A CLINICAL STUDY ON HYPERTENSIVE EMERGENCIES

Dissertation submitted in partial fulfillment of regulation for the award

of

M.D. Degree in General Medicine

The Tamilnadu DR. M.G.R. Medical University

MAY 2012

DECLARATION

I here by declare that this dissertation entitled “A CLINICAL STUDY ON HYPERTENSIVE EMERGANCIES” is a bonafide and genuine research work carried out by me under the guidance of DR.CHANDRASEKARAN.S MD., Associate Professor, Coimbatore medical college, Coimbatore.

Date:

Place: DR.A.ALAGU THIYAGARAJAN

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation “A CLINICAL STUDY ON HYPERTENSIVE EMERGENCIES” is a bonafide research work done by Dr.ALAGU THIYAGARAJAN.A post graduate in M.D General Medicine under my direct guidance and supervision to my satisfaction, in partial fulfilment of the requirements for the degree of M.D General Medicine.

Date: PROFESSOR & UNIT CHIEF M-3

Date: PROFESSOR & HEAD OF DEPT.,

DEPT., OF GENERAL MEDICINE

Date: THE DEAN

COIMBATORE MEDICAL COLLEGE.

ACKNOWLEDGEMENT

I would like to express a hereditary gratitude to my guide and teacher Dr.chandrasekaran M.D Associate Professor of medicine, Coimbatore medical college, Coimbatore.The has been more than a guide to me, a good teacher a mentor and a source of encouragement throughout my course. I also thank him for his able guidance and supervision during the preparation of this dissertation.

It is my privilege and honour to extend my respect, regards and gratitude to Prof Dr.verakesari M.D head of the department of medicine, Coimbatore medical college, Coimbatore for all the encouragement and guidance and for the great teacher he is.

I wish to extend my sincere gratitude to all assistant professors Dr.T.Geetha M.D, Dr.Ushapadmini M.D, Dr.P.Balamurugan, Dr.Vetriveeran M.D in the department of medicine, Coimbatore

medical college for all the encouragement and for all they have taught me. I am indebted to them.

I would like to express my sincere thanks to THE DEAN,PROF. Dr.vimala M.D for his able guidance and encouragement. I am grateful to the director ,administrator and all the officials of Coimbatore medical for their help and permission to carry out this study availing all the required facilities at this institution.

I am grateful to all my colleagues present and past in the department of medicine for being the good friends they are and for the all the cooperation and help I received while preparing this dissertation.

I would like to express my sincere thanks to all patients for their kind co-operation.

Above all I would to thank the almighty for all this guidance and his blessing, to be in this noble profession rendering comfort to the suffering.

Date:

Place: DR.A.ALAGU THIYAGARAJAN

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 descriptive study was done at Coimbatore medical college over the period of one and half years.

The study population included patients admitted in 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 chestpain and dyspnoea, neurological deficit.

Majority of the patients were known hypertensives.

Higher levels of blood pressure at presentation were associated with an adverse outcome. Acute LVF was the commonest target organ damage observed. An in hospital mortality of 12% was observed in the present study.

INTERPRETATION AND CONCLUSION:

Known hypertensive are at a higher risk of presenting with acute target organ damage associated with a chestpain. Acute LVF is the commonest form of target organ damage encountered in the present study.

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ABBREVATIONS

A:	ABSENT
ACEI:	ANGIOTENSIN CONVERTING ENZYME INHIBITOR
ACS:	ACUTE CORONARY SYNDROME
ARB:	ANGIOTENSIN RECEPTOR BLOCKERS
ASMI:	ANTEROSEPTAL MYOCARDIAL INFARCTION
BB:	BETA BLOCKERS
CAHD:	CORONARY ARTERY HEART DISEASE
CCB:	CALCIUM CHANNEL BLOCKERS
CVA:	CEREBROVASCULAR ACCIDENT
COLES:	CHOLESTRELEMIA
LAFB:	LEFT ANTERIOR FASICULAR BLOCK
LVD:	LEFT VENTRICULAR DYSFUNCTION
LVH:	LEFT VENTRICULAR HYPERTROPHY
LVEF:	LEFT VENTRICULAR EJECTION FRACTION
MCA:	MIDDLE CEREBRAL ARTERY
NA:	NOT APPLICABLE
P:	PRESENT
RWMA:	REGIONAL WALL MOTION ABNORMALITY
ICH:	INTRACEREBRAL HAEMORRHAGE
SAH:	SUBARACHNOID HAEMORRHAGE
SHT:	SYSTEMIC HYPERTENSION
TGL:	TRIGLYCEREDEMIA
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INTRODUCTION

Hypertension affects individuals of all classes and across all the groups.The relationship between blood pressure and risk of cardiovascular disease events is continuous and independent of other risk factors.

It is the number one reason for an office visit to a physician; it accounts for the most drug prescriptions, it is a major risk factor for heart disease and stroke, which are the first and third leading causes of death in the developing countries and it is the number one attributable risk for death throughout the world.

At the same time, it is both preventable and treatable in the majority of patients. Despite these impressive statistics, hypertension continues to be neglected.

Hypertension is present in all populations. It has been estimated that hypertension accounts for 6% of deaths worldwide.

Hypertension doubles the risk of cardiovascular diseases, including coronary heart disease (CHD), congestive heart failure (CHF), ischemic and haemorrhagic stroke, renal failure, and peripheral arterial disease.

Hypertensive emergency can be an end result of chronic hypertension,non-compliance of drugs or initial presentation of essential hypertension.

It’s also seen that the incidence of hypertensive emergencies are increasing.

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

AIMS &OBJECTIVES

This study is to find out the various modes of presentation and clinical profile of hypertensive emergencies in Coimbatore medical college.

A number of cardiovascular,pulmonary and neurological symptoms are found to be associated with patients in hypertensive emergency with target organ involvement.

Due to association of hypertensive emergencies with various cerebral,cardiac and renal complications here is an need to recognise this condition so as to reduce the burden associated with it in terms of increased morbidity and mortality.

And probable cause for this hypertensive crisis also going o be evaluated.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Affecting 1 billion people worldwide, hypertension remains the most common, readily identifiable, and reversible risk factor for myocardial infarction, stroke, heart failure, atrial fibrillation, aortic dissection, and peripheral arterial disease.

Recent data from Framingham hert study suggest that individual who are normotensive at 55 years of age have a 90% lifetime risk for developing hypertension.[26]

Because of escalating obesity and population aging, the global burden of hypertension is rising and projected to affect 1.5 billion persons—one third of the world's population—by the year 2025. [2]

It was mahomed of guy's hospital London who in 1879 advocaed of kidney disease based on his personal observations and intuitive arguments.[27] practical measurement of blood pressure by clinician came f=some time later after the invention of the pneumatic arm cuff by riva-roccl of torino in 1896.[28]

Currently, high blood pressure (BP) causes about 54% of stroke and 47% of ischemic heart disease worldwide. Thus, high BP remains the leading cause of death worldwide and one of the world's great public health problems.

The asymptomatic nature of the condition delays diagnosis. The evidence for treaing severe hypertension continues to grow,which clearly shows that overall morbidity and mortality improves with treatment.[29]

Effective treatment requires continuity of care by a knowledgeable physician and frequent medical checkups, which are less common in men and low-income minorities.

For all these reasons, BP is controlled to a value below 140/90 mm Hg in less than one third of affected individuals, even in higher-income countries with the most advanced systems of health care.

The symptoms and signs of hypertensive crises are usually dramatic , likely reflecting acute damage to endothelium and platelet activation. However,

some patients may be relatively asymptomatic, despite markedly elevated pressure and extensive organ damage.

Young black men are particularly prone to hypertensive crisis, with severe renal insufficiency. Even in elderly persons, however, hypertension can initially present in an accelerated-malignant phase. If left untreated, patients die quickly of brain damage or more gradually of renal damage.

Before effective therapy was available, fewer than 25 percent of patients with malignant hypertension survived 1 year and only 1 percent survived 5 years. With therapy, including renal dialysis, more than 90 percent survive 1 year and about 80 percent survive 5 years

Research shows that about 1% of hypertensive patients contract hypertensive crisis,11,12 which could be a life-threatening event.[21,22]

Recent clinical trials have demonstrated that effective BP control can be achieved in most patients who are hypertensive, but the majority will require two or more antihypertensive drugs.[45,46]

Staging of Blood Pressure

According to the 2003 guidelines of the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7), which are still in effect until the publication of JNC 8, BP is staged as normal, prehypertension, or hypertension by the average of two or more readings taken at two or more office visit.[1]

Staging of Blood Pressure (JNC7)

table 1:

BP STAGE	SYSTOLIC BP (mm Hg)	DIASTOLIC BP (mm Hg)
Normal	<120	<80
Prehypertension	120-139	80-89
Stage 1	140-159	90-99
Stage 2	≥160	≥100

Hypertensive emergency:

Hypertensive emergency is defined as the association of an extremely elevated blood pressure with physical or laboratory findings that indicate acute or ongoing target organ damage.[5]

It is important to note that no level of BP is given that automatically demands emergent treatment unless associated with target organ damage.[1]

Clinical Characteristics of Hypertensive Crisis [24]

- Blood pressure—usually > 140 mm Hg diastolic
- Funduscopic findings—hemorrhages, exudates, papilledema
- Cardiac findings—prominent apical impulse, cardiac enlargement, congestive heart failure
- Renal findings—oliguria, azotemia
- Gastrointestinal findings—nausea, vomiting
- Hematological findings—microangiopathichemolysis

Pathogensis: [25]

Early studies in animals and humans by Strandgaard and Paulson have elucidated the mechanism of hypertensive encephalopathy.

First, they directly measured the caliber of pial arterioles over the cerebral cortex in normotensive cats whose BP was varied over a wide range of infusion by vasodilators or A II. As the pressure fell, the arterioles became dilated; as the pressure rose, they became constricted.

Thus, constant cerebral blood flow was maintained by means of autoregulation, which depends on the cerebral sympathetic nerves. However, when mean arterial pressure rose above 180 mm Hg (i.e., 220/110), the tightly constricted vessels could no longer withstand the pressure and suddenly dilated.

Dilation began in an irregular manner, first in areas with less muscle tone and then diffusely, with production of generalized vasodilation. This breakthrough of cerebral blood flow hyperperfuses the brain under high pressure and thereby causes leakage of fluid into the perivascular tissue, resulting in cerebral edema and the syndrome of hypertensive encephalopathy.

Hypertensive urgency:

Hypertensive urgency is a situation with markedly elevated blood pressure but without severe symptoms or progressive target organ damage.[2]

These patients must be carefully evaluated and monitored for hypertension induced heart and kidney damage and for identifiable cause for hypertension.

Chronic hyperesives who do not adhere to therapy may have blood pressures much higher that 230/130 mm Hg without acute target organ damage and therefore may be treated as hypertensive urgency with reinstituting drug therapy.[30]

The term malignant hypertension has been emphasized in reference to the association with encephalopathy or nephropathy with retinopathy.[31]

Incidence:

Zampglione et al in their study on prevalence and clinical presentation of hypertensive urgencies and emergencies noticed that hypertensive crisis account

for 27% of all medical emergencies in an emergency department,out of which 24% fall in the category of hypertensive emergencies. [6]

Sobrino j et al studied prevalence,forms of clinical presentation and treatment of arterial hypertension noted that 19.5% of the patients studied by them presented with an hypertensive emergency. [9]

Martin et al in their study"hypertensive crisis prfile prevalence and clinical presentations",differed in their finding in that hay found the prevalcne of hypertensive crisis to be 1.7% of all clinical emergencies ou of which 39.6% being hypertensive emergencies.[32]

It has been observed taht approximately only less than 1% of the diagnosed hypertensive [atients experiences hypertensive urgency or emergency situations.[33]

Bennet et al in thiestudy on hypertensive emergencies found that 93% of patients presenting in hypertensive emergencies had previously been diagnosed as having chronic hypertension,a dn concluded tah improved management of

pre-existing known hypertension could lower the incidence of hypertensive emergencies.[34]

Adequate controm of hypertension remains elusive one important reason being the asymptomatic nature of the disease for the first 15-20 years even as it hypertension and associated coronary vascular disease seems to be increasing.[35]

The situation in the Indian subcontinent and rest of the developing countries is different from hat of the western world. Where the burden offered by hypertension and associated coronary vascular disease seems to be increasing.[7]

Incidence may be dependent on drug withdrawal,use of intoxicants and due to other precipitating factors.[36]

The number of known hypertensives who discontinued their antihypertensive medications were 12.75% in the study by cerillo et al [13] and 37.5% and 25% in studies by sobrino et al and zampglione et al respectively.

Hypertension in older persons

Hypertension occurs in more than two-thirds of individuals after age 65.[54] This is also the population with the lowest rates of BP control.[53]

Treatment recommendations for older people with hypertension, including those who have isolated systolic hypertension, should follow the same principles outlined for the general care of hypertension. In many individuals, lower initial drug doses may be indicated to avoid symptoms; however, standard doses and multiple drugs are needed in the majority of older people to reach appropriate BP targets.

Hypertension in women

Oral contraceptives may increase BP, and the risk of hypertension increases with duration of use. Women taking oral contraceptives should have their BP checked regularly.

Development of hypertension is a reason to consider other forms of contraception. In contrast, menopausal hormone therapy does not raise BP.[55]

Modes of presentation:

Different manifestation of Hypertensive emergencies: [19]

- Hypertensive encephalopathy
- Acute aortic dissection
- Acute myocardial infarction
- Acute coronary syndrome
- Pulmonary oedema with respiratory failure
- Severe pre-eclampsia, HELLP syndrome, eclampsia
- Acute renal failure
- Microangiopathichemolyticanemia
- HELLP, Hemolysis, elevated liver enzymes, low platelets.

A number of cardiovascular,pulmonary and neurological symptoms are found to be associated with patients in hypertensive emergencies due to the presence of acute ongoing target organ damage.

Focal neurological deficits,dyspnoea,chestpain,headache and loss of vision are considered as the commonest symptoms with which patients in hypertension related acute target organ damage.

Chestpain(27%) was the commonest symptom zampgloine et al noticed in their study,followed by dyspnoea(22%) and neurological deficit(21%).

Study by martin j et al differs from the above mentioned in that they noticed a neurological deficit(48%) as the commonest mode of presentation followed by dyspnoea(25%) and chest pain in(18%).

Hypertensive Heart Disease

Hypertension may contribute to CAD more than is commonly realized because hypertensives have more silent ischemia and unrecognized myocardial infarctions, and patients with acute myocardial infarction often have pre-existing hypertension that evaded detection or treatment.

There have been tremendous advances in our understanding of the cell and molecular mechanisms underlying pressure overload cardiomyocyte hypertrophy.Moreover, the structural abnormalities in the hypertensive heart are not limited to myocyte hypertrophy. They also include medial hypertrophy of the intramyocardial coronary arteries as well as collagen deposition leading to

cardiac fibrosis. These changes result in part from pressure overload and in part from the neurohormonal activation that contributes to hypertension.

In the hypertrophied hypertensive heart, coronary blood flow is normal at rest but vasodilator reserve becomes impaired when myocyte mass outstrips the blood supply. Even in the absence of CAD, coronary vasodilator reserve is blunted or absent, leading to subendocardial ischemia under conditions of increased myocardial oxygen demand. The combination of subendocardial ischemia and cardiac fibrosis impairs diastolic relaxation, leading to exertionaldyspnea and diastolic heart failure.

Before the advent of effective drug therapy for hypertension, beginning in the late 1950s, heart failure caused most deaths from hypertension. With better management of hypertension, there has been a dramatic reduction in hypertension-related deaths from heart failure and a significant delay in its onset.

Hypertension remains the most common cause of diastolic heart failure. In addition, hypertension indirectly leads to systolic heart failure by virtue of its being a major risk factor for myocardial infarction. It is unclear whether mild or

moderate hypertension alone, without myocardial infarction, leads to systolic heart failure.

Pre-existing hypertension increases the case-fatality rate associated with an acute myocardial infarction and substantially increases the risk of haemorrhagic stroke during thrombolytic therapy, especially when systolic BP exceeds 175 mm Hg.

On the electrocardiogram, LVH with strain is a serious harbinger of new-onset heart failure and heart failure death.

Echocardiography detects LVH more sensitively than electrocardiography does.

Large-Vessel Disease

Hypertension also constitutes a major risk factor for majority of patients with aortic dissection (distal more than proximal dissection), abdominal aortic aneurysm, and peripheral arterial disease.

Cerebrovascular Disease

Hypertension is a major risk factor for stroke and dementia.[1]

Hypertensive encephalopathy is thought o be due to cerebral oedema resulting from a failure of cerebral blood flow auto regulation.[15]

Hypertension accounts for 50% of strokes. In hypertensives, 80% of strokes are ischemic (thrombotic or embolic) and 20% are haemorrhagic.

The risk of hemorrhagic stroke is increased in patients with uncontrolled hypertension.[56]

In normal individuals,cerebral blood flow remains fairly constant for a mean arterial pressure from approximately 60mm hg to up to 150mmhg. When the meanarterial pressure decreased to less than the lower limits of autoregulation. The brain becomes hypoperfused and cerebral hypoxia occurs. With symptoms such as dizziness,nausea and syncope.

In chronically hypertensive individuals,the lower limit of autoregulation is increased and autoregulation might fail at mean arterial pressures that are well tolerated in nonhypertensive individuals.[9]

It has been observed that he lower limit of the autoregulation curve tends to be approximately 25% of mean arterial pressure.This has led to the general recommendation that the mean arterial pressure be acutely decreased by no more than 20% to 25%.[10,16]

When diastolic blood pressure exceeds 140 mm Hg, rapidly progressive damage to the arterial vasculature is demonstrable experimentally, and a surge of cerebral blood flow may rapidly lead to encephalopathy. [24,38]

Dementia and cognitive impairment occur more commonly in people with hypertension. Reduced progression of cognitive impairment may occur with effective antihypertensive therapy.[58,59]

Intracerebral haemorrhage is more than twice as common as subarachnoid haemorrhage and is much more likely o result in death or major

disability than cerebral infarction or SAH advancing age and hypertension are the most important risk factors for ICH.[12,17]

There are reports of adverse outcomes with acutely decreasing the blood pressure in hypertensive patients by using short acing anti hypertensive drugs.[39,40]

Chronic Kidney Disease

Hypertension follows only diabetes as a risk factor for CKD.

Microalbuminuria is a sensitive early marker of kidney damage and a powerful independent predictor of cardiovascular complications from hypertension.

Most patients with hypertension-associated CKD die of heart attack or stroke before renal function deteriorates sufficiently to require chronic hemodialysis.

The Benefits Of Treating Hypertension

It is well established that treating hypertension reduces the rate of strokes by 35 to 40%, coronary heart disease events by 20 to 25%, and congestive heart failure by up to 50%, and that the benefits of antihypertensive treatment are more closely related to the change of blood pressure than how it is lowered.[1,48]

In the majority of patients, controlling systolic hypertension, which is a more important CVD risk factor than DBP except in patients younger than age 50.[49]

Effects of Hypertension Treatment on Morbid Events

Event	Average % Reduction
Stroke	35–40%
Myocardial infarction	20–25%
Heart failure	50%

The benefits of treating chronic hypertension were clearly identified by the historic placebo-controlled VA cooperative study published in 1967,showing that blood pressure decreasing drugs markedly reduce

cardiovascular morbidity and mortality[3]. The evidence base for treating severe hypertension continues to grow,which clearly shows that overall morbidity and mortality improves with treatment.[4]

Drug non compliance with prescribed medications and inadequate therapy are the most common causes for hypertensive emergency in the primary hypertension.[20]

Factors Related to Poor Blood Pressure Control

There are multiple reasons, ranging from physiologic to societal, why blood pressure control is not better. They can be divided into three general categories: factors related to the patient, the healthcare provider, and the healthcare system.

FACTORS RELATED TO POOR CONTROL OF HYPERTENSION

Table 2:

Patient	***Physician***	***Health Care System***
Older age	Therapeutic inertia	Poor availability of care
Resistant hypertension	Inappropriate choice of drugs	
Poor adherence	Lack of knowledge	
Poor access to care		

History and Physical Examination and Laboratory Evaluation

The three main goals of the initial evaluation of the hypertensive patient are to

(1) assess the presence of target-organ damage related to hypertension, especially those that might influence choice of therapy

(2) determine the presence of other cardiovascular risk factors and disease

(3) evaluate for possible underlying secondary causes of hypertension. These goals are usually accomplished by a thorough medical history, physical examination, and simple laboratory investigations.

History

The key issues that need to be addressed in the history include:

- Duration, age of onset, and previous levels of high blood pressure
- Previous antihypertensive therapy, its impact on blood pressure and adverse effects
- Symptoms suggestive of secondary causes of hypertension
- Lifestyle factors, such as dietary intake of fat, salt, alcohol, smoking, and physical activity, weight gain since early adult life
- History of symptoms of neurologic dysfunction, heart failure, coronary heart disease, or peripheral arterial target-organ damage
- Use of medications that influence blood pressure such as oral contraceptives, cocaine, amphetamines, steroids, nonsteroidal anti-inflammatory drugs, erythropoietin, and cyclosporine.

Physical Examination:

In addition to blood pressure measurement, the physical examination should search for signs of secondary hypertension and for evidence of organ damage.

Blood pressure measurement technique:[24]

In the office, BP should be measured at least twice after 5 minutes of rest, with the patient seated in a chair, the back supported, and the arm bare and at heart level.

A large adult-sized cuff should be used to measure BP in overweight adults because the standard-sized cuff can spuriously elevate readings.

Tobacco and caffeine should be avoided for at least 30 minutes. BP should be measured in both arms to exclude coarctation of the aorta and after 5 minutes of standing, the latter to exclude a significant postural fall in BP, particularly in older persons and in those with diabetes or other conditions (e.g., Parkinson's disease) that predispose to autonomic insufficiency.

An individual's BP varies widely throughout a 24-hour period and is therefore impossible to characterize accurately except by repeated measurements under various conditions. Out-of-office readings are the only way to obtain a clear picture of a person's usual BP for accurate diagnosis and management.

Ambulatory monitoring provides automated measurements of BP over a 24-hour period while patients are engaging in their usual activities, including sleep. [60]

Prospective outcome studies in both treated and untreated patients have shown that ambulatory BP measurement predicts fatal and nonfatal myocardial infarction and stroke better than standard office measurement.

Other signs, such as carotid bruits; motor or sensory defects; fundoscopic abnormalities; abnormal cardiac rhythms; ventricular gallop; pulmonary rales; dependent oedema; and absence, reduction, or asymmetry of pulses and cold extremities, may suggest end organ damage.

WHITE COAT HYPERTENSION.

Up to one third of patients with elevated office BPs have normal home or ambulatory BPs. If the daytime BP is below 135/85 mm Hg (or preferably below 130/80 mm Hg) and there is no target organ damage despite consistently elevated office readings, the patient has “office-only” or “white coat” hypertension, presumably caused by a transient adrenergic response to the measurement of BP only in the physician's office.

BP self measurements may benefit patients by providing information on response to antihypertensive medication, improving patient adherence with therapy.[47]

Keith et al described the course and prognosis of patients with hypertension according to he degree of retinopathy. These patients were divided into four groups (1-4).Patients i group 1 had survival of 70% at seven years;at the same time. Group 4 had a 90% morality at 1.5 years.[18]

Risks Influencing Prognosis in Patients with Hypertension

- ✓ Risk Factors for Cardiovascular Disease
- ✓ Levels of systolic and diastolic blood pressure
- ✓ Age (yr)—men >55; women >65
- ✓ Smoking
- ✓ Dyslipidemia
- ✓ Family history of premature cardiovascular disease
- ✓ Abdominal obesity
- ✓ Diabetes mellitus
- ✓ C-reactive protein ≥1 mg/dl.

<u>Laboratory Tests:</u>

Routine investigations before initiation of therapy include urine for protein and blood; serum creatinine (estimated glomerular filtration rate [GFR]) and electrolytes; fasting blood glucose; fasting lipid profile; and electrocardiogram (ECG).

Evaluation of renal status may indicate oliguria and azotemia.[37]

Generally, it is not necessary to do more extensive tests unless blood pressure control is not achieved or there are clinical or laboratory clues of secondary hypertension.

Echocardiography is also more sensitive than electrocardiography in identifying left ventricular hypertrophy. Left ventricular hypertrophy is identified by electrocardiography in only 5% o 10% of hypertensives. Whereas LVH is found by echocardiography in nearly 30% of unselected hypertensive adults and in upto 90% of patients with severe hypertension.[11,14]

Risk Assessment:

The key risk factors which need to be considered are age (>55 years male, >65 years female), family history of premature cardiovascular disease (age <55 in men, age <65 years in women), cigarette smoking, dyslipidemia, diabetes, obesity (body mass index >30 kg/m2), reduced GFR (<60 mL/min), or presence of microalbuminuria.

Whereas hypertension is present in 25 percent of adults in the general population, it is present in 75 percent of adults with diabetes and over 90 percent of those with chronic kidney disease. Either of these two comorbidities dramatically increases the cardiovascular risk associated with hypertension, and the presence of hypertension greatly accelerates the progression to end-stage renal disease.

The 2003 JNC 7 guidelines have recommended a usual BP of 140/90 mm Hg as the threshold for initiating antihypertensive medication in most patients, with a lower threshold of 130/80 mm Hg for high-risk patients with diabetes or chronic kidney disease.[50,51]

Serum potassium and creatinine should be monitored at least 1–2 times/year.[52]

Followup and Monitoring[52]

Once antihypertensive drug therapy is initiated, most patients should return for followup and adjustment of medications at approximately monthly intervals until the BP goal is reached. More frequent visits will be necessary for patients with stage 2 hypertension or with complicating comorbid conditions.

Prognosis:

Depending upon risk factors and associated co-morbid conditions and drug compliance of the patients. There are numerous medications available for treatment,these improvement have led to decrease in one year mortality rate.[2]

A patient-centered strategy to achieve the goal and an estimation of the time needed to reach goal are important in the prognosis.[57]

Lip G.Y, bevers D.G investigated the factors affecting the survival in patients with malignant hypertension. Hte most common causes of death were renal failure,sroke which was followed by myocardial infarction and heart failure.[41]

MATERIALS AND METHODS

MATERIALS AND METHODS

The present study was done in patients admitted to Coimbatore medical college,Coimbatore,tamilnadu over a period ofone and half years.

Selection criteria:

Inclusion criteria:

- Patients above the age of 18 years.
- Systolic blood pressure of 180mmhg or diastolic blood pressure of 110mmhg
- Evidence of target organ damage,either clinically or on laboratory findings.

Exclusion criteria:

- Patients less than 18 years.
- Chronic renal failure,valvular heart disease,other secondary causes of hypertension.

Source of data:

Fifty patients admitted to Coimbatore medical college hospital with clinical and laboratory evidence of hypertensive emergency.

Type of study:

Descriptive study

Study protocol:

Data was collected from fifty patients admitted in this hospital from over aperiod of one and half years.

Patients who presented with an elevated blood pressure of systolic blood pressure of >180mmhg or diastolic blood pressure >110mm Hg,with history 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 sympotomalogy,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.

Detailedclinical examination was done in these patients with examination of respiratory system,cardiovascularsystem,abdomen and central nervous system.

Clinical examination also included fundoscopic examination in all the patients.Blood samples of these patients were evaluated for biochemcial abnormalities.

The routine investigation done in these patients were the HB%, totalcount, differential count, ESR, bloodsugar, serumurea,serum creatinine ,serum electrolytes,serum total cholesterol, serum triglycerides, HDL, LDL, microalbunuria and urine analysis.

All the patients also underwent chest x-ray,urineanlaysis and serum electrolyes.

Patients with clinical suspicion of neurological deficits were evaluated with computed tomography of the brain,patients with cardiovascular dysfunction clinically were evaluated with ECHO and patient with renal dysfunction underwent renal sonography.

The collected data was analysed using Microsoft excel software.

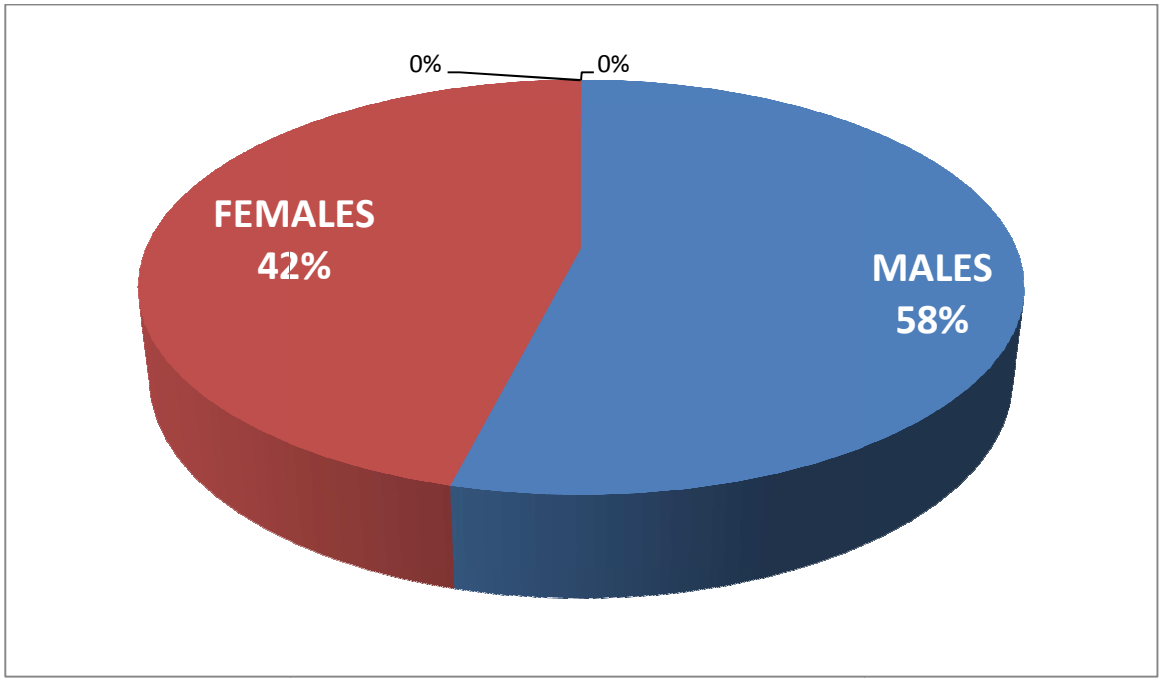
RESULTS & ANALYSIS

RESULTS & ANALYSIS

Results and analysis:

Among the fifty patients in the present study,29 (58%) were males. The male:female ratio is almost 1.4:1.

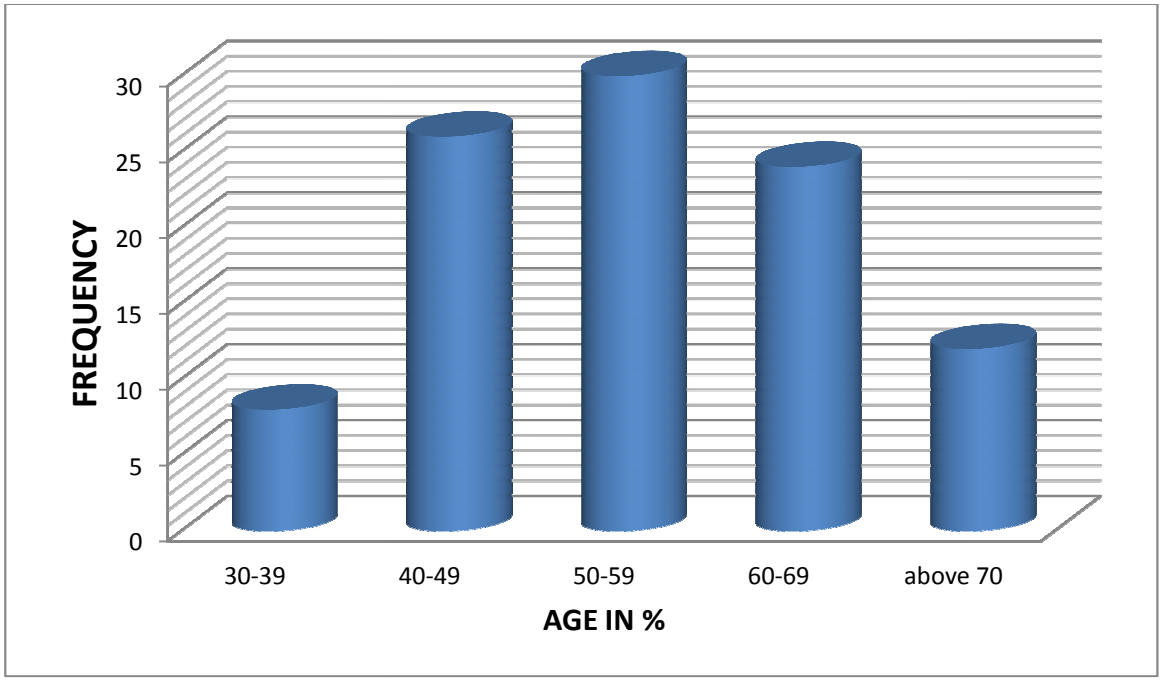
Figure 1:sex distribution



The mean age of the patients was 58.The age varied from 38 to 70 in males and 39 to 78 in females. The mean age for males and females were 54 and 58.5 years respectively. The age distribution is given in table 2 below.

In the age distribution patients,patients less than age of 50 were 34%(17 pts) and more than 50 years were 66%.

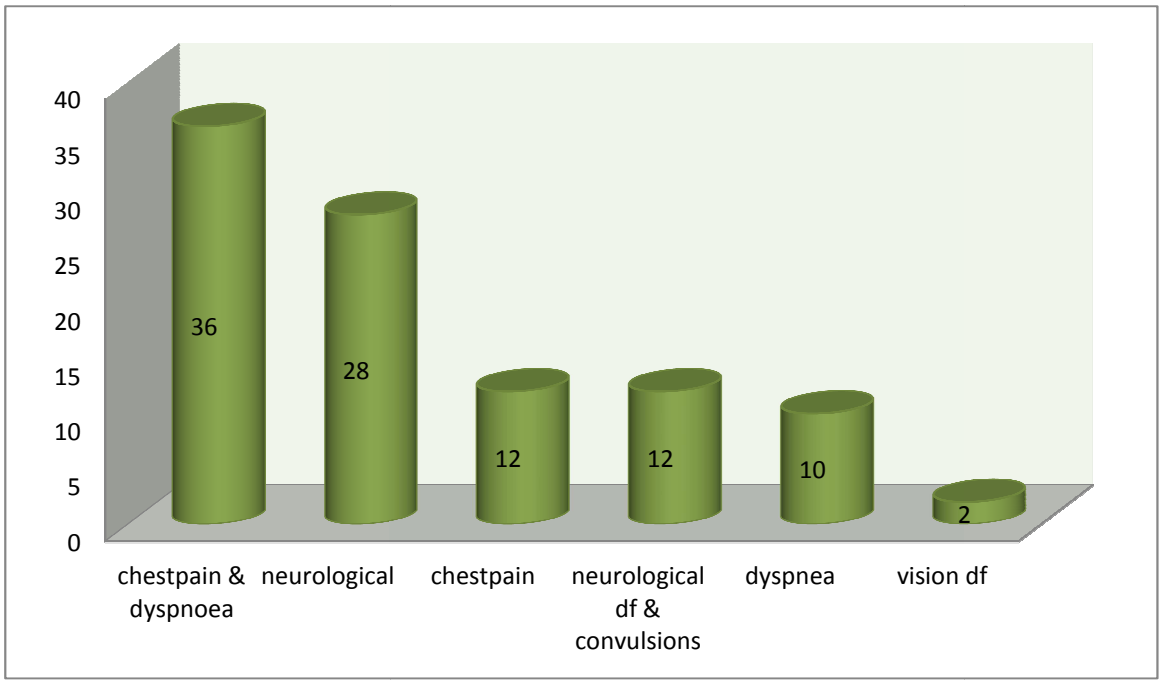
Figure 2: age distribution



In the present study the presenting symptoms in these patients were chestpain& dyspnoea.

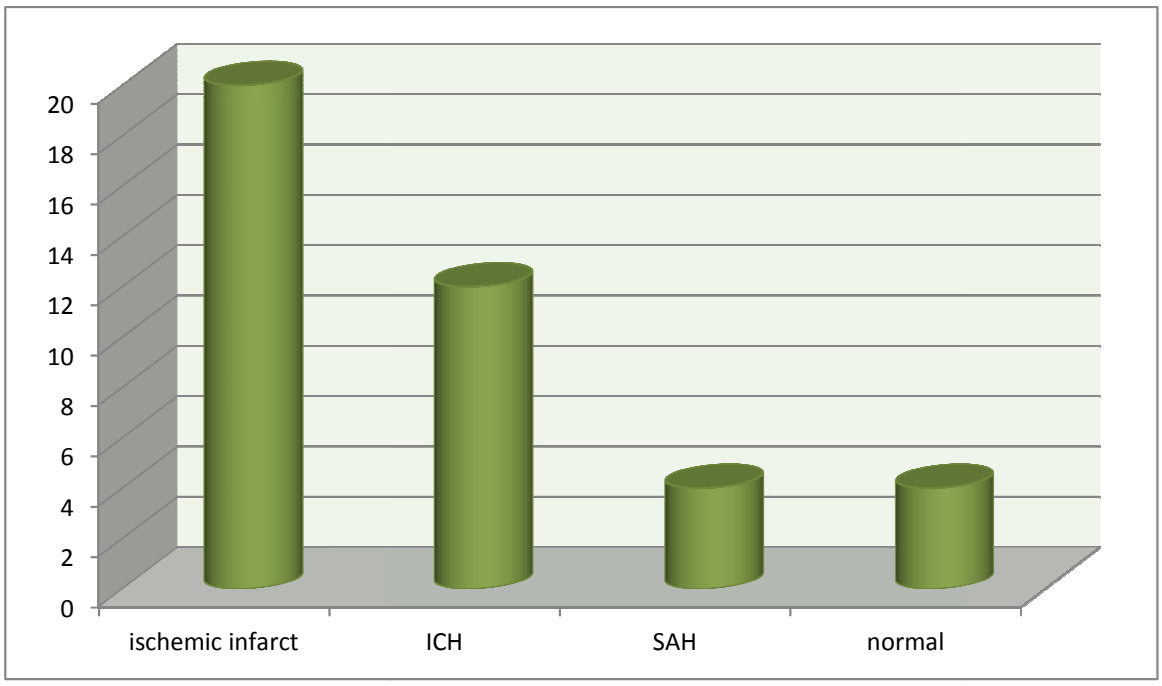
The commonest presenting complaints were chestpain&dyspnoea(36%) and followed by neurological symptom(28%).

Figure 3:presenting symptoms



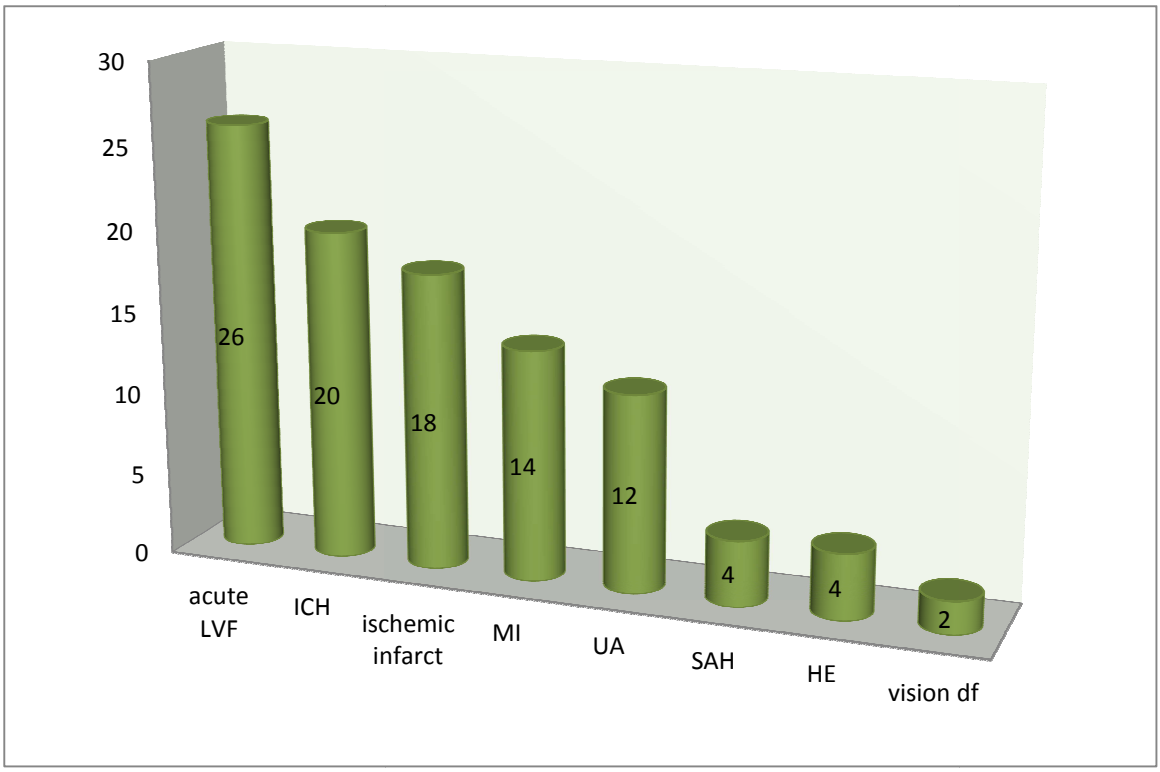
Among patients with neurological deficit,neurological damage included ischemic infarct(20%),intra cerebral haemorrhage(12%),SAH(4%).

Figure 4:neurological manifestation in CT-brain



Among the target organ involvement acute LVF(26%), ICH(20%),ischemic infarct(18%),MI(14%),Unstable angina(12%), SAH(4), hypertensive encephalopathy(4%),vision defect(2%).

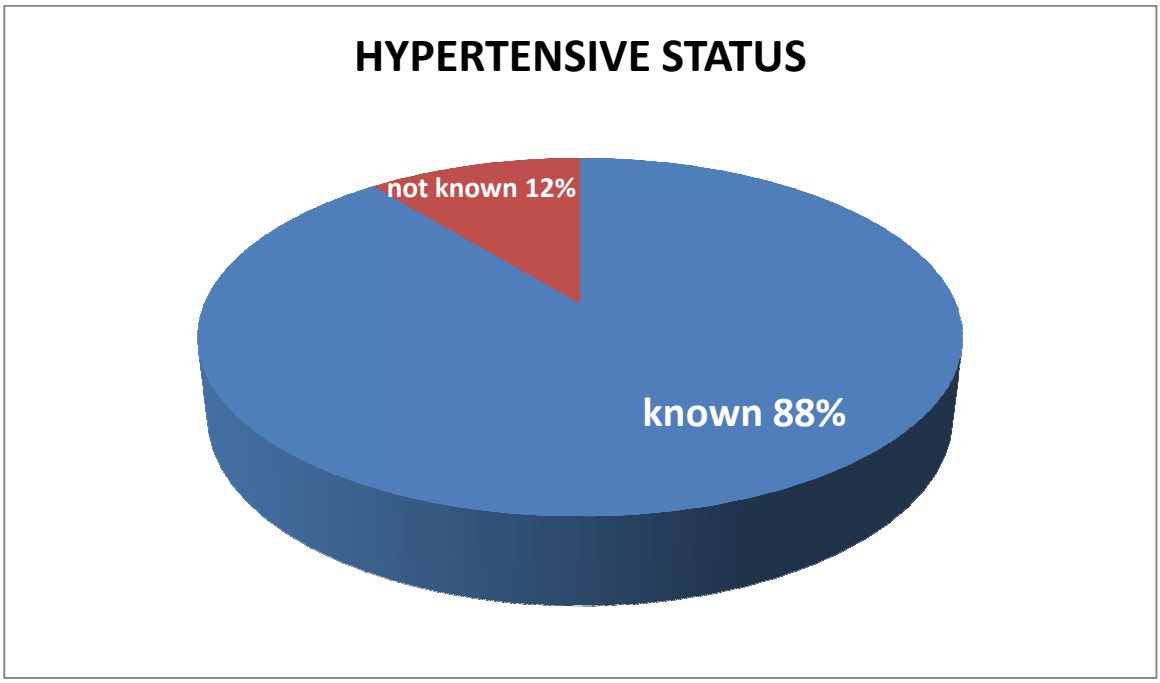
Figure 5:target organ involvement



Hypertensive status:

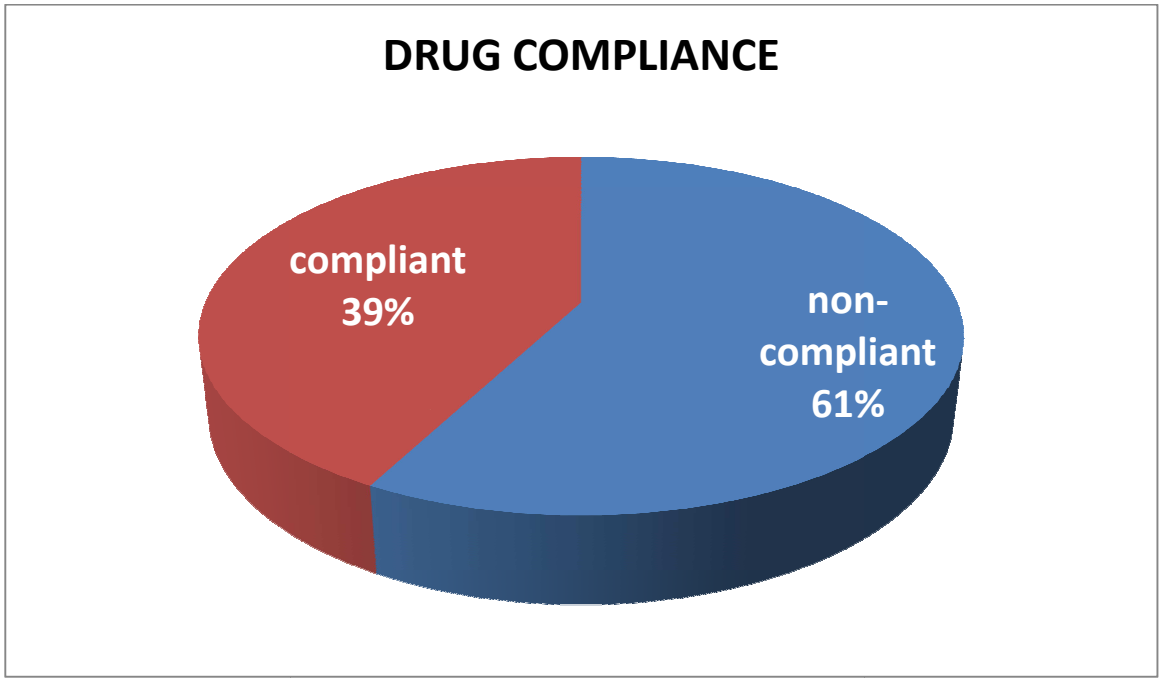
Among the patients 44 patients (88%) were known hypertensives,remaining patients(12%) were not a known hypertensive.

Figure 6:hypertensive status



Of the 50 patients studied 44 patients were known hypertensives,among them 27 patients(61%) were discontinued antihypertensives before the incident,remaining 17 patients(39%) were continued their medication.

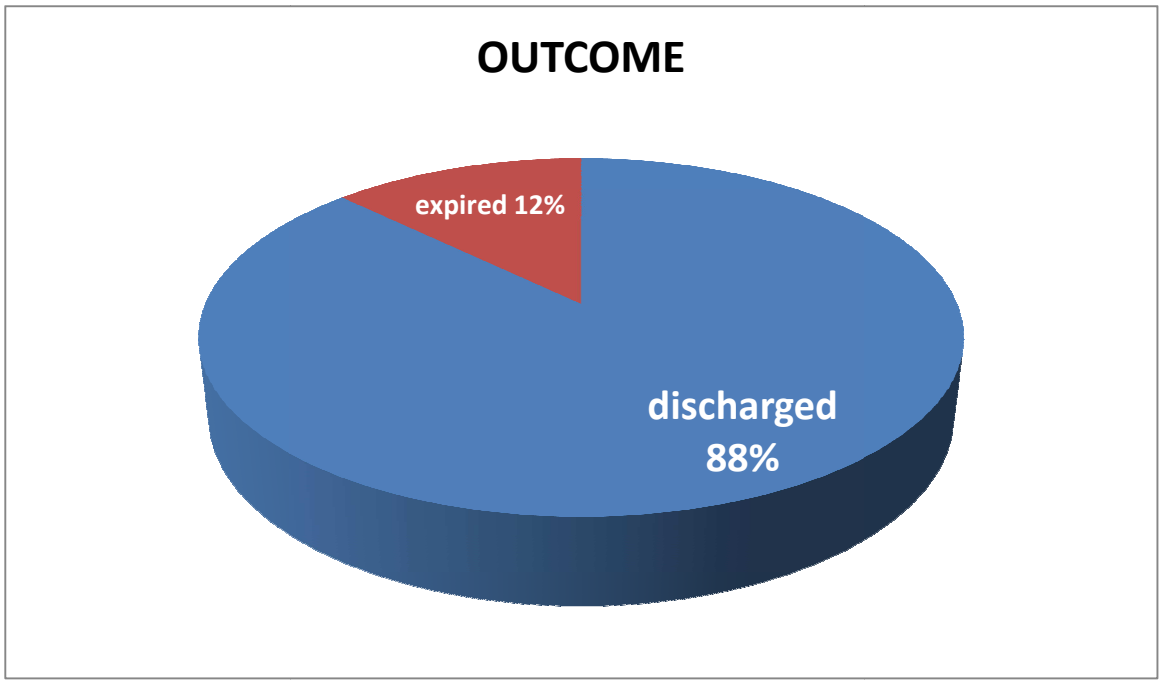
Figure 7: drug compliance



Among the 50 patients 14 patients(28%) were having diabetes mellitus and 14 patients(28%) had dyslipidemias.

Out of 50 patients with hypertensive emergencies,6 patients died before discharge. Hospital mortality was 12%. All expired patients were had intracerbral haemorrhage.

Figure 8:outcome



DISCUSSION

In the present clinical study of hypertensive emergencies in Coimbatore medical college,Coimbatore there is mild male predilection for hypertensive emergencies.

Marin et al in their study on hypertensive crisis observed that 55% of patients were males among patients with hypertensive emergencies. The proportion of males in hypertensive emergencies were also higher in the study by zampoglione et al. This is probably due to an increased susceptibility of males compared with females to hypertension related target organ damage.

This also due to the fact that postmenopausal female hemodynamics is not very mush different from the male profile with regard o blood pressure.[42,43]

The majority of female patients belonged to the postmenopausal age group which shows susceptibility of postmenopausal age to end organ damage.

Decade wise distribution of age shows largest groups belonging o the fifth and sixth decade at the time of presentation with 30% and 26% respectively.

Analysing the presenting symptoms, the largest group of patients in the present study,presented with a chestpain and dyspnoea and followed by neurological deficit.

Zampglione et al in their study had more patients presenting with chestpain(36%) and neurological deficit 28% respectively.

Majority of patients in the present study were previously known hypertensives (88%).martin et al noticed a large a large number of patients (83%) in their study to be previously diagnosed hypertensives.

Zampaglione et al reports a large number, with(92%) of known hyperensives among their patients. This evidence confirms that hypertensive emergencies were higher in patients with previously known hypertension. This also shows that patients with hypertension are at a higher risk of developing a hypertensive emergency,more so if they do not adhere to the antihypertensive therapy.

A. Rafighdoost MD, et al study showed discontinuing of medications to be of utmost importance in precipitating hypertensive crisis. 75% of the subjects had stopped medications and only 25% had continued them.[23]

In the present study 61% among he 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.

Diabetes mellitus and dyslipidemia were the other risk factors present in the present group of patients. Patients with diabetes mellitus and dyslipidemia were 28% and 28% respectively.

Prevalence of arterial hypertension in diabetic patients is greater when compared with that in non diabetic patients(40-50% and 20% respectively).[44]

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

Highest recorded systolic blood pressure was 250mm hg with mean systolic blood pressure of 215mm Hg. The highest diastolic blood pressure recorded was 160mm hg with a mean of 130 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 0f 129+/- 12mm hg.

The higher levels of blood pressure would have added to more severe target organ damage in these patients with an adverse outcome.

Evaluation of fundus revealed changes ranging from hypertensive retinopathy to papilloedema in 50% of patients.Papilloedemawas seen in 12 % of patients which is an evidence of ongoing target organ damage in these patients.

Microalbunuria was seen in 32% 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 18% had left ventricular hypertrophy by echocardiography.

A study done by lip GY et al on complications and survival of 315 patients with malignant hypertension found 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. These findings in patient in hypertensive emergency situation may help in prognosticating these patients.

Evaluation for target organ damage in patients in the present study showed acute LVF(26%) followed by intracerebral haemorrhage(20%),ischemic

infarct(18%),acute myocardial infarction (14%),Sub arachnoid haemorrhage(4%),hypertensive encephalopathy (4%) and vision defect(2%).

Zampglione et al in their study observed target organ damage in the form of intracerebralhaemorrhage(4.5%) left ventricular failure(23%).acute ischemic stroke(24%) in their patients.

Study by Martin et al shows intracerebralhaemorrhage(17%) left ventricular failure (25%),acute ischemic stroke(39%) and acute myocardial infarction in(8%) their patients.

The outcome of the study showed an in hospital mortality of 12% among these patients. All patients who were expired had intracerebral haemorrhage.

CONCLUSION

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

- ✓ Males have higher chances of developing hypertensive emergencies compared in females.

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

- ✓ Presence of diabetes mellitus and dyslipidemia increases the chance of developing hypertensive emergency.

- ✓ Commonest mode of presentation is with a chestpain and dyspnoea.

- ✓ Higher levels of blood pressure at presentation is with a neurological deficit.

- ✓ Acute LVF is the commonest form of target organ damage encountered in the present study.

- ✓ The in-hospital mortality among these patients with hypertensive emergency were 12%. Cause for all was mortality was due to intracranial haemorrhage.

SUMMARY

The clinical profile of patients with hypertensive emergencies presenting in hospitals in a developing country like ours is poorly known.

The present study is a descriptive study done a Coimbatore medical college,Coimbatore,over a period of one and half years.

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.

The commonest presenting symptoms were chestpain and dyspnoea and neurological damage.

Majority of the patients were known hypertensives which was also seen in other similar studies and confirms that known hypertensives have higher risk of developing hypertensive emergencies.

Higher levels of blood pressure at presentation were associated with an adverse outcome. Acute LVF was the commonest target organ damage observed.

An in-hospital mortality of 12% was observed in the present study. Cause for all was mortality was due to intracranial haemorrhage.

BIBLIOGRAPHY

1. Joint national committee on prevention detection and treatment of blood pressure. The seventh report of joint national committee detection and treatment of high blood pressure. JAMA 2003:289:2560-2572.
2. Henry R.B.,George L.B.,William J.systemic arterial hypertension.HURST's-The heart edition,mcgraw-hill 1589-1590.
3. Veterans administration cooperative study group on antihypertensive agents. Effects of treatment on morbidity in hypertension. Results in patients with diastolic blood pressure averaging 115nthrogh 129 mmhg.JAMA.1967:202:1028-1034.
4. Elliot WJ.hypertensive emergencies. In critical care clinics vol 17. Number 2 April 2001 W.B. Saunders company.
5. Singh WG. Aslam N. Hypertensive emergencies-hypertension an international monograph 2001:350.IJPC.
6. Zamapglione et al hypertensive urgencies and emergencies. Hypertension 1996:27:144-147.
7. J .michael gaziane. Global burden of cardiovascular disease in heart disease 6th ed.braunwald,zips,libby,HIEsaunders.PP 1-17.
8. Sobrino j et al prevalence. Forms of clinical presentation and treatment of arterial hypertension at an emergency unit. Rev cli Esp;187(2):56-60 June.

9. Webster j et al. Accelerated HTN-patterns of mortality and factors affecting outcome in treated patients. QM. 1993;86;485-493.
10. Lau et al .cumulative meta analysis of therapeutics trilas for MI; N eng j med 1992;327:248-254.
11. Shapiro LM,beevers DG. Malignant haemorrhage is more than twice as common as subarachnoid haemorrhage. J neurosurg.1993;78:188-191.
12. Murphy C. Hypertensive emergencies. Emerg med clinnorh am. 1995 nov;13(4);973-1007.
13. Cerillo MR et al hypertensive crisis. Prevalence and clinical aspects. rev cli esp 2002;202;255-8.
14. Nadar et al. Echo cardiographic changes in patients with malignant phase hypertension: J humhypertens.2005.
15. Broderick JP et al. Guidelines for the treatment of spontaneous ICH haemorrhage. A statement for health care professionals from special writing group of the stroke council, AHA, stroke 1999;30:905-915.
16. Philip HS. stephen R P. severely increased blood pressure in emergency department. Ann emerg med.2003;513-529.
17. Broderick j e al. Intracerebral haemorrhage is more than twice as common as subarachnoid haemorrhage. J neurosurg. 1993;78:188-191.

18. ventura et al. Desperate disease, desperate measures; tracking malignant hypertension in the 1950s. Am heart j 2001;142;197-203.
19. Andrew R. Haas and Paul E. Marik Division of Critical Care, Pulmonary, Allergy and Immunologic Disease, Jefferson Medical College of Thomas Jefferson University, Philadelphia, Pennsylvania.
20. Recognising and managing hypertensive encephalopathy in the ED by Jean mansfeild,BSN,RN,CRN, and Karen delay,MPH,2000;page no:20.
21. Dommanski M, Mitchell G, Pfeffer M. Pulse pressure and cardiovascular disease- related mortality: Follow-up study of the Multiple Risk Factor Intervention Trial (MRFIT).JAMA 287: 2677, 2002.
22. Burt VL, Whelton P, Roccella EJ. Prevalence of hypertension in the US adult population. Results from the Third National Health and Nutrition Examination Survey. 1988-1991.Hypertension 25: 3050, 1995.
23. A Study of Hypertensive Crisis and Precipitating FactorsA.AliRafighdoost MD, M. Shabestari MD, and T. Bostani MD

A. Rafighdoost MD, et al Received Jul 26, 2005; Accepted for publication Oct. 6, 2006.

24. Libby: Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine, 8th ed;chapter 40;page no.1400-46.

25. Strandgaard S, Paulson OB: Cerebral blood flow and its pathophysiology in hypertension. Am J Hypertens 1989; 2:486.

26. Vasan RS, et al,assestment of frequency of progression to hypertension in joint national committee on prevention detection and treatment of high blood pressure. The normotensive participants in the framinham study heart study. Lancet,2001;358:1682-1686.

27. Cameron JS,hicks J Frederick akbar mahomed and his role in the description of hypertension at guy's hospital. Kidney int.1996:49:1488-1506.

28. Mancia G.scipione riva-rocci.clin cardiol.1997;20:503-504.

29. Elliot WJ.hypertensiive emergencies. In critical care clinics vol 17.number 2 april 200 W.B saunders company.

30. Carl J.V. nasman D.hypertensive emergencies; the lancet july 200 vol356. 411-417.

31. Rudd P.osterberg. G.L. hypertension . topol-cardiovascular medicine.2nd edition lipincott Williams & Wilkins;92-114.

32. Martin J et al: arquios brasleirious de cardiologica-volume 83.no.2,august 2004.

33. Elliot WJ. Current hypertension reports 2003,5:486-492.

34. Bennet NM, shea s. Hypertensive emergency.american journal of public health 1998.78:636-640.

35. Kaplan NM. Primary hypertension: pathogenesis. In clinical hypertension 7th Baltimore,wiiliams and Wilkins,1998. PP41-101.

36. A zhan KA Li RL,pursell RA. Cardiovascular toxicity after consuming herbal ecstasy .J emergency med 17:289-291.1999.

37. Kaplan NM. Hypertensive crisis in Kaplan AM, ed.clinical hypertension,8h ediction. Lippincot willams and Wilkins 2002:339-356.

38. Kawazoe N et al.pathophysiology in malignant hypertension: with special reference to the rennin angioensin system. Clinical cardiology;1987;19-513-518.
39. Impey L. Severe hypotension and fetal distress following sublingual administration of nifedipine to a patient with severe hypertension at 33 weeks Br J obset gynecol.1993;100:959-961.
40. Wacher RM. Symptomatic hypotension induced by nifedipine in the avute treatment of severe hypertension. Arch intern med.1987;147;556-558.
41. Lip GY et al. Complication and survival of 354 patients with malignant pahse hypertension. J hypertens:13(8) 915-24.
42. Messerli FH et al. Disperate cardiovascular findings in men and women with essential hypertension. Ann intern med.1987;107;158-61.
43. Owens JF et al. Menopausal status influences ambulatory blood pressure level and blood pressure changes during mental stress. Circulation 1993;88;2794-802.
44. Sowers JR et al. Diabetes mellitus and associated hypertension,vascular disease and nephropathy. An update. Hypertension 1995;26;869-79.
45. Cushman WC, Ford CE, Cutler JA, et al. Success and predictors of blood pressure control in diverse North American settings: The Antihypertensive and Lipid-Lowering Treatment to Prevent Heart

Attack Trial (ALLHAT). *J Clin Hypertens (Greenwich).* 2002;4:393-404.

46. Black HR, Elliott WJ, Neaton JD, et al. Baseline characteristics and elderly blood pressure control in the CONVINCE trial. *Hypertension.* 2001;37:12-8.

47. American Heart Association. Home monitoring of high blood pressure. Available at http://www.americanheart.org/presenter.jhtml?identifier=576. Accessed April 1, 2003.

48. Neal B, MacMahon S, Chapman N. Effects of ACE inhibitors, calcium antagonists, and other blood-pressure-lowering drugs: Results of prospectively designed overviews of randomized trials. Blood Pressure Lowering Treatment Trialists' Collaboration. *Lancet.* 2000;356:1955-64.

49. Izzo JL Jr, Levy D, Black HR. Clinical Advisory Statement. Importance of systolic blood pressure in older Americans. *Hypertension.* 2000;35:1021-4.

50. American Diabetes Association. Treatment of hypertension in adults with diabetes. *Diabetes Care.* 2003;26(suppl 1):S80-S82.

51. National Kidney Foundation Guideline. K/DOQI clinical practice guidelines for chronic kidney disease: Evaluation, classification, and

stratification. Kidney Disease Outcome Quality Initiative. *Am J Kidney Dis.* 2002;39(suppl 2):S1-S246.

52. Bakris GL, Weir MR, on behalf of the Study of Hypertension and Efficacy of Lotrel in Diabetes (SHIELD) Investigators. Achieving goal blood pressure in patients with type 2 diabetes: Conventional versus fixed-dose combination approaches. *J Clin Hypertens.* 2003;5:201-10.

53. Hyman DJ, Pavlik VN. Characteristics of patients with uncontrolled hypertension in the United States. *N Engl J Med.* 2001;345:479-86.

54. National High Blood Pressure Education Program. The sixth report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Arch Intern Med.* 1997;157:2413-46.

55. Writing Group for the Women's Health Initiative Investigators. Risks and benefits of estrogen plus progestin in healthy postmenopausal women: Principal results from the Women's Health Initiative randomized controlled trial. *JAMA.* 2002;288:321-33.

56. Antithrombotic Trialist Collaboration. Collaborative meta-analysis of randomised trials of antiplatelet therapy for prevention of death, myocardial infarction, and stroke in high risk patients. *BMJ.* 2002;324:71-86.

57. Boulware LE, Daumit GL, Frick KD, et al. An evidence-based review of patient-centered behavioral interventions for hypertension. *Am J Prev Med.* 2001;21:221-32.

58. Staessen JA, Wang J. Blood-pressure lowering for the secondary prevention of stroke. [Commentary]. *Lancet.* 2001;358:1026-7.

59. Di Bari M, Pahor M, Franse LV, et al. Dementia and disability outcomes in large hypertension trials: lessons learned from the systolic hypertension in the elderly program (SHEP) trial. *Am J Epidemiol.* 2001;153:72-8.

60. Pickering T. Recommendations for the use of home (self) and ambulatory blood pressure monitoring. American Society of Hypertension Ad Hoc Panel. *Am J Hypertens.* 1996;9:1-11.

ANNEXURES

PROFORMA

Name: Age: Sex:

Address: DOA: DOD:

Presenting complaints:

Chestpain

Site, radiation:

Character:

Duration:

Dyspnoea:

PND:

Neurological deficit:

Loss of consiousness

Cranial nerve deficit:

Motor/sensory deficit

Pedal oedema:

Oliguria. Anuria

Visual deficit

Others

Past history:

Hypertension

Duration: treatment

Medication discontinued : yes/no

Complications related to hypertension in the past:

Any other associated illness:

Other relevant past history:

General examination

Pallor icterus

Cyanosis

Clubbing

Oedema

Lymphadenopathy

Vital signs:

Pulse rate: Respiratory rate:

Blood pressure on presentation:

Blood pressure after one hour:

Blood pressure after 24 hours:

Blood pressure at discharge:

Respiratory system:

Inspection:

Palpation:

Percussion:

Auscultation:

Cardiovascular system:

Inspection:

Palpation:

Percussion:

Auscultation:

Abdomen:

Inspection:

Palpation:

Percussion:

Auscultation:

Central nervous system:

Higher mental functions:

Cranial nerves

	Right	left
Motor system		
Bulk UL:		
LL:		
Tone UL:		
LL:		
Power UL:		
LL:		

Deep tendon reflexes

Plantar:

Sensory system:

meningeal signs:

Fundus:

Clinical diagnosis:

Investigations:

HB%:

TC: DC: ESR:

Urine analysis: micro/macro albunuria

Random blood sugar:

Serum urea:

Serum creatinine:

Serum cholesterol:

Triglycerides:

LDL cholesterol:

HDL cholesterol:

Serum sodium:

Serum potassium:

Serum chloride:

ECG:

Ultrasound scan(abdomen)

CT scan(head)

Diagnosis:

Master chart

S.NO	*NAME*	*AGE SEX*	*IP NO.*	*CHESTPAIN*	*DYSPNEA*	*NEURO DEFICIT*	*CONVULSIONS*	*VISUAL DEFICIT*	*H/O HTN*	*HTN YRS*	*HTN RX*	*DRUG COMPLINACE*	*DIABETES*	*SENSORIUM*	*BP-0 HOURS MM/HG*
1	**SUBRAMANI**	**45/M**	**24536**	**P**	**A**	**A**	**A**	**A**	**P**	**4**	**CCB**	**P**	**A**	**CONSCIOUS**	**190/120**
2	**SRINIVAS**	**58/M**	**64874**	**A**	**P**	**A**	**A**	**A**	**A**	**A**	**NA**	**NA**	**A**	**CONSCIOUS**	**200/120**
3	**DEIVATHAL**	**65/F**	**34562**	**A**	**A**	**P**	**A**	**A**	**P**	**10**	**CCB**	**NA**	**A**	**STUPEROUS**	**220/140**
4	**MURUGAN**	**45/M**	**26474**	**P**	**P**	**A**	**A**	**A**	**P**	**1**	**NA**	**NA**	**P**	**CONSCIOUS**	**180/110**
5	**VASUDEVAN**	**55/M**	**25378**	**A**	**A**	**P**	**A**	**A**	**P**	**2**	**ACE**	**A**	**A**	**CONSCIOUS**	**190/120**
6	**VELAN**	**55/M**	**34126**	**A**	**A**	**P**	**A**	**A**	**P**	**4**	**CCB**	**P**	**A**	**CONSCIOUS**	**180//110**
7	**DHANALAKSHMI**	**44/F**	**21902**	**P**	**P**	**A**	**A**	**A**	**P**	**5**	**ACEI**	**P**	**A**	**CONSCIOUS**	**190/114**
8	**KALIAPPAN**	**55/M**	**26731**	**P**	**A**	**A**	**A**	**A**	**A**	**A**	**NA**	**A**	**A**	**CONSCIOUS**	**200/124**
9	**PATTAHAL**	**62/F**	**29803**	**A**	**A**	**P**	**P**	**A**	**P**	**2**	**N/A**	**P**	**A**	**DROWSY**	**210/110**
10	**PONNAN**	**70/M**	**23415**	**A**	**P**	**A**	**A**	**A**	**P**	**10**	**ACEI**	**NA**	**P**	**CONSCIOUS**	**190/112**
11	**PONAMMAL**	**78/F**	**56342**	**A**	**A**	**P**	**A**	**A**	**P**	**12**	**CCB**	**NA**	**A**	**UNCONSCIOUS**	**240/120**
12	**CHINNAL**	**70/F**	**42315**	**P**	**P**	**A**	**A**	**A**	**P**	**3**	**ARB**	**NA**	**A**	**CONSCIOUS**	**200/130**
13	**PALANIAPPAN**	**40/M**	**34521**	**P**	**A**	**A**	**A**	**A**	**P**	**2**	**BB**	**A**	**A**	**CONSCIOUS**	**190/110**
14	**THULASI**	**50/F**	**31245**	**A**	**A**	**P**	**P**	**A**	**A**	**A**	**NA**	**NA**	**A**	**CONSCIOUS**	**200/120**
15	**VADIVU**	**74/F**	**21902**	**P**	**P**	**A**	**A**	**A**	**P**	**13**	**BB**	**A**	**P**	**CONSCIOUS**	**190/140**
16	**VIJAYAN**	**58/M**	**45321**	**P**	**P**	**A**	**A**	**A**	**P**	**5**	**BB/ACEI**	**A**	**A**	**CONSCIOUS**	**180/110**
17	**MYLATHAL**	**58/F**	**26735**	**A**	**A**	**P**	**P**	**A**	**P**	**10**	**CCB**	**P**	**A**	**CONSCIOUS**	**200/110**
18	**DHANDAPANI**	**55/M**	**28745**	**P**	**P**	**A**	**A**	**A**	**P**	**2**	**BB**	**A**	**P**	**CONSCIOUS**	**190/110**
19	**SARAVANAN**	**65/M**	**34522**	**A**	**P**	**A**	**A**	**A**	**P**	**4**	**CCB**	**A**	**A**	**CONSCIOUS**	**200/130**
20	**KALIYAMMAL**	**75/F**	**45237**	**A**	**A**	**P**	**A**	**A**	**P**	**6**	**CCB THIAZIDE**	**P**	**A**	**STUPEROUS**	**190/110**
21	**SARAWATHY**	**65/F**	**43266**	**A**	**A**	**P**	**A**	**A**	**P**	**A**	**NA**	**NA**	**A**	**CONSCIOUS**	**190/110**
22	**PUSPHARAJ**	**65/M**	**56332**	**A**	**P**	**A**	**A**	**A**	**P**	**8**	**CCB /BB**	**P**	**P**	**CONSCIOUS**	**180/110**
23	**NEELAVATHY**	**74/F**	**45261**	**P**	**P**	**A**	**A**	**A**	**P**	**4**	**CCB /ARB**	**P**	**P**	**CONSCIOUS**	**170/120**
24	**RAJARAMAN**	**55/M**	**31002**	**A**	**A**	**P**	**A**	**A**	**P**	**2**	**CCB**	**A**	**A**	**STUPEROUS**	**190/120**
25	**PUSPHA**	**63/F**	**25301**	**P**	**P**	**A**	**A**	**A**	**P**	**3**	**ACEI**	**P**	**A**	**CONSCIOUS**	**200/110**
26	**LAKSHMANAN**	**39/M**	**20110**	**A**	**A**	**P**	**A**	**A**	**P**	**2**	**BB**	**A**	**P**	**CONSCIOUS**	**220/110**
27	**MANICKAM**	**45/M**	**31092**	**P**	**A**	**A**	**A**	**A**	**P**	**6**	**ACEI**	**A**	**P**	**CONSCIOUS**	**180/110**
28	**ARUMUGAM**	**55/M**	**59063**	**P**	**P**	**A**	**A**	**A**	**P**	**4**	**ACEI**	**P**	**A**	**CONSCIOUS**	**190/110**
29	**SEENI**	**42/M**	**52617**	**A**	**A**	**P**	**A**	**A**	**P**	**1MON**	**CCB**	**A**	**A**	**SEMI-CONSICOUS**	**220/140**
30	**RANGAN**	**38/M**	**31526**	**A**	**P**	**A**	**A**	**P BLURRING OF VISION NEAR DEFECT**	**P**	**A**	**A**	**A**	**A**	**CONSICOUS**	**220/160**
31	**MUNUSAMY**	**49/M**	**41526**	**P**	**A**	**A**	**A**	**A**	**P**	**5**	**CCB BB**	**P**	**P**	**CONSICOUS**	**210/116**

32	**KANDASAMY**	**38/M**	**31829**	**A**	**A**	**P**	**A**	**A**	**P**	**1**	**ACEI**	**P**	**A**	**CONSCiOUS**	**180/120**
33	**MANIMEGALAI**	**41/F**	**21029**	**A**	**A**	**P**	**A**	**A**	**P**	**2**	**CCB**	**P**	**A**	**CONSICOUS**	**200/110**
34	**KALLIYAMMAL**	**67/F**	**41028**	**P**	**P**	**A**	**A**	**A**	**A**	**NA**	**NA**	**A**	**A**	**CONSICOUS**	**194/102**
35	**MANIKAMMAL**	**43/F**	**50982**	**A**	**A**	**P**	**P**	**A**	**A**	**A**	**NA**	**A**	**A**	**COMATOUS**	**204/106**
36	**MUSTAFA**	**45/M**	**21783**	**P**	**P**	**A**	**A**	**A**	**P**	**1**	**ARB**	**P**	**A**	**CONSICOUS**	**192/106**
37	**PALANATHAL**	**55/F**	**26735**	**A**	**A**	**P**	**A**	**A**	**P**	**10**	**CCB**	**P**	**P**	**COMATOUS**	**184/118**
38	**RAMASAMY**	**45/M**	**20012**	**P**	**P**	**A**	**A**	**A**	**P**	**3**	**PRAZOSIN**	**P**	**P**	**CONSICOUS**	**188/116**
39	**JOSEPH**	**38/M**	**32145**	**P**	**A**	**A**	**A**	**A**	**P**	**6 MON**	**ARB**	**P**	**P**	**CONSICOUS**	**190/126**
40	**THAYAMMAL**	**64/F**	**33980**	**P**	**P**	**A**	**A**	**A**	**P**	**15**	**CCB THIAZIDES**	**P**	**A**	**CONISOIUS**	**240/108**
41	**MEHARUNISHA**	**45/F**	**22274**	**A**	**A**	**P**	**P**	**A**	**P**	**3**	**BB**	**P**	**A**	**SEMI CONSIOUS**	**220/130**
42	**MURUGATHAL**	**50/F**	**21234**	**A**	**A**	**P**	**A**	**A**	**A**	**A**	**NA**	**A**	**A**	**CONSICOUS**	**186/112**
43	**VELUSAMY**	**60/M**	**42516**	**P**	**P**	**A**	**A**	**A**	**P**	**8**	**CCB**	**P**	**A**	**CONSICOUS**	**198/110**
44	**HAMSATH BEGAM**	**54/F**	**33445**	**P**	**P**	**A**	**A**	**A**	**P**	**4**	**ARB THIAZIDES**	**P**	**P**	**CONSICOUS**	**200/104**
45	**RAMU**	**60/M**	**22450**	**A**	**A**	**P**	**P**	**A**	**P**	**10**	**CCB BB**	**P**	**A**	**CONSICOUS**	**190/116**
46	**PALANIAPPAN**	**58/M**	**21135**	**P**	**P**	**A**	**A**	**A**	**P**	**8**	**ACEI**	**P**	**P**	**CONSICOUS**	**250/128**
47	**RANGASMMAL**	**63/F**	**20192**	**A**	**A**	**P**	**A**	**A**	**P**	**5**	**CCB**	**P**	**A**	**STUPEROUS**	**220/114**
48	**HARIKRISHNAN**	**60/M**	**20192**	**P**	**P**	**A**	**A**	**A**	**P**	**16**	**CCB**	**P**	**A**	**CONSICOUS**	**198/120**
49	**KALIMUTHU**	**52/M**	**20921**	**P**	**P**	**A**	**A**	**A**	**P**	**4**	**CCB**	**P**	**A**	**CONSICOUS**	**184/112**
50	**VENGAIYA NAIDU**	**48/M**	**25641**	**A**	**A**	**P**	**A**	**A**	**P**	**^**	**CCB**	**P**	**A**	**CONSICOUS**	**192/112**

NEUROLOGICAL DEFICIT	*MENINGISM*	*FUNDUS*	*CLINICAL LV FAILURE*	*BP-1 HRS MM/HG*	*BP-24 HRS MM/HG*	*BP-AT DISMM/HG*	*UREAMG*	*CREATININE MG*	*URINE MIC. ALB*	*DYSLIP*	*NA*	*K*	*ECG*
A	**A**	**GARDE 2**	**A**	**180/100**	**160/90**	**150/90**	**42**	**1.2**	**1+**	**NIL**	**141**	**3.5**	**LVH ST-T CHANGES**
A	**A**	**GRADE 2**	**P**	**190/110**	**150/90**	**140/80**	**55**	**1.8**	**2+**	**MILD TRIGLY**	**138**	**3.9**	**LVH LAFB**
P	**A**	**PAPILLOEDEMA**	**A**	**180/120**	**170/100**	**170/100**	**38**	**1**	**A**	**NIL**	**139**	**4.1**	**LVH**
A	**A**	**NORMAL**	**A**	**170/100**	**130/90**	**130/80**	**41**	**0.8**	**A**	**NIL**	**142**	**4.4**	**ST-T CHANGES**
P	**A**	**NORMAL**	**A**	**140/100**	**140/100**	**130/90**	**32**	**0.8**	**1+**	**NIL**	**141**	**3.8**	**NORMAL**
P	**A**	**PAPILLOEDEMA**	**A**	**140/100**	**130/100**	**130/90**	**30**	**0.8**	**A**	**NIL**	**140**	**4.0**	**NORMAL**

P	A	GRADE 1	A	150/100	140/90	130/90	30	1.1	1+	NIL	139	4/6	ST-T CHANGES
A	A	NORMAL	A	150/100	140/90	130/90	32	1.2	A	NIL	138	3.9	ST-T CHANGES
P	A	PAPILLO EDEMA	A	180/100	160/100	160/100	34	0.7	A	CHOL	140	4.3	LVH
A	A	GRADE 2	P	190/90	180/100	150/100	32	0.9	1+	NIL	143	3.8	ST-T CHANGES
P	P	NORMAL	A	200/100	190/110	180/110	45	1.2	2+	NIL	140	3.9	NORMAL
A	A	GRADE 2	P	180/100	170/100	140/100	54	1.5	1+	NIL	138	3.8	ST-T CHANGES
A	A	NORMAL	A	160/100	140/90	130/80	34	0.7	A	NIL	145	3.3	ST-T CHANGES
P	P	NORMAL	A	190/110	190/100	160/100	32	1.1	1+	NIL	145	4.3	LVH
A	A	GRADE 3	A	190/90	180/90	160/90	34	1.2	1+	NIL	139	4.5	ST-T CHANGES
A	A	GRADE 1	P	180/100	150/90	140/100	42	1.9	1+	NIL	143	5.2	ST-T CHANGES
P	P	PAPILLO EDEMA	A	170/100	160/100	130/100	50	1.2	A	CHOL ELE	138	4.0	NORMAL
A	A	GRADE 1	P	190/100	160/100	150/90	43	1	A	NIL	139	4.4	NPR + IN V1-V4
A	A	GRADE 1	P	190/120	180/100	140/90	34	0.6	NIL	NIL	145	3.4	NORMAL
P	A	GRADE 1	A	190/100	170/90	150/80	29	0.9	NIL	NIL	145	4.3	LVH
P	A	GARDE 2	A	180/100	160/100	150/100	45	1.2	NIL	NIL	145	3.6	NORMAL ICRBB
A	A	GRADE 2	P	180/100	160/100	150/100	37	0.9	NIL	NIL	139	3.7	NORMAL
A	A	GRADE 1	P	170/110	160/100	140/90	35	1.1	NIL	NIL	140	4.3	NPR IN V1-V4 LBBB PATTERN
P	P	GRADE3	A	190/100	180/110	150/100	38	1.6	NIL	TRI GLY	145	3.9	LVH
A	A	NORMAL	P	180/100	160/110	140/90	35	0.8	NIL	NIL	138	3.3	LAFB
P	A	GRADE 1	A	190/100	160/100	140/90	35	0.9	NIL	NIL	139	3.5	NORMAL
A	A	GARDE1	A	160/100	160/90	150/100	37	1.2	NIL	TGL	142	3.4	ST-T CHANGES ST ELEVATION IN ANTERIOR LEADS WITH RECIPROCAL CHANGES
A	A	GRADE 1	P	160/110	150/100	140/90	37	1.6	NIL	CHOLES	13	3.7	ASYEMMTRICAL T

											7		WAVE INVERSION IN ANTERIOR LEADS
P	A	GRADE 4 PAPILLO EDEMA	A	190/90	140/80	140/90	34	1.7	NIL	CHOLES	138	4.0	NORMAL
A	A	GRADE 4	A	190/90	140/90	140/90	37	1.4	NIL	NIL	140	3.6	LVH
A	A	GARDE 2	A	180/90	140/80	140/70	41	1.2	NIL	TGL	149	3.4	ST ELEVATION IN V1-V4 WITH RECIPROCAL CHANGES
P	A	NORMAL	A	180/90	130/90	120/90	43	1.8	NIL	CHOLES	138	4.3	LOW VOLTGAE COMPLEXES SINUS BRADYCARDIA
P	A	PAPILLO EDEMA	A	178/86	144/92	130/198	35	1.1	NIL	CHOLES	138	4.1	SINUS BRADYCARDIA
A	A	MEDIA HAZY	A	168/82	142/80	130/78	36	1.0	NIL	NIL	139	4.3	ST DEPRESSION IN ANERIOR LEADS AND T WAVE INVERSION IN INFERIOR LEADS
P	P	GRADE1	A	178/88	168/76	130/86	46	1.0	NIL	NIL	140	3.6	NORMAL
A	A	NORMAL	A	164/84	140/68	130/80	45	0.9	NIL	NIL	142	3.4	ST ELEVATION IN V5-V6 & L1/AVL WITH RECIPROCAL CHANGES IN INFERIOR LEADS
P	A	MEDIA HAZY	A	164/98	168/70	140/68	42	0.6	NIL	CHOLES	136	3.6	LOW VOLTAGE COMPLEXES
A	A	GRADE 1	A	148/88	134/96	136/68	34	0.8	1+	NIL	136	3.6	ST DEPRESION IN ANTERIOR LEADS AND T WAVE INVERSION IN SAME LEADS
A	A	NORMAL	A	148/68	138/70	120/70	36	0.9	2+	NIL	142	4.2	GLOBAL T WAVE INVERSIO

													N ST ELEVATION IN AVR
A	A	NORMAL	A	158/88	148/86	138/86	38	0.6	NIL	TGL	146	3.3	LVH WIH STRAIN PATTERN
P	P	GARDE 2	A	198/110	190/100	188/98	36	0.6	1+	NIL	147	4.5	SINUS BRADYCARDIA
P	A	MEDIA HAZY	A	176/80	150/80	140/90	42	2	NIL	NIL	142	4.1	LOW VOLTAGE COMPLEXES ICRBBB
A	A	MEDIA HAZY	P	178/90	168/80	140/90	45	1.5	NIL	NIL	146	3.6	LVH WITH STARIN PATTERN
A	A	NORMAL	P	188/90	168/90	150/90	40	1.4	NIL	NIL	146	3.6	WAVE INVERSION IN ANERIOR LEADS
P	A	PSEUDOPAHKIA	A	180/112	170/108	160/90	38	0.8	2+	NIL	136	3.3	SINUS BRADYCARDIA BIFASICULAR BLOCK
P	A	MEDIA HAZY	A	190/114	188/126	160/110	39	0.9	1+	TGL	139	4.3	SINUS BRADYCARDIA
P	A	PSEUDOPHAKIC	A	190/116	180/120	170/112	40	1.3	1+	CHOLES	140	4.4	LAFB
P	A	MEDIA HAZY	P	180/120	190/114	170/120	46	1.6	NIL	NIL	140	3.3	ST ELEVATION IN L2L3AVF WITH POSTERIOR WALL MI
P	A	GRADE 2	P	190/110	180/98	140/86	44	1.2	NIL	TGL	142	3.6	ASYMMETRICAL T WAVE INVERSION IN ANTERIOR LEADS

CXR	*RENAL USG*	*ECHO*	*CT SCAN HEAD*	*DIAGNOSIS*	*OUTCOME*
MILD CARDIOMEGALY	**NA**	**LVH**	**NA**	**SHT/CAHD/UA**	**DISCHARED**
NORMAL	**NA**	**LVH**	**NA**	**ACC.SHT/ACUTE PULMONARY EDEMA**	**DISCHARGED**
NORMAL	**GRADE 1 MRD**	**LVH**	**LEFT CAPSULO GANGLIONIC HAEMMORRAHGE**	**SHT/CVA/HAEMORRHAGE STROKE**	**EXPIRED**
NORMAL	**NORMAL**	**HYPOKINESIA OF LV APEX**	**NA**	**SHT/CAHD/ACUTE AWMI/KILLIP CLASS 2**	**DISCHARGED**
NORMAL	**NA**	**NA**	**INFARCT IN LEFT SUBCORTICAL AREA**	**SHT/CVA/R HEMIPARESIS**	**DISCHARGED**
NORMAL	**NA**	**NA**	**INFARCT IN RIGHT INTERNAL CAPSULE**	**SHT/CVA/L HEMIPARESIS**	**DISCHARGED**
NORMAL	**NA**	**HYPOKINESIA OF LV APEX LVEF 45%**	**NA**	**SHT/ACUTE LVF**	**DISCHARGED**
NORMAL	**NA**	**HYPOKINESIA OF POSTERIOR WALL**	**NA**	**SHT/ACUTE AWMI**	**DISCHARGED**
NORMAL	**NA**	**NA**	**LEFT ICH/INTRA VENTRICULAR EXTENSION**	**SHT/CVA/HAEMORRHAGIC STROKE**	**EXPIRED**
MILD CARDIOMEGALY PULMONARY CONGESTION +	**NA**	**HYPOKINESIA OF LV**	**NA**	**SHT/LVD**	**DISCHARGED**
NORMAL	**NA**	**NA**	**NA**	**SHT/SAH**	**DISCHARGED**
MILD CARDIOMEGALY	**NA**	**HYPOKINESIA OF RWMA LVEF 60%**	**NA**	**SHT/CAHD/ACUTE LVF**	**DISCHARGED**
NORMAL	**NA**	**RWMA+**	**NA**	**SHT/CAHD/ACUTE AWMI**	**DISCHARGED**
NORMAL	**NA**	**NA**	**LEFT THALAMIC INFARCT**	**SHT/L THALAMIC INFARCT /LEFT HEMIPARESIS**	**DISCHARGED**
NORMAL	**NA**	**CAHD/GRADE 1 DIASTOLIC DYSFUNCTION**	**NA**	**SHT/CAHD/LVD**	**DISCHARGED**
NORMAL	**NA**	**NA**	**HAEMMORAHGE IN RIGHT FRONTAL REGION**	**SHT/CVA/ICH**	**EXPIRED**
MOD.CARDIMEGALY	**NA**	**RWMA+/LV DIASTOLIC DYSFUNCTION**	**NA**	**SHT/TYPE2 DM/CAHD/LVD**	**DISCHARGED**
NORMAL	**NA**	**MILD DIASTOLIC DYSFUNCTION**	**NA**	**SHT/ACUTE PULMONARY EDEMA**	**DISCHARGED**
NORMAL	**NA**	**NA**	**HAEMARRHAGIC INFRACT SEEN IN LEFT THALAMUS & LEFT CUPSULOGANGLIONIC REGION**	**SHT/CVA/ICH**	**DISCHARGED**
NORMAL	**NA**	**NA**	**RIGHT BASAL GANGLIA INFARCT**	**SHT/CVA/LEFT HEMIPARESIS**	**DISCHARGED**
NORMAL	**NA**	**GRADE 2 DIASTOLIC DYSFUNCTION**	**NA**	**SHT/ACUTE PULMONARY EDEMA**	**DISCHARGED**
NORMAL	**NA**	**GRADE 1 DIASTOLIC DYSFUNCTION**	**NA**	**SHT/TYPE 2 DM/CAHD OLD ASMI**	**DISCHARGED**
NORMAL	**NA**	**NORMAL**	**HAEMORRHAGIC**	**SHT/LEFT**	**DISCHARG**

			INFARCT IN LEFT CEREBELLUM	CEREBELLAR HAEMORRHAGE	ED
NORMAL	NA	RWMA+/LVEF 45%	NA	SHT/CAHD/LV D	DISCHARGED
NORMAL	NA	NA	NORMAL STUDY	SHT/CVA/RIGHT HEMIPARESIS	DISCHARGED
NORMAL	NA	NA	INFARCT SEEN RIGH INERNAL CAPSULE	SHT/CVA/LEFT HEMIPARESIS	DISCHARGED
NORMAL	NA	TACHYCARDIA DURING STUDY LV SYSTOLIC FUNCTION NORMAL DIASTOLIC DYSFUNC	NA	SHT/ACUE PULMONARY EDEMA	DISCHARGED
NORMAL	NA	NA	HAEMMRAHGE PRESENT IN THE LEFT EMPORAPARIETAL REGION WIH MIDLINE SHIFT	ACCELERATED SHT/CVA/ICH	DISCHAGRED
NORMAL	NA	MILD DIASTOLIC DYSFUNCTION	PERIVENTRICULAR REVERSIBLE LEUOENCEPHALOPATHY	ACCELERATED SHT	DISCHARGED
NORMAL	NA	HYPOKINESIA OF LW ANTERIOR WALL LVEF ADEQUATE	NA	ACCELERATED SHT/CAHD/ACS/ACUTE AWMI?KILLIP CLASS 1	DISCHARGED
NORMAL	NA	NA	NORMAL STUDY	ACELERATED SHT/CVA/ISCHEMIC INFARCT /LEFT HEMIPARESIS	DISCHARGED
NORMAL	NA	NA	CEREBRAL EDEMA NO OBVIOUS HAMMOARHGE R INFARCT	ACCELERATED SHT/HYPERETNSIVE ENCEPHALOPATHY	DICHARGED
MILD CARDIOMEGALY	NA	GRADE 3 DIASTOLIC DYSFUNCTION LVEF 50% RWMA +	NA	ACCELERATED SHT/CAHD/UNSTABEL ANGINA	DISCHARGED
NORMAL	NA	NA	ICH PRESENT IN THE BRAIN STEM	ACCELERATED SHT/CVA/ICH WITH INTRAVENTICULAR HAEMORRHAGE	EXPIRED
NORMAL	NA	RWMA + IN LV APEX LVEF 56% DIASTOLIC FUNCTION NORMAL	NA	ACCELERATED SHT?CAHD/ACS/ACUTE LAERAL WALL MI	DISCHARGED
NORMAL	NA	NA	MULTIPLE INFARCT PERIVANTRICULAR TRANSLUCENCY	SHT/DYSLIPIDEMIA /CVA/MULTI INFARCT STATE WITH DEMENTIA/RIGHT HEMIPARESIS	DISCHARGED
NORMAL	NA	LVH GRADE 2 DIASTOLIC DYSFUNCTION LV SYSTOLIC FUNCTION ADEQUATE	NA	SHT/CAHD/ACS/UNSTABLE ANGINA/KILLIP CLASS 2	DISCHARGED
NORMAL	GRADE IN MRD	LV SYSOLIC FUNCTION ADEQUATE GLOBAL HYPOKINESIA	NA	SHT/CAHD/ACS/UA/?RIPLE VESSEL DISEASE	DISCHARGED

NORMAL	**NA**	**LEFT VENTRICULAR HYPERTROPHY**	**NA**	**SHT/ACUTE PULMONARY EDEMA**	**DISCHARGED**
NORMAL	**NA**	**NA**	**SAH WITH CEREBRAL EDEMA**	**SHT/SAH/CEREBRAL EDEMA**	**DISCHARGED**
NORMAL	**NA**	**NA**	**INFARCT IN RIGHT MCA TERRITORY**	**SHT/CVA/LEFT HEMIPARESIS**	**DISCHARGED**
NORMAL	**NA**	**LEFT VENRICULAR HYPERTROPHY GRADE 1 DIASTOLIC DYSFUNCTION NO RWMA LV SYSOLIC FUNCTION ADEQUATE**	**NA**	**SHT/ACUTE PULMONARY EDEMA**	**DISCHARGED**
NORMAL	**NA**	**HYPOKINESIA OF LV FREE WALL LV SYSTOILC FUNCTION ADEQUATE**	**NA**	**SHT/CAHD/ACS/ACUTE LATERAL WALL MI**	**DISCHARGED**
NORMAL	**NA**	**NA**	**MASSIVE INFARCT IN LEFT HEMISPHERE INO HAEMORRHAGIC TRANSMISSION**	**SHT/CVA/HAEMAORGHIC INFARCT**	**EXPIRED**
MILD CARDIOMEGALY	**GARDE 1 MRD**	**LVH + DIASTOLIC DYSFUNCTION GRADE 2**	**CEREBRAL EDEMA**	**SHT/UA/HYPERTENSIVE ENCEPHALOPATHY**	**DISCHARGED**
NORMAL	**NA**	**NA**	**ICH IN RIGHT ABSAL GANGLIA**	**SHT/CVA/ICH**	**EXPIRED**
NORMAL	**NA**	**LVH HYPOKINESIA SEEN IN LV POSERIOR WALL**	**NA**	**SHT/CAHD/ACS/ACUTE INFEROPASTERO WALL MI?KILLIP CLASS 2**	**DISCHARGED**
NORMAL	**NA**	**LVH LV SYSOLIC FUNCTION ADEQUATE**	**NA**	**SHT/CAHD/ACS/UA**	**DISCHARGED**
NORMAL	**NA**	**NA**	**ICH IN LEFT HEMISPHERE MASSIVE HAEMORHHAGE**	**SHT/CVA/ICH**	**DISCHARGED**

A:	ABSENT
ACEI:	ANGIOTENSIN CONVERTING ENZYME INHIBITOR
ACS:	ACUTE CORONARY SYNDROME
ARB:	ANGIOTENSIN RECEPTOR BLOCKERS
ASMI:	ANTEROSEPTAL MYOCARDIAL INFARCTION
BB:	BETA BLOCKERS
CAHD:	CORONARY ARTERY HEART DISEASE
CCB:	CALCIUM CHANNEL BLOCKERS
CVA:	CEREBROVASCULAR ACCIDENT
COLES:	CHOLESTRELEMIA
LAFB:	LEFT ANTERIOR FASICULAR BLOCK
LVD:	LEFT VENTRICULAR DYSFUNCTION
LVH:	LEFT VENTRICULAR HYPERTROPHY
LVEF:	LEFT VENTRICULAR EJECTION FRACTION
MCA:	MIDDLE CEREBRAL ARTERY
NA:	NOT APPLICABLE
P:	PRESENT
RWMA:	REGIONAL WALL MOTION ABNORMALITY
ICH:	INTRACEREBRAL HAEMORRHAGE
SAH:	SUBARACHNOID HAEMORRHAGE
SHT:	SYSTEMIC HYPERTENSION
TGL:	TRIGLYCEREDEMIA
UA:	UNSTABLE ANGINA