intracerebral Hemorrhage

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 so.

What Blood Pressure Level Is Considered To Be Too High And Requiring Immediate Reduction?

Despite absence of definitive supportive evidence, some 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.

What Is The Appropriate Target Blood Pressure In Patients With Ich?

Immediately after an ICH, it is perhaps more appropriate to tailor the target BP to each patient rather than using a "one size fits all" approach. The possibility of increased ICP and a history of chronic untreated hypertension should be considered while choosing the target. Recognizing the absence of definitive data, 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.

How Fast Should Blood Pressure Be Lowered?

Results of small studies suggest 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 than 20% in the first 24 hours.

What Antihypertensive Agents Are Appropriate For Use In The Acute Setting?

Short and rapidly acting intravenous antihypertensive agents are preferred. In the United States, labetalol, hydralazine, esmolol, nicardipine, enalapril, nitroglycerin, and nitroprusside

have been recommended. Intravenous urapidil is also used in Europe. Large studies comparing various antihypertensives are not available. 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.

Blood Pressure Management after Acute Ischemic Stroke

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 being associated with poor post-stroke outcome. Early recurrent stroke has been suggested as one possible mechanism by which elevated BP may be associated with poor outcome. 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. High pre-thrombolysis BP has also been shown to be associated with poor recanalization and sustained hypertension may contribute to worsening cerebral edema and hemorrhagic transformation following acute ischemic stroke. Cardiovascular complications as well as early stroke recurrence in patients with elevated post-stroke BPs have been proposed as possible mechanisms for poor outcome.

Dilemma on Bp Management Immediately After an Acute Ischemic Stroke

Apart from managing the ischemic penumbra, the management of other parameters like hyperglycemia, hyperthermia, raised ICP and the accelerated HT are of paramount importance, of which the high blood pressure leads the situation.

Should Blood Pressure Be Lowered In Patients With Elevated Bp After An Ischemic Stroke?

As per the AHA/ASA guidelines, it is recommended that before intravenous thrombolytic treatment, BP should be lowered if >185 mm Hg systolic or >110 mm Hg diastolic. After thrombolytic treatment, SBP should be kept <180 mm Hg and DBP <105 mm Hg. Intravenous labetalol, nitropaste, nicardipine infusion, and, if BP remains elevated, sodium nitroprusside are the recommended agents. 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. 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%.

Should Blood Pressure Be Elevated to Improve Cerebral Perfusion in Patients With Ischemic Stroke?

A few small case series have shown neurological improvement with induced hypertensive therapy. Studies are underway to assess the usefulness of this form of therapy in patients with a diffusion-perfusion mismatch on MRI. In the meantime, it is reasonable to try volume expansion and/or vasopressors in patients with hypotensive stroke or in patients who have had a worsening of the neurological deficit in association with a drop in BP.

Should Patients on Antihypertensive Agents Have Their Medications Held or Continued?

The AHA/ASA guidelines recommend restarting antihypertensives at 24 hours in previously hypertensive neurologically stable patients unless contraindicated.

At the end, the optimum post-stroke BP, and how to achieve it, is yet to be identified. Current clinical guidelines do not advocate the active reduction of hypertension in the immediate post-stroke period unless there is a concurrent indication to do so. If elevated BP is to be lowered in the acute post-stroke period, the reduction should be cautious.

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How to cite this article:

Ansh Chaudhary *et al* (2022) 'Management of Blood Pressure in Acute Stroke: Guidelines and Practical Tips', *International Journal of Current Advanced Research*, 11(08), pp. 1415-1417. DOI: <http://dx.doi.org/10.24327/ijcar.2022.1417.0314>
