Original Research Article

Role of blood pressure control in all acute cerebrovascular accidents with hypertension

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Abstract

Various risk factors have been implicated in causation of stroke but hypertension remains the single most important treatable risk factor in all age groups, and it is the one which seems to have a direct relation to incidence. Furthermore antihypertensive therapy has been observed to be effective in reducing the incidence of CVD (cerebrovascular disease). The interaction between hypertension, antihypertensive treatment and stroke is a complex one. Nevertheless the conclusion is that antihypertensive treatment has definite beneficial effects on stroke. Limited data are available to guide the choice of a target for the systolic blood-pressure level when treating acute hypertensive response in patients with intra cerebral hemorrhage. In this study, a total of 55 patients who presented with CVD and with hypertension (stage 1 and 2 of JNC 7) were included in this study. Out of 55, 37 (67%) cases are ischemic and 18 (33%) cases were due to cerebral hemorrhage. In ischemic stroke 35 (63.5%) cases are thrombotic, 2 (3.5%) are embolic strokes. All patients were given treatment with oral Nifedipine retard preparation 10-20mg q 8th hourly or Angiotensin converting enzyme inhibitors (which also stabilizes endothelium of the vasculature), along with other conservative treatment and physiotherapy. Patients with intra cerebral haemorrhage were treated with Nimodipine 60 mg q 8th hourly along with antihypertensive treatment. In our study, the reduction of blood pressure improved the outcome, and their Blood Pressure normalized without any complications. Blood pressure reduction in acute stroke, irrespective of the etiology improved the outcome. In our study, we reduced the BP in all acute CVA patients with

hypertension to normal within 24-48 hours. This study suggested BP control improves the outcome, when compared to fatalities in other studies, where BP is not reduced acutely.

Key words

Blood pressure control, Acute cerebrovascular accidents with hypertension, Nifedipine, Angiotensin converting enzyme inhibitors.

Introduction

Acute stroke remains one of the most devastating and feared neurological disorder and a leading cause of mortality and morbidity throughout the world. While acute stroke is the most severe form of cerebrovascular disease, chronic forms and end-disability are areas of major medical and socioeconomic concerns.

Community based surveys from West and Japan indicate average annual incidence of stroke as 111-180 per 100,000 population [1] and 9 – 28 per 10,000 in young persons below 45 years. But no specific age, incidence or mortality data for Indian population is available as yet. Some information on crude prevalence rates based on community surveys point towards an annual incidence of around 3% [2]. Abraham, et al. [3] from Vellore reported a survey of 2,50,576 population and reported that 25 per cent of patients with stroke were of less than 40 years of age. The incidence has shown a decreasing trend in some western countries with better management of risk factors.

According to current understanding, stroke should not be considered as a disease in itself but a heterogeneous group of disorders that affect the vasculature of brain. There are two major subdivisions – hemorrhagic and ischemic have got definite diagnostic, therapeutic and prognostic implications.

Significant advances in our understanding of pathophysiology of stroke lead to emergence of new rational approaches of its therapy. The concept of ischemic penumbra, recognition of therapeutic window concept of first six hours [4], need for specialized stroke units and various thrombolytic and neuroprotective therapies, are all steps in the direction of improving not only mortality but also the quality of survival after stroke.

Various risk factors have been implicated in causation of stroke but hypertension remains the single most important treatable risk factor in all age groups. Besides the hypertension, other important risk factors are age, hyperglycemia, tobacco use, recent ischemic stroke and low content of normal haemoglobin [5]. Certain other risk factors like disturbed equilibrium in coagulation and fibrinolysis or subacute central nervous system infections or autoimmune diseases have been postulated to be associated with stroke in the young. Hyperlipidemia particularly low High density lipoprotein (HDL) and high Low density lipoprotein (LDL) is an important risk factor for stroke in young adults.

In fact from the analysis of many case control studies the concept of profile of a 'stroke prone' individual has emerged [6]. But hypertension is the one which seems to have a direct relation to incidence. Furthermore antihypertensive therapy has been observed to be effective in reducing the incidence of CVD.

A metaanalysis of various trials found a linear relationship between high blood pressure and risk of stroke (a 7.5mm rise in diastolic blood pressure may double the likelihood of stroke) [7, 8]. It has also been shown that risk of stroke in a patient with a diastolic pressure greater than 110 mm of Hg is fifteen fold that of a patient with diastolic pressure of less than 80 mm of Hg but even borderline hypertension is associated with increased stroke risk. The interaction between hypertension, antihypertensive treatment and stroke is a complex one. Nevertheless the

conclusion is that antihypertensive treatment has definite beneficial effects on stroke [9].

The management of BP during an acute stroke remains controversial. BP is often elevated in the immediate post stroke period and is thought by some to be a compensatory physiological response to improve cerebral perfusion to ischemic brain tissue. As a result, it has been common practice after acute cerebral infarction to reduce or withhold BP treatment until the clinical condition has stabilized. There are no large clinical studies upon which to base definitive recommendations [10-14].

Pathogenesis and pathology of stroke
- Cerebrovascular disease is caused by one of the several pathological processes involving the blood vessels of brain. The process may be intrinsic to the vessel as atherosclerosis, lipohyalinosis, inflammation, amyloid deposition, arterial dissection, developmental malformation, aneurysmal dilatation or venous thrombosis [15, 16].
- It can occur when an embolus from the heart in extracranial circulation lodges in intracranial vessels.
- Due to decreased perfusion pressure or increased blood viscosity with adequate cerebral blood flow [17].
- Due to rupture of a vessel into subarachnoid space or intra cerebral tissue.

A stroke is acute neurological injury occurring as a result of one of these pathological processes and is manifested as brain infarction or hemorrhage within hours to days. The grey matter becomes congested with enlarged dilated blood vessels and minute petechial hemorrhages. When an embolus blocking a major vessel migrates, lyses or disperses within hours. A primary intra cerebral hemorrhage damages the brain directly at the site of hemorrhage by compressing the surrounding tissue [12, 13]. The brain is unable to repair itself and only forms fibrogliotic scar tissue at the site of infarction or hemorrhage. The advent of CT and MRI coupled with carotid Doppler, MRA, catheter angiography etc., have revolutionized the evaluation of patient with stroke both in terms of parenchymal lesion as well as delineation of cerebrovascular tree [12, 13].

Atherosclerosis and its sequelae is the cause of cerebral thromboembolism in over 90% of cases in the head and neck, atherosclerosis occurs most commonly at the internal carotid artery origin or the carotid bifurcation [12, 13]. Recently non-invasive techniques like MRA have become important in the evaluation of carotid bifurcations.

Recent studies on molecular and metabolic events leading to cerebral injury have shown that there is a dense central core, surrounded by a less dense zone of ischemia and neuronal death occurs unless perfusion is immediately restored. On the other hand, nerve cells in the zone of ischemic penumbra remain viable for at least 3 hours ('therapeutic window') and can be salvaged by reperfusion (oxygen and glucose), neuroprotective agents, etc. [15, 18, 19].

lschemic Cascade Hypothesis [15, 18]
Major factors which enhance nerve cell injury are an increase in intracellular cytosolic calcium concentration (from failure of ionic pump functions or 'leaks'), changes in Na/K gradient, acidosis, release of glutamate as well as 'excitotoxic' substances, free radicals and many unknown factors which in turn disrupt the blood brain barrier & cell membrane functions.

The energy depletion resulting from ischemic-hypoxia is one of the key events that fail to maintain normal concentrations of cellular ATP leading to delay in re-synthesis of macromolecular proteins essential for cell structure. Such energy failures also induce

proteolysis and lipolysis, in addition to production of arachidonic acid, platelet activating factors, and free radicals etc. which in turn cause further neuronal damage.

The role of leukocyte-endothelial interaction, receptor activation, post-ischemic hypo/ hyperperfusion damage (injury), role of nitric oxide and nerve growth factors and gene expression are under study. Thus, severity of cerebral injury is not the mere result of hypoxia from impaired perfusion but end result of several highly complex factors.

Cerebral Edema [12]
This is a most important aspect of tumor growth, cerebral trauma, infarction, abscess, hypoxia, and other toxic and metabolic states. There are three categories of edema, vasogenic, cytotoxic and interstitial edema. So called interstitial (hydrocephalic) edema is a recognizable condition but is probably of less clinical significance than cytotoxic or cellular edema.

Ischemic cerebral oedema [13]
Cerebral ischemia causes not only reversible and then irreversible loss of brain function if ischemia prolonged, but also causes cerebral oedema. Ischemic oedema is partly 'cytotoxic' and partly 'vasogenic'. Cytotoxic oedema starts early, within minutes of stroke onset, and affects the grey more than the white matter. Vasogenic oedema, which starts rather later, within hours of stroke onset, affects the white matter more than the grey; the damaged blood–brain barrier allowing plasma constituents to enter the extracellular space. Ischemic cerebral oedema reaches its maximum in 2–4 days and then subsides over a week or two. It certainly complicates large infarcts, being visible on CT and at post-mortem, but whether it occurs to any extent in small infarcts (e.g. lacunar) is not known.

Clinical features
It can present with features of raised ICT like, headache, vomiting, altered sensorium and coma. A large infra tentorial infarct may cause upward herniation through the tentorium or downward medullary coning at the foramen magnum. Death in the first week after cerebral infarction is usually due to these mass effects.

Treatment of Brain Edema and Raised ICP
The definitive treatment of any given case (excision of a tumor, treatment of intracranial infection, placement of a shunt, etc.) will, of course, be governed by the underlying disease.

The use of high-potency glucocorticosteroids has a beneficial effect on the vasogenic edema associated with tumors, both primary and metastatic, sometimes beginning within hours.

In addition, drugs such as dexamethasone reduce the vasogenic edema associated with brain abscess and head injury, but their usefulness in cases of large cerebral infarctions, contusions, and hemorrhage is less clear.

In patients who require intravenous fluids, Normal saline (314 m osmol/L) is preferable and Ringer lactate solution (osmolarity 289 mosmol/L) is acceptable, but dextrose solutions alone, in any concentration (except D5/NS), are to be avoided because of their hypo-osmolar nature. The parenteral administration of hypertonic solutions, to which the brain is only partially permeable (mannitol, hypertonic saline, urea, glycerol), by shifting water from brain to plasma, is an effective means of reducing brain volume and lowering ICP.

Edema, however, is actually little affected by these agents. Mannitol is the most widely used; a 25% solution is administered parenterally in a dose of 0.5 to 1.0 g/kg body weight over a period of 2 to 10 min. Hypertonic saline solutions (3 or 1.5%) are equally effective. A single administration of these solutes has only a short-lived effect, a matter of several hours or less as the brain extracellular space reaches an equilibrium concentration, but repeated use on a regular schedule can lead to a reduction in headache and stabilization of some of the deleterious effects of a mass. Acetazolamide and

furosemide, are said to be helpful in special instances (interstitial edema, pseudotumor cerebri) by virtue of creating a hyperosmolar state and by reducing the formation of CSF. However, their effects are usually transient.

Furthermore, with repeated administration of hyperosmolar solutions such as mannitol or with diuretics, the brain gradually increases its osmolality - the result of added intracellular solute; these agents are therefore not suitable for long-term use [20-24].

Controlled hyperventilation is another method of rapidly reducing brain volume by producing respiratory alkalosis and cerebral vasoconstriction; it is used mainly in brain trauma with high ICP, during intracranial surgery, and in the management of patients who have become acutely comatose from the mass effect of a tumor, but its effect is brief.

Modifiable risk factors associated with stroke
There is only limited potential for successful medical treatment to reverse the neurological sequelae of a completed stroke; therefore interventions aimed at stroke prevention are extremely important. Age, race, sex and family history are all important biological indicators of enhanced stroke susceptibility, but these are inherent characteristics and cannot be altered [25].

Modifiable risk factors for stroke
- Hypertension
- Hyperlipidemia
- Heart Disease
- Diabetes Mellitus
- Elevated Fibrinogen Levels
- Smoking
- Obstructive sleep apnea

Of the modifiable risk factors listed above, hypertension is the most important for stroke. The degree of risk increases with higher levels of blood pressure and becomes particularly strong with levels higher than 160/95 mm Hg. Systolic hypertension and high mean arterial pressure represent parallel risks. In Framingham Heart Study [26], a sevenfold increased risk of cerebral infarction was observed in patients who were hypertensive. Hypertension increases the risk of several stroke types including thrombotic, lacunar, haemorrhagic stroke and subarachnoid haemorrhage.

The International society of Hypertension labels hypertension as the most important modifiable risk factor for stroke. This association is stronger for hemorrhagic stroke as compared to ischemic stroke and rises with an increase in BP, with adequate treatment of hypertension this risk decreases.

Successful long term treatment of hypertension results in substantial risk reduction. Early diagnosis and effective management of hypertension limits the secondary changes of hypertensive vascular disease. Treatment of hypertension after a patient has had a stroke is much less effective in reducing the risk of future vascular events. The Seventh Report of the Joint National Commission (JNC 7) [27] defines hypertension as a systolic blood pressure (SBP) >140 mm Hg and a diastolic BP (DBP) > 90 mm Hg.

Hypertension in stroke
Post stroke Hypertension
Elevated blood pressure is a very common clinical finding (80%) in patients of stroke during acute phase. A significant number (1/3rd) of these cases give history of previous hypertension. There may be hourly fluctuation in BP during first 24 hrs of admission and there after BP tends to fall spontaneously [28]. Extreme elevation in BP with slow decline is more often associated with cerebral haemorrhage than infarction [29].
It has also been stated that hypertension immediately after acute stroke is associated with early 30 day mortality, particularly in patients whose conscious level is impaired, which itself is

a bad prognostic feature following stroke [30]. There is no clue whether antihypertensive treatment was given by the author or not.

Early management of post stroke Hypertension

One of the most controversial aspects pertains to management of hypertension after stroke. A dilemma continues to exist regarding reduction of BP during acute stage. If BP remains elevated, this may worsen the arterial blood vessel wall injury resulting in cerebral edema. However, if BP is reduced, this may decrease cerebral perfusion and worsen neurological deficit. Thus a judicious use of antihypertensives is to be stressed in the concomitant occurrence of hypertension and stroke [31].

The observation of Lavine [32]: The protective rise in BP immediately following the ischemic stroke is an attempt to maintain adequate perfusion pressure to ischemic penumbra ; thus to artificially lower systemic BP by pharmacological means except in extreme circumstances, may lead to further neurological damage " is worth remembering while treating this dynamic variable.-BP [33]. Increase in BP routinely increases the risk of stroke, then why not increase in BP in acute stroke worsens the stroke.

Cerebral autoregulatory mechanism

MAP (mean arterial pressure) = <u>diastolic BP+ pulse pressure/ 3</u>

CPP (Cerebral perfusion pressure) = MAP- ICP (intra cranial pressure)

CBF (Cerebral blood flow) = CPP/CVR (Cerebral vascular resistance)

Normal values:

ICP = 15 mmHg or less

CPP = 70 -100 mmHg (ischemia<40 mm)

CBF = 5 0ml/min (irreversible damage <18 ml)

Chronic hypertension leads to adaptive changes in cerebral circulation. There is structural remodelling leading to narrowing and wall thickening in resistance vessels with a consequent shift of autoregulatory curve to rightwards so that a decrease in CBF occurs at a BP higher than in healthy normal individuals [34]. Thus in chronic hypertension, the tolerance of cerebral circulation to increases in pressure is improved, while acute hypertension may lead to cerebral ischemia at blood pressure levels well tolerated by normotensive individuals [35].

In injured brain, the cerebral blood flow passively follows mean arterial pressure. Due to this impaired autoregulation, any drastic decrease in MAP will lead to compromised circulation in collaterally perfused areas around the infarcted zone. After acute stroke, due to vasoparalysis, the cerebral microvasculature around the infarcted zone would dilate in response to an increase in MAP and thereby augmented CBF. It may convert a pale ischemic infarct into haemorrhagic one.

Review of literature
To treat or not to treat?

The debate regarding treatment of elevated blood pressure during acute stroke is still not conclusively settled. It is pertinent to know the salient and key points in favour or against it.

Hypertension in acute phase of ischaemic strokes should not be treated, why?

According to Yastu and Zivin (1985) [35], the justification and rationale for not treating Hypertenion in acute ischemic strokes is summerized as below:

- In persons who were chronic hypertensive prior to the stroke, the autoregulation is impaired and the curve is shifted. In these patients autoregulation starts at higher BP than in normal individuals and below that level CBF is depenedent on MAP. Hence, BP reduction may impair CBF further.
- Stroke itself further impairs autoregulation during acute phase. Hence CBF is dependent on MAP and if hypertension is corrected the marginal ischemic penumbra may suffer.
- There may be certain 'watershed'

(compromised) lesions in such patients which may get infracted with hypotensive therapy.

- Partial occlusion of a vessel may create decreased flow in remaining patent portion of the artery. Further decreasing flow by reducing MAP will facilitate thrombus formation and potentially extend the original occlusion. Proponent of this theory have advised to wait for a period of 7-10 days and evaluate the patient constantly for antihypertensive therapy [36-48].

Lavin P (1986) [36] also advocated withholding of antihypertensive medication in acute cerebral infarction except in situations like, vital organ compromised (cardiac or renal), DBP>130 mmHg, Cardiovascular emergency, and hypertensive encephalopathy.

Prattichizzo, et al. (1994) [37] have found that BP variability is much greater in thromboembolic stroke than hemorrhagic stroke, hence have suggested BP monitoring and restrain for early therapy. They also suggested that hypertension in stroke is more due to adrenergic activity and have advocated use of anti adrenergic agents (clonidine) [49] to reduce not only BP but also BP variability in ischemic stroke.

Presence of hypertensive encephalopathy, ICH or cardiovascular emergency will require early and prompt correction of BP. Algorithm for anti-hypertensive treatment after stroke (infarction and hemorrhage) - Brott and Reed recommendations as per **Table - 1**[41].

Table - 1: Algorithm for anti-hypertensive treatment after stroke (infarction and hemorrhage) Brott and Reed recommendations [41].

BP mmHg	Mode of treatment
<180/115	No antihypertensive drugs
180/115 to 230/120 (duration >60min)	Oral Nifedipine, Captopril or Labetolol
>230/120 (duration >20min)	IV Labetolol
DBP >120, SBP slightly increased	Nitroglycerine 5 mg IV or 10 mg oral
Diastolic BP >140	IV Sodium nitroprusside infusion

Hypertension in acute stroke should be treated

The prognostic significance of Hypertension in haemorrhagic stroke has been discussed. It has also been observed that intracranial hemorrhage, acting as a mass producing lesion, can result in increased ICP and can augment the marked elevation of BP typically seen on presentation. Persistent marked elevation of BP can promote rebleeding as well as increase CBF and blood volume with further elevation in ICP. Hence it is derived that markedly elevated BP on admission and persistent inadequate BP control in patients of hypertensive intracerebral haemorrhage adversely affect the prognosis, and effective hypotensive therapy improved outcome in such patients. Also recurrence of ICH is seen more

frequently in patients whose BP is not adequately controlled after 1st episode [39].

Certain conditions tend to make a person more prone to have very high BP after stroke and they need immediate attention. It includes younger age, more severe previous hypertension, alcohol abuse, severe mental stress etc. The type, size and location of brain lesion itself could not be statistically related to high BP but very large lesions particularly haemorrhages, might be associated with a reactive blood pressure response [40].

Guidelines for treatment [41, 42, 44]
A randomized, double blind placebo controlled study has suggested that cerebral perfusion

becomes impaired when MAP falls by 16% of its baseline. Therefore aim of treatment should be to reduce MAP by less than 20% only. In hypertensive encephalopathy this reduction should be attempted in 2-3 hrs and in infarction or haemorrhage reduction should be slower (6-12 hours) [43].

Nevertheless, the American Stroke Association has provided the following guidelines [44]:

In patients with recent ischemic stroke whose Systolic BP is >220mmHg or Diastolic BP 120–140 mmHg, cautious reduction of BP by about 10–15 % is suggested, while carefully monitoring the patient for neurologic deterioration related to the lower pressure [44].

SBP >185 mmHg or diastolic pressures >110 mmHg are contraindications to the use of tissue plasminogen activator (tPA) within the first 3 hours of an ischemic stroke [45].

Table - 2 and **Table - 3** depict the brief clinical pharmacology of important antihypertensive drugs used in acute stroke patients [46].

Table - 2: Oral drugs for hypertensive urgency (stroke).

Drugs	Class	Dose	Onset	Duration
Nifedipine	Calcium channel blocker	5-10 mg S/L or swallowed	5-10 min	3-5 hours
Captopril	ACE inhibitor	12.5-50 mg	15 min	4-6 hours
Labetolol	Alpha –beta blocker	200-400 mg	½-2 hours	8-12 hours
Clonodine	Central alpha agonist	0.2 mg stat then 0.1 mg/hr (total 0.8 mg)	½-2 hours	6-8 hours

Table - 3: Parenteral drugs for treatment of hypertensive emergency in stroke.

Drugs	Dosage	Onset of action	Duration of action	Side effects
Nitroprusside	0.25-10 μg/kg/min as IV	Instant	1-2 min	Nausea, vomiting, muscle twitching, sweating thiocyanaye and cyanide intoxication
Esmolol	200-500 μg/kg/min for 4 min, then 50-300 μg/kg/min IV	1-2 min	10-20 min	Hypotension nausea
Labetolol	20-80 mg IV bolus every 10 min, 2 mg/min IV infusion	5-10 min	3-6 hours	Postural hypotension nausea, vomiting, dizziness, burning in throat
Nitroglycerine	5-100 μg/min as infusion	2-5 min	3-5 min	Headache, vomiting tolerance on prolonged

In advanced centers with specialized stroke units, the favoured drugs are short acting vasodilators e.g. sodium nitroprusside and labetolol. But in majority of centers in our country it may not always possible to monitor therapy carefully and in case of cerebral haemorrhage it may be catastrophic. Hence the use of short acting arteriolar vasodilators such as Nifedipine or captopril may be the choice of therapy even in presence of cerebral hemorrhage.

Nifedipine [48] is the most popular drug followed by captopril-both sublingually or orally. These agents can be administered repeatedly every 30 minutes till desirable effect is achieved. These drugs cause leftward shift of lower limit of autoregulation, thus chances of cerebral ischemia are rare. These agents in different ways, both dilate large cerebral arteries and increase downstream pressure, causing smaller – resistance vessels to constrict, thus during a fall in pressure these vessels provide more autoregulatory dilatory capacity than normal [47].

Aim

- To study the effect of blood pressure control and outcome in all acute cerebrovascular accident patients i.e. thrombotic, embolic and hemorrhagic strokes with hypertension (stage 1 and 2 according to JNC 7) and to end the controversy regarding control of hypertension in acute stroke.

Materials and methods

Selection of Patients

The study was conducted in patients who presented clinically with acute storke (proven by CT Scan and or MRI brain) and with hypertension and admitted to the Medical wards of Gandhi Hospital, Secunderabad.

Period of study: From May 2008 to November 2010.

Inclusion criteria

- CVA patients- thrombotic, embolic and hemorrhagic stroke
- Hypertension at presentation
- Both male and female patients included
- All age groups
- The diagnosis was supported in every patient by axial CT and or MRI brain.

Exclusion criteria

- CVA without hypertension

Sample Size

After fulfilment of both exclusion and inclusion criteria, a total number of 55 cases were considered for study. Out of 55 cases 35 are with thrombotic, 2 with embolic and 18 cases hemorrhagic with hypertension.

Study protocol

A detailed history was taken either from the patient himself or the attendants and the various risk factors like Hypertension, Smoking, Diabetes, and Alcoholism were considered. A detailed general examination including height, weight, BMI, markers of atherosclerosis and vital data including Pulse, Blood Pressure were noted. Detailed systemic examination was done to rule out any other systemic disease. A detailed neurological examination based on proforma was done and recorded. Patients were assessed based on EVM score.

All the exclusion criteria were taken into consideration and relevant data recorded in the proforma.

CT scan was performed for every subject to confirm cerebrovascular accident (either infarct or hemorrhage).Other investigations like Complete blood picture, Complete Urine examination, ESR. Random blood sugar, Blood urea, Serum Creatinine, Serum electrolytes, Liver function tests, ECG, Chest X-ray and 2D Echo were done for all subject.

Treatment

All patients were given treatment with oral Nifedipine retard preparation 10-20 mg q 8th hourly with or without Angiotensin converting enzyme inhibitors, along with other conservative treatment and physiotherapy. Patients with EVM score <11 and with features suggestive of raised ICT and cerebral edema were treated with Inj. Mannitol 20%, 100 ml q 8th hourly. Patients with intra cerebral hemorrhage were treated with Nimodepine 30 mg q 6th hourly along with antihypertensive treatment.

All patients were monitored for their clinical condition and blood pressure daily for 15 days.

Results

A total of 55 patients who presented with CVA and with hypertension (stage 1 and 2 of JNC 7) were included in this study. Out of 55, 37 (67 %) cases are ischemic and 18 (33%) cases were due to cerebral hemorrhage. In ischemic stroke 35 (63.5%) cases were thrombotic, 2 (3.5%) were embolic strokes. Of all 35 (64%) were males and 20 (36%) were females. (**Table - 5, 6, 7**) Age distribution was as per **Table – 8**.

In these 33 patients had EVM score >10 (hemorrhagic and are due to ischemia), 17 were drowsy/semiconscious with EVM score 6-10 (hemorrhagic and ischemic) and 5 were unconscious with EVM <5 (3 hemorrhagic and 2 ischemic) at presentation.

The patients who presented with coma (5cases) had worst prognosis and resulted in death in 4 cases. Of this 2 patients are with hemorrhagic stroke, with EVM <5 at admission, 2 were due to thrombotic stroke with EVM score < 5. The remaining patients recovered with no adverse effects on their general condition. (**Table - 9, 10**)

All these patients with hypertension were treated with oral antihypertensive drugs i.e Nifedipine retard preparation 10 mg q 8th hourly OR ACE inhibitor i.e. Ramipril 5 mg 1 OD. Oral Nimodepine 30 mg q 6th hourly was given to patients with hemorrhagic stroke. Inj. Mannitol 20% 100 ml q 8th hrly for patients with features of raised ICT along with other conservative measures. All patients whose blood pressure is controlled irrespective of type of stroke recovered. It is interesting that cerebral hemorrhage cases also recovered whose blood pressure is controlled, which was thought earlier fatal. (**Table - 4**)

Table – 4: Outcome of CVA's with bp control.

	Type of stroke	BP - Normalised		BP - High	
		Survived	Died	Survived	Died
All CVA's with HTN (55)	**C. thrombosis (35)**	33	01	00	01
	C. embolism (2)	02	00	00	00
	C. hemorrhage (18)	16	00	00	02

Table – 5: Type of stroke.

Type of Stroke	No (%)
Hemorrhagic	18 (33%)
Thrombotic	35 (63.5%)
Embolic	2 (3.5%)

Discussion

Stroke remains a major cause of human mortality and morbidity. This is in spite of our increasing understanding of the pathophysiology of stroke and continuing advances in its prevention and treatment. By and large, impaired cerebral perfusion by occlusion (e.g. thromboembolism) is responsible for 'Ischemic cerebrovascular disease' whereas 'Hemorrhagic cerebrovascular disease' results from leakage of blood from damaged cerebral vessels.

Table – 6: Type of stroke.

Type of stroke	Males	Females	Total
Hemorrhagic	15 (27.2%)	3 (5.4%)	18 (33%)
Thrombotic	20 (36.4%)	15 (27.2%)	35 (63.6%)
Embolic	0	2 (3.5%)	2 (3.5%)

Table – 7: Sex distribution.

Males	Females
35 (64%)	20 (26%)

Table – 8: Age distribution.

Age group (Years)	Thrombotic	Embolic	Hemorrhagic	Total
30-40	2	1	2	5
41-50	9	0	6	15
51-60	11	0	4	15
61-70	9	1	6	16
> 70	4	0	0	4

Table – 9: EVM score and outcome.

EVM Score	Improved	Death	Total
< 5	1	4	5
6-10	17	0	17
>10	33	0	33

Table – 10: Analysis of mortality (EVM < 5).

EVM < 5	Thrombotic	Hemorrhagic
Total	2	3
Improved	0	1
Death	2	2

Various risk factors have been implicated in causation of stroke but hypertension remains the single most important treatable risk factor in all age groups and has a direct relation to the incidence. There may be hourly fluctuation in BP during first 24 hours of admission and there after BP tends to fall spontaneously.

Extreme elevation in BP with slow decline is more often associated with cerebral haemorrhage than infarction [29]. It has also been stated that hypertension immediately after acute stroke is associated with early 30 day mortality, particularly in patients whose conscious level is impaired, which itself is most important prognostic feature following stroke [30]. It is therefore unclear whether post stroke hypertension causes increased mortality or is simply a marker of a more severe stroke. In one study, the treatment of participants with intra cerebral hemorrhage to achieve a target systolic blood pressure of 110 to 139 mm Hg did not result in a lower rate of death or disability than

standard reduction to a target of 140 to 179 mm Hg [49].

In our study also extreme elevation in BP is more common in hemorrhagic stroke than in ischemic. And mortality is more in patients who are unconscious with EVM < 5 at admission, which itself is a poor prognostic factor.

One of the most controversial aspects pertains to management of hypertension after stroke. A dilemma continues to exist regarding reduction of BP during acute stage. If BP remains elevated, this may worsen the arterial blood vessel wall injury resulting in cerebral edema. However if BP is reduced, this may decrease cerebral perfusion and worsen neurological deficit. Thus a judicious use of BP is to be stressed in the concomitant occurrence of hypertension and stroke [31]. But there are no large scale clinical studies upon which to base definitive recommendations.

Recently AHA/ASA recommended the following, BP: Recommendations
- For ICH patients presenting with SBP between 150 and 220 mmHg and without contraindication to acute BP treatment, acute lowering of SBP to 140 mmHg is safe (Class I; Level of Evidence A) and can be effective for improving functional outcome (Class IIa; Level of Evidence B). (Revised from the previous guideline)
- For ICH patients presenting with SBP >220 mmHg, it may be reasonable to consider aggressive reduction of BP with a continuous intravenous infusion and frequent BP monitoring (Class IIb; Level of Evidence C). (New recommendation) [50].

In our study, we tried to reduce BP in all acute CVA pts with hypertension to normal within 24-48 hours, but this didn't affect the outcome of patients compared to other studies where BP is not reduced acutely.

Summary
Cerebrovascular disease is a common cause of morbidity and mortality, especially in the elderly. Out of 55 cases of CVA, 35 (63.6%) cases are due to thrombosis, 2 (3.6%) cases are due to embolism and 18 (32.7%) are due to hemorrhage. Stroke is more common in males (64%) than in females (36%). For all cases it is useful to score on Glasgow coma scale, as score of >10 have a good recovery or only mild disability and score of < 5 is associated with very high mortality (80%) and morbidity. Mortality is high in hemorrhagic stroke with massive bleed and mass effect or intra ventricular extension and also in ischemic stroke with low (<5) Glasgow coma scale at admission. In our study reduction of blood pressure improved the outcome and their BP normalized without any complications.

Conclusion

Blood pressure reduction in acute stroke, irrespective of the etiology improved the outcome and BP normalized. Mortality is more in patients with hemorrhagic stroke with massive bleed with intra ventricular extension, mass effect and with low Glasgow coma scale i.e. <5 at admission. Mortality is high in ischemic stroke with low Glasgow coma scale i.e. <5 at admission. The drugs used are Nifedipine and Angiotensin converting enzyme inhibitors, which also stabilizes endothelium of the vasculature. We recommend these drugs for patients who present with any stroke and hypertension.

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