



A CLINICAL STUDY OF HYPERTENSIVE EMERGENCIES FROM A TERTIARY CARE CENTRE

KEYWORDS

Arterial hypertension, Hypertensive emergency.

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ABSTRACT

Background and Objectives: The clinical profile of patients with hypertensive emergencies presenting to hospitals in a developing country like ours is poorly known. The objective of the present study was to evaluate the modes of presentations, clinical profile and spectrum of target organ damage in patients with hypertensive emergencies. **Method:** This prospective study was done at Osmania General Hospital, Hyderabad, over a period of two years from Jan 2014 to Dec 2015. 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. **Results:** Males had higher chances of developing a hypertensive emergency compared to females. The commonest presenting symptoms were neurological deficit, dyspnoea, chest pain followed by convulsions and loss of vision. Majority of the patients were known hypertensives. Higher levels of blood pressure at presentation were associated with an adverse outcome. Laboratory abnormalities noticed in these patients include hyponatremia, hypokalemia and hyperkalemia. Intracerebral haemorrhage was the commonest target organ damage observed. An in-hospital mortality of 22% was observed in the present study. **Conclusion:** Known hypertensives are at a higher risk of presenting with acute target organ damage associated with hypertensive emergency. Commonest mode of presentation is with a neurological deficit. Acute intracerebral haemorrhage is the commonest form of target organ damage encountered in the present study.

INTRODUCTION

Hypertension affects individuals of all classes and across all age groups. The relationship between blood pressure and risk of cardiovascular disease events is continuous, consistent and independent of other risk factors. 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 patients in hypertensive emergency with target organ involvement. Focal neurological deficits, dyspnoea, chest pain, headache, loss of vision, are considered as the commonest symptoms with which patients in hypertension related acute target organ damage present.² The physician should perform an extensive evaluation in a patient who presents with any of these symptoms and with an elevated blood pressure to exclude a hypertensive emergency.

Although great strides have been made in the treatment of hypertension, patients still present in hypertensive crises and emergencies. This account for more than one fourth of all medical urgencies and emergencies.³ A hypertensive emergency is characterized by rapid deterioration of target-organs and poses an immediate threat to life. These conditions were invariably fatal before the advent of anti hypertensive drugs.⁴ It is also seen that the incidence of hypertensive emergencies are increasing.⁵

Defining abnormally high blood pressure is extremely difficult and arbitrary.⁶ Based on recommendations of the Sixth Report of the Joint National Committee of Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC VI)^{1,6,7}, the classification of blood pressure (expressed in mm Hg) for adults aged 18 years or older is as follows: The classification^{3,5,6} is based on the average of 2 or more properly measured, seated BP readings on each of 2 or more office visits.

- Optimal - **Systolic lower than 120 and diastolic lower than 80.
- Normal - Systolic lower than 130 and diastolic lower than 80.
- High normal (Prehypertension), - Systolic 130-139 or 85-89.
- Hypertension *** Stage 1 - Systolic 140-159 or diastolic 90-99
- Stage 2 - Systolic 160-179 or diastolic 100-109.
- Stage 3 - systolic > 180 or diastolic > 110.

Hypertensive crisis^{8,9,10} occurs when critically elevated blood pressure is accompanied by diastolic pressure greater than 120 to 130 mmHg. There are two types.

Hypertensive emergencies¹¹ are characterized by severe elevations in BP (>180/120 mmHg) complicated by evidence of impending or progressive target organ dysfunction. The systems primarily involved include the central nervous system, cardiovascular system, and the kidneys, which require reduction of blood pressure within minutes to hours to avoid catastrophic outcomes.

Malignant hypertension¹² severe hypertension accompanied by certain retinal findings such as hemorrhages, exudates and papilledema associated with diastolic pressures generally greater than 140-mmHg and end organ damage.

Accelerated hypertension⁹ is defined as a recent significant increase over baseline blood pressure that is associated with target organ damage. This is usually vascular damage on funduscopic examination, such as flame-shaped hemorrhages or soft exudates, but without papilledema. The pathogenesis of *accelerated hypertension* is not fully understood. However, at least two independent processes^{6,7,8,9,11} dilatation of cerebral arteries and generalized arteriolar fibrinoid necrosis contribute to the associated signs and symptoms. The characteristic vascular lesion is fibrinoid necrosis of arterioles and small

arteries, Endothelial injury^{6,8} from increased arterial wall tension leads to the release of prostaglandins and other inflammatory mediators, which causes a subsequent myointimal proliferation (referred to as fibrinoid necrosis). Other vasoactive compounds such as angiotensin II are usually involved as well. Red blood cells are damaged as they flow through vessels obstructed by fibrin deposition, resulting in microangiopathic hemolytic anemia.^{6,8}

Accelerated/ Malignant hypertension(WHO Definition): Severe hypertension plus bilateral fundal hemorrhages and exudates. Papilloedema not required for the diagnosis. DBP>130 mm Hg, Renal failure, Microangiopathic hemolysis may be present but not diagnostic.

Hypertensive urgency^{12,13} must be distinguished from emergency. Urgency is defined as severely elevated blood pressure (i. e., systolic >220 mmHg^{6,8,13} or diastolic >120 mmHg) with no evidence of target organ damage, which is generally treated over 24 to 48 hours in a closely monitored outpatient setting and associated with severe headache, shortness of breath, epistaxis, or severe anxiety. The majority of these patients present as noncompliant or inadequately treated hypertensives, often with little or no evidence of target organ damage.

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. 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 was to evaluate the modes of presentations, clinical profile and spectrum of target organ damage in patients with hypertensive emergencies.

MATERIALS AND METHODS

The present study was done in patients admitted to Osmania General Hospital, a tertiary referral centre situated in the city of Hyderabad, Afzulgunj, Andhra Pradesh over a period of two years from Jan 2014 to Dec 2015.

Inclusion criteria

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

Source of data

50 patients admitted to Osmania General Hospital with clinical and laboratory evidence of hypertensive emergency.

Type of study

Prospective study

Study protocol

Data was collected from fifty patients admitted to this hospital from 01-05-2010 to 20-10-2012 over a period of two and a half years. Patient who presented with an elevated blood pressure of Systolic blood pressure of >180 or diastolic blood pressure of >110 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 with which included presenting symptomatology, hypertension related history with emphasis on drug compliance.

The information thus obtained was recorded in the proforma, a copy of which is furnished in the annexure. 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. Blood samples of these patients were evaluated for biochemical abnormalities.

The routine investigations done in these patients were the haemoglobin, total count, differential count, erythrocyte sedimentation rate, blood sugar, serum urea, serum creatinine serum electrolytes, serum total cholesterol, serum triglycerides, high density lipoprotein, low density lipoprotein, microalbuminuria and urine analysis. All patients also underwent chest x- ray and electrocardiography. Patients with clinical suspicion of neurological deficits were evaluated with computed tomography of the brain. Patients with cardiovascular dysfunction clinically were evaluated with echocardiography and patient with renal dysfunction underwent renal sonography.

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 2.3:1.

Table 1 : Age Distribution

Age - Years	Frequency (N=50)	Percentage(%)
30-39	4	8
40-49	10	20
50-59	14	28
60-69	13	26
70 years and more	9	18

The mean age of the patients was 59.36 years. The age varied from 38 to 80 in males and 43 to 75 years in females. The mean age for males and females were 57.65 and 63.33 years respectively. The age distribution is given in table 2 below.

The commonest presenting complaints were neurological deficits in 25 patients (50 %) followed by dyspnoea in 17 (34 %) and chest pain in 15 patients(30 %). 3 (6%) presented with convulsions and visual deficits each.

Among patients with neurological deficit, 20 patients (80 %) had hemiparesis and one patient had monoparesis (four percent) 24 patients (98 %) were in altered sensorium. Three patients (12 %) presented with convulsions and visual deficits each.

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

Out of the known hypertensives, 22 (73 %) were still continuing their antihypertensive medications while eight patients (27 %)

had discontinued medications (Figure 4)
Of the 50 patients studied six patients (12 %) had diabetes mellitus and (36 %) had dyslipidemia. (Table 4)

Table 2. Blood pressure measurements

	MEAN SBP	MEAN DBP
AT ADMISSION	216±24.74	125±18.41
AT ONE HOUR	197±22.20	111±22.29
AT 24 HOURS	163±22.01	96±10.99
AT DISCHARGE	136±10.81	85±5.08

Mean variation in blood pressure at one, 24 hours and at the time of discharge are given in the table 2.

The mean blood pressure readings in patients who were discharged from the hospital are shown in the Table 3.

Table 3. Blood pressure at presentation- Patients who were discharged from the hospital and those who expired

	Mean SBP	Mean DBP
Discharged	212	112
Expired	226	137

Fundoscopy examination was done in every patient and the results are shown in Figure 1.

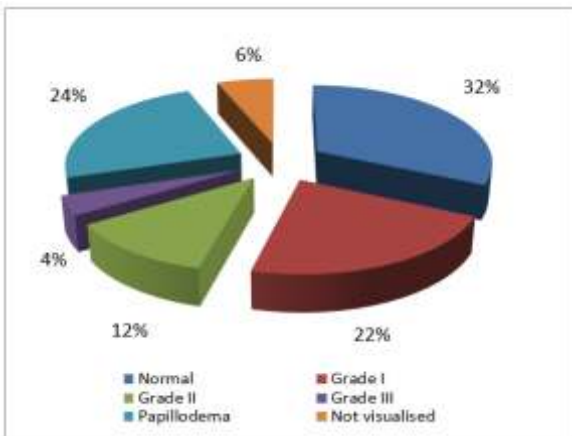


Figure 1 Fundus examination findings

Clinical examination revealed evidence of left ventricular failure in the form of left ventricular S3 or basal crepitations or frank pulmonary oedema in 25 patients (50 %).

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 findings are shown in Figure 2.

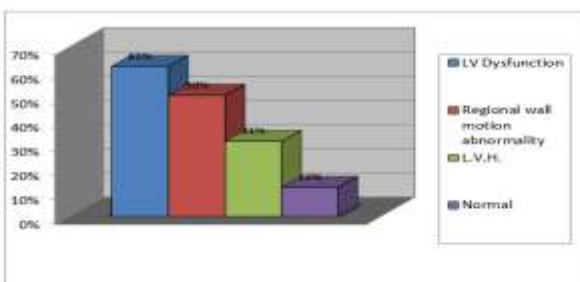


Figure 2 Echocardiographic findings

Serum urea was abnormal with levels above 40mg/ dl in 12 (24 %) of patients Serum creatinine level were above 1.4 mg/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.

Hyponatremia with serum sodium levels less than 135 meq/l were seen in 16 patients (32 %) while 6 patients (12 %) had hypokalemia with serum potassium levels less than 3.5 meq/l and 2 (4 %) had hyperkalemia with potassium levels more than 5.0 meq/l.

Table 3: Electrolyte abnormalities

	FREQUENCY (N=50)	PERCENTAGE
Hyponatremia	16	32
Hypokalemia	6	12
Hyperkalemia	2	4

Microalbuminuria was seen in 18 patients (36 %)

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.

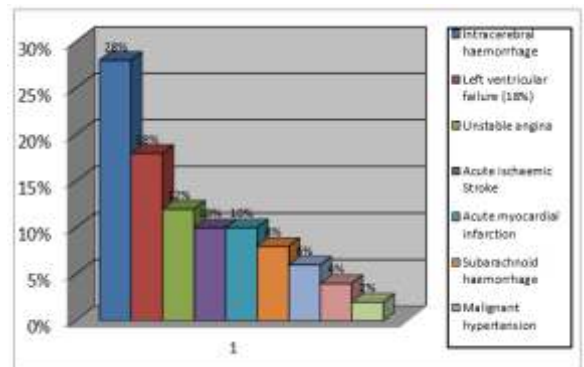


Figure 3 Target organ damage

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%).(Figure 10) Accelerated hypertension was seen in 18 patients (36 %) in the present study.

Out of 50 patients with hypertensive emergencies, 11 patients died before discharge. In- hospital mortality was (22 percent) (Figure 12).

DISCUSSION

In the present clinical study of hypertensive emergencies in Osmania General Hospital, Hyderabad, the number of males presenting with hypertensive emergencies were more than the number of females. 70% of the patients were males.

The proportions of males were higher when studying the group of patients less than 50 years of age. Majority of female patients belonged to the postmenopausal age group which shows susceptibility of postmenopausal age to end organ damage.

This is also due to the fact that postmenopausal female hemodynamics is not very much different from the male profile with regard to blood pressure.^{14,15}

Decade wise distribution of age shows largest groups belonging to the fifth and sixth decade at the time of presentation with 28% and 26% respectively.

Analyzing the presenting symptoms, the largest group of patients in the present study, presented with a neurological deficit, (50%) followed by dyspnoea (34 %) and chest pain. (30%) . This was similar to the study by Martin et al¹⁶, who in their study found presenting symptoms of neurological deficits, dyspnoea and chest pain in 48%, 25 % and 18 % of their patients. Zampaglione et al in their study had more patients presenting with chest pain (27 %) followed by dyspnoea (22 %) and neurological deficits (21%).

Neurological deficits in the present study varied from hemiparesis (80%), altered sensorium (96%), convulsions (12%), and visual deficits (12%). Hemiparesis accounted for the largest group of patients with neurological deficit.

In the present study 27% among the known hypertensives ignored their hypertensive status and discontinued antihypertensive medications which would have put them at a higher risk for acute target organ damage and hypertensive emergency.

These risk factors would have added to premature atherosclerosis and coronary artery disease in these patients predisposing them to acute target organ damage.

Metabolic abnormalities (hyperglycemia, hyperinsulinemia, and dyslipidemias) may play a role in the pathogenesis and complications of arterial hypertension, as seen in the present study.

Highest recorded systolic blood pressure was 280 mm Hg with mean systolic blood pressure of 216 ± 25 mm Hg. The highest diastolic blood pressure recorded was 180 mmhg with a mean of 126 ± 18 mm Hg. Martin et al in their study reports a mean systolic blood pressure of 193 ± 26 mm Hg in their patients and a mean diastolic blood pressure of 129 ± 12 mm Hg.

The mean reduction in blood pressure in one hour after admission to the hospital was 19 mm Hg of systolic blood pressure and 14 mm Hg of diastolic blood pressure. Blood pressure levels at admission were higher in the group of patients who expired compared to those who were discharged from the hospital. The higher levels of blood pressure would have added to more severe target organ damage in these patients, with an adverse outcome.

This indicates worse prognosis with a higher levels of blood pressure at presentation.

Evaluation of fundus revealed changes ranging from hypertensive retinopathy to papilloedema in 62 percent of patients. Papilloedema was seen in 24% of patients which is an evidence of ongoing target organ damage in these patients. Renal dysfunction in the form of raised serum urea and creatinine were seen in 24% and 18% of patients respectively. Renal changes amounting to hypertensive target organ

damage was seen in five patients. Hyponatremia was observed in 32 % of patients. 12 % of the patients had hypokalemia compared to 4% with hyperkalemia reflecting secondary aldosteronism from increased renin secretion induced by intra renal ischemia.

Microalbuminuria was seen in 36% of the patients which puts these patients at a higher risk for hypertension related renal disease compared to the patients without proteinuria.

Computed tomography of the brain showed intracerebral haemorrhage as the commonest cause for the neurological target organ damage followed by cerebral infarct and subarachnoid haemorrhage. Voltage criteria suggestive of left ventricular hypertrophy on ECG was seen in 20% of patients and 31% had left ventricular hypertrophy by echocardiography.

A study done by Lip GY et al¹⁷ showed low median survival time in patients with proteinuria and high serum urea and serum creatinine levels at presentation and if left ventricular hypertrophy was detected on electrocardiogram.¹⁶ Evaluation for target organ damage in patients in the present study showed intracerebral haemorrhage as the commonest cause (28 %) followed by left ventricular failure (18 %), unstable angina (12 %), acute myocardial infarction (10 %) acute ischaemic stroke (10 %), subarachnoid haemorrhage (8 %), malignant hypertension (6 %), acute myocardial infarction with left ventricular failure (4 %) and hypertensive encephalopathy (2 %).

Zampaglione et al³ in their study observed target organ damage in the form of Intracerebral haemorrhage (4.5%) left ventricular failure (23 %), Acute ischaemic stroke (24 %) in their patients.

Study by Martin et al⁷ shows Intracerebral haemorrhage (17%) left ventricular failure (25 %), Acute ischaemic stroke (39 %) and acute myocardial infarction in (8%) their patients.

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. Commonest mode of presentation is with a neurological deficit. Higher levels of blood pressure at presentation points towards a more adverse outcome. Hyponatremia and hypokalemia were common in patients with hypertensive emergencies. 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 was 22%.

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 emergency department patients. American journal of emergency medicine- vol 23(2) march 2005; 106110
3. Zampaglione et al. Hypertensive urgencies and emergencies. Hypertension 1996; 27: 144-147
4. Ventura et al. Desperate disease, desperate measures: Tackling malignant hypertension in the 1950s. Am Heart J 2001; 142: 197-203.
5. Elliot WJ. Hypertensive emergencies. In Critical Care Clinics Vol 17. Number 2 April 2001 W.B.Saunders company.
6. Veterans Administration Cooperative Study Group on Antihypertensive Agents. Effects of treatment on morbidity in hypertension. Results in patients with diastolic blood pressures averaging 115 through 129 mm Hg. JAMA. 1967; 202:1028-1034.
7. Martin J et al: Arquivos Brasileiros de Cardiologia - Volume 83, No 2, August 2004

8. Med 1992; 327: 248- 254.33. Philip H S, Stephen R P. Severely increased blood pressure in emergency department. *Ann Emerg Med.* 2003; 41:513-529.
9. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, et al. The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 Report. *JAMA.* 2003; 289: 2560-2572.
10. Leeman EP, Bodmen HG, Velta W, Epstein RH. Studies of blood pressure in school children of Zurich, Switzerland (abstract) International symposium on juvenile hypertension, Parma, June 4-6th 1979.
11. Harrison's Textbook of Internal Medicine, 18th edition.
12. Kawazoe N et al. Pathophysiology in malignant hypertension: with special reference to the rennin-angiotensin system. *Clin Cardiol;* 1987; 19:513-518
13. Platt, R.: Hypertension and Unilateral Kidney Disease. *Ort Jr Med,* 16:143.1947
14. Messerli FH et al. Disparate cardiovascular findings in men and women with essential hypertension. *Ann Intern Med* 1987; 107: 158-61.
15. Owens JF et al. Menopausal status influences ambulatory blood pressure levels and blood pressure changes during mental stress. *Circulation* 1993; 88: 2794-802.
16. Martin J et al: *Arquivos Brasileiros de Cardiologia* - Volume 83, No 2, August 2004.
17. Lip GY et al. Complication and Survival of 354 patients with malignant phase hypertension. *J Hypertens* Aug; 13(8) 915-24.