A STUDY ON CLINICAL PROFILE AND OUT-COME OF HYPERTENSIVE EMERGENCIES IN A TERTIARY CARE CENTRE

## Dr. G. Swarnalatha Devi

HOD, Dept Of General Medicine Asram, Eluru.

## Dr.Tadi

 Srujanitha*
## Dr.R.Bhargavi

Post Graduate -IIIRD Year, Dept Of General Medicine, Asram *Corresponding Author

ABSTRACT Hypertension being more prevalent now a days, it is very crucial for the physician in the emergency department to identify the hypertensive emergencies and to manage them through blood pressure lowering medi-cations in order to avoid further in our hospital.

## KEYWORDS : Heart Failure, Hypertensive Encephalopathy, Malignant Hypertension,myocardial Infarc-tion, Stroke

## INTRODUCTION

Hypertension affects individuals of all classes and all age groups. Target organ damage resulting from hypertension includes those affecting the brain, heart, kidneys and the eyes.[1].A number of cardiovascular, pulmonary and neurological symptoms are found to be associated with hypertensive emergency with target organ involvement.[2].This account for more than one forth of all medical urgencies and emergencies.[3]The incidence of hypertensive emergencies are increasing.[4]A hypertensive emergency is characterized by rapid deterioration of target-organs and poses an immediate threat to life.[5]

This study is done to find out various modes of presentation and clinical profile of hypertensive emergencies in our hospital.

## AIMS AND OBJECTIVES

The aim of the present study is to evaluate the modes of presentations, clinical profile and spectrum of target organ damage in patients with hypertensive emergencies

## MATERIALSAND METHODS

## STUDYAREA

The study is conducted in the Dept of General Medicine ASRAM Hospital, ELURU.

## PERIOD OFSTUDY

The study was conducted between December 2016 and July 2018.

## DESIGN OFSTUDY

Prospective study.

## SAMPLESIZE

The study population included patients admitted to this hospital with severely elevated blood pressure with clinical or laboratory evidence of acute target organ damage. The clinical and laboratory profile of 50 of these patients were evaluated.

## SELECTION CRITERIA

## INCLUSION CRITERIA

Patients above 18 years of age.
Systolic blood pressure of 180 mm Hg or diastolic blood pressure of 110 mmHg .

Evidence of target organ damage, either clinically or on laboratory findings.

## EXCLUSION CRITERIA

Patients less than 18 years of age.
Chronic renal failure.
valvular heart diseases.

## CONSENT

Informed consent is obtained from the patients.

## STUDY PROTOCOL

Data was collected from fifty patients admitted to this hospital during December 2016 to July 2018

Patient who presented with an elevated blood pressure of Systolic blood pressure of $\geq 180$ or diastolic blood pressure of $\geq 110 \mathrm{mmhg}$, with history suggestive of acute target organ damage or with a laboratory evidence of acute target organ damage were included in the study. A detailed history was taken which included presenting symptom atology, hypertension related history with emphasis on drug compliance. Blood pressure was recorded in these patients at the time of admission, after one hour, after 24 hours and at the time of discharge. Detailed clinical examination was done in these patients with examination of respiratory system, cardiovascular system, abdomen and central nervous system. Clinical examination also included Fundoscopic examination in all the patients.

The routine investigations done in these patients are hemogram renal function tests, random blood sugar, lipid profile, urine analysis, chest xray and electrocardiography. Patients with neurological deficits are evaluated with computed tomography and MRI of the brain. Patients with cardiovascular dysfunction are evaluated with echocardiography and patient with renal dysfunction underwent renal sonography.

## STATISTICALANALYSIS

The collected data was analyzed using Microsoft Excel software.

## RESULTS

Among the fifty subjects in the present study,
35 ( 70 percent) were males.
The male - female ratio was 2.33:1.
The mean age of the patients was 59.36 years. The mean age for males and females were 57.65 and 63.33 years respectively.

Among the fifty patients studied 30 patients ( $60 \%$ ) were previously known hypertensives.

Of the 50 patients studied six patients ( $12 \%$ ) had diabetes mellitus and (36\%) had dyslipidemia.

The mean blood pressure readings in patients who were discharged from the hospital were systolic blood pressure of 212 mm Hg and diastolic blood pressure of 112 mm Hg . Similar readings in patients who expired were systolic blood pressure of 225 mm Hg and diastolic blood pressure of 137 mm Hg respectively.

|  | MEAN SYSTOLIC BP | MEAN DIASTOLIC BP |
| :--- | :--- | :--- |
| DISCHARGED | 212 mm hg | 112 mm hg |
| EXPIRED | 226 mm hg | 137 mm hg |

The commonest presenting complaints were neurological deficits in 25 patients ( $50 \%$ ) followed by dyspnoea in 17 ( $34 \%$ ) and chest pain in 15 patients ( $30 \%$ ). Three (six percent) presented with convulsions and visual deficits each.

Neurological evaluation in symptomatic patients with computed tomography scan of brain revealed acute intracerebral hemorrhage in 14 patients ( $28 \%$ ) subarachnoid hemorrhage was seen in four patients ( $8 \%$ ), acute cerebral infarct in five patients ( $10 \%$ ) and normal study of the brain in two patients.


Neurological target organ damage included intracerebral haemorrhage ( $28 \%$ ), subarachnoid haemorrhage ( $8 \%$ ), acute ischaemic stroke ( $10 \%$ ), cardiac target organ damage were acute myocardial infarction (10\%), unstable angina (12\%), left ventricular failure (18\%), acute myocardial infarction with left ventricular failure (4\%) hypertensive encephalopathy ( $2 \%$ ) malignant hypertension ( $6 \%$ ).


Chest radiograph was suggestive of cardiomegaly in seven patients and five patients had signs of pulmonary oedema. Chest radio-graph was normal in 38 patients.

Out of 50 patients 26 patients ( $52 \%$ ) had ST segment or T wave abnormalities, 10 ( $20 \%$ ) had ECG with voltage criteria suggestive of LVH and two had both the changes.

Echocardiography done in 26 patients with evidence of cardiac dysfunction showed regional wall motion abnormality in 13 patients ( $50 \%$ ), left ventricular dysfunction in 16 patients ( $62 \%$ ) left ventricular hypertrophy In 8 patients ( $31 \%$ ) and normal echocardiographic study in 3 patients ( $12 \%$ ).

Serum urea was abnormal with levels above $40 \mathrm{mg} / \mathrm{dl}$ in $12(24 \%)$ of patients Serum creatinine level were above $1.4 \mathrm{mg} / \mathrm{dl}$ in $9(18 \%)$ of patients Serum urea and creatinine were elevated in 5 patients ( $10 \%$ ) and 7 patients out of 17 patients had elevated serum urea levels alone.

Patients with laboratory evidence of renal dysfunction were subjected to renal sonogram. Out of 17 patients 3 patients had grade I changes, Two patients had grade II changes.

Microalbuminuria was seen in 18 patients ( $36 \%$ )
Fundoscopic examination was done in every patient and Fundoscopic evaluation was normal in 16 patients ( $32 \%$ ) 11 had grade I changes ( 22 $\%$ ), 6 had grade II changes ( $12 \%$ ), two patients had grade III changes ( $4 \%$ ), 12 of the patients had evidence of papil-lodema ( $24 \%$ ). Fundus could not be visualized in 3 patients ( $6 \%$ )

Out of 50 patients with hypertensive emergencies, 11 patients died before discharge

In-hospital mortality was ( 22 percent)

## DISCUSSION

Hypertension is the leading cause for global burden
6 INDIAN JOURNAL OF APPLIED RESEARCH

The WHO first reported the GBD for 1990 using disability-adjusted life years (DALY), which is a time-based measure that combines years of life lost due to morbidity and premature mortality.[6]

## DEFINITION OF HYPERTENSION[7]

*Individuals with SBP and DBP in 2 categories should be designated to the higher BP category.

| CATEGORY | SBP |  | DBP |
| :--- | :--- | :--- | :--- |
| NORMAL | $<120 \mathrm{~mm} \mathrm{hg}$ | AND | $<80 \mathrm{~mm} \mathrm{hg}$ |
| ELEVATED | $120-129 \mathrm{~mm}$ hg | AND | $<80 \mathrm{~mm} \mathrm{hg}$ |
| HYPERTENSION- |  |  |  |
| STAGE 1 | $130-139 \mathrm{~mm} \mathrm{hg}$ | OR | $80-89 \mathrm{~mm} \mathrm{hg}$ |
| STAGE 2 | $>140 \mathrm{~mm} \mathrm{hg}$ | OR | $>90 \mathrm{~mm} \mathrm{hg}$ |

Hypertensive crises are acute, severe elevations in blood pressure that may or may not be associated with target-organ dysfunction.

Hypertensive crisis includes both hypertensive urgencies and emergencies. Hypertensive emergency can be an end result of chronic hypertension, non compliance of drugs, or new presentation of unrecognized essential hypertension.

Hypertensive emergencies are characterized by acute, severe elevations in blood pressure, often greater than $180 / 110 \mathrm{~mm} \mathrm{Hg}$ (SBP greater than 200 mm Hg and/or DBP greater than 120 mm Hg ) associated with the presence or impendence of target-organ dysfunction. Hypertensive urgencies are characterized by a similar acute elevation in blood pressure but are not associated with target-organ dysfunction.[8]

Focal neurological deficits, dyspnoea, chest pain, headache, loss of vision are considered as the commonest symptoms.Due to the association of hypertensive emergencies with various cerebral, cardiac and renal complications, there is an urgent need to recognize this condition so as to reduce the burden associated with it in terms of increased morbidity and mortality in the society.

Worldwide, recent estimations indicate that HTN is the responsible cause for approximately 7.1 million deaths per year[9]

In hypertensive crisis, there is a lack of autoregulation in vascular bed and blood flow and so an abrupt increase of BP and systemic vascular resistance can occur, which often leads to mechanical stress and endothelial injury [10].The second mechanism is the activa-tion of renin-angiotensin system, leading to further vasoconstriction and thus generating a vicious cycle of continuous injury and subsequently ischemia.

In cases with hypertensive urgency BP control should be managed with the use of low doses of oral antihypertensive medications, where a gradual decrease of BP over hours to days is expected.

On the other hand, hypertensive emergencies require rapid BP control with a parenteral antihypertensive medication. The BP should be reduced within minutes to an hour to about $20-25 \%$ in the first hour and then to $160 / 100$ or $160 / 110 \mathrm{mmHg}$ within the next $2-6 \mathrm{~h}$.

## CONCLUSION

Majority of patients presenting in hypertensive emergency belonged to the fifth and sixth decades of age.

Males have higher chances of developing hypertensive emergencies compared to females.

Known hypertensives are at a higher risk of presenting with acute target organ damage associated with hypertensive emergency.

Presence of diabetes mellitus and dyslipidemia increases the chance of developing hypertensive emergencies

Commonest mode of presentation is with a neurological deficit. Higher levels of blood pressure at presentation points towards a more adverse outcome.

Acute intracerebral haemorrhage is the commonest form of target organ damage encountered in the present study

The in-hospital mortality among these patients with hypertensive emergency were 22 percent.

## REFERENCES

1. Joint national committee on prevention detection and treatment of high blood pressure. The seventh report of Joint national committee on prevention detection, and treatment of high blood pressure. JAMA 2003; 289: 2560-2572.
2. Karras et al. lack of relationship between hypertension associated symptoms and blood pressure in hypertensive emergen-cy department patients. American journal of emergency medicine- vol 23(2) march 2005; 106-110
3. Zamapaglione et al. Hypertensive urgencies and emergencies. Hypertension1996; 27: 144-147
4. Elliot WJ. Hypertensive emergencies. In Critical Care Clinics Vol 17. Number 2 April 2001 W.B.Saunders company.
5. Ventura et al. Desperate disease, desperate measures: Tackling malignant hypertension in the 1950s. Am Heart J 2001; 142: 197-203
6. WHO . Global Burden of Disease (GBD) World Health Organization; [Accessed January 27, 2013]. 2012
7. A report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines J Am Coll Cardiol. Sep 2017, 23976; DOI: 10.1016/j.jacc.2017.07.745
8. Parati G, Ochoa JE, Lombardi C, et al. Assessment and management of blood pressure variability. Nat Rev Cardiol 2013;10:143-55.
9. Papadopoulos DP, Mourouzis I, Thomopoulos C, Makris T, Papademetriou V. Hypertension crisis. Blood Press (2010) 19(6): 328-36.10. 3109/ 0803 7051. 2010. 488052
10. Taylor DA. Hypertensive crisis: a review of pathophysiology and treatment. Crit Care Nurs Clin North Am (2015) 27(4):439-47.10.1016/j.cnc.2015.08.003.
