A STUDY ON PREVALENCE OF MICROALBUMINURIA IN ESSENTIAL HYPERTENSION AND ITS RELATIONSHIP WITH DURATION AND SEVERITY OF HYPERTENSION AND TARGET ORGAN DYSFUNCTION

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DECLARATION

I, **Dr. KESAVAN G** solemnly declare that this dissertation, "**A STUDY ON PREVALENCE OF MICROALBUMINURIA IN ESSENTIAL HYPERTENSION AND ITS RELATIONSHIP WITH DURATION AND SEVERITY OF HYPERTENSION AND TARGET ORGAN DYSFUNCTION**" is a bonafide record of work done by me in the Department of General Medicine, Thanjavur medical college, Thanjavur under the guidance of **Dr. K. NAMASIVAYAM** MD., Professor & HOD Department of General Medicine, Thanjavur medical college, Thanjavur. This Dissertation is submitted to the Tamil Nadu Dr. M.G.R Medical University in partial fulfilment of the rules and regulations for the award of M.D GENERAL MEDICINE DEGREE BRANCH–I examination to be held in MAY 2020.

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CERTIFICATE II

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Instances where selected sources appear:

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CONTENTS

INTRODUCTION

Systemic Hypertension is one of the most important and most common public health problem in all countries. It is an asymptomatic, easily diagnosed, easily treatable non communicable disease and often leads to lethal complication if left untreated. It affects more than one billion individuals and causes an estimated 9.4 millions death per year. In 90 to 95% of hypertensive cases the etiology is still unknown and is termed as Essential hypertension. It may be due to interaction of genetic factors and environmental factors.

In United states, systolic blood pressure is greater for men than women during early adulthood. Among older individuals the age related rate of rise is steeper for women. Consequently among individuals aged greater than 6o years systolic blood pressure of women are higher than those of men.

Among individuals diastolic BP tends to rise progressively with age until 55 years after which it tends to decrease. Obesity and weight gain are important and strong independent risk factors for essential hypertension. Increased dietary intake of sodium chloride and decreased dietary intake of calcium and potassium contribute to risk of hypertension.

Untreated systemic hypertension increases the incidence of target organ dysfunctions. Main organs affected are heart, brain, kidney, retina and peripheral vessels. The major target organ dysfunctions are left ventricular

hypertrophy, coronary heart disease, congestive cardiac failure, Ischemic and haemorrhagic stroke, hypertensive encephalopathy, nephropathy, retinopathy and peripheral vascular disease.

Microalbuminuria is a marker of wide spread vascular damage in essential hypertension. Hypertensive patients with microalbuminuria were found to have significantly higher prevalence of target organ dysfunction when compared to their normoalbuminuric counter parts. At present microalbuminuria is emerged as an early marker of target organ damage in essential hypertension.

In persons with mild hypertension and no cardiovascular complications, the main determinant of albumin excretion in urine is seems to be hemodynamic load. But in persons with more severe hypertension and associated end organ dysfunction, albumin excretion is seems to be due to glomerular damage.

AIM & OBJECTIVES

1) To determine the prevalence of microalbuminuria in patients with essential hypertension.

2) To study the relationship of microalbuminuria with duration and severity of hypertension and target organ dysfunction

REVIEW OF LITERATURE

Historical review :

The history of hypertension begin with the understanding of cardio vascular system based on the work of physician WILLIAM HARVEY (1578 – 1657) who described about circulation of blood. During 1853, VIERORDT a German scientist, was the first person introduced an instrument to measure systemic blood pressure.

In 1896, SCIPIONE RIVA-ROCCI invented the cuff based sphygmo manometer which allowed blood pressure to be measured in clinics. In 1905, NIKOLAI KORTOKOFF improved the technique by describing kortokoff sounds. In 1981, DONAL NUNN invented an accurate fully automated oscillometric sphygmomanometer. In 1911, EBERHARD FRANK described the essential hypertension as elevated blood pressure for which no cause could be found.

Definitions :

Hypertension is defined as that level of blood pressure at which institution of therapy reduces blood pressure related morbidity and mortality. In children and adolescents, hypertension is defined as systolic and/or diastolic blood pressure consistently greater than 95th percentile for age, sex

and height. Blood pressure between 90th and 95th percentile are considered prehypertension.

Classification of BP for adults aged 18 years and older :

As per the JNC VII report on prevention, detection, evaluation and treatment of high BP.

Category	Systolic BP (mmHg)	Diastolic BP (mmHg)
Normal	<120	<80
Prehypertension	120 – 139	80 - 89
Stage 1	140 – 159	90 - 99
Stage 2	>160	>100
Isolated SBP	>140	<90

Essential hypertension :

Also called as primary or idiopathic hypertension. There is no identifiable cause for hypertension.

Secondary hypertension :

A form of hypertension caused by one or more identifiable underlying primary cause.

Hypertensive emergency :

Defined as conditions with severe elevations in BP often higher than

220/140mmHg complicated by clinical evidence of progressive target organ dysfunction. Also called as malignant or accelerated hypertension.

Hypertensive urgency :

Also called as severe hypertension. Defined as condition with marked elevation of blood pressure usually higher than 180/110 mmHg without non progressive target organ damage.

Whitecoat hypertension :

Condition in which blood pressure is elevated on casual measurement taken in physicians clinic but normal at other times. Long term outcome of these individuals are more similar to normotensive individuals.

Masked hypertension :

Defined as condition in which blood pressure is normal in office but elevated in out of office situation. It is nearly equivalent to that of sustained hypertension.

Guidelines for measurement of blood pressure :

Patients conditions :

Posture :

For patients older than 60 years, on antihypertensive therapy, having diabetes, check for postural changes in BP by taking reading after 5 minutes of supine, then immediately 2 minutes after standing is necessary to rule out postural hypotension.

For routine follow up, a patient should sit quietly for 5 minutes with arm supported at the level of the heart and the back resting against a chair.

Circumstances:

Relax the patient for atleast 5 minutes before measuring BP.

Talking shouldn't be encouraged during relaxation.

No caffeine, exercise and smoking allowed atleast 30 minutes before.

Patients urinary bladder should be empty.

The location of cuff placement shouldn't be covered with clothing.

Equipment :

Cuff & Bladder size :

The proper cuff and bladder size used in measurement of blood pressure is important for accuracy. Cuff that is too short and narrow for a patients arm results in high blood pressure measurement. Use of a too large cuff results in low pressure measurement.

The length of the bladder should be 80 percent of the patients arm circumference. The width should be 40 percent of arm circumference.

Manometer :

Mercury spyghmomanometer – available since more years.

Oscillometric devices – use a sensor that detects pulsations in the flowing blood vessels as the cuff inflates and deflates.

The BP measurement device should be validated and calibrated periodically.

Stethoscope :

Bell of the stethoscope should be used to avoid interference.

Technique :

Number of readings :

Take 2 to 3 readings at an interval of 5 minutes.

For initial diagnosis should measure 3 sets of readings, each atleast 1 week apart.

Procedures for recording blood pressure :

At the first visit, measure blood pressure in both arms. If the pressure differs, use the arm with higher pressure. If the arm pressure is elevated, then also record pressure in legs particularly in younger patients with age below 30 years.

Use a palpated estimate of radial pulse obliteration pressure to estimate systolic blood pressure. Inflate the cuff 20 – 30mm Hg above this level for an auscultatory determination of blood pressure level. Deflate the cuff pressure at the rate of 2mmHg per second and listen for korotkoff sounds.

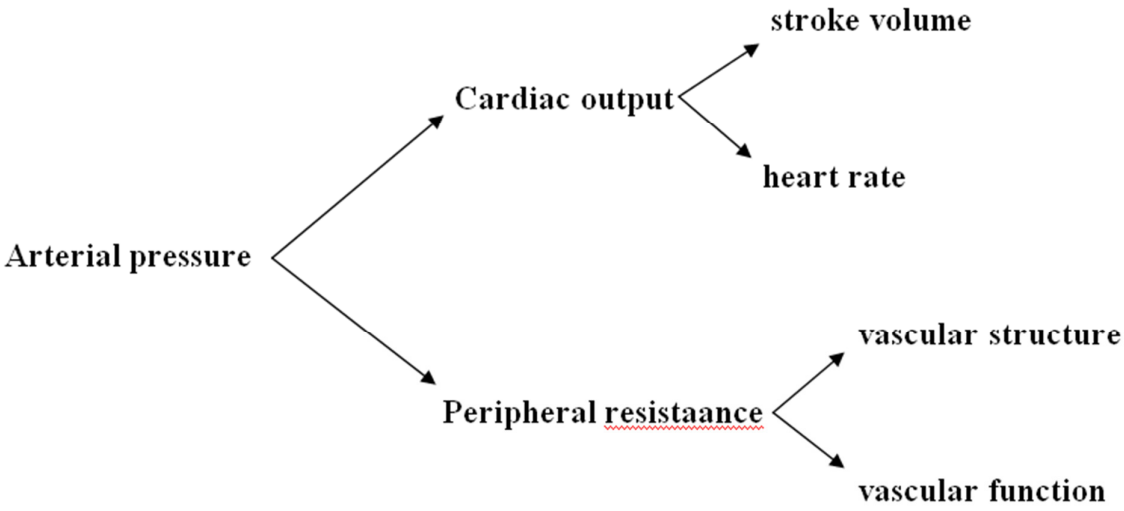
The reading at onset of the first korotkoff sound is recorded as systolic blood pressure. The reading at disappearance of all korotkoff sounds is recorded as diastolic blood pressure.

Recordings :

After measuring the blood pressure, should note the recorded pressure, patients position, measured limb, cuff size etc..(Example – BP : 130/80 mmHg , sitting position, right arm, large adult size cuff).

Mechanism of systemic hypertension :

Determinants of arterial pressure :



The initial elevation of blood pressure in response to vascular volume expansion may be related to an increase of cardiac output. But when times goes peripheral vascular resistance increases and cardiac output reverts towards normal.

Sodium is a primary determinant of intravascular volume. Sodium can activate a number of neural, endocrine and vascular mechanism all of which

increase arterial pressure. Sodium with chloride affects blood pressure dominantly.

Pressure natriuresis :

Increased urinary sodium excretion when arterial arterial pressure increases due to high sodium chloride intake. This phenomenon involves increase in glomerular filtration rate, decrease in reabsorbing capacity of renal tubules and effect atrial natriuretic factor.

Sodium chloride dependent hypertension may be a consequence of a decreased capacity of the kidney to excrete sodium due either to intrinsic renal disease or to increased production of a salt retaining hormone.

Autonomic nervous system :

Adrenergic reflexes modulate blood pressure over the short term whereas humoral and volume related factors contributes to long term regulation of arterial pressure.

Alpha adrenergic receptors are located on postsynaptic cells in smooth muscle cells and elicit vasoconstriction.Those receptors present in kidney increases renal tubular reabsorption of sodium.

Alpha 2 receptors are located on presynaptic membranes of postganglionic nerve terminals that synthesize norepinephrine.

Beta1 receptors are present in myocardium and responsible for increase in heart rate, strength of cardiac contraction which increases cardiac output and finally increases blood pressure.

Beta 2 receptors are present in smooth muscles and are responsible for vasodilation. Baroreceptors are present in aortic arch and carotid sinuses. They are stimulated by increased arterial pressure which leads to decrease in sympathetic outflow. This results in decrease in heart rate and blood pressure

This is the mechanism responsible for acute fluctuation of arterial pressure that occur during postural changes, behavioural changes, psychological stress and changes in blood volume. However the activity of baroreflex declines or adapts to sustained increase in arterial pressure.

Rennin angiotensin aldosterone axis :

Rennin is an aspartyl protease and its secretion is stimulated by,

1) Decreased Nacl transport in distal portion of the thick ascending limb of loop of Henle.

2) Decreased pressure or stretch within the renal afferent arteriole

3) Sympathetic nervous system stimulation of rennin secreting cells via Beta 1 adreno receptors.

4) Obstruction of the renal artery leads to decreased renal perfusion pressure thereby stimulating rennin secretion.

Rennin secretion is inhibited by,

1) Increased Nacl transport in the thick ascending limb of loop of Henle.

2) Beta 1 receptor blockade.

Angiotensin II type 1 receptor is a potent pressor substance and is a primary factor for secretion of aldosterone by adrenal zona glomerulosa. It play a role in pathogenesis of atherosclerosis.

Angiotensin II type 2 receptors has opposite effects of the Angiotensin II type 1 receptors. It induces vasodilation ,sodium excretion, inhibition of cell growth and matrix formation. Excess tissue angiotensin II may contribute to atherosclerosis, cardiac hypertrophy and renal failure.

Aldosterone is secreted by zona glomerulosa of adrenal cortex. Its synthesis and secretion is regulated by angiotensin II. Its synthesis is decreased in potassium depleted individuals.

Aldosterone is a potent mineralocorticoid that increase sodium reabsorption by amiloride sensitive epithelial sodium channels on the apical surface of principal cells of the renal cortical collecting duct by exchanging with potassium and hydrogen ions.

Increased aldosterone secretion may result in hypokalemia and alkalosis. Aldosterone has deleterious effects on cardiovascular system by inducing fibrosis, endothelial dysfunction, inflammation and oxidative stress.

Vascular mechanism :

Vascular radius and compliance of resistance arteries are the determinants of arterial pressure. Vascular stiffness is one of the main causes of hypertension. Na+ H- exchange in vascular smooth muscle is increased in hypertensive patient and results in,

1) Increased vascular tone by means of increased sodium entry which leads to activation of Na+ Ca2+ exchange, finally increased intracellular calcium level and increased vascular tone occurs.

2) Increased Na+ H- ion exchange also stimulates growth of vascular smooth muscle by increasing sensitivity to nitrogen.

3) Vascular tone is also modulated by vascular endothelial function.

Vascular endothelial cells synthesis and release nitric oxide which cause vasodilation. Vascular endothelial cells also secrete endothelin which is a vasoconstrictor. Aerobic exercises and weight loss improves vascular compliance and endothelium dependent vasodilation.

Mechanism involving Immunity, Inflammation, Oxidative stress :

Inflammation and immune system are involved in pathogenesis of vascular injury and hypertension.

Inflammation results in generation of reactive oxygen species which modifies T cell function and further enhance inflammation. Oxidative stress

in renal medulla affects pressure - natriuresis and leads to development of hypertension.

Other conditions associated with hypertension :

Obesity :

Obesity is a major modifiable risk factor for hypertension. Framingham study has showed that 1.25 kg weight gain leads to 1mmHg rise of systolic blood pressure.

Abdominal obesity is measured by waist circumference.Values of greater than 80cms in women and 90cms in men is considered as abdominal obesity. It is a main risk factor for development of hypertension when compared to truncal obesity.

Morbid obesity usually leads to development of sleep apnea syndrome. Sleep apnea is characterised by development of hypoxemia during sleep which leads to increased sympathetic activity and release of endothelin. Both these factors finally causes increased systemic arterial hypertension.

Physical inactivity :

Physical inactivity leads to 20% to 50% increased risk of development of hypertension.

Regular 30 to 45 minutes of aerobic exercise atleast 5 days per week may reduce the pressure in hypertensive individuals.

Alcohol intake :

Alcohol intake causes increased sympathetic activity which leads increased cardiac output and increased heart rate.

Excessive alcohol intake increases the risk of development of hypertension upto 5% to 30% when compared to non alcoholic.

Ingestion of small amount of alcohol may reduce the incidence of coronary heart disease as it mobilize cholesterol from tissue to liver for excretion.

Recommended amount of alcohol intake in men is 30gms/day and in women is 20gms/day.

Smoking :

Smoking habit raises the blood pressure by releasing nicotine. Nicotine cause sympathetic nerve stimulation, increased release of epinephrine and norepinephrine and thereby vasoconstriction.

Diabetes mellitus :

Approximately 50% of Type 1 diabetes patients develop hypertension due to renal damage. But 80% of Type 2 diabetes develop hypertension as a component of metabolic syndrome.

JNC VII has recommended target blood pressure for a diabetes patient is 130/80mmHg.

Polycythemia :

Polycythemia is more frequently associated with hypertension. It increases the viscosity of blood which leads to high blood pressure because there is increase need of force to pump thicker blood through circulatory system.

High hematocrit levels interfere with the vasodilatory effects of nitric oxide which leads to development of hypertension.

Types and causes of systemic hypertension :

Types :

Broadly classified into

1) Both systolic and diastolic hypertension

2) Isolated systolic hypertension

Both systolic and diastolic hypertension :

Elevation of both systolic and diastolic blood pressure.

It is further classified into,

1) Primary hypertension

2) Secondary hypertension

Primary hypertension :

Also called essential hypertension. It includes 80 – 95% of total hypertensive cases. There is no identifiable cause for essential hypertension. It

may be due to interaction between environmental and genetic factors. The prevalence of essential hypertension increases with age.

Secondary hypertension :

It includes 5 – 20% of total hypertensive cases. There is known specific underlying disorder which causes high blood pressure.

Causes of secondary hypertension :

1) Renal causes :

a) Parenchymal diseases :

Acute glomerulonephritis

Chronic glomerulonephritis

Diabetic nephropathy

b) Cystic diseases :

Polycystic kidney disease

c) Obstuctive uropathy :

Hydronephrosis

d) Vascular causes :

Renal artery stenosis

Arteriosclerosis

Fibromuscular dysplasia

e) Tumors :

Reninoma – rennin producing tumour

f) Syndromes causing sodium retention :

Liddle's syndrome

Gordon's syndrome

2) Endocrine disorders :

a) Adrenal disorders :

Cortical causes :

Cushing syndrome

Congenital adrenal hyperplasia

Primary aldosteronism

Medullary causes :

Tumor – pheochromocytoma

b) Acromegaly

c) Hyperparathyroidism

d) Hyperthyroidism

e) Hypothyroidism

3) Coarcation of aorta

4) Obstructive sleep apnea

5) Pregnancy induced hypertension

a) Preeclampsia

b) Eclampsia

6) Neurologic disorders :

a) Increased intracranial pressure

Brain tumor

Encephalitis

b) Acute spinal cord section

c) Diencephalic syndrome

d) Acute porphyria

e) Lead poisoning

7) Acute stressful conditions :

a) Perioperative

b) Alcohol withdrawal

c) Psychogenic hyperventilation

8) Drugs :

a) High dose estrogen

b) Adrenal steroids

c) Appetite suppressants

d) Tricyclic antidepressants

e) Monoamine oxidase inhibitors

f) Erythropoietin

g) Cocaine

h) NSAIDS

i) Decongesants

j) Cyclosporine

Causes of Isolated systolic hypertension :

1) Increased cardiac output :

a) Aortic regurgitation

b) Thyrotoxicosis

c) Thiamine deficiency - Beriberi

d) Arteriovenous fistula

e) Patent ductus arteriosis

2) Decreased compliance of aorta

a) Atherosclerosis

Arterial disease in hypertension :

There are three layers in blood vessels.

1) Tunica intima

2) Tunica media

3) Tunica externa

Classification of arterial disease :

1) Disease involving large arteries :

a) Intimal layer disorder

Fatty streaks

Nodular atherosclerosis

- fatty plaques

- fibrous plaques

b) Medial layer disorder

Aneurysm

Hypertrophy

Fatty degeneration

Senile involution

Calcification

2) Disease involving small arteries :

a) Intimal layer disorder

Elastosis

Thickening

b) Medial layer disorder

Aneurysm

Hypertrophy

Degeneration

Atrophy

3) Disease involving smallest arteries and arterioles :

a) Arteriosclerosis

b) Fibrinoid arteriolar necrosis

Essential hypertension :

It is defined as a condition in which arterial blood pressure is raised for that no specific cause can be found.

It is diagnosed by exclusion of all known causes of hypertension. Its clinical manifestations represent the effect of increased blood pressure on central nervous system, cardiovascular system, renal system and eye.

Features of secondary hypertension :

Essential hypertension is specially characterised by rise of blood pressure when age increases. Therefore young adults with gross hypertension always have a secondary hypertension.

Features of specific diseases causing secondary hypertension are,

1) Coarctation of aorta-characterised by presence of high blood pressure in upper limb, low pressure and weak pulses in lower limbs.

2) Chronic Nephritis - H/o recurrent urinary tract infections, protienuria, presence of red cells and casts in urine

3) Polycystic kidney palpable- kidney, confirmation by imaging

4) Renal artery stenosis - uncontrolled hypertension, confirmation by aortography

5) Pheochromocytoma - urinary excretion of catecholamines

6) Conns syndrome - muscle weakness due to low potassium levels muscle cramps due to low calcium levels and polyuria.

Secondary hypertension may cause rapid rise of blood pressure in a short period. For example – renal artery stenosis, pyelonephritis.

Clinical features of essential hypertension :

Age of onset :

Prevalence of essential hypertension increases with age.

Possible mechanisms are,

1) Reduction in vascular compliance due to stiffening

2) Decrease in glomerular filtration rate while age increases.

Symptoms :

Most of the patients with essential hypertension have no specific symptoms. Usually it is identified during routine physical examination.

1) Headache

Headache may be present in some patient. But it occurs only in severe hypertension. It is commonly occurs when patient arises in morning and subsides spontaneously after several hours.

Other possible symptoms may be,

2) Chest pain and breathlessness due to cardiac failure

3) Giddiness due to transient ischemic attack

4) Blurring of vision due to retinopathy

5) Epistaxis and altered sensorium

Clinical evaluation of essential hypertension :

Asking history about,

1) Hypertension in other family members favours diagnosis.

2) Features of causes of secondary hypertension

3) Risk factors – smoking, alcohol, diabetes, kidney disease, diet, physical activity and family status.

Physical examination

1) General appearance Cushing syndrome -Round face and truncal obesity, Coartation of aorta -Muscular development of upper extremities is out of proportion to lower limbs

2) Measuring 3 BP recordings with interval of 2 minutes in all extremities, in supine or sitting position with feet on ground and after standing for atleast 2 minutes to rule out Coarctation of aorta, Postural hypotension.

3) Height, weight, Hip waist ratio measurement to find out risk factor Obesity

4) Examine the pulse in all extremities to find out delayed or absent femoral pulses.

5) Look for arcus senalis, Xanthelasma and Xanthoma

6) Fundus examination – for ascessing of retinal changes which gives clues regarding duration of hypertension.

7) Look for Pedal edema, JVP to rule out congestive heart failure.

CVS examination :

1) 3rd heart sounds – favours congestive heart failure

2) 4th heart sounds – favours left ventricular hypertrophy

RS examination :

Rales and Rhonchi are signs of pulmonary congestion seen in CCF.

Abdominal examination :

Look for abdominal bruits - Renal artery stenosis

Palpation of enlarged kidneys, masses – Polycystic kidney

CNS examination :

Look for any evidence of past or ongoing cerebrovascular disease.

Laboratory investigations :

There are three lines of investigations for evaluating hypertension.

1) Basic investigations

2) Additional investigations

3) Investigations specific for secondary hypertension

1) Basic investigations :

a) Hemoglobin

b) Hematocrit

c) Blood urea

d) Serum creatinine

e) Random blood sugar

f) Total cholesterol

g) Urine for protein, sugar, red cells, sediments

h) Electrocardiography

2) Additional investigations :

a) Fasting lipid profile

b) Chest x ray PA view

c) Echocardiogram

3) Investigations specific for secondary hypertension :

a) Renal artery stenosis

ACE inhibitor radionucleotide renal scan

USG Doppler study of renal vessels

Renal artery arteriogram

b) Cushing syndrome

24 hrs urine cortisol

Overnight dexamethasone suppression test

c) Primary aldosteronism

CT abdomen for adrenal mass

Plasma aldosterone to rennin activity ratio

d) Pheochromocytoma

CT or MRI head, neck, chest and abdomen for localising the tumor

24 hrs urine for catecholamine, metanephrine and creatine

Target organ dysfunction in hypertension :

The main target organs usually involved in hypertension are heart, brain, kidney, retina. Target organ dysfunctions mainly occurs in hypertensive patients who left untreated or undertreated.

1) Heart

Systemic hypertension causes both structural and functional changes in heart. They are,

a) Myocyte hypertrophy – LVH

b) Microvascular disease

c) Epicadial large coronary vessel disease

d) Congestive cardiac failure

e) Cardiac arrhythmia

Hypertension leads to both systolic and diastolic dysfunction.

Central nervous system :

Untreated severe hypertension may cause,

1) stroke

2) Hypertensive encephalopathy

1) Stroke

Incidence of stroke rises progressively with increasing blood pressure particularly systolic BP. (cerebral haemorrhage (15%) and cerebral ischemia (85%))**.** Cerebral haemorrhage is due to increased arterial pressure and formation of microaneurysms. Cerebral ischemia is due to atherosclerosis.

2) Hypertensive encephalopathy

Cerebral blood flow remains unchanged over a wide range of arterial pressure (MBP – 50 to 150mmHg) because of autoregulation of blood flow. But in malignant hypertension, failure of autoregulation of blood flow to brain occurs and resulting in vasodilation and hyperperfusion.

Symptoms include severe headache, nausea, projectile vomiting, focal neurological signs, alteration in mental status.

Untreated hypertensive encephalopathy may progress to stupor, coma, convulsion, death within a hour.

Retina :

Systemic hypertension causes retinopathy, neuropathy and choroidopathy. But most common presentation is retinopathy.

Hypertensive retinopathy includes,

1) acute effect due to arterial vasospasm

2) chronic effect due to arteriosclerosis, vascular aneurysm or macroaneurysm which leads to visual loss.

Grading of hypertensive retinopathy :

Keith & Wegner's grading :

Grade 1	Thickening & tortuosity of arteries showing silver wire appearance
Grade 2	Grade 1 changes + arterio venous nipping
Grade3	Grade II changes + flame shaped haemorrhage & cotton wool exudates
Grade4	Grade III changes + papilloedema

Flame shaped haemorrhage - due to bleed into nerve fibre layer

Dot blot haemorrhage – bleed into inner retina

Cotton wool spots – ischemia in nerve fibre layer secondary to fibrinous necrosis and luminal narrowing

Papilledema – due to leakage & ischemia of arteriole supplying optic disc.

Kidney :

Renal dysfunction is closely related to systolic than to diastolic blood pressure. In systolic hypertension the afferent, efferent arterioles and the

glomerular capillary tufts are affected due to atherosclerosis, focal segmental sclerosis which results in decreased glomerular filteration rate and tubular dysfunction. Micro and macroalbuminuria are early markers of renal injury.

Microalbuminuria (MAC) :

The term Microalbuminuria (MAC) is first coined Guy's Hospital group in London in 1982. It is defined as an abnormal elevated excretion of albumin in urine but without clinical protienuria.

It is an early sign and marker of vascular damage. It represent a abnormal change in generalised vascular system, not just confined only to renal microvascular system.

Epidemiology of microalbuminuria in essential hypertension :

Prevalence of microalbuminuria appears to be higher in non Europeans (8% - 28%) than in Europeans (2% - 10%). In US, approximately 6% of men and 9.7% of woman have microalbuminuria.

Albuminuria below 2mg/day should be considered as normal. Microalbuminuria should be assessed annually in all patients and every 6 months within the first year of treatment.

Definition of microalbuminuria :

The median excretion of albumin during daytime is 4 to 6 µg/min.

Microalbuminuria is defined as urinary excretion of albumin of 20 to 200 µg/min (or) 30 to 300 mg/24 hours.

Macroalbuminuria is defined as urinary excretion of albumin greater than 300mg /24 hrs.

Transient protienuria is characterized by protein excretion of < 1gm /24 hours and is seen in normal individuals.

Sustained protienuria is characterized by protein excretion of >1 - 2 gm/24 hours and is associated with glomerular disease.

Orthostatic protienuria occurs only with upright posture.

The concentration of albumin and creatinine excreted in morning first sample is similar to the concentration in 24 hours urine sample. If an overnight urine albumin to creatinine ratio is more than 2mg/mmol, then it predicts that the urinary albumin excretion is in the range of microalbuminuria.

Diagnostic range of various types of albuminuria :

	24 Hrs albumin(mg/24hrs)	ACR (mg/gm)	Dipstick protienuria	24 Hrs urine protein(mg/24hrs)
Normal	8 -10	<30	-	< 150
Microalbuminuria	30- 300	30-300	- /Trace/+	-
Macroalbuminuria	>300	>300	Trace 3+	>150

Mechanism of relationship between microalbuminuria and hypertension

There are three mechanism for microalbuminuria,

1) Raised intraglomerular pressure

2) Intrinsic glomerular damage with alteration in the glomerular filtration barrier.

3) Tubular function abnormalities

1) Increased intraglomerular pressure :

Glomerular pressure is determined by the balance between the relative vasoconstriction and vasodilation of the afferent and efferent of the arterioles.

Arteriolar tone is regulated by a number of mechanisms and presence of pressor and depressor substances.

Normally, the afferent arteriole protects the glomerulus against raised systemic arterial pressure by means of vasoconstriction. But vasoconstriction of efferent arterioles leads to increase in intraglomerular pressure.

If this autoregulatory function of arterioles becomes defective, then increase in intraglomerular pressure occurs. The raised intraglomerular pressure causes protein leakage and renal dysfunction.

Factors like stimulation of rennin angiotensin system, deficiency of vasodilatory hormones, increased sympathetic activation and hyperinsulin levels disturbs this autoregulation of arterioles.

ACE inhibitor drugs causes relaxation of the efferent arteriole which

leads to reduction in protein leakage and finally protects renal function. So it is clearly observed that systemic hypertension is directly related to the renal functional changes facilitating protein leakage.

Changes in glomerular filtration barrier :

Charge selectivity of glomerular basement membrane is important for normal glomerular function. In diabetes patients glycation of tissue proteins may cause loss of anionic charge leading to albumin leakage.

This association between impairment of charge selectivity of glomerular membrane and microalbuminuria has also been shown in individuals with no diabetes.

Microalbuminuria is the renal manifestation of generalized vascular endothelial dysfunction and is strongly correlated with development of cardio vascular disease.

Microalbuminuria is strongly associated with decreased size and charge selectivity of the glomerular vessel wall and is an independent markers of systemic vascular protein leakage.

Microalbuminuria is strongly correlates with von willebrand's factor, factor VIII hyperactivity, factor fibrinogen level and endothelial cell damage.

Methods to detect microalbuminuria :

Microalbuminuria cannot be detected by routine urine investigations.

The variability of microalbuminuria reported in hypertensive patients is

likely due to differences in

1) Techniques used to detect microalbuminuria

2) Criteria used for patient's selection

3) Modality of urine collection (spot versus 24 hours)

4) No of urine samples collected in each patient

Lower values have been reported in

1) patients taking antihypertensive drugs especially those drugs inhibiting rennin angiotensin system.

2) In patients having mild degree of hypertension.

Available screening test for microalbuminuria :

1) Albumin creatinine ratio in early morning sample

2) 24 hrs urine collection

3) Timed urine collection

Variable presentations of microalbuminuria :

The urinary albumin excretion rate can vary from day to day upto 40% in normal persons. Early morning urine sample have been reported to have smaller variations. Measurement of 24 hour urine albumin have been lowest variability.

Transient increase in urine albumin excretion occurs in acute illness, stressful conditions, prolonged upright posture, exercises etc.Increased urine

albumin excretion was noted in asian peoples, obese persons, increasing age and daytime urine samples.

Microalbuminuria can also be found in diabetes, nondiabetes persons with hypertension, cardiac failure, renal failure and urinary tract infection.

Methods available for measurement of human albumin in urine :

Urine albumin to creatinine ratio in a early morning sample has became a widely accepted tool for assessing urine albumin excretion in clinical practice. The AC ratio seems to be as sensitive as other methods for measurement of urinary albumin excretion.

Several methods are available with varying sensitivity and measurement time.

Method	**Time requirement for result**
Electroimmunoassay	4 to 6 hours
Radioimmunoassay	1 to 2 days
Immuno nephalometry	6 hours
Fluorescent immunoassay	4 to 6 hours
Immunoturbidometricassay	20 to 30 minutes
Zoneimmunoelectrophoresis	16 to 18 hours
Radioimmunodiffusionassay	1 to 2 days
Enzyme linked immunosorbantassay	5 to 6 hours

Physiology of microalbuminuria :

The glomerular transport depends on number of factors like**,**

1) Glomerular membrane status

2) Electric charge of the molecule

3) Size of the molecule

The fractional excretion of albumin is low due to its negative charge and large molecular size.

Mechanism of nondiabetic microalbuminuria :

Glomerular permeability changes is mainly responsible for development of microalbuminuria in hypertensive patient. The exact mechanism for the defect in glomerular filtration barrier is not known till now**.** The possible mechanism are,

1) renal vessel endothelial dysfunction

2) increased activity of rennin angiotensin system

3) lipid abnormalities

4) prothrombotic factors

5) systemic inflammation.

Finally, haemodynamic abnormality and structural changes in the kidney can be considered as causes of microalbuminuria.

Several data suggests that, irrespective of exact pathogenesis, microalbuminuria in hypertensive patient can be regarded as a specific, sensitive and cost effective tool for detection of patients at high risk to develop target organ dysfunction.

So screening for microalbuminuria can be considered as a part of initial workup of every hypertensive patient for effective management.

Only 5% of proteins filtered by the kidney are detectable by conventional immunochemical assays. The remaining >95% is degraded and undetectable by routine immunochemical assays.

Some studies have suggested that albumin excretion in healthy persons is associated with degradation lysosomes in renal cells distal to glomerular basement membrane. The degradation of filtered albumin before its excretion by lysosomes is a rapid process.

This process involves lysosomal uptake and exocytosis of peptide products back to the tubular lumen by renal cells distal to the glomerular basement membrane and occurs within minutes.

Some recent studies have shown that hypertension causes increased stretching forces in vessel walls which leads to upregulation of TGF- β levels. This upregulation increases lysosomal activity and induces albuminuria.

Microalbuminuria and cardiovascular disease :

Microalbuminuria is a marker of subclinical organ damage in nondiabetic hypertensive patients. It is associated with increased incidence of cardio vascular complications like left ventricular hypertrophy, myocardial ischemia, hypertensive heart failure, increased thickness of the coronary artery.

Microalbuminuria and cerebrovascular disease :

In some persons with essential hypertension, urine albumin excretion correlates well with development of increased carotid intimal & medial layer thickness and other peripheral arterial atherosclerosis. Microalbuminuria has been shown to be a important predictor of ischemic stroke which may be due to carotid artery atherosclerosis.

Microalbuminuria and kidney damage :

Even mild increase in arterial pressure for long duration of time may increase the development of end stage renal disease. The main mechanism behind that is development of nephrosclerosis.

Microalbuminuria and dyslipidemia :

The combined presence of microalbuminuria and dyslipidemia is frequent in patients with essential hypertension. Both has been regarded as a predictor of cardiovascular and renal dysfunction in hypertension.

Abnormal lipid profile like high total cholesterol and high triglycerides could cause tubulointerstitial damage by means of infiltration and deposition of fats in renal tubules.

In hypertensive patients, increased arterial pressure causes pulsatile stress to the glomeruli and contributes to glomerular damage. Hypertension accelerates renovascular atherosclerosis.

Microalbuminuria and insulin resistance :

Several studies has suggested that hypertensive patients having hyperbilirubinemia excrete greater amounts of albumin in urine. The presence of increased urinary albumin excretion in subject without diabetes predicts the future development of NIDDM.

The pathophysiology relationship between microalbuminuria and hyperinsulinemia is unclear. It may be genetically determined. Hyperinsulinemia may cause both microalbuminuria and hypertension probably by altering membrane permeability.

Microalbuminuria and endothelial dysfunction :

Microalbuminuria is a marker of generalised endothelial damage. Transmembrane albumin passage is due to reduced production of heparan sulphate and consequent loss of negative charge. Poor glycemic control inhibits the enzyme N – deacetylase which is responsible for heparin suphalte production.

The level of markers of endothelial dysfunction like VWF, activated factor VII (VIIa), thrombomodulin, are increased in patients having MAC.

Measures to reduce microalbuminuria in hypertension :

1) Antihypertensive therapy

ACE inhibitors is the first line drug to reduce blood pressure. It also has antiprotienuric effect.

Mechanisms for antiprotienuric effect of ACE inhibitors are,

a) Growth inhibiting effect on messengial cell and extracellular matrix

b) Reduction of intraglomerular pressure by means of dilating effect on efferent arterioles.

c) Increased production of bradykinin in renal circulation causes vasoprotection at the level of endothelium.

Several studies has suggested that ACE inhibitors should be stared in even normotensive diabetic patients to retard the progression of diabetic nephropathy.

2) Low protein diet :

Nitrogenous waste products causes afferent arteriolar dilatation and increases osmotic activity in glomerular apparatus.

So reduced intake of protein diet causes reduction in protienuria by means of decreasing glomerular hyperfilteration.

Scope of microalbuminuria in future :

Microalbuminuria is an index of generalised vascular damage.

It is an early marker for undiagnosed end organ damage in essential hypertension. To measure microalbuminuria easly available, simple, cheap and more reliable screening tests are available. Every hypertensive patients should do these test atleast annually once for better management.

METHODOLOGY

Patients with essential hypertension admitted to medicine wards of Goverment Thanjavur medical college, Thanjavur were chosen as subjects for this study during the period of January 2018 to October 2019.

Essential hypertension was diagnosed as per protocol and severity of hypertension has been classified as per Joint National Committee (JNC) VII report on prevention, detection, evaluation and treatment of high blood pressure.

Category	**Systolic BP (mmHg)**	**Diastolic BP (mmHg)**
Normal	<120	<80
Prehypertension	120 – 139	80 - 89
Stage 1	140 – 159	90 - 99
Stage 2	>160	>100
Isolated SBP	>140	<90

Two or more reading with two minutes interval were averaged for patient taking antihypertensive medication. Secondary hypertension was diagnosed and ruled out by clinical examination and appropriate investigations

Inclusion crieteria :

1) Patients with essential hypertension admitted in medical wards of Government Thanjavur medical college, Thanjavur.

2) Age – 25 yrs to 65 yrs

3) Sex – Both male and female

4) History – Patient having H/O hypertension

Exclusion crieteria :

1) Patient's refusal

2) Age - < 25 yrs and > 65 yrs

3) Proven case of secondary hypertension

4) Urinary tract infection

5) Congestive cardiac failure

6) Chronic kidney disease

7) Diabetes mellitus

8) Pregnancy

9) Drugs like steroids

10) Stressful conditions

During this study, a total of 100 patients were studied. All subjects were asked for detailed history about symptoms, duration of hypertension, drug intake, previous blood pressure recordings and thoroughly examined for signs suggestive of target organ damage and investigated with complete urine

analysis, haemogram, blood urea, serum creatinine random blood sugar, fasting lipid profile, electro and echocardiography, CT brain in suspected stroke patients.

Fundus examination was done in all 100 cases in this study, and retinopathy was classified as per KEITH - WAGNER - BAKER classification of hypertensive retinopathy. First morning urine sample for albumin creatinine ration estimation was done by photometric method.

Left ventricular hypertrophy was diagnosed by applying voltage criteria on ECG and confirmed by ECHO screening.Ischemic heart disease was considered to present based on past history and confirmed by ECG and ECHO screening. Chest x ray was taken if there was suspicision of cardiomegaly.

Echocardiography:

Echocardiogram remains the method of choice for diagnosing left ventricular hypertrophy, regional motion abnormality, systolic and diastolic dysfunction. Cardiac structures were evaluated with an M-mode and 2-dimensional echocardiography, patient in left lateral position and slight rotation of the chest.

Normal adult values of septal thickness and left ventricular posterior wall thickness (mean 9mm, range 6-11 mm in both). At the end of diastole in parasternal view at mitral chordate level, measurements were made.

Normal ventricular wall thickness slowly increases with age from a

mean of 7.7 mm in 30 years to 8.9 mm in 50 years. Normal septal thickness increases from a mean of 7.6mm in youths to 9.3mm in middle aged adults.

Left ventricular hypertrophy can be diagnosed by the presence of an thick ventricular septum and left ventricular posterior wall (> 11mm). Left ventricular hypertrophy quantification is usually obtained by estimating left ventricular mass.

Volume of left ventricular myocardial shell is defined as difference between the external volume of left ventricle and the left ventricle chamber volume. Left ventricular mass is usually calculated by multiplying the estimated left ventricle myocardial volume by 1.04 which is the specific gravity of myocardium.

Devereux and Reichek were the first two firson to correlate echo left ventricular mass estimates with LV specimen of the same hearts at autopsy. Both found that anatomical LV mass correlated best with LV measurements by penn conversion (r = 0.96), using the following equation,

LV mass = 1.04 { (IVST + LVID + PWT)3 – (LVID)3 } – 13.6gms

IVST - Interventricular septal thickness

PWT – Left ventricular posterior wall thickness

LVID – Left ventricular dimension (end diastolic)

Devereux suggested a cut off value of 134 gms / m2 for men and

110gms /m2 for women. Left ventricular hypertrophy is diagnosed when the values are greater than above said cut off vaue.

Concentric remodelling:

Normal LV mass and increased LV wall thickness

Eccentric remodelling:

Increased LV mass and normal LV wall thickness

Concentric LVH:

Increase in both LV mass and LV wall thickness

RESULTS AND INTERPRETATION

The present study **"Prevalence on microalbuminuria in essential hypertension and its relationship with duration and severity of hypertension and target organ dysfunction"** was conducted in Government Thanjavur medical college, Thanjavur during the period of January 2018 to October 2019. During this study after satisfying the selection criteria 100 essential hypertensive patients were included in this study.

Stasistical analysis:

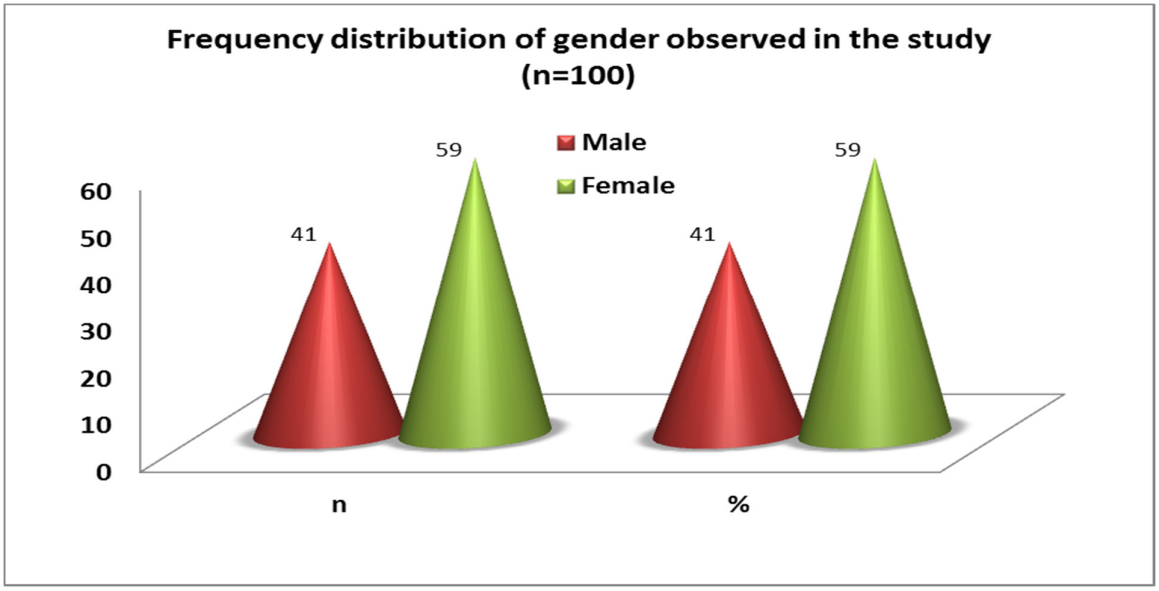
The data were coded and entered in MS excel office 2010. The data were analysed using graph pad prism version 5.

The categorical data were represented as n(%) and numerical data in mean standard deviation.

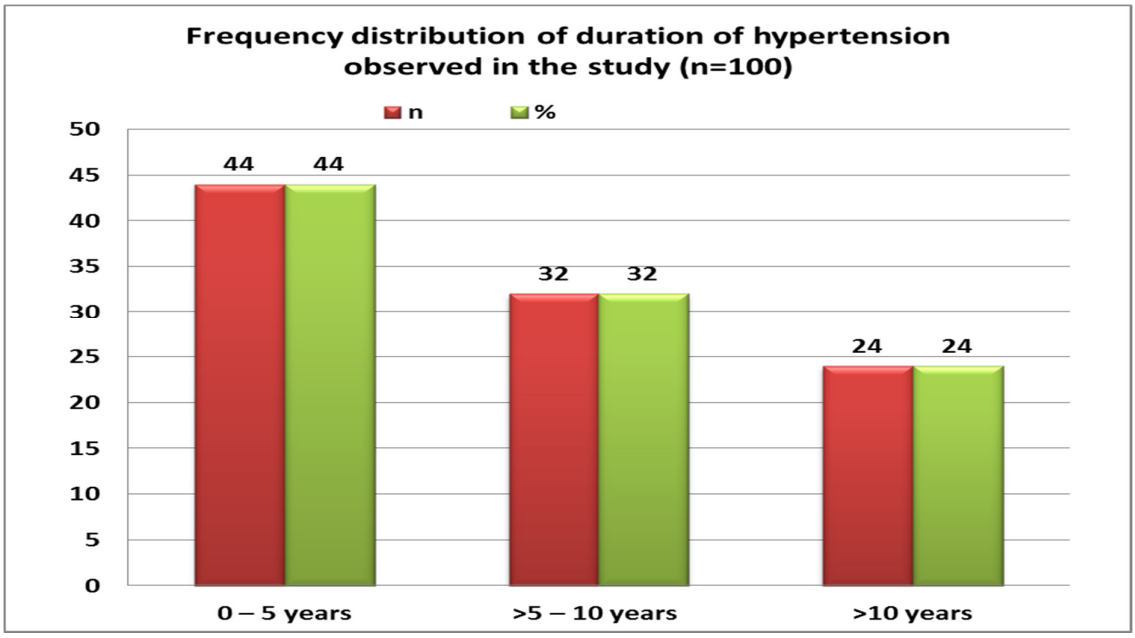
Fisher's exact test was used to compare the proportion between the groups for sample less than 30.

Unpaired 't' test was used to compare the mean between the normal and microalbuminuria groups.

Pearson's correlation was used to determine the strength of association between ACR and different variables. $P < 0.05$ was considered stasitically significant.



Vertical cone diagram depicting the frequency distribution of gender observed in the study (N=100)



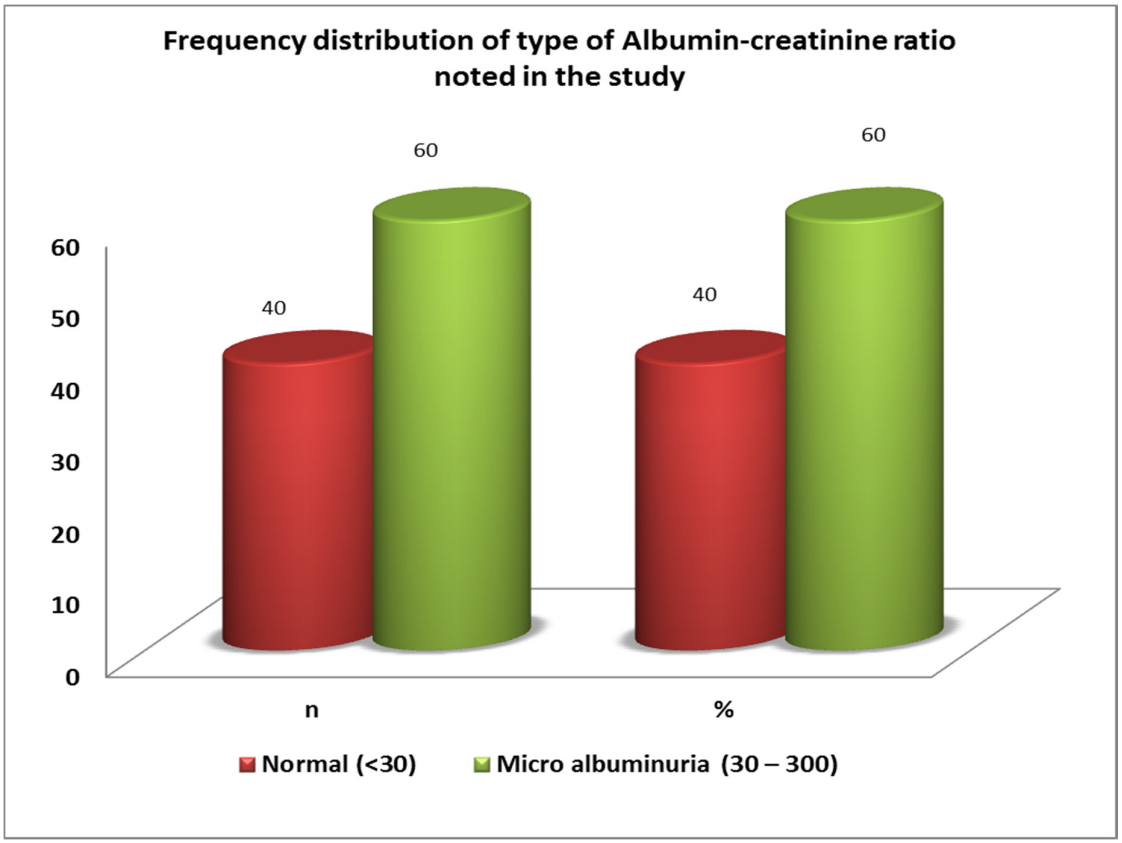
Vertical bar diagram representing the frequency distribution of duration of hypertension observed in the study. N=100

S.No	Parameter		n	%
1	Age category	25 – 30 years	1	1
		30 – 45 years	28	28
		46 – 60 years	53	53
		61 – 65 years	18	18
2	Gender	Male	41	41
		Female	59	59
3	Duration of hypertension	0 – 5 years	44	44
		6 – 10 years	32	32
		>10 years	24	24

Description of various baseline parameters in the study (overall N =100). Data are expressed as n with %. The total N=100

In this study, out of 100 cases with essential hypertension 1% had age in the range of 25 -30 years. 28 % of cases had age in the range of 30 – 45 years. 53% had age around 46 – 60 years.18% had age around 61 – 65 years.

Out of 100 cases participated, 41 members were male and 59 members were female. The no of cases with h/o duration of hypertension in years (0-5) was 44 cases. 32 cases had h/o duration in the range of 6 – 10 years. No of cases with duration of hypertension > 10 years was 24.



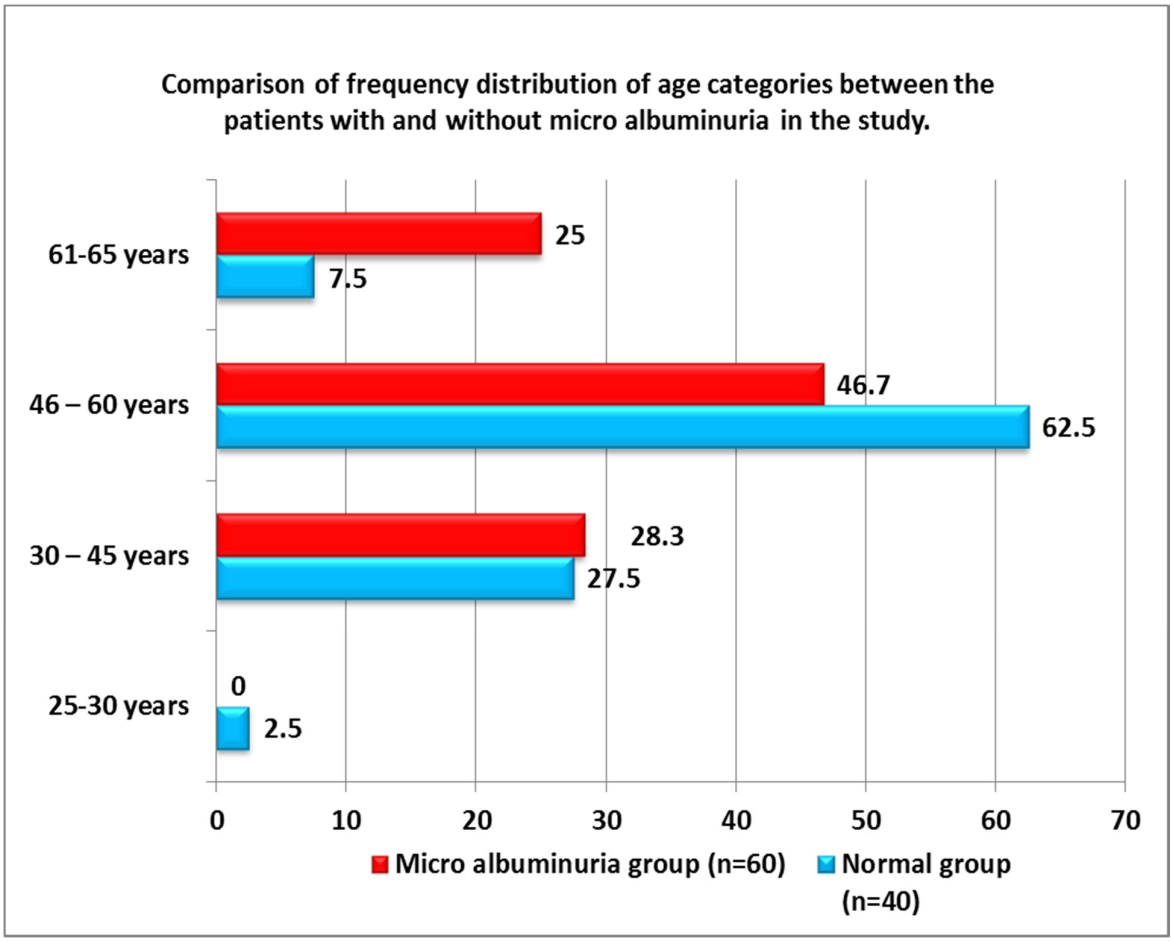
Cylindrical bar diagram depicting the frequency distribution of type of albumin-creatine ratio noted in the study.

S. No	Urine albumin-creatinine ratio (mg/g)	n	%
1	Normal (<30)	40	40
2	Micro albuminuria (30 – 300)	60	60

Frequency distribution of type of Albumin-creatinine ratio noted in the study. Data are expressed as n with %. The total N=100.

In this study, out of 100 essential hypertension cases 40% had normoalbuminuria. The remaining 60% had microalbuminuria. So it was found that the number of microalbuminuria cases is more than the number of normoalbuminuria cases in our study.

Horizontal bar diagram representing the comparison of frequency distribution of age categories between the patients with and without micro-albuminuria in the study. Data are expressed as percentages.



Comparison of frequency distribution of age categories between the patients with and without micro albuminuria in the study.

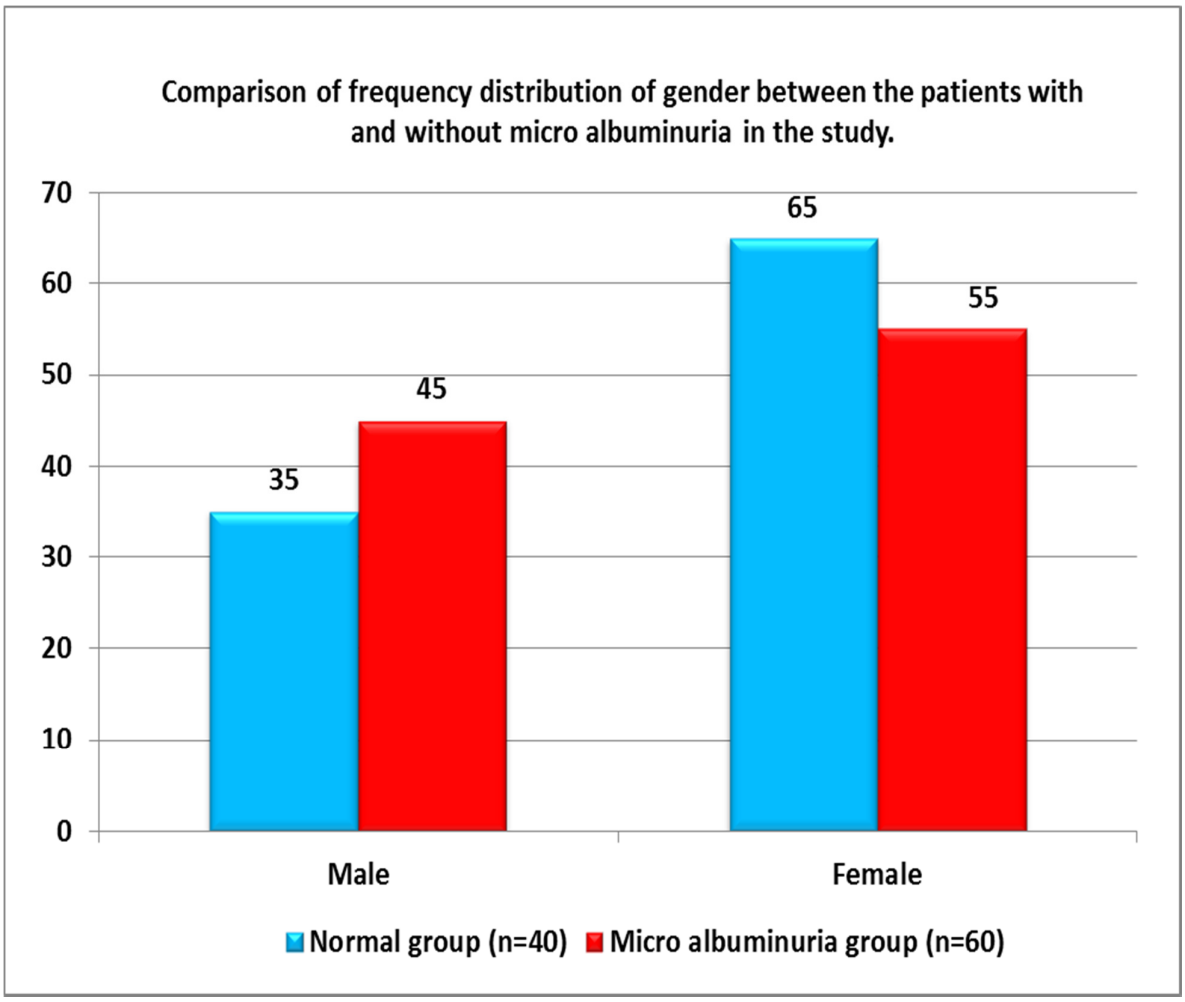
S. No	Age category	Normal group (n=40)		Micro albuminuria group (n=60)		Chi square value	df	p value
		n	%	n	%			
1	25 – 30 years	1	2.5	0	0	6.725	3	0.056 (NS)
2	30 – 45 years	11	27.5	17	28.3			
3	46 – 60 years	25	62.5	28	46.7			
4	61 – 65 years	3	7.5	15	25			

Data are expressed as n with %. Fisher's exact test was used to compare the frequencies between the groups. NS = Not significant.

In this study, the no persons with age between 25 – 30 yrs had microalbuminuria was 1(2.5%) and those had microalbuminuria was 0(0%). The no of persons with age between 30 – 45 yrs had normo albuminuria was 11(27.5%) and those had microalbuminuria was 17(28.3%). The no of persons with age between 46 – 60yrs had normo albuminuria was 25(62.5%) and those had microalbuminuria was 28(46.7%). The no of persons with age between 61 – 65yrs had normo albuminuria was 3(7.5%) and those had microalbuminuria was 15(25%).

The observed p value is 0.056 which is not significant. It indicates age has no relationship with microalbuminuria in patients with essential hypertension.

Vertical bar diagram representing the comparison of frequency distribution of gender between the patients with and without micro albuminuria in the study.



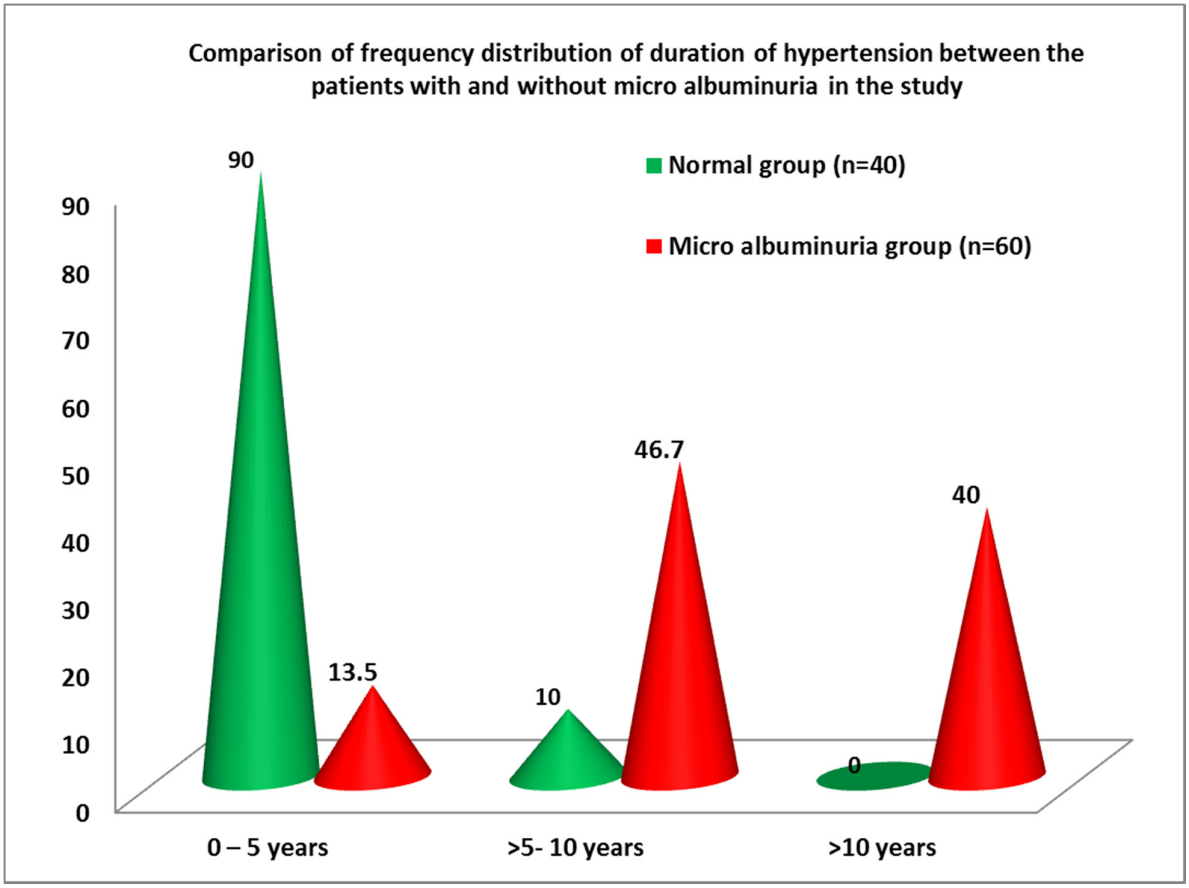
Data are expressed as percentages.

Comparison of frequency distribution of gender between the patients with and Without microalbuminuria in this study

	Gender	Normal group (n=40)		Micro albuminuria group (n=60)		Chi square value	df	p value
		n	%	n	%			
1	Male	14	35	27	45	0.992	1	0.407 (NS)
2	Female	26	65	33	55			

Data are expressed as n with %. Fisher's exact test was used to compare the frequencies between the groups. NS = Not significant

Among 41 males included in this study, 14(35%) patients had normo albuminuria and 27(45%) had microalbuminuria. Among 59 females, 26(65%) patients had normoalbuminuria and 33(55%) had micro albuminuria. P value is 0.407 which is not significant and indicates there is no relationship between gender and microalbuminuria in essential hypertension.



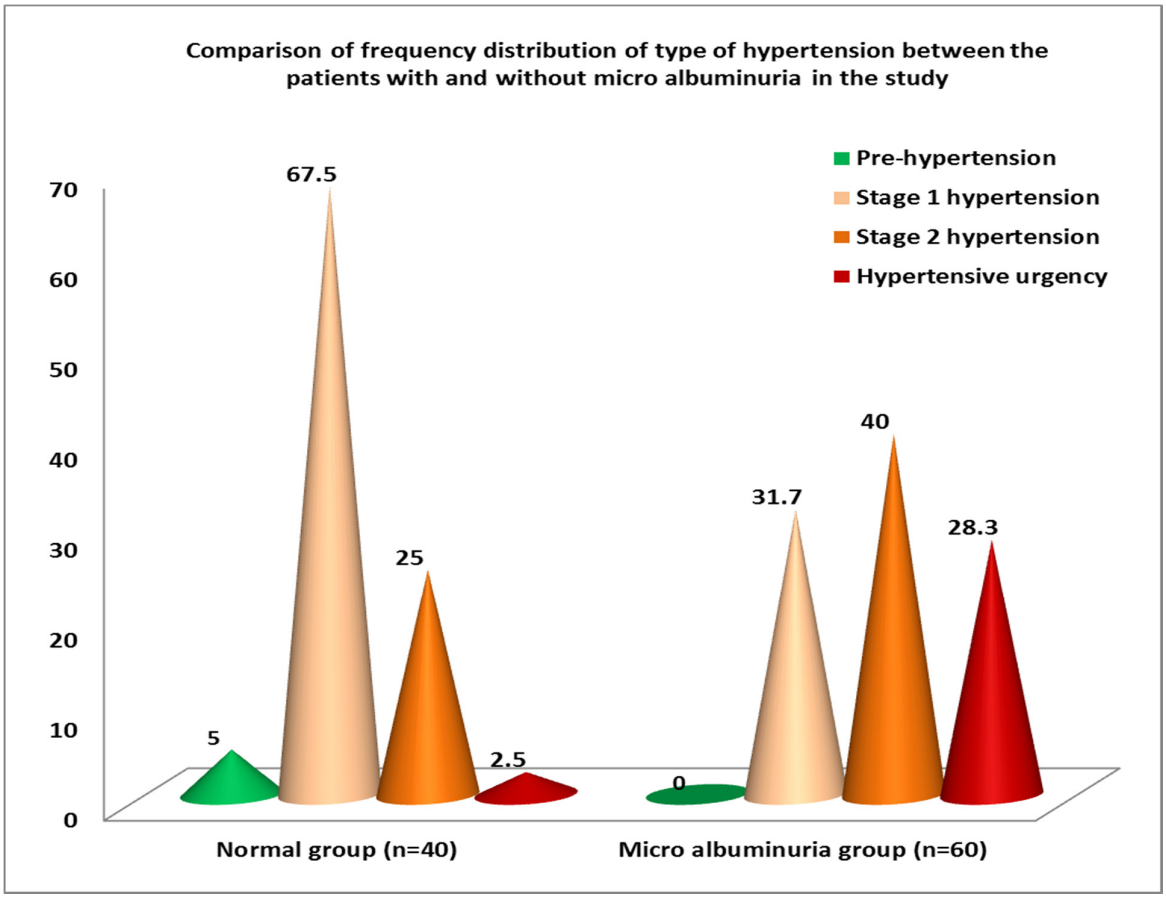
Vertical cone diagram representing the comparison of frequency distribution of duration of hypertension noted between the patients with and without micro-albuminuria in the study. Data are expressed as percentages.

Comparison of frequency distribution of duration of hypertension between the patients with and without micro albuminuria in the study.

S. No	Duration of hypertension	Normal group (n=40)		Micro albuminuria group (n=60)		Chi square value	df	p value
		n	%	n	%			
1	0 – 5 years	36	90*	8	13.3	58.14	2	<0.00001*
2	>5- 10 years	4	10	28	46.7*			
3	>10 years	0	0	24	40*			

Data are expressed as n with %. Fisher's exact test was used to compare the frequencies between the groups. *indicates p<0.05 and considered statistically significant.

In this study, among patients with h/o hypertension of duration 0 - 5 years 36(90%) persons had normoalbuminuria and 8(13.3%) persons had microalbuminuria. Among patients with h/o hypertension of duration 6 -10 years, 4(10%) persons had normoalbuminuria and 28(46.7%) persons had microalbuminuria. There was no persons with duration of hypertension > 10yrs had normoalbuminuria but 24(40%) persons had microalbuminuria. The observed P value is < 0.00001 which clearly indicates that when the duration of hypertension increases prevalence of albuminuria also increases.



Vertical cone diagram representing the comparison of frequency distribution of type of hypertension observed between the patients with and without micro-albuminuria in the study. Data are expressed as percentages.

Comparison of frequency distribution of type of hypertension between the patients with and without micro albuminuria in the study.

S. No	Type of hypertension	Normal group (n=40)		Micro albuminuria group (n=60)		Chi square value	df	p value
		n	%	n	%			
1	Pre-hypertension	2	5	0	0	20.18	3	0.000155*
2	Stage 1 hypertension	27	67.5 *	19	31.7			
3	Stage 2 hypertension	10	25	24	40*			
4	Hypertensive urgency	1	2.5	17	28.3*			

Data are expressed as n with %. Fisher's exact test was used to compare the frequencies between the groups. *indicates p<0.05 and considered statistically significant.

In this study the no of cases with prehypertension had normo albuminuria was 2(5%) and no of those had microalbuminuria was 0. The no of cases with stage 1 hypertension had normoalbuminuria was 27(65.5%) and no of those had microalbuminuria was 19(31.7). The no of cases with stage 2 hypertension had normoalbuminuria was 10(25%) and no of those had microalbuminuria was 24(40%). The no of cases with hypertensive urgency had normoalbuminuria was 1(2.5%) and no of those had microalbuminuria was 17(28.3%). The P value observed was 0.000155. It clearly indicates that the prevalence of microalbuminuria is increases when the severity of hypertension increases.

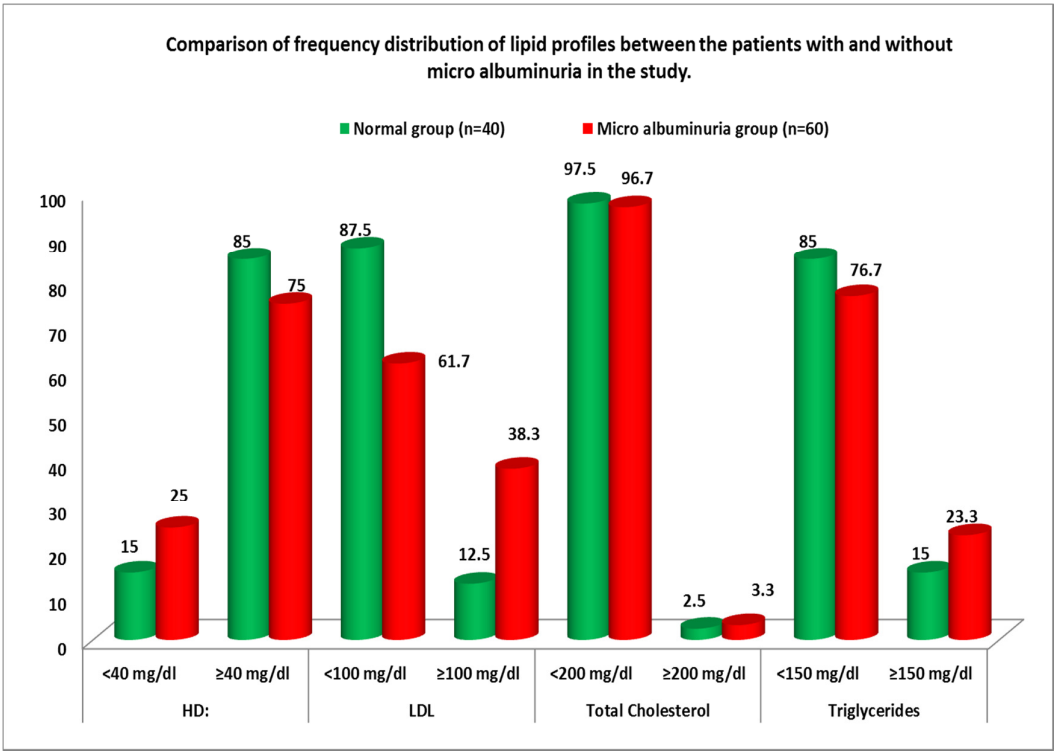
Comparison of mean SBP and DBP between the patients with and without micro albuminuria in the study.

S. No	Parameter	Normal group (n=40)		Micro albuminuria group (n=60)		't' value	df	p value
		Mean	SD	Mean	SD			
1	SBP (mm of Hg)	147.4	12.1	168.2	23.2	5.215	98	<0.00001*
2	DBP (mm of Hg)	92.2	6.9	99.4	12.9	3.224	98	0.002*

Data are expressed as n with %. Unpaired 't' test was used to compare the means between the groups. *indicates p<0.05 and considered statistically significant.

In this study, it was observed when the mean SBP was147.4mmHg, patients had normoalbuminuria. And in those patients with mean SBP 168.2 mmHg, microalbuminuria was present. When the mean DBP was 92.2mmHg, the patients had normoalbuminuria. In those patients with mean DBP 99.4mmHg, microalbuminuria was present. It was also observed that in this table, SBP is more significantly associated with microalbuminuria when compared with DBP.

Vertical cylindrical diagram depicting the comparision of various lipid profile between the groups in this study. Data are expressed as percentages.

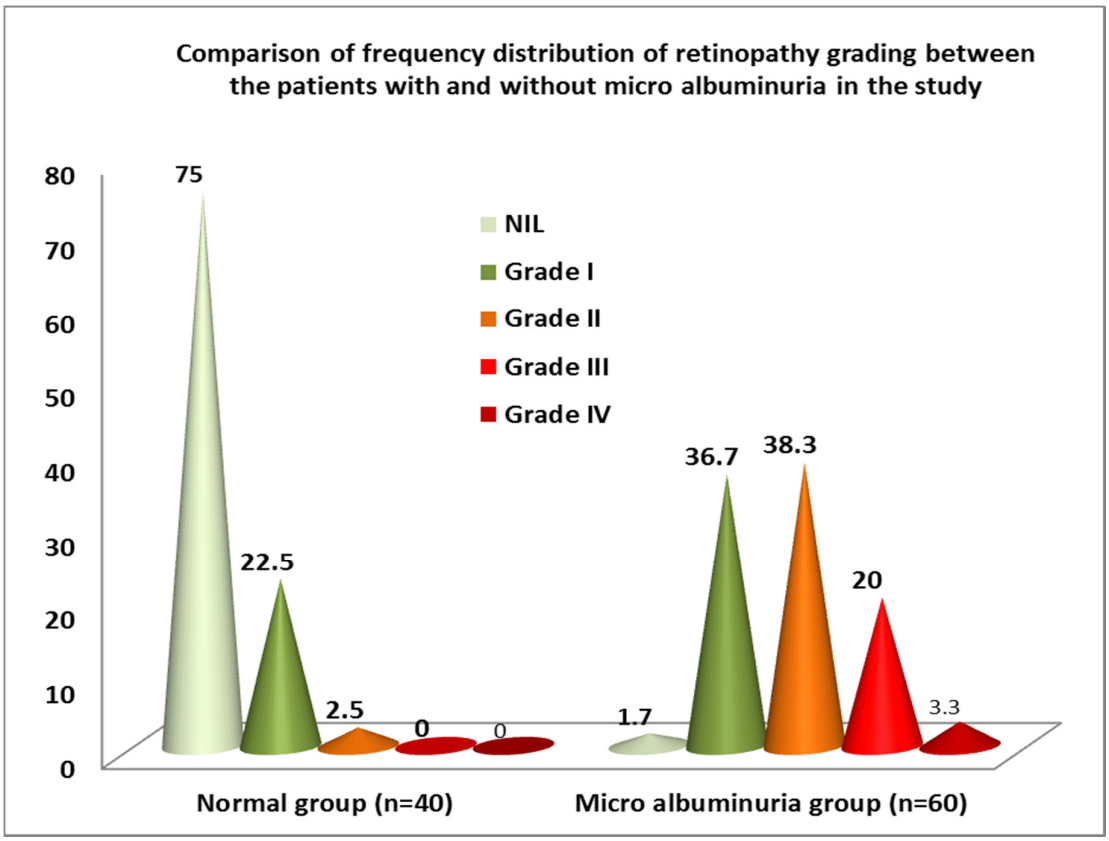


Comparison of frequency distribution of lipid profiles between the patients with and without micro albuminuria in the study.

S. No	Lipid profiles	Normal group (n=40)		Micro albuminuria group (n=60)		Chi square value	df	p value
		n	%	n	%			
1	HDL cholesterol					1.447	1	0.317 (NS)
	<40 mg/dl	6	15	15	25			
	≥40 mg/dl	34	85	45	75			
2	LDL cholesterol					7.945	1	0.006*
	<100 mg/dl	35	87.5*	37	61.7			
	≥100 mg/dl	5	12.5	23	38.3			
3	Total cholesterol					0.057	1	>0.999 (NS)
	<200 mg/dl	39	97.5	58	96.7			
	≥200 mg/dl	1	2.5	2	3.3			
4	Triglycerides					1.042	1	0.445 (NS)
	<150 mg/dl	34	85	46	76.7			
	≥150 mg/dl	6	15	14	23.3			

Data are expressed as n with %. Fisher's exact test was used to compare the frequencies between the groups. *indicates p<0.05 and considered statistically significant. NS = Not significant.

In this study, it was found that, LDL cholesterol level had strong relationship with prevalence of microalbuminuria. In patients with low HDL level and high Triglycerides and high Total cholesterol the prevalence of microalbuminuria seems to be little high.



Vertical cone diagram depicting the comparison of frequency distribution of retinopathy grading between the patients with and without micro-albuminuria in the study. Data are expressed as percentages.

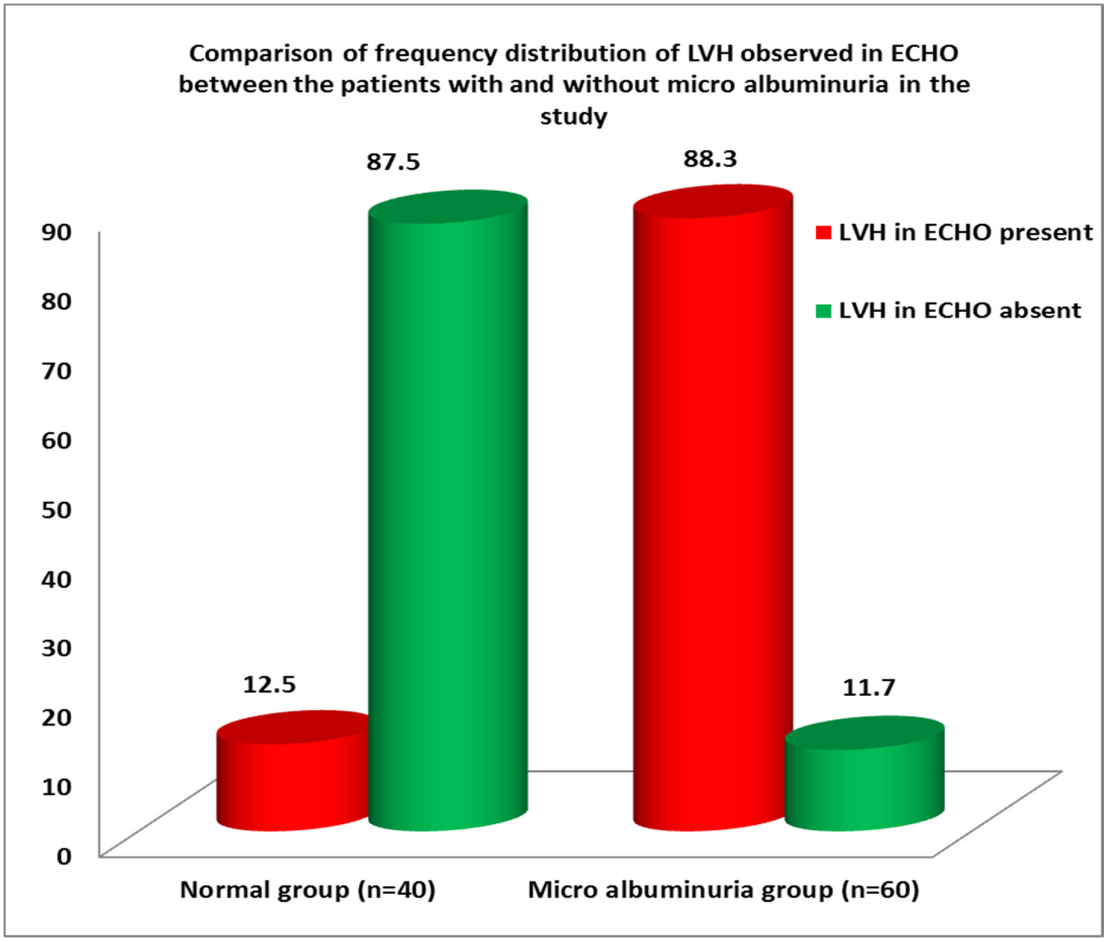
Comparison of frequency distribution of retinopathy grading between the patients with and without micro albuminuria in the study.

S. No	Grades of retinopathy	Normal group (n=40)		Micro albuminuria group (n=60)		Chi square value	df	p value
		n	%	n	%			
1	NIL	30	75*	1	1.7	65.36	4	<0.00001*
2	Grade I	9	22.5	22	36.7			
3	Grade II	1	2.5	23	38.3*			
4	Grade III	0	0	12	20*			
5	Grade IV	0	0	2	3.3			

Data are expressed as n with %. Fisher's exact test was used to compare the frequencies between the groups. *indicates p<0.05 and considered statistically significant.

Out of 100 patients, 30 cases with no retinopathy had normo albuminuria. Only one case with no retinopathy had microalbuminuria. 9 cases with grade 1 retinopathy had nomoalbuminuria. 22 cases with grade 1 retinopathy had microalbuminuria. Only one case with grade 2 retinopathy had normo albuminuria. 23 cases with grade 2 retinopathy had microalbuminuria. But no cases with grade grade 3 retinopathy had normoalbuminuria. 12 cases with grade 3 retinopathy had microalbuminuria. No cases with grade 4 retinopathy had normo albuminuria. 2 cases with grade 4 retinopathy had microalbuminuria.

The P value observed was <0.00001 and clearly indicates that patient with essential hypertension and microalbuminuria are more prone to develop retinopathy.



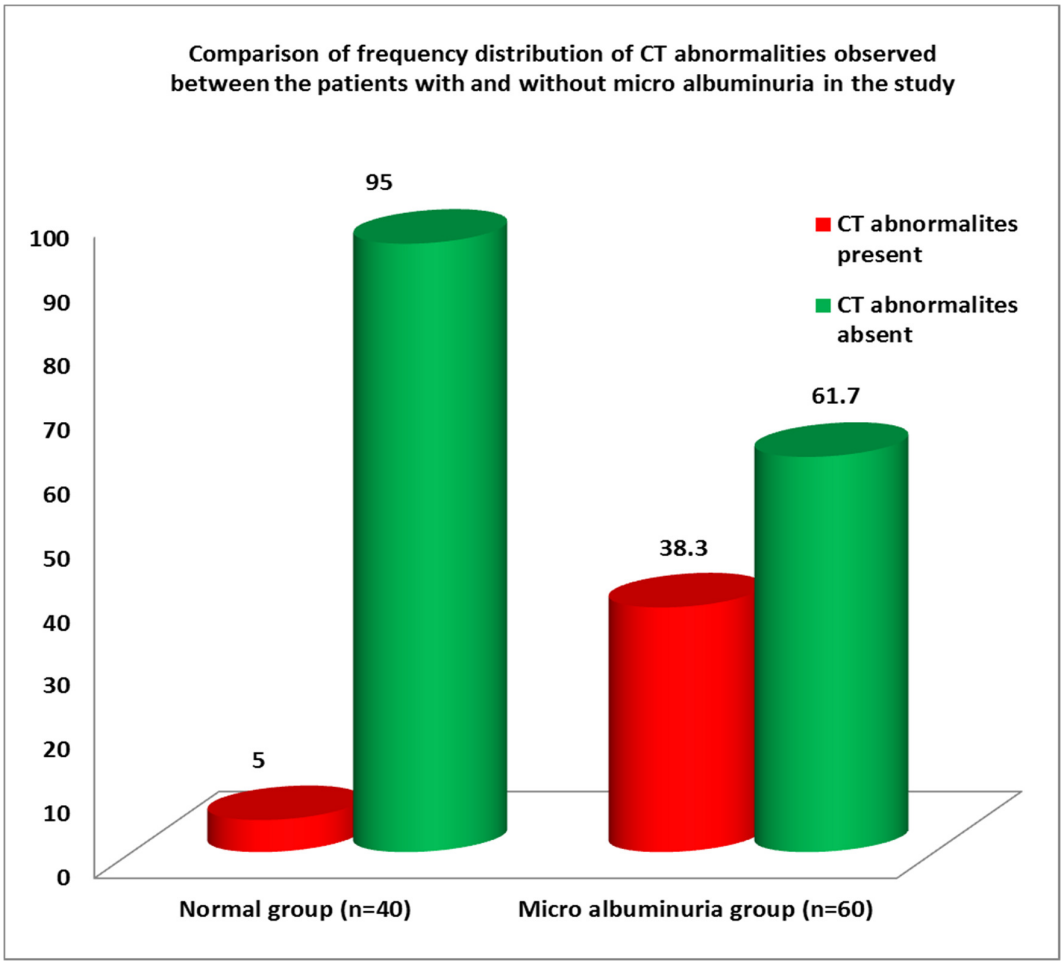
Vertical cylindrical diagram depicting the comparison of frequency distribution of LVH in ECHO observed between the patients with and without micro-albuminuria in the study. Data are expressed as percentages.

Comparison of frequency distribution of LVH observed in ECHO between the patients with and without micro albuminuria in the study.

S. No	LVH in ECHO	Micro albuminuria group (n=60)		Normal group (n=40)		Chi square value	df	p value
		n	%	n	%			
1	Present	53	88.3	5	12.5	56.65	1	<0.00001*
2	Absent	7	11.7	35	87.5			

Data are expressed as n with %. Fisher's exact test was used to compare the frequencies between the groups. The relative risk is 5.483 with confidence interval of 2.7 to 10.8. *indicates p<0.05 and considered statistically significant.

In this study, out of 100 patients 53 patients with microalbuminuria had LVH. 5 patients with normoalbuminuria had LVH. 7 patients with micro albuminuria had no LVH. 35 patients with normoalbuminuria had no LVH. Observed P value is < 0.00001 which indicates that persons having systemic hypertension and microalbuminuria are more prone to develop to LVH.



Vertical cylindrical diagram depicting the comparison of frequency distribution of CT abnormalities observed between the patients with and without micro-albuminuria in the study. Data are expressed as percentage

Comparison of frequency distribution of CT abnormalities observed between the patients with and without micro albuminuria in the study.

S. No	Abnormalities in CT brain	Micro albuminuria group (n=60)		Normal group (n=40)		Chi square value	df	p value
		n	%	n	%			
1	Present	23	38.3	2	5	14.22	1	0.00016*
2	Absent	37	61.7	38	95			

Data are expressed as n with %. Fisher's exact test was used to compare the frequencies between the groups. The relative risk is 1.865 with confidence interval of 1.44 to 2.41. *indicates p<0.05 and considered statistically significant.

Out of 100 study groups, 23 patients with microalbuminuria had abnormalities in CT brain. 2 persons with normoalbuminuria had abnormalities in CT brain. 37 persons with microalbuminuria had no CT brain abnormalities. 38 persons with normoalbuminuria had no CT brain abnormalities. The observed P value is 0.00016 which was significant and indicates that patient with essential hypertension and microalbuminuria are more prone to have stroke.

Correlation of urine-albumin-creatinine-ratio with the various parameters measured in the study.

S.No	Correlation of Urine albumin creatinine ratio with	Pearson's r value	R square	P value	Interpretation
1	Systolic blood pressure	0.502	0.252	<0.00001*	Positive association with moderate strength
2	Diastolic blood pressure	0.394	0.155	0.00005*	Positive association with weak strength
3	Duration of hypertension	0.598	0.357	<0.00001*	Positive association with negligible strength
4	Age	0.152	0.023	0.132 (NS)	No correlation

Correlation was done using Pearson's correlation test. * indicates p<0.05 and considered statistically significant. NS = Not significant

This table shows that in this study,

1) Systolic blood pressure has more significant positive association with prevalence of microalbuminuria.

2) Diastolic blood pressure also has significant positive association with prevalence of microalbuminuria but less than systolic blood pressure association with the same.

3) Duration of hypertension also has significant positive relationship with prevalence of microalbuminuria.

DISCUSSION

Protienuria is due to renal dysfunction. In this study, it was observed that the people with microalbuminuria are at increased risk to develop cardio vascular, cerebrovascular morbidity and mortality and retinal dysfunction.

Hypertension is commonly associated with microalbuminuria. The mechanism of microalbuminuria in hypertensive patient was found to be renal manifestation of generalized vascular endothelial dysfunction.

Most of the retrospective and cross sectional studies have reported that prevalence of cardiovascular disease is significantly higher among hypertensive patients with microalbuminuria than hypertensive patients with normoalbuminuria.

Therefore by means of early detection of significant microalbuminuria in patients with essential hypertension, the future development of target organ dysfunction can be prevented. With this aim and objective, this cross sectional study was conducted as per protocol to find out the prevalence of microalbuminuria in essential hypertension and its relationship with duration and severity of hypertension and target organ dysfunction.

Totally 100 cases admitted in medicine ward of TMCH, Thanjavur were included in this study after following the inclusion and exclusion criteria.

In this study, it was found that out of 100 patients with essential hypertension, 60 patients had microalbuminuria. This prevalence is greater when compared to the prevalence of microalbuminuria in various other studies ranging from 24% to 46%.

It is very clear that the prevalence of microalbuminuria in this study is higher than the observation from other studies which may be due to greater sensitivity of early morning urine ACR detection method used in this study.

Among 41 males and 59 females included in this study, 45% of males and 55% of females had microalbuminuria which is stasistically insignificant. It is clearly observed that there is no correlation between microalbuminuria in hypertensive patients and gender of that patients.

In this study, mean diastolic pressure in microalbuminuric cases was 99.4mmHg and in normoalbuminuric cases was 92.2mmHg. The observed P value was(P - 0.002) which is stasistically significant.

In this study, mean systolic pressure observed was 168.2 mmHg in microalbuminuric cases and 147.4mmHg in normoalbuminuric cases. By this observation it is clear that microalbuminuria correlates well with severity of hypertension particularly with systolic blood pressure.

Several studies conducted previously has reported that more number of hypertensives are microalbuminuric. These patients with microalbuminuria had significantly longer duration of hypertension and higher degree of hypertension.

This implies that vascular damage occurs with increasing severity and duration of hypertension and is probably reflected as microalbuminuria. All microalbuminuric essential hypertensives patients showed a higher prevalence of coronary artery diseases, stroke and retinopathy which indicates a indirect reflection of wide spread vascular damage.

Out of 100 patients in this study, 46 cases had stage 1 hypertension. Among these 46 cases, 19 (31.7%) cases were microalbuminuric and 27(67.5%) cases were normoalbuminuric. Then 34 cases had stage 2 hypertension. Out of these 34 cases, 24(40%) were micro albuminuric and 10(25%) cases were normo albuminuric. Then out of remaining 18 patients, having hypertensive urgency, 1(2.5%) cases had normoalbuminuria and 17(28.3%) cases had normoalbuminuria.

These clearly shows that as the stage of hypertension increases, the prevalence of microalbuminuria also increases which is stasistically significant. The observed P value is p (0.000155).

This study showed that microalbuminuria have significant correlation with duration of the disease. The prevalence of microalbuminuria for first 5 yrs was 13.3%. It increases to 46.7% at around 6 to 10 yrs. When the duration of hypertension is more than 10 yrs the prevalence of microalbuminuria the prevalence is almost 100%.

In general patients having hypertension of less than 10 yrs of duration 60% of cases had microalbuminuria. In cases had h/o duration of hypertension more than 10 yrs, almost all 100% had microalbuminuria. In this study, the mean duration of hypertension in patients with microalbuminuria observed was 9.5 years and in patients with normoalbuminuria the duration observed was 3.96 years.This clearly showed that longer the duration of hypertension, greater the prevalance of microalbuminuria. The observed P value is (p – 0.0001).

In this study out of 31 cases with grade 1 retinopathy, 22(36.7%) cases had microalbuminuria. Among 24 cases with grade 2 retinopathy, 23(38.3%) cases had microalbuminuria. All the 12(100%) cases with grade 3 retinopathy and 2 (100%) cases with grade 4 retinopathy had microalbuminuria. This clearly indicates that microalbuminuria in hypertension is strongly associated with retinopathy.

In this study, out of 100 cases, 58 cases had left ventricular hypertrophy (LVH). 42 cases had no evidence of LVH. Among those cases with LVH, 53(88.3%) had microalbuminuria. Among those patients with no LVH, 7(11.7%) cases had microalbuminuria.

P value observed is < 0.00001. So this study showed that more patient having microalbuminuria were found to have LVH when compared to cases had normoalbuminuria with LVH.

In this study, out of 100 patients 25 cases had ischemic heart disease(IHD). Among these 25 cases, 19 had microalbuminuria. The remaining 6 cases had normoalbuminuria. This observation clearly showed that cases with hypertension and microalbuminuria had more prevalence of IHD when compared to those with hypertension and normoalbuminuria.

Out of 100 patients in this study, 28 patients had CT brain abnormalities. Among these 28 patients, 23 cases had microalbuminuria and remaining 5 patients had normoalbuminuria. This observation indicates that patient with hypertension and microalbuminuria had more prevalence of stroke than the cases with hypertension and normoalbuminuria.

In this study, 72 patients had normal LDL cholesterol level. Among these 72 cases only 37 cases had microalbuminuria. 28 cases had elevated LDL level and among these 28 cases 23 cases had microalbuminuria.

Out of 100 cases, 80 cases had normal Triglycerides(TGL) level. Among these 80 cases, 46 cases had microalbuminuria. 20 cases had elevated TGL level. Among these 20, 14 cases had microalbuminuria.

Among 100 cases in this study, 79 patients had normal HDL level. Out of these 79 patients, 45 had microalbuminuria. The remaining 21 patients had low HDL level. Among these 21 , 15 patients had microalbuminuria.

By means of this observation in this study, it is very clear that more percentage of patients with microalbuminuria have high level of triglycerides and LDL and low level of HDL.

CONCLUSION

In this study, the prevalence of microalbuminuria in essential hypertension was found to be 60%. Microalbuminuria in essential hypertension does not have any correlation with sex of the patient. When the duration and severity of hypertension increases, the prevalence and degree degree of micro albuminuria increase. Increase in systolic blood pressure has better correlation with micro albuminuria when compared to normoalbuminuria. In this study, among patients with essential hypertension and microalbuminuria, 100% had retinopathy , 88.3% had LVH, 31.6% had IHD and 38.3% had CVA.

SUMMARY

This study on prevalence of microalbuminuria in essential hypertension and its relationship with duration and severity of hypertension and target organ dysfunction was conducted in Thanjavur medical college, Thanjavur.

Totally 100 patients of essential hypertension were included in this study after using inclusion and exclusion criteria as per protocol. Among 100 patients, 40 patients had normoalbuminuria and 60 patients had microalbuminuria.

Participants with longer duration of hypertension (> 10 years) , stage 2 hypertension and high systolic blood pressure (168.2mmHg) were found to have high mean range of micro albuminuria which were found to be stasistically more significant.

Diastolic blood pressure was found to have significant correlation with degree of microalbuminuria but not much that of systolic blood pressure correlation.

Level of LDL cholesterol showed a positive correlation with microalbuminuric hypertensive patient which were stasistifically significant.

Hypertensives with microalbuminuria were found to have have significantly higher prevalence of hypertensive retinopathy(100%), left ventricular hypertrophy(88.3%), cerebrovascular disease(38.3%) and IHD(31%).

Gender of the patient with essential hypertension has found to have no correlation with degree of microalbuminuria.

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INFORMED CONSENT FORM

Mr/Miss/Mrs. _____

You are invited to participate in our research study that is, 'Study on prevalence of microalbuminuria in essential hypertension and its relationship with duration and severity of hypertension and target organ dysfunction, a cross sectional study at Thanjavur medical college'.

Why am I being asked to participate in this research?

Patients admitted in Thanjavur medical college with essential hypertension are eligible for inclusion into the study group. Participation is entirely voluntary, as you_____are a patient with essential hypertension. Since you are eligible for this research, you are asked to participate in the same.

Why is this research being done?

Purpose of this research :

a. To determine the prevalence of Microalbuminuria is patients with essential hypertension
b. To correlate micro albuminuira with the clinical profile in patients with essential hypertension
c. To evaluate Microalbuminuria inrelation to complications of essential hypertension
d. Better management of hypertension patients using Microalbuminuria as a prognostic indicator.

Procedures Involved

a. Biochemical analysisof early spot urine sample to meaure albumin creatinine ratio,

Are there any potential risks or discomforts?

Yes, there are potential risks or discomfort involved.

Are there benefits to taking part in this research?

On the basis of new information there could be a better patients management as a long term prognostic indicator. There will be no extra benefits to patients otherwise.

What other options are there?

The patient has the option to decline participation in this study without any discrimination and the patients will treated as per the existing protocol for the condition.

Will I be told about the new information that may affect my decision to participate?

All information collected during study from patient will be told to her/him.

What about privacy and confidentiality?

The privacy of the individual will be respected and all information of the patients in the study will be kept strictly confidential

What if I am injured as a result of my participation?

There will neither by any compensation to or for the patient and his/her relatives nor would there be any monetary benefits for the damage incurred.

What are the costs for participating in this research?

Does not apply to this research

Will I be reimbursed for any of my expenses for participation in this research?

No

Can I withdraw or be removed from the study?

To start with as the participation was voluntary, patient can be with draw from the study. Such a step will not alter the patient management by any of the staff in the hospital.

Whom should I contact if I have any questions?

At any time during or after the study, Dr. G.KESAVAN can be called on 9003743174 for any questions pertaining to the study.

Signature of the participant or legally authorized representative :

Participant's Name : _____

Signature: _____

Experimenters / Witness's name: _____

Signature: _____

Date : _____

Place : _____

PROFORMA

Name : Address :

Age:

Sex:

Occupation : D.O.A :

Education : D.O.D. :

Marital Status : I.P. No. :

Unit : Ward :

PRESENTING COMPLAINTS

1. Headache
2. Palpitation
3. Chest pain
4. Breathlessness
5. Oedema
6. Giddiness
7. Blurring of vision
8. Stroke

HISTORY OF PRESENT ILLNESS

Headache

Duration -

Onset -

Site -

Type -

Radiation -

Aggravating factors -

Relieving factors -

Associated symptom -

Any other -

Chest Pain

Duration -

Onset -

Site -

Type -

Associated with -

Any other -

Radiation -

Aggravating factors -

Relieving factors -

Palpitation

Onset -

Progression -

Regular or irregular -

How does it subside -

Associated symptoms -

Any other -

Breathlessness

Duration -

Onset sudden or insidious -

Severity at the height -

Progress, if exertional -

grade of breathlessness -

Orthopnea -

PND Attacks -

Oedema

Duration -

Unilateral / bilateral -

Where did it appear first -

Progression -

Painful / painless -

Diurnal variation -

Any other -

History of (CVA) : Present /Absent

History of renal disorder : Present / Absent

History of liver disorder : Present / Absent

PAST HISTORY

Diabetes: Present / Absent

Duration -

Treatment -

Hypertension : Present / Absent

Duration -

Treatment -

IHD: Present / Absent :

Renal Disease : Present/Absent

Drugs intake : Present/Absent

FAMILY HISTORY

Hypertension

Diabetes

PERSONAL HISTORY

Diet -

Appetite -

Sleep -

Micturation / Bowels -

Habits : Smoking - Quantity

Alcohol - Quantity

Mode of life : Sedentary / Executive / Moderately active / very active

GENERAL PHYSICAL EXAMINATION

Height: Tall / Average / Short

Weight: Expected / Over weight / Underweight

Built : Obese / Average / Thin

Nourishment : Good / Moderate / Poor

Subcutaneous fat :

Mental State : Conscious / Altered / Drowsy / Emotional

Anemia : Present / Absent

Cyanosis : Present / Absent

Clubbing : Present / Absent

Jaundice : Present / Absent

Lymphadinopathy : Present / Absent

Thyroid : Present / Absent

Edema: Present / Absent

VITAL SIGNS

Pulse

Respiration

B.P.

SYSTEMIC EXAMINATION

CARDIO VASCULAR SYSTEM

Peripheral C.V.S.

1) Pulse

Rate	Rhythm
Volume	Tension
Force	Condition of vessel wall
Character of pulse	Radial to radial comparison
Radial – femoral comparison	

Pulsations in peripheral vessels : Right Left

Dor Pedis -

Post tibial -

Popliteal -

Femoral -

Brachial -

Carotid -

2) Signs of Congestive cardiac failure

Jugular venous pressure

Liver

Dependent oedema

Hepato jugular reflux

Blood pressure : Upper limb - Standing / Sitting

Lower limb -

CENTRAL CARDIOVASCULAR EXAMINATION

Inspection

Pre-cordial bulge

Apical impulse location

Pulsation other than apical impulse

Other areas

Palpation

Apical impulse : location character

Left parasternal heave

Epigastric pulsation

Diastolic shock

Supra clavicular pulsations

Thrills

Any other pulsations

Treacheal tug

Percussion :

Rt. Border

Lt. Border

Rt. 2nd space

Lt. 3rd space

Sternum : Upper / Lower

Ausculation

(Heart sounds : Splits, added sounds, loudness etc)

First heart sound

Second heart sound

Splitting

Murmurs : Site where best heard

Timing -

Character -

Grade -

Pitch -

Conduction -

Which body position best heard -

Relation to phase of respiration -

With bell or diaphragm -

Special manouers to alter the murmur -

Pericardial Rub -

PER ABDOMEN

Inspection

Palpation : Renal Mass / Aorta / Liver / Spleen

Percussion : Free fluid

Auscultation : Bruit over aorta / Renal arteries

RESPIRATORY SYSTEM

Breath sounds :

Added sounds :

CNS EXAMINATION:

Normal / Abnormal

FUNDUS EXAMINATION

Retinopathy – Present / Absent

Grade -

Urine – early morning spot ACR

Hemoglobin -

Hematocrit -

Blood urea -

Serum creatinine -

Fasting lipid profile -

Random Blood Sugar

CT Brain -

Chest X –ray

ECG

ECHO

Fundus Examination

KEY TO MASTER CHART

Acc HTN	Accelerated hypertension
ACRT MCA	Acute right middle cerebral artery
ALD with PHT	Alcoholic liver disease with portal hypertension
APD	Acid peptic disease
ASMI	Anterior wall myocardial infarction
AW ischemia	Anterior wall ischemia
BP sys/dia	Blood pressure, systolic /diastolic
Br. Asthma	Bronchial asthma
CABG	Coronary artery by pass grafting
CT	Computerized tomography
CVA	Cerebrovascular accident
DVD	Double vessel disease
ECHO	Echocardiography
Ess HTN	Essential hypertension
F	Female
FBS	Fasting blood sugar
GBS	Gullain barre syndrome
HDL	High density lipoprotein
IHD	Ischemic heart disease
IP No	Inpatient number
IWMI	Inferior wall myocardial infarction
LDL	Low density lipoprotein

LL	Lower limb
Lt	Left
M	Male
MCA	Middle cerebral artery
MRI	Magnetic resonance imaging
PCA	Posterior cerebral artery
Prox myopathy	Proximal myopathy
RHD	Rheumatic heart disease
Rt.	Right
RVD	Retrovial disease
SAH	Subarachnoid hemorrhage
Sl No.	Serial number
TG	Triglycerides
TIA	Transient Ischemic attack
Total	Total cholesterol
TVD	Triple vessel disease
UL	Upper limb
UMN	Upper motor neuran
VBI	Vertebrobaslar insufficiency
VII N Pl	7th nerve palsy

SL.NO	NAME	AGE	SEX	IP NO	DOA	ALBUMIN CREATINIE RATIO(mg/gm)	CREA TININE	RBS	TOTAL CHOLESTEROL	HDL	LDL	TGL	RETINOPATHY GRADE I - IV	ECHO-LVH	ECHO-RMWA	CT BRAIN	OTHERS
1	SARASU	55	FEMALE	53546	1.10.18	90	1	88	166	44	88	168	II	PRESENT	NIL	LT CG HAEMORRAGE	
2	BANUMATHI	55	FEMALE	53657	10.10.18	138	0.9	86	144	42	83	94	II	PRESENT	NIL	LT MCA INFARCT	
3	ANANTHAVALLI	53	FEMALE	60916	6.11.18	52	1	96	135	40	55	196	III	PRESENT	NIL	RT CG HAEMORRAGE WITH IVH	
4	VASUDEVAN	57	MALE	67359	3.12.18	22	0.8	102	168	34	107	138	NIL	ABSENT	NIL		COPD
5	SUCILA	58	FEMALE	68788	10.12.18	24	1..1	94	140	44	66	154	NIL	ABSENT	NIL		UNKNOWN BITE
6	GUNASEKARAN	36	MALE	70174	16.12.18	110	0.7	102	212	48	139	126	I	PRESENT	OLD ASMI	RT MCA INFARCT	
7	MAHAMAYEE	61	FEMALE	70185	16.12.18	280	1.1	106	148	46	82	98	III	PRESENT	NIL	RT MCA INFARCT	
8	NAGARAJ	58	MALE	70439	18.12.18	38	1	110	170	40	108	104	I	PRESENT	OLD IWMI		
9	LAKSHVANAN	60	MALE	70439	18.12.18	22	1	126	164	46	94	120	I	ABSENT	NIL		URTICARIA
10	KATHIRVEL63	63	MALE	70500	17.12.18	278	0.9	122	182	38	116	142	III	PRESENT	NIL		ACCELERATED SHTN
11	SAHAYAMARY	50	FEMALE	73388	31.12.18	20	1	108	154	48	76	134	NIL	ABSENT	NIL		APD
12	ARBUSBEGAM	62	FEMALE	1368	7.1.19	86	0.8	128	160	42	90	140	II	PRESENT	NIL		BPPV
13	RAMAYEE	64	FEMALE	4370	21.1.19	92	1	118	174	38	108	138	I	PRESENT	OLD AWMI		
14	PUSHPA	55	FEMALE	5696	27.1.19	22	0.9	110	146	36	80	146	I	PRESENT	OLD IWMI		
15	LATHA	45	FEMALE	5803	28.1.19	36	1	130	182	46	114	110	I	PRESENT	OLD ASMI		
16	THANGAPONNU	52	FEMALE	5800	28.1.19	14	0.9	122	156	42	89	128	NIL	ABSENT	OLD IWMI		
17	SUCILA	60	FEMALE	6086	28.1.19	84	1.1	102	190	38	121	146	II	PRESENT	OLD IWMI		
18	BHAVANI	42	FEMALE	14459	3.3.19	19	1	96	140	42	72	134	NIL	ABSENT	NIL		BPPV
19	MINNALKODI	59	FEMALE	15668	11.3.19	14	0.9	128	186	40	118	140	II	PRESENT	NIL		ACCELERATED SHTN
20	MUMTHAJ BEGAM	47	FEMALE	18227	25.3.19	176	1	132	186	44	118	122	II	PRESENT	OLD IWMI		
21	SAVITHRI	55	FEMALE	20498	4.4.19	108	1.1	128	168	44	96	140	II	PRESENT	OLD ASMI		
22	SARINABANU	46	FEMALE	21886	11.4.19	26	1	96	154	48	80	123	NIL	ABSENT	NIL		URI
23	KALAISELVI	53	FEMALE	21948	12.4.19	78	1.1	130	174	36	112	134	II	PRESENT	NIL		ACUTEGASTROENTRITIS
24	MANGALAM	60	FEMALE	22718	15.4.19	286	1.2	124	186	38	117	154	III	PRESENT	NIL	LT THALAMIC HAEMORRAGE	
25	THILAGAVATHY	40	FEMALE	24299	22.4.19	18	0.9	126	159	45	88	132	I	ABSENT	NIL	LT CG INFARCT	
26	KAVERI	44	FEMALE	25112	26.4.19	16	1	112	172	48	97	136	NIL	ABSENT	NIL		APD
27	PATTU	55	FEMALE	27519	6.5.19	24	0.8	108	182	32	110	202	NIL	ABSENT	OLD ASMI		
28	MANONMANI	45	FEMALE	27597	6.5.19	58	1.1	134	192	36	124	162	II	ABSENT	NIL	LT MCA INFARCT	
29	MALARKODI	64	FEMALE	28235	9.5.19	84	1.1	122	178	34	121	90	I	PRESENT	NIL	RT MCA INFARCT	
30	VALLI	45	FEMALE	29270	13.5.19	88	1	128	168	42	98	140	III	PRESENT	NIL		EPISTAXIS
31	SAROJA	65	FEMALE	29911	16.5.19	12	1	132	202	40	135	136	I	ABSENT	OLD IWMI		
32	LAKSHMI	60	FEMALE	30912	20.5.19	26	0.9	122	190	38	117	174	NIL	ABSENT	OLD ASMI		
33	MUTHU	45	FEMALE	33251	31.5.19	14	1	98	146	46	72	140	NIL	ABSENT	NIL		ACUTE GASTRITIS
34	MEENA	43	FEMALE	34135	3.6.19	100	0.9	120	168	40	98	146	I	PRESENT	NIL	RT CG HAEMORRAGE WITH IVH	
35	GANESAN	60	MALE	34725	6.6.19	16	1	118	140	42	70	132	NIL	ABSENT	NIL		MIGRAINE
36	VISHWANATHAN	55	MALE	34810	7.6.19	246	1.2	134	190	36	126	142	III	PRESENT	OLD AWMI		
37	KANNAGI	44	FEMALE	34837	7.6.19	22	1	112	148	42	78	138	NIL	ABSENT	NIL		GIDDINESS
38	SURESH	32	MALE	36438	13.6.19	68	1.1	107	180	40	110	152	II	PRESENT	NIL	LT CG HAEMORRAGE	

39	MOHANDOSS	60	MALE	37318	17.6.19	11	1	130	158	38	96	120	NIL	ABSENT	NIL	LT CG HAEMORRAGE	
40	CHANDRASEKAR	62	MALE	37432	18.6.19	6.5	1.1	138	146	40	76	152	I	ABSENT	NIL		
41	INDRA	62	FEMALE	37421	18.6.19	64	1.1	102	145	46	74	127	II	ABSENT	NIL		GIDDINESS
42	THENDRAL	40	FEMALE	37401	18.6.19	11	0.8	130	142	52	63	136	NIL	ABSENT	NIL		CENTIPEDE BITE
43	SENTHIL KUMAR	28	MALE	37710	18.6.19	10	0.7	102	132	48	60	128	NIL	ABSENT	NIL		APD
44	SANKAR	49	MALE	37853	19.6.19	7.5	1	132	153	46	79	140	NIL	ABSENT	NIL		GIDDINESS
45	KASIYA	52	MALE	37886	19.6.19	270	1.1	124	164	40	93	158	1V	PRESENT	TVD		
46	PITCHAIYAMMAL	40	FEMALE	38029	20.6.19	282	1.2	116	146	44	75	134	IV	PRESENT	NIL		ACCELERATED SHTN
47	SENTHIL KUMAR	37	MALE	38113	20.6.19	78	1.1	132	166	38	98	149	II	PRESENT	OLD ASMI		
48	PANEER	58	MALE	38085	20.6.19	134	0.9	108	162	47	87	141	II	PRESENT	NIL	LT CG HAEMORRAGE	
49	PAPPA	60	FEMALE	38986	24.6.19	24	1	126	154	48	72	163	I	PRESENT	NIL		ACUTE GASTRITIS
50	JAYANTHI	45	FEMALE	39039	25.6.19	96	0.9	98	184	36	117	154	II	PRESENT	NIL		HYPOTHYROIDISM
51	RANJITH	40	MALE	39902	28.6.19	82	0.8	118	168	44	96	140	II	PRESENT	NIL	LT CG HAEMORRAGE	
52	THILAGAVATHI	48	FEMALE	40648	1.7.19	21	0.9	106	142	48	62	160	NIL	PRESENT	OLD IWMI		
53	REMINA BEEVI	55	FEMALE	40798	1.7.19	10	1	98	128	46	55	136	NIL	ABSENT	NIL		LRI
54	PARVATHI	55	FEMALE	40756	1.7.19	92	1.1	130	162	42	96	120	I	PRESENT	NIL	LT CG HAEMORRAGE	
55	MAYAMBAL	60	FEMALE	42384	11.7.19	11.7	1	128	148	40	80	140	I	ABSENT	NIL		GIDDINESS
56	RAJU	52	MALE	42394	11.7.19	61.5	0.8	106	126	46	52	138	I	ABSENT	NIL		UNKNOWM BITE
57	SHIEK ABDUL	41	MALE	42390	11.7.19	197	1.1	98	172	43	98	156	I	PRESENT	NIL		ACCELERATED SHTN
58	BALAKRISHNAN	45	MALE	43072	11.7.19	34	0.8	122	206	38	140	138	II	PRESENT	TVD		
59	KALLIYAPPAN	53	FEMALE	43082	11.7.19	80	1	112	168	42	99	136	II	PRESENT	NIL	LT THALAMIC INFARCT	
60	SELVARAJ	45	MALE	43184	12.7.19	284	1.1	132	174	38	103	164	III	PRESENT	NIL	LT THALAMIC HAEMORRAGE	
61	PUSHPAM	64	FEMALE	43690	14.7.19	286	1.2	94	160	42	90	148	III	PRESENT	NIL	LT MCA INFARCT	
62	PUSHPAVALLI	50	FEMALE	46432	25.7.19	144	1.1	134	174	38	109	136	I	PRESENT	OLD IWMI		
63	GOVINDHAMMAL	45	FEMALE	46485	25.7.19	18	0.9	120	134	46	60	140	NIL	ABSENT	NIL		APD
64	DHARMARAJ	50	MALE	46487	25.7.19	84	1	88	158	42	85	154	II	PRESENT	NIL		EPISTAXIS
65	SENTHAMARAI	44	FEMALE	47411	29.7.19	8	0.8	124	136	50	66	100	NIL	ABSENT	NIL		GIDDINESS
66	SEDHU	57	FEMALE	48018	1.8.19	86	1	130	154	46	83	124	II	PRESENT	ACUTE AWMI		
67	CHELLADURAI	40	MALE	48058	1.8.19	116	0.9	124	148	42	81	148	I	PRESENT	ACUTE IWMI		
68	NOORJAHAN BEEVI	64	FEMALE	48702	4.8.19	38	0.7	132	160	46	87	136	I	ABSENT	NIL		COPD
69	MARIYAMMAL	62	FEMALE	48932	5.8.19	96	0.9	94	152	42	82	142	II	PRESENT	NIL	RT MCA INFARCT	
70	GANESAN	61	MALE	48998	5.8.19	54	0.8	136	148	48	63	186	I	PRESENT	ACUTE IWMI		
71	MURUGANANTHAM	49	MALE	52914	22.8.19	34	1	130	160	38	96	130	I	PRESENT	NIL		
72	GUNASEKARAN	60	MALE	48800	5.8.19	22	0.9	132	128	46	57	122	NIL	ABSENT	NIL		SNAKE BITE
73	VETRISELVAN	53	MALE	49093	8.8.19	54	1.1	104	154	44	85	126	I	PRESENT	NIL		
74	MEENATCHISUNDARAM	52	MALE	49710	9.8.19	34	0.8	108	176	45	104	134	I	PRESENT	NIL		
75	JAGADEESAN	45	MALE	56806	13.8.19	37	1	116	198	42	104	160	I	PRESENT	ACUTE IWMI		
76	MURUGANANTHAM	42	MALE	50997	14.8.19	55	0.7	130	189	40	114	172	II	PRESENT	ACUTE AWMI		
77	PADMAVATHY	59	FEMALE	50998	14.8.19	63	1	110	164	40	97	138	I	PRESENT	NIL		BPPV
78	PARVATHY	59	FEMALE	50934	14.8.19	38	0.8	130	142	46	67	138	I	ABSENT	NIL		LRI
79	SELVARAJ	50	MALE	51151	15.8.19	31	1.2	126	158	40	90	140	NIL	ABSENT	NIL		AFI
80	SAKTHIVEL	37	MALE	57186	15.8.19	26	0.8	126	144	50	69	125	NIL	ABSENT	NIL		HORNET STING

81	PATHMAVATHY	60	FEMALE	51244	15.8.19	26	0.6	94	124	48	55	106	NIL	ABSENT	NIL		UNKNOWN BITE
82	UMAVATHY	58	FEMALE	51271	15.8.19	24	0.9	114	142	42	75	126	NIL	ABSENT	NIL		AFI
83	RENGARAJ	63	MALE	51267	15.8.19	276	1.1	100	176	44	104	140	III	PRESENT	NIL	LT CG HAEMORRAGE	
84	DEVENDRAN	30	MALE	51312	16.8.19	28	0.9	126	140	48	67	127	I	ABSENT	NIL		ACUTE GASTRITIS
85	THIYAGARAJAN	49	MALE	51433	16.8.19	1.6	1	122	160	39	98	114	NIL	ABSENT	NIL		
86	SAHAYAMARY	54	FEMALE	51448	16.8.19	224	1	106	178	36	112	147	III	PRESENT	NIL	RT MCA INFARCT	
87	POYHIYAPPAN	58	MALE	51290	16.8.19	247	1.1	94	164	40	90	172	III	PRESENT	NIL	LT MASSIVE ICH	
88	MURUGANANTHAM	38	MALE	52914	22.8.19	18	0.9	88	130	46	63	104	NIL	ABSENT	NIL		APD
89	BALAMURUGAN	48	MALE	53475	25.8.19	21	1	104	132	46	61	126	NIL	ABSENT	NIL		UNKNWON BITE
90	MARIAYYA	63	MALE	53508	26.8.19	18	1	98	126	50	56	122	NIL	ABSENT	NIL		BRONCHIAL ASTHMA
91	SELVARAJ	40	MALE	53551	26.8.19	86	1.1	128	128	45	54	148	I	ABSENT	NIL		ACUTE GASTROENTERITIS
92	KALAIYARASI	56	FEMALE	54736	31.8.19	4	0.8	75	132	42	68	109	I	PRESENT	NIL		GIDDINESS
93	RAMALINGAM	63	MALE	54968	1.9.19	114	1	112	168	41	99	140	II	PRESENT	NIL	MULTI INFARCT	
94	JAYA	60	FEMALE	55150	2.9.19	24	1	106	134	44	64	132	NIL	ABSENT	NIL		SNAKE BITE
95	SAROJA	64	FEMALE	55235	2.9.19	54	1	104	146	48	75	118	III	PRESENT	NIL	RT CG INFARCT	
96	RAJAMMAL	65	FEMALE	55187	2.9.19	295	1	96	158	41	95	112	II	PRESENT	NIL	MULTI INFARCT	
97	ACHIPONNU	58	FEMALE	55259	2.9.19	109	0.9	99	154	46	81	138	I	PRESENT	NIL		CHEST PAIN
98	ALAGARSAMY	55	MALE	55476	3.9.19	58	1	68	188	40	118	148	I	PRESENT	OLD ASMI		
99	SOUNDARAVALLI	65	FEMALE	55463	3.9.19	68	0.9	130	148	42	81	132	II	PRESENT	OLD ASMI		
100	MARIYAMMAL	48	FEMALE	55482	3.9.19	24	0.9	136	140	40	74	128	NIL	ABSENT	NIL		APD