# A STUDY ON PREVALENCE OF LIPID AND GLYCAEMIC ABNORMALITIES IN PATIENTS WITH ESSENTIAL HYPERTENSION


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# CERTIFICATE

This is to certify that this dissertation entitled " **A STUDY ON THE PREVALENCE OF LIPID AND GLYCAEMIC ABNORMALITIES IN PATIENTS WITH ESSENTIAL HYPERTENSION**" is a bonafide record of work done by Dr.P.Meenakumari, during the period of her post graduate study from 2006 to 2009 under our direct guidance and supervision in the Department of Medicine, Tirunelveli Medical College Hospital, in partial fulfillment required for the award of M.D. Degree Branch I – General Medicine, submitted to the faculty of Medicine, Tamil Nadu Dr.M.G.R. Medical University.

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# AIMS AND OBJECTIVE

**1.** To determine the prevalence of undiagnosed type 2 diabetes mellitus and impaired glucose tolerance in newly diagnosed patients with uncomplicated essential hypertension.

**2.** To determine the prevalence of abnormal lipid profile in patients with uncomplicated essential hypertension in relation to age and sex.

**3.** To determine the prevalence of metabolic syndrome in patients with essential hypertension.

# INTRODUCTION

Hypertension is an emerging health problem in India. It is a cardinal risk factor for coronary artery disease (CAD) and the risk of CAD further increases in presence of dyslipidemia. Both hypertension and dyslipidemia coexist more often than by chance alone.[1]

The Diabetes Atlas published by the International Diabetes Federation shows that India currently leads the world in number of people with diabetes and is currently home to over 40 million diabetics and is predicted to increase to 69 million by 2025. Further India occupies the second position with number of subjects with Impaired Glucose Tolerance (IGT).[2]

Insulin resistance and hyperinsulinemia are considered as pathogenetic factor in both hypertension and dyslipidemia, both by themselves are risk factors for Coronary Artery Disease.

The co-existence of hypertension dyslipidemia and diabetes mellitus substantially increases the risk of macro

vascular complications, including stroke, coronary disease and is responsible for increased cardio vascular mortality.

Thus, it becomes essential to screen the patients with essential hypertension for lipid abnormalities and impaired glucose tolerance.

# REVIEW OF LITERATURE

## HYPERTENSION:

### Definition:[3]

Clinically, hypertension might be defined as Systolic blood pressure ≥ 140 mm Hg and diastolic blood pressure ≥ 90mm Hg based on average of two or more readings. A single reading is sufficient if systolic BP is ≥ 210 mm Hg or diastolic BP ≥ 120 mmHg.

### JNC VII classification [3]

**Blood pressure classification for aduts aged 18 years and older**

| | Systolic BP( mm Hg) | Diastolic BP (mmHg) |
|---|---|---|
| Normal | < 120 | and < 80 |
| Pre hypertension | 120-139 | or 80-89 |
| Stage 1 hypertension | 140-159 | or 90-99 |
| Stage 2 hypertension | ≥ 160 | or ≥ 100 |

* Isolated systolic hypertension is defined as systolic BP ≥ 140 mmHg and diastolic BP ≥ 90 mmHg and staged appropriately.

## FACTORS AFFECTING BLOOD PRESSURE

### Smoking:

Smoking causes an increase in both Systolic BP and Diastolic BP lasting for about 30 minutes.

### Caffeine:

It causes an increase in BP by increasing both renin and catecholamines levels.

### Alcohol:

Modest consumption of alcohol (< 30 g of ethanol a day) is generally not associated with increase in BP. Larger amounts of alcohol ingestion have a dose related effect on BP both in hypertensives and normotensive subjects.[4]

### Genetic determinants:

Concordance of blood pressures is higher in families than in unrelated individuals , higher between monozygoyic than dizygotic twins.

### Salt intake:

Excess sodium intake increases BP by increasing fluid volume and preload, thereby increasing cardiac

output.Chloride and not just sodium may be involved in hypertension. BP rises more with NaCl (sodium chloride salt) than with nonchloride salts of sodium.[5]

**PSEUDO HYPERTENSION:**

In some elderly patients with very rigid calcified arteries the bladder of the BP apparatus may not be able to collapse the brachial artery giving rise to falsely high reading or pseudo hypertension (Osler's sign)

**WHITE COAT HYPERTENSION: [6]**

Approximately 15-20% of patients with stage I hypertension based on office blood pressures have average ambulatory readings < 135/85 mm Hg. This phenomenon called " White coat hypertension" may also be associated with an increased risk of target organ damage. These individuals are also at increased risk for developing sustained hypertension.

**AMBULATORY BP MONITORING: [6, 8]**

Home blood pressure and average 24 hour ambulating blood pressure measurements are generally lower than clinic blood pressures. Because ambulatory blood pressure recordings yield multiple readings throughout the day and night, they provide a more comprehensive assessment of the vascular burden of hypertension than a limited number of office readings. They also more reliably predict target organ damage than office blood pressures.

**Technical Advantages of ABPM vs Clinic BP measurement**

Twenty-four hours average BP values are more reproducible than isolated office recordings.

- ABPM does not prevent the occurrence of physiological nocturnal BP variations.
- ABPM does not trigger any alerting reaction and pressure rise in the patients; and therefore avoids the " white coat effect ".
- ABPM is also largely unaffected by any placebo effect.

**Indications for ABPM**

The following are the situations in which ABPM may be useful:

- ❖ Office or white coat hypertension
- ❖ Evaluation of drug resistance / drug trials for effective control of BP
- ❖ Evaluation of nocturnal BP changes
- ❖ Episodic hypertension
- ❖ Hypertensive symptoms associated with antihypertensive medication or automatic dysfunction
- ❖ Carotid sinus syncope and pacemaker syndromes
- ❖ Hypertension in pregnancy
- ❖ Hypertension in children and adolescents.

**Day Vs Night BP: [8]**

Night time blood pressures are generally 10 - 20% lower than day time blood pressure. Investigators have defined hypertensive patients as 'dippers' or 'non dippers' based on the reduction of night time BP, respectively greater or smaller than 10% as compared to day time values. An

attenuated night time blood pressure "dip" is associated with increased cardiovascular disease risk.

Blunting of day night blood pressure pattern occurs in several conditions like sleep apnea, autonomic neuropathy and in certain population including African Americans diabetes, obesity and renal insufficiency.[7,8]

Recommended criteria for a diagnosis of hypertension by this method are awake blood pressure ≥ 135/85 mm Hg and asleep blood pressure ≥ 120/75 mm Hg. These levels approximate a clinic blood pressure of 140/90 mm Hg [6]

**HYPERTENSIVE CRISIS:**

Includes hypertensive urgencies and emergencies.

**Hypertensive urgencies** are defined as a substantial increase in BP usually with a diastolic BP of 120-130 mmHg (including upper levels of stage 2 hypertension , optic disc edema , progressive end organ complications rather than damage and severe perioperative hypertension ) warranting BP reduction within hours.[3]

**Hypertensive Emergencies** –include **accelerated hypertension**- defined as systolic BP > 210 and diastolic BP > 130 mmHg presenting with headache, blurred vision or focal neurological deficits, and **malignant hypertension** which requires the presence of papilledema.[9]

**CAUSES OF HYPERTENSION :[6]**

**ESSENTIAL HYPERTENSION:**

80 – 95% of hypertensive patients are diagnosed as having essential hypertension also referred to as primary or idiopathic hypertension. In the remaining 5 – 20 % a specific underlying disorder causing elevation of BP is identified.

**SECONDARY HYPERTENSION**

In individuals with secondary hypertension ,a specific mechanism for the blood pressure elevation is often apparent.

**Systolic hypertension with wide pulse pressure:**

1. Decreased vascular compliance (arteriosclerosis)
2. Increased cardiac output.
    (a) Aortic regurgitation

(b) Thyrotoxicosis

(c) Hyperkinetic heart syndrome

(d) Arteriovenous fistula

(e) Patent ductus arteriosus

**Systolic and diastolic hypertension**

1. Renal

A. Parenchymal renal disease

B. Polycystic kidney disease

C. Renin producing tumors

D. Obstructive uropathy

2. Renovascular : Arterio sclerotic

Fibromuscular dysplasia

3.Adrenal :

Primary aldosteronism

Cushing's syndrome

17 α hydroxylase deficiency

11 β hydroxylase deficiency

11 hydroxy steroid dehydrogenase deficiency

Pheochromocytoma.

4. Coarctation of aorta

5. Obstructive sleep apnea

6. Pre eclampsia / eclampsia

7. Neurogenic :

Psychogenic

Diencephalic syndrome

Familial dysautonomia

Polyneuritic (acute porphyria, lead poisoning)

Increased intracranial pressure

Acute spinal cord section

8. Endocrine – Hypothyroidism

Hyperthyroidism

Hypercalcemia

Acromegaly.

9. Medications -

High dose estrogens

Adrenal steroids

Decongestants

Appetite suppressants

Cyclosporine

Tricyclic antidepressants

Monoamine oxidase inhibitors

Erythropoietin

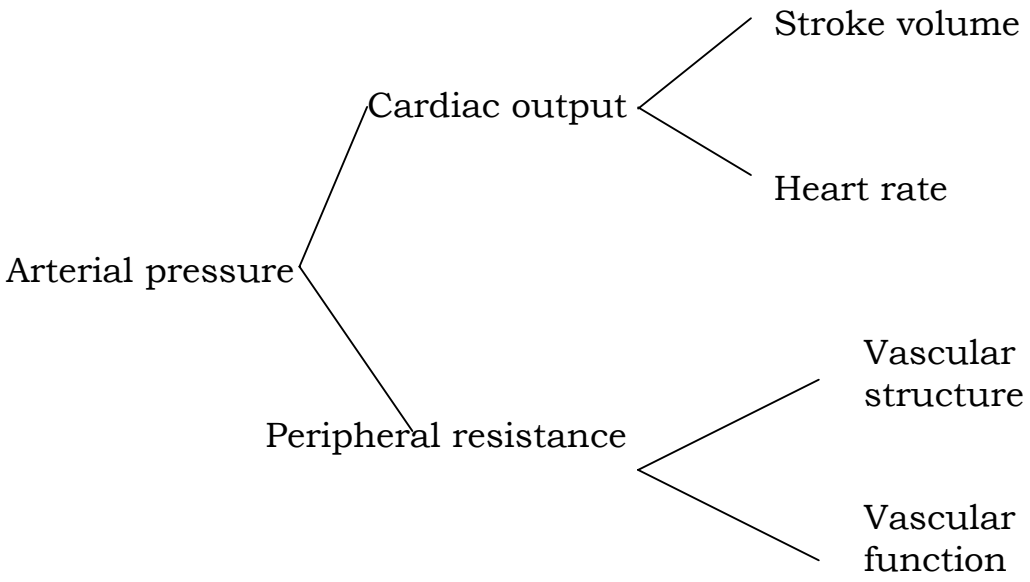
Cocaine

Non steroidal anti – inflammatory agents

10. Mendelian forms of hypertension

## MECHANISMS OF HYPERTENSION[6]

### DETERMINANTS OF ARTERIAL PRESSURE




**1. Intravascular volume**

Blood flow = Pressure across the vascular bed

Vascular resistance

When NaCl intake exceeds the capacity of the kidneys to excrete sodium, vascular volume initially expands and

cardiac output increases. However overtime, peripheral resistance increases and cardiac output reverts to normal.

As arterial pressure increases in response to high NaCl intake, urinary sodium excretion increases and sodium balance is maintained at the expense of an increase of an increase in arterial pressure, a phenomenon called " pressure – natriuresis ".

**2. Autonomic nervous system**

Norepinephrine , epinephrine and dopamine are the three endogenous catecholamines acting through α and β receptors . α receptors are more activated by norepinephrine(NE) than by epinephrine and the reverse is true for β receptors . $\alpha_1$ receptors located on post synaptic cells in smooth muscle elicit vasoconstriction .$\alpha_2$ receptors localized on presynaptic membranes of post ganglionic nerve terminals that synthesize norepinephrine act as negative feed back controllers inhibiting further NE release.

$\beta_1$ receptor activation stimulates cardiac contraction and increases cardiac output and stimulates renin release

from kidneys. $\beta_2$ receptor activation by epinephrine release relaxes the vascular smooth muscle cells resulting in vasodilatation.

Arterial baro reflex modulate blood pressure; mediated by stretch sensitive sensory nerve endings located in the carotid sinuses and aortic arch. As BP increases , baroreceptors are stimulated which decreases the sympathetic outflow resulting in decrease of arterial pressure and heart rate.

**3. Renin Angiotensin Aldosterone system**

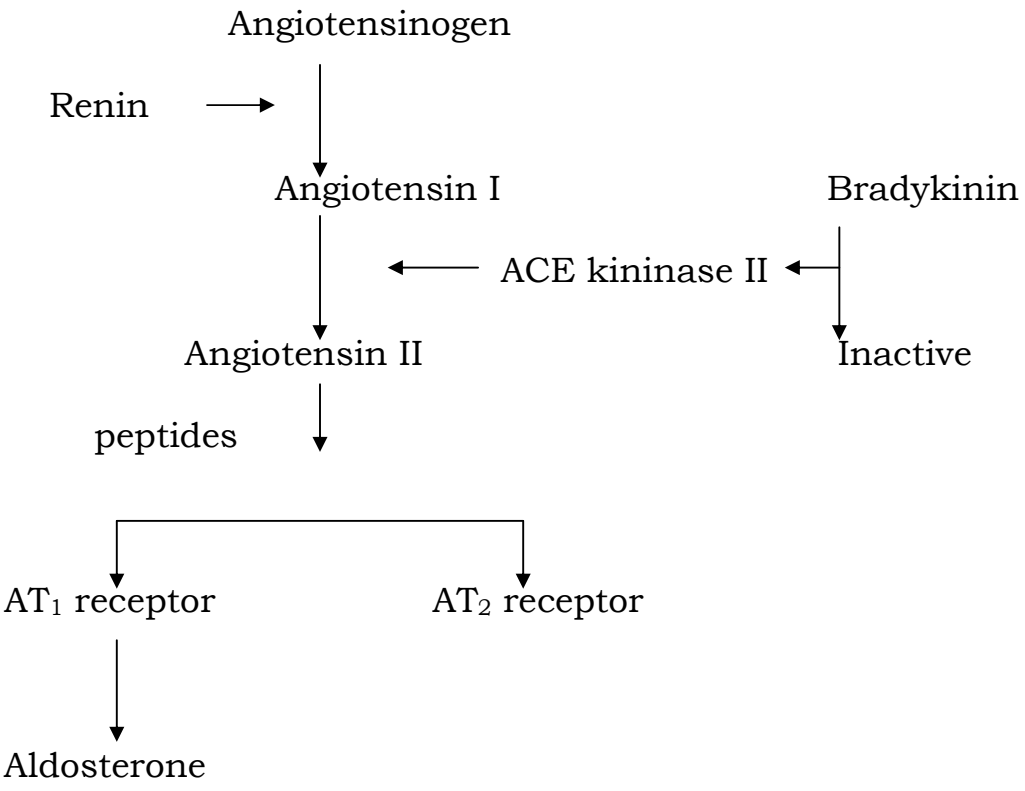
Renin is synthesized by the juxtaglomerular cells. There are three primary stimuli for renin secretion

(1) Decreased NaCl transport in the thick ascending limb of loop of henle (Macula densa mechanism).

(2) Decreased pressure or stretch within the renal afferent arteriole (baroreceptor mechanism)

(3) Sympathetic nervous system stimulation of renin – secreting cells via $\beta_1$ adreno receptors.

**Renin Angiotensin – Aldosterone system**




Once released into the circulation active renin cleaves angiotensinogen to form an inactive decapeptide, angiotensin I. A converting enzyme, located primarily but not exclusively in the pulmonary circulation converts angiotensin I to the active octapeptide, angiotensin II. The same enzyme cleaves a number of other peptides including bradykinin and thereby inactivating them.

Acting primarily through angiotensin II type I ($AT_1$) receptor located on cell membranes, angiotensin II is a potent pressor substance, the primary trophic factor for the secretion of aldosterone by the adrenal zona glomerulosa, and a potent mitogen stimulating vascular smooth muscle and myocyte growth.

An angiotensin II type 2 ($AT_2$) receptor widely distributed in the kidneys has opposite effects of $AT_1$ receptor and induces vasodilation, sodium excretion and inhibition of cell growth and matrix formation.

The renin-angiotensin aldosterone system contributes to arterial pressure primarily via the vasoconstrictor properties of angiotensin II and the sodium retaining properties of aldosterone.

**4. Vascular mechanisms**

Resistance to flow varies inversely with the fourth power of radius. In hypertensive patients,mechanical,

structural and functional changes decrease the lumen diameter of small arteries and arterioles and the peripheral vascular resistance increases.

Activity of $Na^+$ - $H^+$ exchanger is increased in hypertension and this may result in increased vascular tone by two mechanisms- first, increased sodium entry may lead to increased vascular tone by activating $Na^+$ - $Ca^{2+}$ exchange and thereby increasing intra cellular calcium.

Second, increased intracellular pH enhances calcium sensitivity of the contractile apparatus, leading to an increase in contractility for a given intra cellular calcium concentration. Additionally, increased $Na^+$ - $H^+$ exchange might stimulate vascular smooth-muscle cell growth by enhancing sensitivity to mitogens.

Vascular endothelial function also modulates vascular tone. The vascular endothelium synthesizes and releases vasoactive substance including Nitric oxide, a potent

vasodilator. Endothelium dependent vasodilatation is impaired in hypertensive patients.

## REVIEW OF LITERATURE – DIABETES MELLITUS

Diabetes mellitus refers to a group of common metabolic disorders that share the phenotype of hyperglycemia. The factors contributing to hyperglycemia include reduced insulin secretion, decreased glucose utilization and increased glucose production.

### Criteria for diagnosis of Diabetes:[6]

- Symptoms of diabetes plus random blood glucose ≥ 200 mg/dL
- Fasting plasma glucose ≥ 126 mg/dL
- Two hour plasma glucose ≥ 200 mg /d L during an oral glucose tolerance test.

Glucose tolerance is classified into three categories based on FPG [10]

1. FPG < 100 mg/dl – Normal.

2. FPG :100 – 125 mg /dl – Impaired Fasting Glucose

3. FPG > 126 mg/dl– Diabetes

**Based on OGTT ,**

IGT is defined as plasma glucose levels between 140 – 199 mg / dl and diabetes is defined as glucose > 200 mg/dl 2h after 75g oral glucose load.[10]

| | **Normal** | **Prediabetes (IFG/IGT)** | **Diabetes** |
|---|---|---|---|
| FPG (mg/d L) | < 100 | 100 - 125 | > 126 |
| 2h PG(mg/d L) | < 140 | 140 - 199 | > 200 |

**Risk factors for type 2 diabetes mellitus [6]**

1. Family history of diabetes

2. Obesity-BM1 > 25 kg/$m^2$

3. Habitual physical inactivity
4. Race / ethnicity (African Americans, Asian Americans, Pacific Islanders)
5. Previously identified IFG or IGT
6. History of GDM or delivery of baby > 4Kg
7. **Hypertension (blood pressure ≥ 140/90mm Hg)**
8. HDL – C < 35mg /dl and / or Triglyceride > 250mg/dl
9. Polycystic ovary syndrome or acanthosis nigricans
10. History of vascular disease.

Note: IFG – impaired fasting glucose

IGT – impaired glucose tolerance

GDM – Gestational diabetes mellitus.

Hypertension can accelerate other complications of Diabetes, particularly cardiovascular disease and nephropathy. The target goal of BP is < 130/80 mm Hg in diabetic individuals without proteinuria and < 125/75 mm Hg in for individuals with micro albuminuria or macro albuminuria.

**Dyslipidemia**

Dyslipidemia is an important risk factor for coronary artery disease.The NCEP : ATP III (National Cholesterol Education Program : Adult Treatement Panel had published guidelines for the diagnosis and evaluation of high blood cholesterol in adults [11]

The normal lipid profile is defined as :

LDL –cholesterol <130mg%, total cholesterol <200mg/dl , Triglycerides <150mg/dl .Desirable HDL – C: > 40 mg/dl (Males) and > 50 mg/dl (Females).

The most common pattern of dyslipidemia encountered in diabetes is hypertriglyceridemia and reduced HDL cholesterol levels. Diabetes itself does not increase levels of LDL, but the small dense LDL particles found in type 2 DM are more atherogenic because they are more easily glycated and susceptible to oxidation.

## THE METABOLIC SYNDROME[11,12, 13]

It is a constellation of factors including abdominal obesity, insulin resistance or diabetes and atherogenic lipid profile. According to the NCEP : ATP III 2001 criteria : [11,12,14]

Patients are considered to have the metabolic syndrome if they have three or more of the following:

1. Abdominal obesity :

   Waist circumference > 102 cm in men

   > 88 cm in women

2. Triglyceride ≥150 mg / dl.

3. HDL – C < 40 mg / dl in men and < 50 mg / dl in women

4. Blood pressure ≥ 130 / 85 mm Hg

5. Fasting plasma glucose ≥ 100 mg / dl.

## METABOLIC SYNDROME AND HYPERTENSION

Hypertension is seen in 30 - 50% patients with type 2 DM and 20 – 40% of individuals with IGT: conversely,

about 50% of hypertensives have impaired insulin sensitivity.[13]

Hypertension is one of the defining characteristics of the metabolic syndrome. The etiology of hypertension in the metabolic syndrome is multifactorial – genetic factors, hyperinsulinemia and obesity.

The risk factors for developing metabolic syndrome include : obesity, sedentary life style, ageing, diabetes mellitus, coronary heart disease and lipodystrophy.

**ROLE OF INSULIN IN THE PATHOGENESIS OF ESSENTIAL HYPERTENSION [13]**

1) Insulin physiologically lowers the blood pressure in humans by inducing vasodilatation mediated by increased synthesis of nitric oxide .

Insulin exerts both vasodilator as well as pressor effects on vascular beds. The vasodilator action is mainly in skeletal muscles. Insulin directly stimulated endothelial

nitric oxide synthetase (eNOS) activity and releases NO. Nitric oxide causes vascular smooth muscle relaxation.

Insulin directly stimulates the calcium pump in vascular smooth muscle cells, decreasing the intracellular calcium. In the insulin resistant state, intracellular cytosolic calcium is increased; enhancing vascular smooth muscle response to vasoconstrictors.

2) Normally insulin release after food intake stimulates glucose uptake in the ventromedial thalamus. This is turn increases symphathetic activity and plays an important physiological role in dietary thermoregulation.

3) Insulin also acts in the distal renal tubule causing sodium and water retention. Renal tubular sensitivity to insulin is preserved in hyperinsulinemia. In hyperinsulinemia there is greater increase in sympathetic activity resulting in hypertension through its effects on blood vessels and heart.

4) Insulin may also stimulate proliferation of vascular smooth muscle cells, leading to medial hypertrophy and increased peripheral vascular resistance.

5) Insulin resistance is characterized by impairment of phosphatidyl- 3- kinase signalling pathway causing imbalance between nitric oxide and endothelin – 1 production. Enhanced endothelin activity prevents vasodilation to insulin in insulin resistance[15]. Resistin also impairs insulin induced vasodilation[16].

Thus while insulin normally has a vasodilatory effect, hyperinsulinemia in insulin resistant states produces hypertension.

Several studies prove that decreased sensitivity to insulin is involved in the pathogenesis of essential hypertension. The hyperinsulinemia is caused by beta-cell hypersecretory response to the defective peripheral action of the hormone and by a decreased hepatic insulin clearance.[17.] Essential hypertension is an insulin resistant state and insulin resistance correlates directly with the severity of hypertension.[18]

## PATHOPHYSIOLOGY OF METABOLIC SYNDROME

### Insulin resistance: [12]

An early major contributor to the development of insulin resistance is over abundance of free fatty acids, derived from adipose tissue triglyceride stores released by hormone sensitive lipase.

Fatty acids are also derived through the lipolysis of triglyceride – rich lipoproteins in tissues by lipoprotein lipase. Insulin mediates both antilipolysis and the stimulation of lipoprotein lipase in adipose tissue.

The antilipolytic action in adipose tissue is the most sensitive pathology of insulin action. Thus when insulin resistance develops, increased lipolysis produces more fatty acids, which further decrease the antilipolytic effect of insulin. Fatty acids impair insulin mediated glucose uptake and accumulate as triglycerides in both skeletal and cardiac muscle whereas increased glucose production and triglyceride accumulation are seen in liver.

In the oxidative stress hypothesis studies in insulin resistant subjects with obesity or type 2 DM, a defect is identified in mitochondrial oxidative phosphorylation leading to accumulation of triglycerides in muscle which is associated with insulin resistance.

Adipose tissue produces inflammatory cytokines – C-Reactive protein, interleukin-6 and tumour necrosis factor (TNF-α.). CRP activates complement, decreases NO production and increases plasminogen activator inhibitor(PAI-1) levels.

Adiponectin is a cytokine released by adipose tissue with insulin sensitizing effects. Decreased adiponectin plays important role in metabolic syndrome. Leptin from mature adipocyte binds to hypothalamic receptor and induces satiety. Hypothalamic mediated leptin resistance perpetuates a vicious cycle of hyperinsulinemia, more fat, more leptin.[19]

**CAUSES OF HYPERTENSION IN A PATIENT WITH DIABETES[20]**

- Association
  - Cardio metabolic syndrome
  - Nephropathy
- Hypertension and diabetes due to common endocrine pathology
  - Acromegaly
  - Cushing's syndrome
  - Conn's syndrome
  - Pheochromocytoma
- Drugs inducing both hypertension and diabetes
  - Steroids
  - Oral contraceptive pills
- Diabetogenic anti hypertensive drugs
  - Diuretics- chlorthalidone, thiazides
  - B-blockers
- Chance occurrence of hypertension along with diabetes
  - Essential hypertension
  - Isolated systolic hypertension
  - Primary renal pathology

## EFFECTS OF ANTI HYPERTENSIVE AGENTS ON GLUCOSE METABOLISM[21]

### Angiotensin converting enzyme inhibitors

ACEIs are the initial drug of choice for patient who has both diabetes and hypertension especially if associated with albuminuria. There is increasing data that ACEIs improve insulin sensitivity. A 34% relative risk reduction for developing diabetes was observed in HOPE trial.[22]

### Angiotensin receptor blockers (ARB)

ARBs like ACEIs have shown to reduce the risk of new-onset diabetes.

### Thiazide diuretics[23]

The adverse effects on lipid and carbohydrate metabolism are uncommon with low dose thiazide therapy; however thiazides should be used judiciously in hypertensives with elevated fasting glucose.

**Calcium channel blockers**

CCBs have been shown to reduce insulin resistance or new onset diabetes in people with cardiometabolic syndrome and increase HDL.

**β blockers**

β blockers have adverse effects on glucose and lipid profile and are implicated in new onset diabetes in obese patients. However non selective β blockers like carvidelol reduce cardiovascular mortality and microalbuminuria without adversely affecting the glucose or lipid profiles.

Despite their adverse effects, they have significant favourable effects on cardiovascular disease outcome in hypertensive diabetic patients and are included in therapy.

**α Antagonists:**

Selective α1 blockers like prazosin, terazosin lower LDL cholesterol, raises HDL cholesterol and improves insulin sensitivity.

# MATERIALS AND METHODS

This study was carried out with the aim of studying the prevalence of impaired glucose metabolism and dyslipidemia among newly detected essential hypertensive patients. The study was conducted in the Hypertension clinic, Tirunelveli Medical College Hospital over a period of 6 months from July 2007 to December 2007.

## METHODOLOGY

Newly diagnosed patients with hypertension in the out patient department and in the hypertension clinic are included in this study. The hypertension was diagnosed based on JNC VII criteria, i.e. systolic blood pressure ≥ 140 mm Hg and or diastolic blood pressure ≥ 90 mm of Hg.

The selected individuals underwent general physical examination to rule out any systemic illness.

**Study design** : Cross sectional study

**Patient selection** : Simple random selection

**SELECTION**

**INCLUSION CRITERIA**

1. Patients having blood pressure ≥140 /90 mmHg were selected.
2. Patients with sustained elevation of blood pressure ≥ 140/90 mmHg on 3 separate occasions were selected.
3. Patients of age group ranging from 30 – 70 years were included in the study.

**EXCLUSION CRITERIA:**

1. Patients with secondary hypertension were excluded.
2. Patients who are known diabetes were excluded.
3. Individuals with renal failure, heart disease, stroke, liver disease and other systemic illness were excluded from the study.
4. Patients with paroxysms of hypertension were excluded.

5. Patients with target organ damage like retinopathy, nephropathy, cardiovascular disease were excluded.
6. Patients taking drugs for other illnesses were excluded.

**DOCUMENTATION OF BLOOD PRESSURE:[7]**

The standard sphygmomanometer with prescribed cuff size was used.

1. The same instrument was used for all the patients throughout the study.
2. Calibration of blood pressure was done frequently to ensure the mercury level at zero.
3. Three readings were taken for a single patient to exclude labile hypertension or paroxysmal hypertension.
4. Cuff of proper size was used for the patient.
5. The centre of the cuff should be at heart level and the length & width of the bladder cuff used was 25 x 10 cm.
6. The bladder should encircle atleast 80% of the circumference and cover two- thirds of the arm.

7. Bladder has to be inflated quickly to a pressure 20 mmHg above the systolic BP , recognized by disappearance of radial pulse , to avoid an auscultatory gap.
8. Bladder has to be deflated at 3 mmHg/sec
9. Korotkoff phase I (appearance) and phase V (disappearance) are recorded.
10. Before taking measurement, the individual was seated quietly for 15 min in a quiet room with a comfortable room temperature.
11. Patients were advised to empty bladder, reduce anxiety; to avoid exercise, coffee , tea ,smoking for half an hour preceding the measurement.

**BLOOD INVESTIGATIONS:**

- Fasting blood for lipids and blood sugar was collected.
- Fasting is defined as no caloric intake for at least 8 h.
- Oral glucose tolerance test was done in all the 50 patients selected for the study.

- The test was performed using a glucose load containing the equivalent of 75g anhydrous glucose dissolved in water.
- Two hours later plasma blood glucose was estimated.
- Venous plasma was taken for the estimation.
- Blood glucose was estimated by glucose oxidase method; the value expressed in mg / dl.
- The total cholesterol (TC), Triglyceride (TG) and HDL-C were measured by enzymatic method.
- The LDL-Cholesterol was derived by the Friedwald's formula[24]

$$LDL - C = TC - ( HDL + TG/5)$$

**Waist circumference:**[14]

Is measured at the level of the tip of the right iliac crest and measurement is made at the end of expiration.

# OBSERVATION AND RESULTS

**Table 1**

**Pattern of distribution of patients with respect to the stage of hypertension.**

| **Hypertension** | **No of patients** | **Percent %** |
|---|---|---|
| Stage 1 | 21 | 42% |
| Stage 2 | 29 | 58% |

**Table 2**

**Prevalence of undiagnosed diabetes mellitus and impaired glucose tolerance in patients with newly detected essential hypertension**.

| | **No of patients** | **Percent %** |
|---|---|---|
| $T_2DM$ | 5 | 10% |
| IGT | 16 | 32% |
| NGT | 29 | 58% |

## Table 3

**Prevalence of impaired glucose metabolism (DM /IGT) in newly detected patients with essential hypertension in relation to age.**

| **Age in years** | **No of patients** | **DM** | | **1GT** | | **Normo Glycaemia** | |
|---|---|---|---|---|---|---|---|
| | | **No** | **%** | **No** | **%** | **No** | **%** |
| 31-40 | 8 | - | 0% | 2 | 25% | 6 | 75% |
| 41-50 | 18 | 2 | 11.11% | 5 | 27.78% | 11 | 16.11% |
| 51-60 | 20 | 3 | 15% | 7 | 35% | 10 | 50% |
| 61-70 | 4 | 0 | - | 2 | 50% | 2 | 50% |
| **Total** | **50** | **5** | **10%** | **16** | **32%** | **29** | **58%** |

**Table 4**

**Prevalence of impaired glucose metabolism (DM +1GT) in patients with newly detected essential hypertension in relation to sex.**

| **Sex** | **No. of patient** | **DM** | | **1GT** | | **NGT** | |
|---|---|---|---|---|---|---|---|
| | | **No** | **%** | **No** | **%** | **No** | **%** |
| Males | 30 | 3 | 10% | 9 | 30% | 18 | 60% |
| Females | 20 | 2 | 10% | 7 | 35% | 11 | 55% |

**Table 5**

**Prevalence and Pattern of Dyslipidemia in patients with essential hypertension**

| | **↑ Total cholesterol** | **↑ TGL** | **↓ HDL < 40(men) < 50(women)** | **↑ LDL > 130** |
|---|---|---|---|---|
| No of patients out of 50 | 12 | 16 | 19 | 12 |
| Percent | 24% | 32% | 38% | 24% |

↑-increased

↓-decreased

## Table 6

**Prevalence of dyslipidemia associated with impaired glucose metabolism in patients with essential hypertension**

| | **No.of patients With dyslipidemia** | **Percent (%)** | **Dyslipidemia Associated with IGM/DM** | |
|---|---|---|---|---|
| | | | **No. of patients** | **% out of patients with abnormal lipid value** |
| ↑ TC | 12 | 24% | 7 | 58.8% |
| ↑ TGL | 16 | 32% | 9 | 56.22% |
| ↓ HDL | 19 | 38% | 12 | 63.16% |
| ↑ LDL | 12 | 24% | 7 | 58.3% |

↑-increased

↓-decreased

**Table 7**

**Prevalence of dyslipidemia in patients with essential hypertension in relation to sex**

| | **No of patients** | **Patients with normal lipid profile** | **Percent** | **Patients with dyslipidemia** | **Percent** |
|---|---|---|---|---|---|
| Males | 30 | 16 | 53.33% | 14 | 46.67% |
| Females | 20 | 7 | 35% | 13 | 65% |
| **Total** | **50** | **23** | **46%** | **27** | **54%** |

# DISCUSSION

In this study, out of the total 50 patients 21 were found to be in stage 1 hypertension (42% ) and 29 patients were in stage 2 hypertension (58 %) (Table 1) (Fig 1)

Out of the 50 patients with uncomplicated hypertension, 5 patients (10%) had diabetes, 16 patients (32%) had impaired glucose tolerance, 29 patients (58%) had normal glucose tolerance. 42% of the patients among the hypertensives had impaired glucose metabolism, either diabetes or impaired glucose tolerance , which was previously undiagnosed.(Table 2) (Fig 2)

The study proves that impaired glucose metabolism is common in patients with essential hypertension and impaired glucose tolerance (32% ) is more prevalent than diabetes (10%) .

The results of our study are similar to other studies done in the past. The San Antonio Heart study [25] done in 1993 showed a prevalence of undiagnosed type 2 diabetes mellitus and impaired glucose tolerance to be 8.9% and 25.2% respectively.

Johnson et al [26]found a prevalence of 11.5% and 41% of $T_2$DM and 1GT respectively. In studies in India by Joglekar and Nanivadeker [27] 36% of patients with essential hypertension had impaired glucose metabolism.

In the present study,in patients above 50 years of age 37.5% had impaired glucose metabolism and 12.5% had diabetes. In hypertensive patients less than 50 years ,the values were 26.9% and 7.69% respectively. Thus the prevalence of abnormal glycaemic metabolism is more in patients above 50 years of age (Table 3) (Fig 3)

The prevalence of diabetes was equal (10%) in both males and females and that of impaired glucose tolerance was 30% in males; 35% in female hypertensives (Table 4) (Fig 4)

According to the NCEP / ATP III criteria, the prevalence of dyslipidemia was: high cholesterol – 24%, hypertriglyceridemia 32% ,low HDL 38%, high LDL 24% in this study .(Table 5).The common forms of dyslipidemia noted in our study were low HDL and hypertriglyceridemia .

7 out of 12 patients (58.8%) with abnormal lipid profile had associated impaired glucose metabolism. 9 out of 16 patients (56.22%) with raised TG, 12 out of 19 patients (63.16%) with decreased HDL and 7 out of 12 patients with raised LDL had associated impaired glucose metabolism.

Thus more than 50% of patients with any one form of dyslipidemia had associated abnormal glyaemic metabolism (Table 6)

The prevalence of dyslipidemia in female patients (65%) was more than in males (46.67%).Totally 54% of patients with essential hypertension had some form of dyslipidemia (Table 7) (Fig 5). Previous study by Joglekar and Nanivadekar [27] and Malhotra et al[28] in India had shown similar results.Studies from western countries also show[29,30,32] similar prevalence.

24 out of 50 patients (48%) fulfilled the criteria for metabolic syndrome.The prevalence of metabolic syndrome in this study was 48% and studies in the Western countries [31,32] show similar prevalence.

The prevalence of metabolic syndrome increases with age, as does the incidence of diabetes. Metabolic syndrome is strongly predictive of future diabetes and vascular complications. The high incidence of abnormal glycaemic metabolism in hypertensive patients signals the need to perform an Oral glucose tolerance test in them.[33] Thus we can aim at halting vascular impairment in hypertensives

with type 2 DM and delay the development of type 2 diabetes in patients with glucose intolerance.

Though physical activity is higher among rural population, they have increased prevalence of smoking and the Indian diet is rich in carbohydrate. The urban population consumes high fat diet which increases the LDL-C levels. Other confounding factors for dyslipidemia like smoking and alcohol consumption were not included in the study. The common causes of low HDL-C are carbohydrate rich diet, smoking, sedentary activity.

The proposed mechanism of these lipid abnormalities are likely to be due to hyperinsulinemia. Syndrome X is known to predispose hypertensive individuals to higher risk of coronary artery disease.[34]

# CONCLUSION

1. Prevalence of previously undiagnosed impaired glucose metabolism in patients with essential hypertension is 42%. Hence OGTT should be done for all patients with essential hypertension.
2. Prevalence of type 2 diabetes is 10% and impaired glucose tolerance is 32%.
3. Abnormal glycaemic metabolism is more prevalent in patients older than 50 years of age.
4. Abnormal glycaemic metabolism is more common in females 35% .
5. Prevalence of dyslipidemia in hypertensive patients is 54%.
6. Dyslipidemia is more common in females (65%).
7. Prevalence of metabolic syndrome in the study group is 48%.
8. Impaired glucose metabolism and dyslipidemia are associated with hypertension.

## RECOMMENDATIONS AND PREVENTIVE ASPECTS[35]

1. Smoking cessation is the single –most important lifestyle modification in all patients with hypertension.
2. Patients should be encouraged to adopt Dietary Approaches to stop Hypertension DASH eating plan – diet rich in plant food, moderate amounts of meat, poultry, fish and low total fat and poly and monounsaturated fats. It is rich in potassium and calcium content[35].
3. Salt reduction reduces BP by 5 –7mm Hg when sodium intake is reduced below 100 mmol/day (2.4g of sodium)[36,37]
4. Regular physical activity lowers systolic and diastolic BP. 30-45 minutes of moderate aerobic exercise per day, is recommended for most days of the week. Yoga and meditation also reduces BP.

## Life style interventions for BP reduction

| **Intervention** | **Recommendation** | **Expected systolic BP reduction (range)** |
|---|---|---|
| Weight reduction | Maintain ideal BMS (20-25 kg/$m^2$) for adults | 5-10 mm Hg per 10kg weight loss |
| DASH eating plan | Consume diet atleast five portions a day rich in fruits vegetables, low-fat dairy products with reduced content of saturated and total fat | 8-14 mm Hg |
| Physical activity | Engage in regular aerobic physical activity, for example brisk walking for at least 30 min most days a week | 4-9 mm Hg |
| Alcohol moderation | Men ≤21 U/week<br>Women ≤14 U/week | 2-4mm Hg |

Obesity is the driving force behind metabolic syndrome. Thus weight reduction and physical activity are very important in increasing insulin sensitivity.

Early detection of impaired glucose metabolism and dyslipidemia, and proper institution of life style modifications and pharmacological therapy decrease the morbidity and mortality in patients with essential hypertension.

# SUMMARY

The prevalence of undiagnosed impaired glucose metabolism is 42% among patients with essential hypertension ,and hence oral glucose tolerance test should be done in all patients with essential hypertension.The prevalence of dyslipidemia in hypertensive patients is 54%. Dyslipidemia and metabolic syndrome are common in hypertensive patients.

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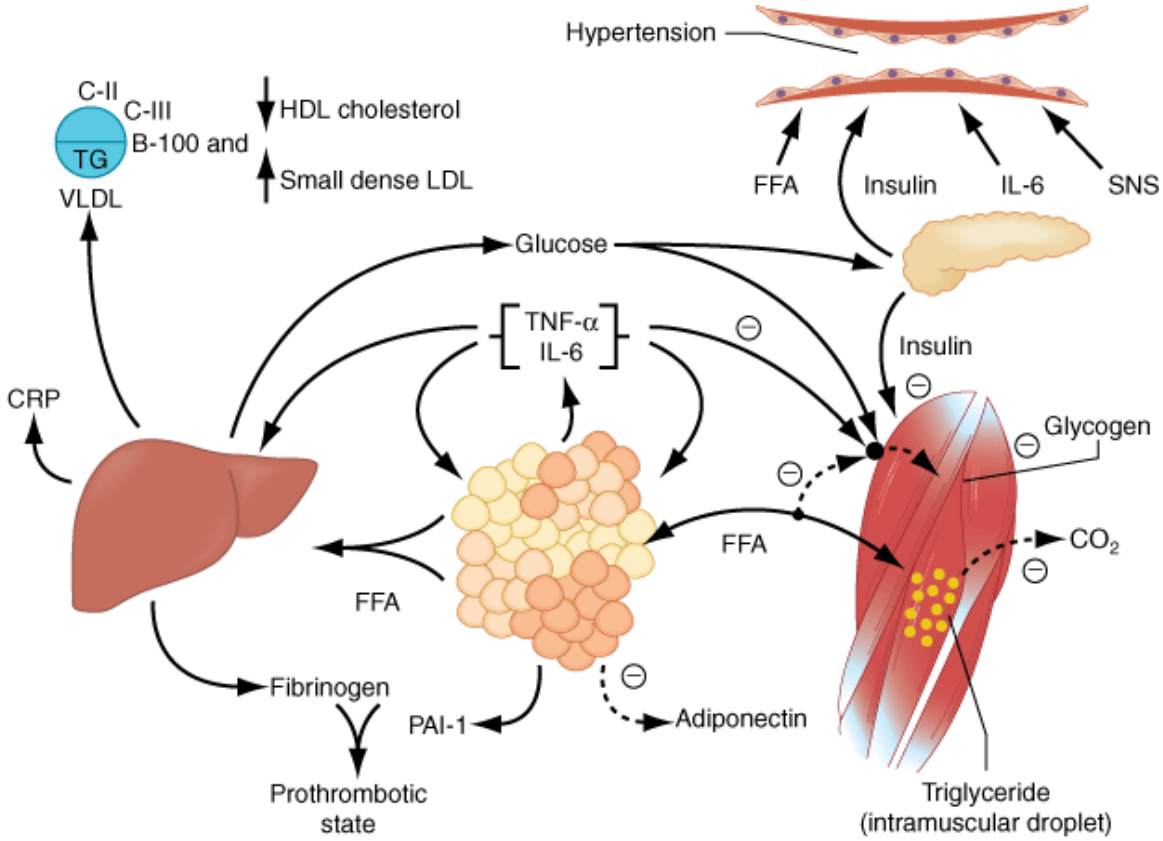
| S.No | Name | Age | Sex | BPmmHg | Waist circumference (CM) | Blood glucose fasting mg% | Glucose 2hr post prandial (mg%) | Lipid profile (mg%) | | | | IGT/DM | Dys lipidemia | Metabolic syndrome |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | TC | TG | HDL | LDL | | | |
| 1 | Subbiah | 36 | M | 160/90 | 93 | 65 | 110 | 125 | 78 | 46 | 94 | - | - | - |
| 2 | Annalakshmi | 56 | F | 150/100 | 90 | 135 | 162 | 195 | 120 | 47 | 124 | IFG | - | + |
| 3 | Arunachalam | 55 | M | 180/100 | 96 | 75 | 160 | 211 | 101 | 55 | 136 | IGT | + | - |
| 4 | Guruvammal | 45 | F | 170/90 | 95 | 98 | 115 | 214 | 176 | 43 | 136 | - | + | + |
| 5 | Muthusamy | 57 | M | 160/100 | 103 | 135 | 210 | 192 | 129 | 36 | 97 | DM | + | + |
| 6 | Mari | 46 | M | 160/96 | 94 | 83 | 132 | 162 | 192 | 40 | 84 | - | + | - |
| 7 | Tamilselvi | 52 | F | 150/90 | 90 | 90 | 134 | 185 | 334 | 37 | 92 | - | + | + |
| 8 | Stephen | 49 | M | 140/90 | 96 | 93 | 101 | 145 | 125 | 48 | 62 | - | - | - |
| 9 | Mymoon | 48 | F | 140/90 | 95 | 128 | 206 | 191 | 243 | 41 | 91 | DM | + | + |
| 10 | Thangaraj | 42 | M | 150/94 | 93 | 93 | 129 | 214 | 196 | 34 | 131 | - | + | + |
| 11 | Karuppasamy | 55 | M | 170/100 | 91 | 76 | 130 | 165 | 130 | 40 | 68 | - | - | - |
| 12 | Vasuki | 45 | F | 146/94 | 95 | 110 | 162 | 177 | 161 | 45 | 100 | IGT | + | + |
| 13 | Rajadurai | 54 | M | 150/94 | 104 | 84 | 114 | 142 | 127 | 51 | 86 | - | - | - |
| 14 | Thanraj | 69 | M | 164/90 | 88 | 90 | 103 | 185 | 144 | 50 | 106 | - | - | - |
| 15 | Mani | 55 | M | 170/90 | 95 | 147 | 188 | 222 | 158 | 42 | 148 | DM | + | + |
| 16 | Kunaseelan | 47 | M | 146/96 | 103 | 82 | 124 | 163 | 128 | 43 | 94 | - | - | - |
| 17 | Vaiyali | 48 | M | 150/90 | 104 | 129 | 105 | 161 | 113 | 45 | 103 | IGT | - | + |
| 18 | Mariappan | 49 | M | 146/90 | 89 | 209 | 310 | 241 | 187 | 38 | 166 | DM | + | + |
| 19 | Malayandi | 58 | M | 190/120 | 92 | 96 | 162 | 186 | 132 | 42 | 118 | IGT | - | - |
| 20 | Thilagavathi | 55 | F | 150/80 | 89 | 75 | 103 | 151 | 111 | 50 | 79 | - | - | - |
| 21 | Kamala | 52 | F | 190/110 | 93 | 160 | 192 | 229 | 162 | 44 | 153 | IGT | + | + |
| 22 | Thangam | 56 | F | 150/90 | 91 | 142 | 204 | 186 | 182 | 53 | 98 | DM | + | + |
| 23 | Saraswathi | 48 | F | 180/90 | 92 | 84 | 214 | 172 | 126 | 51 | 96 | IGT | - | + |
| 24 | Bakkirmydeen | 55 | M | 168/92 | 98 | 96 | 137 | 157 | 144 | 40 | 88 | - | - | - |
| 25 | Velpandi | 45 | M | 150/96 | 91 | 81 | 122 | 142 | 126 | 48 | 69 | - | - | - |

| S.No | Name | Age | Sex | BPmmHg | Waist circumference (CM) | Blood glucose fasting mg% | Glucose 2hr post prandial (mg%) | Lipid profile (mg%) | | | | IGT/DM | Dys lipidemia | Metabolic syndrome |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | TC | TG | HDL | LDL | | | |
| 26 | Tamilselvi | 52 | F | 150/96 | 92 | 134 | 190 | 192 | 134 | 50 | 115 | IGT | - | + |
| 27 | Jeyalakshmi | 57 | F | 170/100 | 90 | 95 | 197 | 225 | 232 | 37 | 142 | IGT | + | - |
| 28 | Madhiyarasi | 46 | F | 160/90 | 91 | 72 | 109 | 186 | 149 | 50 | 106 | - | - | - |
| 29 | John | 51 | M | 146/96 | 89 | 84 | 136 | 152 | 120 | 35 | 93 | - | + | - |
| 30 | Murugan | 37 | M | 160/92 | 93 | 95 | 166 | 245 | 146 | 38 | 178 | IGT | + | + |
| 31 | Mangalam | 43 | F | 150/100 | 89 | 78 | 102 | 136 | 112 | 42 | 72 | - | - | + |
| 32 | Subbu | 47 | F | 170/96 | 91 | 84 | 121 | 162 | 146 | 51 | 82 | - | - | - |
| 33 | Kathiresan | 36 | M | 144/90 | 98 | 120 | 169 | 140 | 121 | 48 | 68 | IGT | - | - |
| 34 | Janaki | 47 | F | 146/90 | 96 | 121 | 205 | 190 | 238 | 41 | 94 | IGT | + | + |
| 35 | Muniraj | 38 | M | 150/90 | 86 | 90 | 126 | 140 | 123 | 40 | 75 | - | - | - |
| 36 | Megalai | 41 | F | 166/92 | 93 | 98 | 132 | 156 | 142 | 52 | 76 | - | - | - |
| 37 | Daniel | 53 | M | 176/92 | 97 | 132 | 186 | 235 | 210 | 34 | 159 | IGT | + | + |
| 38 | Nagarajan | 34 | M | 170/90 | 95 | 77 | 136 | 149 | 127 | 42 | 82 | - | - | - |
| 39 | Muniyandi | 61 | M | 180/100 | 96 | 118 | 179 | 184 | 146 | 35 | 120 | IGT | - | + |
| 40 | Subramani | 51 | M | 156/90 | 90 | 89 | 123 | 191 | 245 | 43 | 99 | - | + | - |
| 41 | Chellapandi | 39 | M | 160/94 | 103 | 93 | 131 | 179 | 148 | 46 | 105 | - | - | - |
| 42 | Stella | 38 | F | 150/90 | 91 | 86 | 124 | 242 | 207 | 51 | 150 | - | + | + |
| 43 | Kathija | 55 | F | 160/90 | 92 | 82 | 136 | 153 | 122 | 53 | 76 | - | - | - |
| 44 | Abdullah | 59 | M | 156/92 | 94 | 98 | 128 | 178 | 144 | 39 | 110 | - | + | - |
| 45 | Venkatesan | 44 | M | 150/90 | 96 | 128 | 156 | 237 | 149 | 37 | 170 | IGT | + | + |
| 46 | Arputham | 51 | F | 170/98 | 93 | 90 | 132 | 287 | 223 | 52 | 171 | - | + | + |
| 47 | Sadagopan | 34 | M | 160/100 | 96 | 82 | 115 | 156 | 138 | 54 | 95 | - | - | - |
| 48 | Saravanan | 62 | M | 180/100 | 97 | 126 | 177 | 99 | 150 | 41 | 128 | IGT | + | + |
| 49 | Meenatchi | 66 | F | 166/92 | 86 | 76 | 136 | 257 | 215 | 46 | 173 | - | + | + |
| 50 | Mayilraj | 42 | M | 158/94 | 95 | 88 | 131 | 262 | 221 | 42 | 182 | - | + | - |

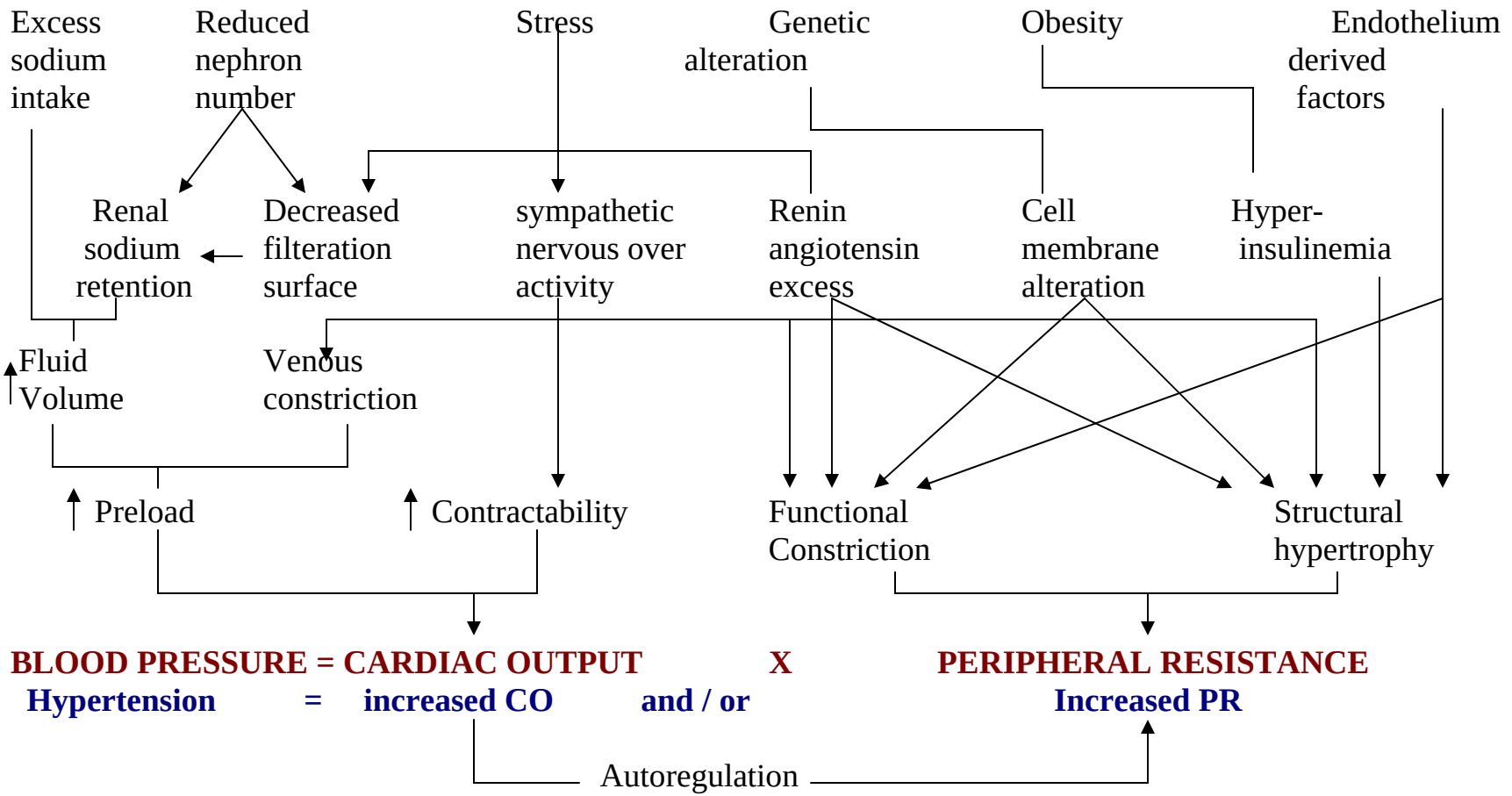
# PATHOGENESIS OF METABOLIC SYNDROME






(FFA-Free fatty acid)

# FACTORS CONTROLLING BLOOD PRESSURE



**PREVALENCE OF LIPID AND GLYCAEMIC ABNORMALITIES IN PATIENTS WITH ESSENTIAL HYPERTENSION**

# PROFORMA

Name: No:

Age/Sex:

**HISTORY:**

Hypertension : CAHD :

CVA:

Diabetes : Renal disease:

**EXAMINATION:**

Pulse: Weight: Height: Waist circumference:

BP:

CVS: Abdomen:

RS: CNS:

Fundus:

**INVESTIGATIONS:**

Blood: TC : Hb%:

DC: ESR:

Urine: Albumin-

Sugar -

Deposit -

Blood: Glucose: Fasting- Urea:

2 Hr Postprandial- Creatinine:

**LIPID PROFILE:**

Total Cholesterol:

Triglyceride :

HDL :

LDL :

VLDL :

**REMARKS**

Impaired glucose tolerance

Dyslipidemia

Metabolic syndrome