

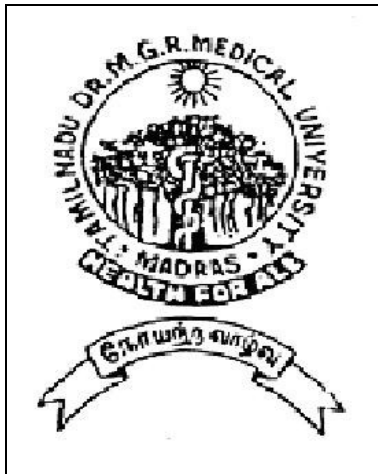
Dissertation on

**A HOSPITAL BASED CROSS-SECTIONAL STUDY ON THE
INCIDENCE OF MICRO-ALBUMINUREA AND LEFT
VENTRICULAR HYPERTROPHY AMOUNG HYPERTENSIVE
PATIENTS.**

Submitted in partial fulfillment for the degree of

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M.D., GENERAL MEDICINE



**DEPARTMENT OF MEDICINE
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DECLARATION

I solemnly declare that this dissertation was prepared by me at Chengalput medical college, Chengalput under the guidance and supervision of Prof. **DR.K.H.NOORUL AMEEN, M.D.**, professor and HOD of medicine, Chengalput medical college, Chengalput. This dissertation is submitted to the Tamilnadu Dr MGR medical university, Chennai in partial fulfillment of the university regulation for the award of degree of M.D., in general medicine.

Place:

Date:

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BONAFIDE CERTIFICATE

This is to certify that this dissertation titled “A hospital based cross-sectional study on the incidence of micro albumin urea and left ventricular hypertrophy among hypertensive patients” in, Chengalput medical college, Chengalput is a bonafide work done by Dr.D.Damodaran in general medicine during the period of March 2009 to Dec 2009. This was a regular and systematic study done under my general guidance and supervision and submitted for the M.D., examination in March 2010 of DR.M.G.R. Medical university, Chennai, TamilNadu.

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PROFORMA

MASTER CHART

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ABBREVIATIONS

ACE	–	Angiotensin Converting Enzyme
ACR	–	Albumin Creatinine Ratio
ALLHAT	–	Antihypertensive and Lipid-Lowering Treatment to
ARB	–	Angiotensin Receptor Blocker
BMI	–	Body Mass Index
BP	–	Blood Pressure
		Cardiovascular End Points trial.
CHD	–	Coronary Heart Disease
CONVINCE	–	Controlled Onset Verapamil Investigation of
CV	–	Cardio Vascular
CVD	–	Cardiovascular Disease
DBP	–	Diastolic Blood Pressure
ECG	–	Electro Cardio Grapy
HDL	–	High Density Lipoprotein
HF	–	Heart Failure
HOPE	–	Heart Outcomes Prevention Evaluation
JNC	–	Joint National Committee
LDL	–	Low Density Lipoprotein
LIFE	–	Losartan Intervention for Endpoint Reduction
LVH	–	Left Ventricular Hypertrophy

MA	–	Microalbuminuria
MAGIC	–	Microalbuminuria: A Genoa Investigation on Complications.
MAU	–	Microalbuminuria
OR	–	Odd Ratio
P	–	Probability
PP	–	Pulse Pressure
PREVEND	–	Prevention of Renal and Vascular End Stage Disease Prevent Heart Attack Trial
RR	–	Relative Risk
SBP	–	Systolic Blood Pressure
SD	–	Standard Deviation
UAC	–	Urinary Albumin Concentration
UAE	–	Urinary Albumin Excretion
UCAR	–	Urine Albumin Creatinine Ratio
WHO	–	World Health Organizat

INTRODUCTION

Hypertension provides both despair and hope. Despair because it is quantitatively the largest risk factor for cardiovascular diseases, is growing in prevalence, and is poorly controlled virtually everywhere. Hope because prevention is possible and because treatment can control hypertension in almost all patients with consequent marked reduction in stroke and heart attack.

It should be noted that cardiovascular diseases are the leading cause of death worldwide, not only in economically developed countries but also in the developing world. In turn, hypertension is overall the major contributor to the risks for cardiovascular diseases. The over all world wide prevalence of hypertension is approximately 26% of the adult populationⁱ, with marked differences between countries. The increasing prevalence of hypertension is primarily a consequence of the population becoming older and obese. Despite overwhelming evidence that effective treatment of hypertension is associated with a significant reduction of cardiovascular events, the number of patients who are aware of their condition and who achieve adequate BP control remains unacceptably low.

Albumin excretion and microalbuminuria are currently drawing a great deal of

attention in the medical literature. Much of this attention derives from the fact that albumin excretion is a risk factor for kidney failure, stroke, and cardiovascular and all-cause mortality, particularly for persons with diabetes and/or hypertensionⁱⁱ.

Based on the data from the Heart Outcomes Prevention and Evaluation Study (HOPE STUDY)ⁱⁱⁱ, it is clear that the presence of microalbuminuria is a signal from kidney that cardiovascular risk is increased and that vascular responses are altered. Microalbuminuria is a highly specific predictor of the simultaneous occurrence of both cardiac and vascular abnormalities.

Data from the LIFE^{iv} study show that the ECG strain pattern of ST-segment depression and T-wave inversion in the lateral precordial leads is a predictor of heart failure. This strain pattern has been associated with increased left ventricular mass and depressed left ventricular function.

This study focuses on the incidence of microalbuminuria and left ventricular hypertrophy among hypertensive patients along with demographic analysis.

AIMS AND OBJECTIVES

TO FIND OUT THE INCIDENCE OF MICRO-ALBUMINURIA AND LEFT VENTRICULAR HYPERTROPHY AMONG HYPERTENSIVE PATIENTS WITHOUT ASSOCIATED CONDITIONS LIKE DIABETES MELLITUS, STROKE, ISCHAEMIC HEART DISEASE AND RENAL DISEASES.

STUDY POPULATION

A hospital based cross-sectional study was conducted among hypertensive patients who attend the out-patient department of cardiology in Chengalpattu medical college hospital, Chengalpattu from march 2009 to December 2009 to find out the incidence of microalbuminuria and left ventricular hypertrophy Among hypertensive patients. All patients between the age of 35 and 65 who attend the hypertensive clinic were screened for the following diseases and excluded from the study.

1. Diabetes mellitus.
2. Cerebro-vascular accident.
3. Coronary artery disease.
4. Kidney diseases (creatinine above 2 mg/dl).
5. Urinary tract infection.

Diabetes mellitus was ruled out by fasting and post prandial blood sugar. Coronary artery disease and stroke was ruled out by history, physical examination and appropriate investigation. Urinary tract infection was ruled out by urine culture and kidney diseases by blood urea and serum creatinine. Patients who were on ACE/ARB drugs were excluded from the study. Study subjects who were anemic and who had fever were excluded from the study. *Only patients with raised blood pressure without the above conditions were the study subjects.*

BLOOD PRESSURE MEASUREMENT^v

Once the patient was selected by the above criteria the BP has been recorded in the following manner. Several steps have been taken to minimize the variability.

1. Having the subjects rest silently and comfortably (with back and arm support if seated) for about 5min before measurement.
2. Abstaining from drinking caffeine or alcohol containing beverages or tobacco use within 30 min before a BP measurement.
3. Questioning the subject regarding the most recent meal or evacuation of bowl or bladder.
4. Centering the bladder of cuff over the brachial artery with its lower edge within 2.5 cm of the antecubital fossa.
5. Assuring that the arm was supported at the level of the heart.
6. Listening over the brachial artery by using the bell of stethoscope with minimal pressure exerted over the skin.
7. BP measurements, the cuff typically should be inflated 20mm hg higher than the pressure at which the palpable pulse at the radial artery disappears.
8. Attempting to avoid “terminal digit preference,” there should be an equal number of readings ending in 0,2,4,6, or 8 mmhg.
9. Measurement of BP in both arm typically are obtained at the initial visit, and the

arm with higher BP was used thereafter if the difference was greater than 10/5 mmhg.

10. On each occasion at least two readings were taken with as much as time as it was practicable separateable. If reading more than 5mmhg additional readings were taken until they where close.

11. For diagnosis at least three sets of readings at one week apart.

CLASSIFICATION:

Hypertension was defined based on seventh joint national committee criteria^{vi}

JNC 7	SBP/DBP
NORMAL	<120/<80
PRE HYPERTENSION	120-139/80-89
STAGE 1 HYPERTENSION	140-159/90-99
STAGE 2 HYPERTENSION	>160/>100

ANTHROPOMETRIC METHODOLOGY

Patients nutritional status was quantified by comparing weight and height-the

Quetelet index.

Body mass index=weight in Kg/(height in meters)²

WHO classification of obesity^{vii}.

CATEGOR Y	BMI
UNDER WEIGHT	<18.5
HEALTHY WEIGHT	18.5-24.9
OVERWEIGHT	25-29.9
MODERATE OBESE	30-34.9
SEVERE OBESE	35-39.9
MORBID OBESE	>40

ELECTROCARDIOGRAPHY METHODOLOGY

Left ventricular hypertrophy was diagnosed by ecg-romhilt and estes point score system.

ROMHILT AND ESTES POINT SCORE SYSTEM^{viii}.

1. INCREASED QRS MAGNITUDE=3 POINTS.
2. ST-T ABNORMALITY=3 POINTS.
3. LEFT ATRIAL ENLARGEMENT=3 POINTS.
4. LEFT AXIS DEVIATION=2 POINTS.
5. INCREASED VENTRICULAR ACTIVATION TIME=1 POINT.

A SCORE OF 5 POINTS OR MORE INDICATES LVH.

MICROALBUMINUREA MEYHODOLOGY METHOD^{ix}

This test was based on antigen antibody reaction. The polyclonal goat anti-human albumin present in the reagent reacts with albumin present in the urine and forms precipitate which can be measured by immunoturbidity method. The precipitate formed is proportional to the amount of albumin present in urine.

REAGENTS

Antiserum consist of polyclonal goat anti human albumin,phosphate buffer saline and sodium azide. Buffer consist of saline,accelerator and sodium azide.

Calibrator contains defibrinated human plasma diluted with phosphate buffered saline.

SAMPLE

Spot midstream urine sample was collected. Study subjects were advised not to smoke and exercise before sample collection.

PROCEDURE

50 micro liter of urine sample was taken in a test tube to this 900 micro liter of buffer and 100 micro liter of antiserum was added. The mixture was incubated at 37 degree centigrade for 5 min. the precipitate obtained was read immune turbidometrically at 340 nanometer.

ESTIMATION OF URINARY CREATININE

PRINCIPLE

Creatinine present in the urine reacts with alkaline picrate in the reagent to form reddish orange colored complex known as creatinine picrate which can be measured calorimetrically. This method was known as Jaffes reaction.

REAGENTS

0.75 N sodium hydroxide

0.04 M picric acid

Creatinine standard of various concentrations.

SAMPLE

The urine sample was diluted with distilled water in 1:20 dilution.

PROCEDURE

250 micro liter of picric acid and 250 micro liter of sodium hydroxide was taken in a test tube to this 50 micro liter of urine was added. This mixture was kept at room temperature. Then the reading was taken at 515 nanometer calorimetrically.

ESTIMATION OF ALBUMIN CREATININE RATIO^x

Albumin creatinine ratio = micro albumin in mg/L x 1000.

Urine creatinine in mg/L

The ratio gives the value of micro albumin in milligram per gram of urinary creatinine.

ACR IN Mg PER Gm OF CREATININE	REFERENCE RANGE
<30	NORMAL
30-300	MICROALBUMIN UREA
>300	MACROALBUMIN UREA

The chi-square test was used in the statistical analysis. Informed consent from the study subjects and ethical clearance from the institute was obtained for this project.

REVIEW OF LITERATURE

Affecting 1 billion people worldwide, systemic hypertension remains the most common, readily identifiable, and reversible risk factor for myocardial infarction, stroke, heart failure, atrial fibrillation, aortic dissection, and peripheral arterial disease. Because of escalating obesity and population aging in developed and developing countries, the global burden of hypertension is rising and projected to affect 1.5 billion persons, one third of the world's population, by the year 2025^{xi}. Thus, hypertension remains the leading cause of death worldwide and one of the world's great public health problems.

The asymptomatic nature of the condition delays diagnosis. Hypertension currently is defined as a usual BP of 140/90 mm^{xii} Hg or higher, BP levels for which the benefits of pharmacological treatment have been definitively established in randomized placebo-controlled trials.

LIFETIME RISK OF HYPERTENSION

Hypertension is an increasingly important medical and public health issue. The prevalence of hypertension increases with advancing age to the point where more than half of people aged 60 to 69 years old and approximately three-fourths of those aged 70 years and older are affected^{xiii}. The age-related rise in SBP is primarily responsible for an increase in both incidence and prevalence of hypertension with increasing age^{xiv}.

Framingham Heart Study investigators recently reported the lifetime risk of hypertension to be

approximately 90% for men and women who were nonhypertensive at 55 or 65 years old and survived to age 80 to 85^{xv}.

BLOOD PRESSURE AND CARDIOVASCULAR RISK

Data from observational studies involving more than 1 million individuals have indicated that death from both ischemic heart disease and stroke increases progressively and linearly from BP levels as low as 115 mm Hg systolic and 75 mm Hg diastolic upward^{xvi}. For every 20 mm Hg systolic or 10 mm Hg diastolic increase in BP, there is a doubling of mortality from both ischemic heart disease and stroke.

Basis for Reclassification of Blood Pressure

Because of the new data on lifetime risk of hypertension and the impressive increase in the risk of cardiovascular complications associated with levels of BP previously considered to be normal, the JNC 7 report has introduced a new classification that includes the term "prehypertension" for those with BPs ranging from 120 to 139 mm Hg systolic and/or 80 to 89 mm Hg diastolic blood pressure (DBP). This new designation is intended to identify those individuals in whom early intervention by adoption of healthy lifestyles could reduce BP, decrease the rate of progression of BP to hypertensive levels with age, or prevent hypertension entirely.

CARDIOVASCULAR DISEASE RISK

The relationship between BP and risk of CVD events is continuous, consistent,

and independent of other risk factors. The higher the BP, the greater is the chance of heart attack, HF, stroke, and kidney diseases. The presence of each additional risk factor compounds the risk from hypertension^{xvii}. Management of these other risk factors is essential and should follow the established guidelines for controlling these coexisting problems that contribute to overall cardiovascular risk.

IMPORTANCE OF SYSTOLIC BLOOD PRESSURE

Impressive evidence has accumulated to warrant greater attention to the importance of SBP as a major risk factor for CVDs. Changing patterns of BP occur with increasing age. The rise in SBP continues throughout life, in contrast to DBP, which rises until approximately 50 years old, tends to level off over the next decade, and may remain the same or fall later in life. Diastolic hypertension predominates before 50 years of age, either alone or in combination with SBP elevation. The prevalence of systolic hypertension increases with age, and above the age of 50 years, systolic hypertension represents the most common form of hypertension. DBP is a more potent cardiovascular risk factor than SBP until age 50; thereafter, SBP is more important^{xviii}.

Clinical trials have demonstrated that control of isolated systolic hypertension reduces total mortality, cardiovascular mortality, stroke, and HF events^{xix}. Both observational studies and clinical trial data suggest that poor SBP control is largely responsible for the unacceptably low rates of overall BP control^{xx}.

In the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack

Trial (ALLHAT) and Controlled Onset Verapamil Investigation of Cardiovascular End Points (CONVINCE) trial, DBP control rates exceeded 90%, but SBP control rates were considerably less (60 to 70%)^{xxi}. Poor SBP control is at least in part related to physician attitudes. Most physicians have been taught that the diastolic pressure is more important than SBP and thus treat accordingly. Greater emphasis must clearly be placed on managing systolic hypertension.

PREVENTION OF HYPERTENSION: PUBLIC HEALTH CHALLENGES

The prevention and management of hypertension are major public health challenges. If the rise in BP with age could be prevented or diminished, much of hypertension, cardiovascular and renal disease, and stroke might be prevented. A number of important causal factors for hypertension have been identified, including excess body weight; excess dietary sodium intake; reduced physical activity; inadequate intake of fruits, vegetables, and potassium; and excess alcohol intake^{xxii}. The prevalence of these characteristics is high.

Because the lifetime risk of developing hypertension is very high, a public health strategy that complements the hypertension treatment strategy is warranted. In order to prevent BP levels from rising, primary prevention measures should be introduced to reduce or minimize these causal factors in the population, particularly in individuals with prehypertension. A population approach that decreases the BP level in the general population by even modest amounts has the potential to substantially reduce morbidity and mortality or at least delay the onset of hypertension. For example, it has been

estimated that a 5 mm Hg reduction of SBP in the population would result in a 14% overall reduction in mortality due to stroke, a 9% reduction in mortality due to CHD, and a 7% decrease in all-cause mortality^{xxiii}.

PREVALENCE OF MICROALBUMINUREA

In 1974, Parving *et al.*^{xxiv} demonstrated the presence of microalbuminuria in patients with untreated essential hypertension. The Losartan Intervention for Endpoint Reduction (LIFE) trial in hypertensive patients with electrocardiographic signs of left ventricular hypertrophy (LVH) the mean age of the 8029 patients was 66 years, 54% were women. Microalbuminuria was found in 23% and macroalbuminuria in 4% of patients.

General population study such as Prevention of Renal and Vascular End Stage Disease (PREVEND) show an 8 to 11.5% prevalence of microalbuminuria in individuals with hypertension^{xxv}.

Microalbuminuria: A Genoa Investigation on Complications [MAGIC]^{xxvi} Study From the Department of Internal Medicine and the Institute of General Pathology (G.S.), University of Genoa; and the Division of Medicine, Galliera (C.C.) and S. Martino (G.G.) Hospitals, Genoa, Italy.

The prevalence of microalbuminuria and its relationship with several cardiovascular risk factors and target organ damage were evaluated in a cohort of 787 untreated patients with essential hypertension. Albuminuria was measured as the

albumin-to-creatinine ratio in three nonconsecutive, first morning urine samples. The was 6.7%. Albuminuric patients were more likely to be men and to be characterized by higher blood pressure, body mass index, and uric acid levels and lower HDL cholesterol and HDL cholesterol-to-LDL cholesterol ratio. The log of ACR showed a positive although weak correlation with age and a stronger correlation with BP (DBP and mean BP), but it was unrelated to BMI and the duration of hypertension. Patients with major ECG changes were more likely to have microalbuminuria (OR, 1.76; 95% CI, 1.06 to 2.95; $P < .04$) and showed higher levels of albuminuria and BP, older age, and a longer duration of hypertension compared with patients with normal ECGs.

The prevalence of microalbuminuria in the general population is in the range of 5 to 7% according to several large cohort studies: PREVEND, Nord-Trøndelag Health Study (HUNT), AusDiab^{xxvii} .

The prevalence of microalbuminuria in patients with hypertension is less consistent in large population or cohort studies, varying from 8 to 23%.

Microalbuminuria can be detected in up to 40% of the population with established hypertension, particularly in those patients not controlled satisfactorily by anti-hypertensive therapy^{xxviii}. Even blood pressure levels between 130 and 139/80 and 89 mmhg are significantly associated with microalbuminuria^{xxix} .

ASSOCIATION OF MICROALBUMINUREA WITH AGE AND SEX

The Nord-Trøndelag Health Study (HUNT)^{xxx}, Norway, 1995 to 1997 (n=65 258)

conducted a 4.3-year follow-up of 2307 men and 3062 women (20 years old) with self-reported treated hypertension. The main outcome measures were relative risk (RR) of all-cause mortality according to increasing albuminuria, defined at different levels of albumin-to-creatinine ratio (ACR). There was a consistent positive association between increasing ACR and all-cause mortality in men. The adjusted RR for ACR in the fourth quartile (1.70 mg/mmol) was 1.6 (95% CI, 1.0 to 2.6), compared with ACR in the first quartile (<0.55 mg/mmol). The corresponding RR in women was 1.5 (95% CI, 0.8 to 3.1). They found a positive association between mortality and increasing number of urine samples with ACR above different cutoff levels, especially in men. In 3 urine samples, the lowest ACR level associated with mortality in men was 0.86 mg/mmol, RR 1.6 (95% CI, 1.1 to 2.4). The sex differences persisted after exclusion of those who died during the first year of follow-up, those with hypertension not treated optimally, and those with known cardiovascular disease.

The association between ACR and all-cause mortality was stronger in treated hypertensive men than in women. The persistent sex differences indicate that hypertensive women tolerate MA better than men and that MA in women should be interpreted differently than in men.

The American Diabetes Association defines microalbuminuria as excretion of 30–299 mg of albumin in a 24-hour collection, with values above 300 being defined as macroalbuminuria^{xxxii}.

The gender-specific ACR cutoffs of 17–249 mg/g for men and 25–354 mg/g for women that have been proposed by Warram et al^{xxxii}. B Hitha, JM Pappachan et al from Kottayam Medical College, Kottayam, Kerala, India conducted an cross-sectional cohort study in patients with essential hypertension attending the medical outpatient clinic and those admitted to the medical wards at the Kottayam Medical College, between May 2005 and October 2006.the sample size was 150.It was found that the patients with longer duration of hypertension, older age and adverse lipid profile are more prone to develop MA.

EVALUATION OF MEASURES OF URINARY ALBUMIN EXCRETION IN EPIDEMIOLOGIC STUDIES

INTERMAP,(International Study of Macronutrients and Blood Pressure)^{xxxiii}

Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL. June 22, 2004 evaluated the Measures of Urinary Albumin Excretion.

. Twenty-four-hour urinary albumin excretion (UAE) is considered the gold standard for determining albumin level in epidemiologic studies, but this measure is inconvenient and often unavailable. Simpler alternatives include the albumin: creatinine ratio (ACR) and urinary albumin concentration (UAC) obtained from a single sample. The authors assessed the strengths and weaknesses of ACR and UAC as alternatives to UAE using albumin measurements from two 24-hour urine samples collected in 1996–

1999 from 4,678 participants aged 40–59 years in the International Study of Macronutrients and Blood Pressure (17 population samples from four countries). The authors compared ACR and UAC with regard to correlations with UAE, daily within-person variability, and associations with known predictors of UAE. Rank-order correlations of ACR with UAE were 0.949 and 0.942 for men and women, respectively, versus 0.881 and 0.816 for UAC. Mean within-person coefficients of variation were 34.0–40.0% for the three measures, with the smallest values being observed for UAC. Average correlations with blood pressure were similar for UAE, ACR, and UAC, but the correlation with body mass index was lower for ACR (0.118 for ACR and 0.188 for UAC vs. 0.211 for UAE) because of high correlation between body mass index and creatinine level. Thus, UAC and ACR are acceptable alternatives to the more complex UAE, and the simpler UAC may be preferable to ACR in some respects.

ECG AND LEFT VENTRICULAR HYPERTROPHY

Ary L Goldberger, MD et al^{xxxiv} found the sensitivity of the various criteria for moderate to severe LVH was in the 30 to 60 percent range, with specificities in the 80 to 90 percent range. Although it is relatively insensitive, the ECG does have prognostic significance. Hypertensive patients with echocardiographically proven LVH who also meet ECG criteria have a greater left ventricular mass than those without the expected ECG changes^{xxxv}.

Echocardiography is the procedure of choice for diagnosing LVH. It can also

permit quantitation of LV mass and give important information about the etiology of LVH (such as aortic or mitral valve disease, or hypertrophic cardiomyopathy). However, the ECG may be used when echocardiography is unavailable or too expensive^{xxxvi} .

The electrocardiographic diagnosis of LVH is quite reliable when very prominent voltage is seen in conjunction with left atrial and ST-T abnormalities, leftward axis, or widening of the QRS. As noted above, however, false negative and false positive diagnoses are not uncommon. Patients with LVH may fail to show voltage criteria, especially if they have only mild hypertrophy or underlying obstructive lung disease. The sensitivity is also reduced in women and in subjects with obesity^{xxxvii} . On the other hand, increased voltage is a common normal variant, particularly in young adult males^{xxxviii} .

The electrocardiogram is a useful but imperfect tool for detecting LVH. The utility of the ECG relates to its being relatively inexpensive and widely available. The limitations of the ECG relate to its moderate sensitivity or specificity depending upon which of the many proposed sets of criteria are applied^{xxxix} .

MICROALBUMINURIA AS AN EARLY MARKER FOR CARDIOVASCULAR DISEASE

Excretion of albumin in the urine is highly variable, ranging from nondetectable quantities to milligrams of albumin and even grams of albumin. Microalbuminuria is defined as low levels of urinary albumin excretion of 30 to 300 mg/^{xl}. It is interesting that microalbuminuria also is found frequently in seemingly healthy individuals (5 to 7%)^{xli}. The variable excretion of albumin in the urine is related to the risk for the individual to develop cardiovascular (CV) disease: Absence or very low levels of albuminuria is associated with low CV risk, whereas the CV risk increases markedly with increasing amount of albumin in the urine (even within the now considered normal range). The predictive power of urinary albumin levels for CV risk is independent of other CV risk factors and not only is present in individual with diabetes and/or hypertension but also in healthy individuals. Treatments that lower albuminuria are associated with CV protection, as demonstrated in randomized, controlled trials of patients with diabetes as well as in patients with hypertension. There is preliminary evidence that albuminuria lowering is CV protective in healthy individuals with an elevated albumin excretion rate. Higher levels of urinary albumin seem to reflect the ordinary inter individual variability in (renal and systemic) endothelial function. In conclusion, albuminuria seems to be a sensitive marker very early in life for the susceptibility of an individual to CV disease. It therefore may be an ideal target for early primary prevention using CV-protective therapy regimens.

Microalbuminuria is defined as small quantities of albumin in the urine, ranging from 30 to 300 mg/d. Below 30 mg/d (or 20 mg/L) is considered normal, and above 300 mg/d (or 200 g/L) is considered to be macroalbuminuria (also called overt albuminuria).

MICROALBUMINUREA AND CARDIOVASCULAR RISK

Framingham Offspring Study^{xlii} the association of urinary albumin excretion (spot urine albumin indexed to creatinine [UACR] and the incidence of CVD events and all-cause mortality in 1568 non-hypertensive, non-diabetic participants (mean age, 55 years; 58% women) free of CVD was examined. On follow-up (median, 6 years), 54 participants (20 women) developed a first CVD event, and 49 (19 women) died. After adjustment for established risk factors, increasing UACR was associated with greater risk of CVD (hazards ratio [HR] per SD increment in log UACR, 1.36; 95% CI, 1.00 to 1.87) and death (HR per SD increment in log UACR, 1.55; 95% CI, 1.10 to 2.20). Participants with UACR greater than or equal to the sex-specific median (3.9 µg/mg for men, 7.5 µg/mg for women) experienced a nearly 3-fold risk of CVD (adjusted HR, 2.92; 95% CI, 1.57 to 5.44; $P<0.001$) and a borderline significantly increased risk of death (adjusted HR, 1.75; 95% CI, 0.95 to 3.22; $P=0.08$) compared with those with UACR below the median. The increased CVD risk associated with UACR at or above the median remained robust in analyses restricted to individuals without microalbuminuria (n=1470) and in subgroups with intermediate (n=1469) and low (n=1186) pretest probabilities of CVD.

Brantsma *et al.*^{xliii} showed that individuals with microalbuminuria had an approximately four-fold increase in the risk for developing subsequent new-onset diabetes than those with low normal urinary albumin levels, even after correcting for baseline glucose and insulin levels or after excluding those with impaired fasting glucose or metabolic syndrome. Brantsma *et al.* similarly found that microalbuminuria increased the risk for *de novo* hypertension by two-fold as compared with normal albuminuria levels.

In the patient with hypertension, microalbuminuria has been discovered as an important factor, although it has not penetrated all guidelines. The groups of Bigazzi and Campese *et al.*^{xliv} reviewed the importance of microalbuminuria as a CV risk predictor. Larger cohort studies confirmed this to be independent from other risk markers in the general hypertensive population (MONICA)^{xlv}, a hypertensive cohort with LVH (LIFE)^{xlvi}, and in individuals with already increased CV risk (Heart Outcomes Prevention Evaluation [HOPE])^{xlvii}.

In the above studies, microalbuminuria was associated and clustered with other widely known CV risk factors (age, diabetes, hypertension, LVH, overweight, metabolic syndrome, etc.) that could explain the increased CV risk. Careful correction for such factors and *post hoc* selection of "healthy" individuals in the large general population cohorts did still reveal the marked and overwhelming independent predictive power of microalbuminuria. This was confirmed by the recent Framingham publication of Arnlov

et al.^{xlviii} that showed elegantly that in normotensive individuals without diabetes and with normal renal function, microalbuminuria remains a strong predictor for CV outcome.

LOWERING MICROALBUMINURIA AND CV PROTECTION

Albuminuria seems to be an independent and strong predictor for CV disease. Several strategies are available to lower urinary albumin excretion in the microalbuminuric range. Widely known is the albuminuria-lowering effect of antihypertensive agents, in particular those that intervene in the renin-angiotensin-aldosterone system. However, statins and glucose-aminoglycans also have been proved to lower albuminuria^{xlix}. Asselbergs *et al.*^l indeed showed that the lowering of albuminuria with the angiotensin-converting enzyme inhibitor fosinopril tended to be cardio protective. A recent *post hoc* analysis of the LIFE trial found similar results in hypertensive patients: The more the angiotensin II antagonist losartan lowered albuminuria, the more the patient was cardio protected, irrespective of the effect on other CV risk factors^{li}.

Future CV trials involving drugs that target albuminuria more specifically are needed to resolve the issue of whether specific lowering of albuminuria results in CV protection and whether this is a cost-effective health care approach. In this issue's *Frontiers in Nephrology*, De Jong and Curhan^{lii} review the public health perspectives of screening and monitoring of urine albumin excretion in relation to CV disease

prevention.

LEFT VENTRICULAR HYPERTROPHY AND HEART FAILURE IN HYPERTENSIVE PATIENTS

One well-known risk factor for heart failure is hypertension, with 39% of new cases in men and 59% in women ascribed to hypertension^{liii}.

Independent of blood pressure, electrocardiographic (ECG) left ventricular hypertrophy (LVH) is also associated with an increased risk of new heart failure^{liv}. This risk is decreased by the prevention of ECG LVH. Antihypertensive therapy that reduced blood pressure has been shown to decrease ECG LVH, and regression of ECG LVH has been correlated with a significant reduction in cardiovascular death, myocardial infarction (MI), and stroke, regardless of antihypertensive treatment or degree of decrease in blood pressure^{lv}.

A very recent publication from Leoncini *et al*^{lvi} examined the presence of sub clinical CV damage, localized in the heart and in the carotid artery, in hypertensive patients with either microalbuminuria and a creatinine clearance <60 ml/min/m. After adjusting for duration of hypertension, mean blood pressure, smoking habits and age, the risk of left ventricular hypertrophy and/or carotid atherosclerosis was increased by 43% with each SD reduction in creatinine clearance and by 89% with each SD increase in microalbuminuria. Mildly increased serum creatinine has been shown to be associated with angiographic coronary artery disease in women^{lvii}. An increased left ventricular

mass has been amply demonstrated in patients with microalbuminuria^{lviii} . These and other recently reviewed^{lix} data indicate that microalbuminuria can be considered as an integrated marker of sub clinical organ damage starting with the early stages of arterial hypertension. Microalbuminuria is then a useful tool for risk profiling.

RESULTS AND DISCUSSION

AGE WISE DISTRIBUTION

Age	No. Of Persons	%
35-45	14	14.4
46-55	36	37.1
56-65	47	48.5
TOTAL	97	100

Table No: 1



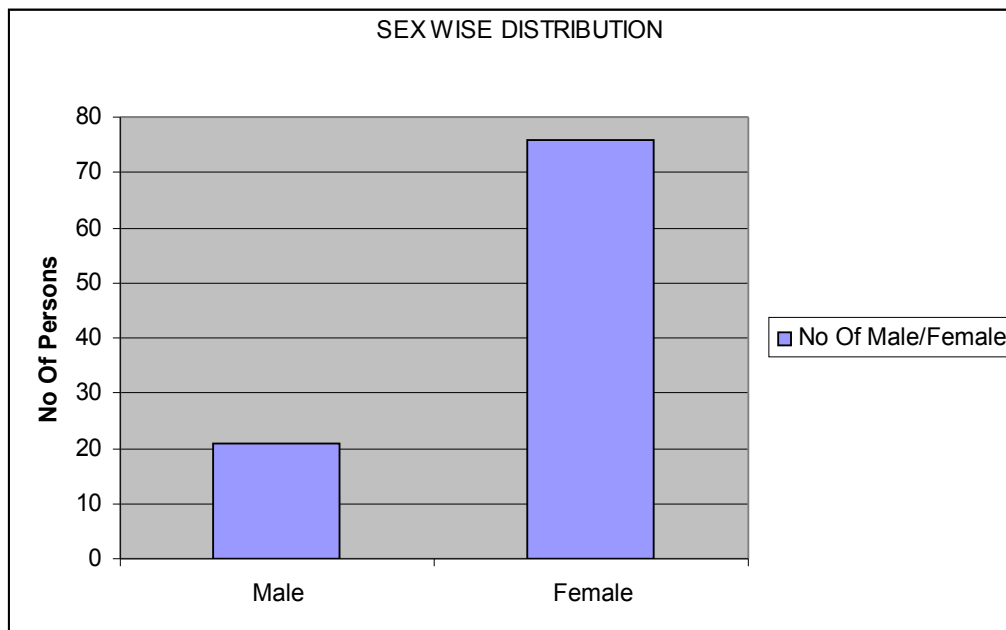
AGE WISE DISTRIBUTION

After applying the exclusion criteria the majority (48.5 %) of the study subjects were in the age group between 56 and 65 yrs. 37 % of study subjects were between 46 and 55 yrs. Only 14% were younger than 46 yrs.

SEX WISE DISTRIBUTION

Sex	No Of Male/Female	%
Male	21	21.6
Female	76	78.4
TOTAL	97	100

Table No: 2



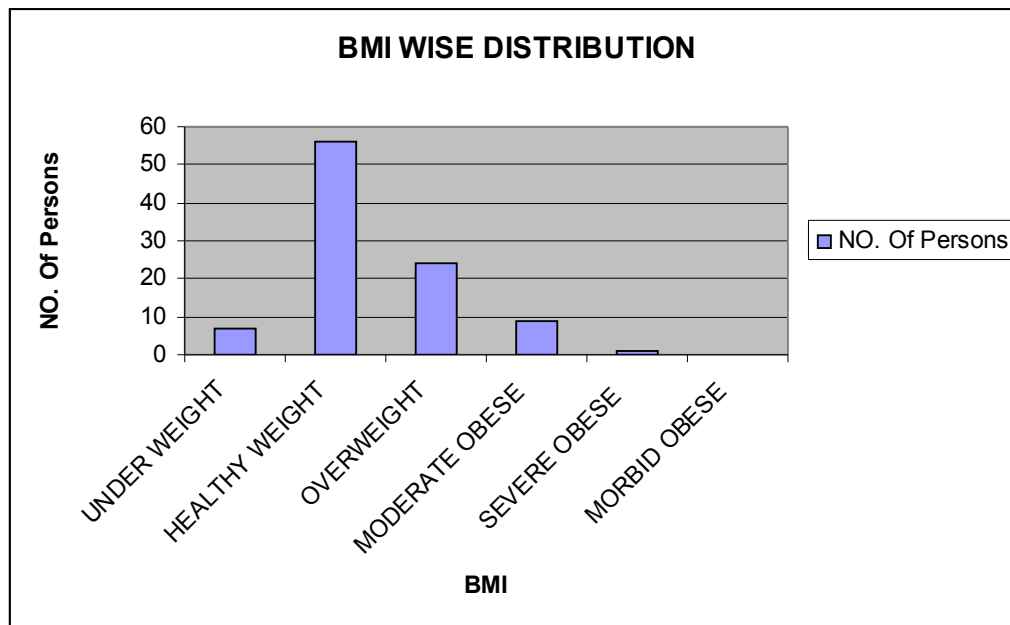
SEX WISE DISTRIBUTION

78 % of the study subjects were female and remaining were males.

BMI WISE DISTRIBUTION

BMI	NO. Of Persons	%
UNDER WEIGHT	7	7.2
HEALTHY WEIGHT	56	57.8
OVERWEIGHT	24	24.8
MODERATE OBESE	9	9.2
SEVERE OBESE	1	1
MORBID OBESE	0	0
TOTAL	97	100

Table No: 3



BMI WISE DISTRIBUTION

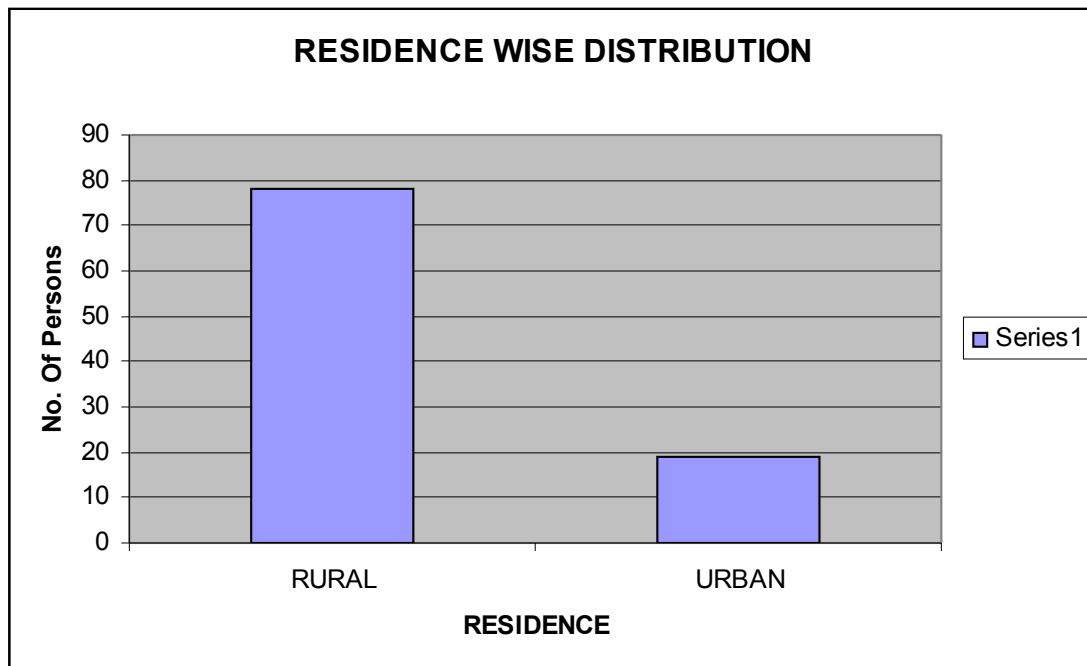
As per the WHO classification of obesity the majority (57.8%) of the study

subjects were in the BMI (Body Mass Index) between 18.5 and 24.9(healthy weight).34.8% were over weight by BMI. Only minimal study subjects were moderate and severe obese.

RESIDENCE WISE DISTRIBUTION

RESIDENCE	No. Of Persons	%
RURAL	78	80.4
URBAN	19	19.6
TOTAL	97	100

Table No: 4

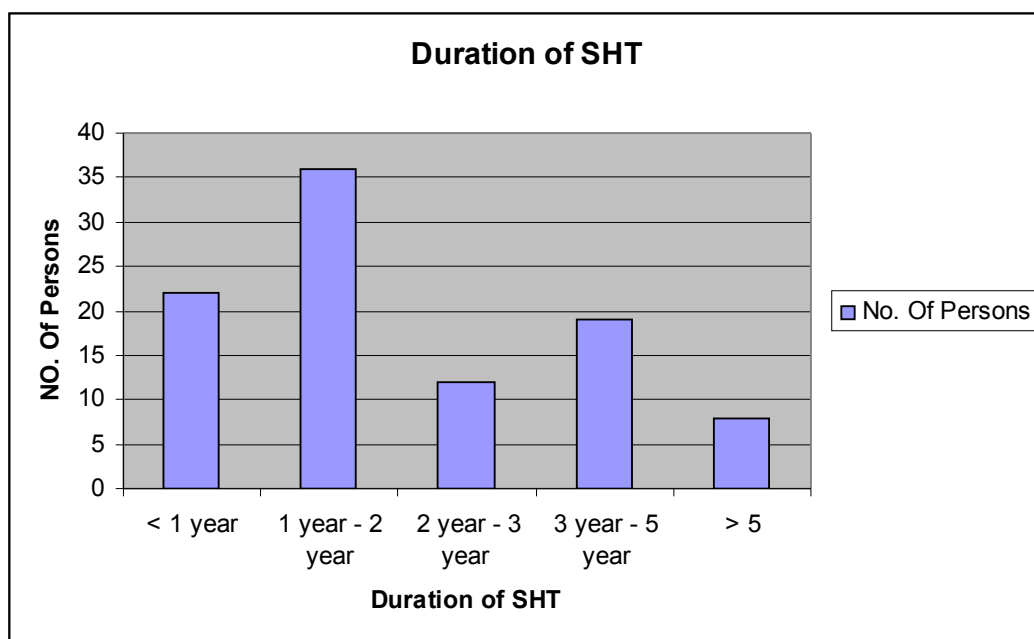


Majority (80.4 %) of selected subjects were from rural area.

DURATION OF HYPERTENSION AND ITS DISTRIBUTION

Duration of SHT	No. Of Persons	%
< 1 year	22	22.7
1 year - 2 year	36	37.1
2 year - 3 year	12	12.4
3 year - 5 year	19	19.6
> 5	8	8.2
Total	97	100

Table No: 5



DURATION OF HYPERTENSION AND ITS DISTRIBUTION

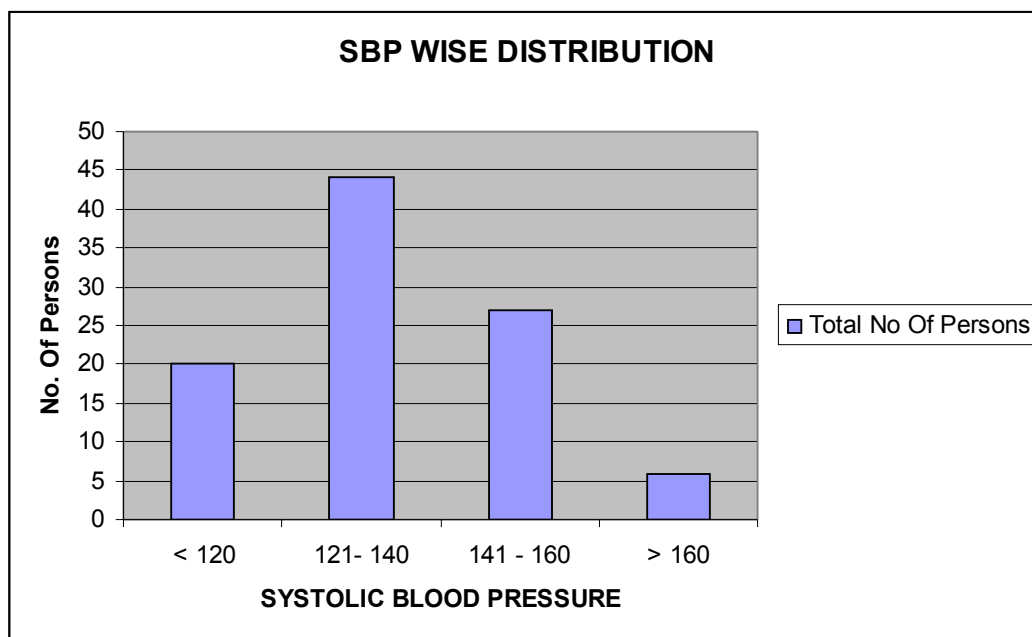
From the history obtained from the study subjects majority (37.1 %) admitted that they were taking treatment for hypertension between one to two yrs.19 % of the

study subjects were taking treatment for 3 to 5 yrs as per the history. Only meager were in the category of more than 5 yrs duration.

SYSTOLIC BLOOD PRESSURE AND ITS DISTRIBUTION

SBP	Total No Of Persons	%
< 120	20	20.6
121- 140	44	45.4
141 - 160	27	27.8
> 160	6	6.2
TOTAL	97	100

Table No: 6



SYSTOLIC BLOOD PRESSURE AND ITS DISTRIBUTION

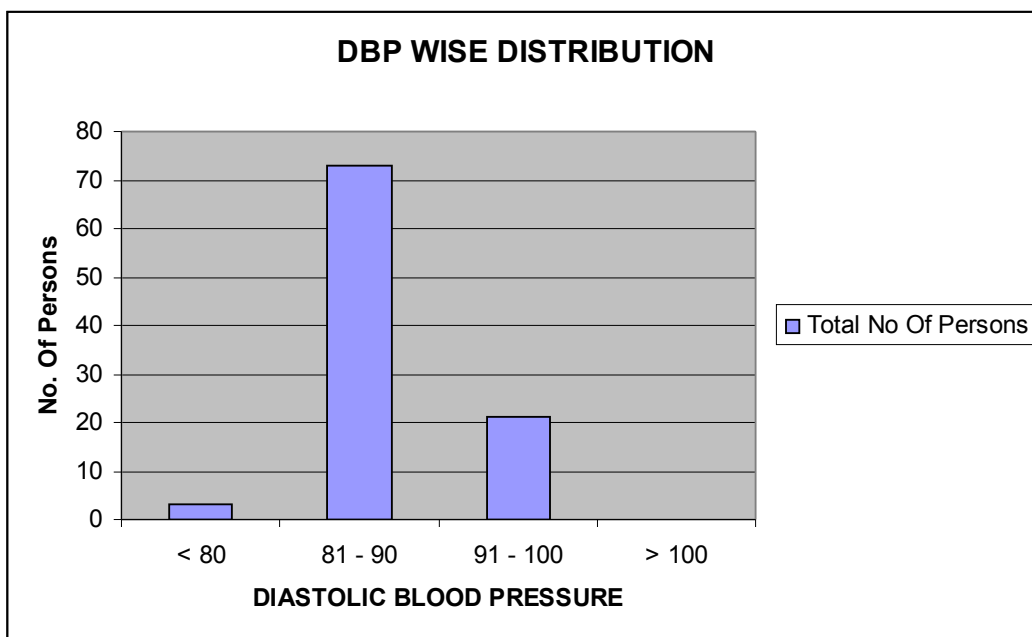
45.4 % of the study subjects had systolic blood pressure (SBP) reading between

121 to 140 at the time of examination after following the necessary procedure. 27.8 % of study subjects had SBP between 141 and 160. Negligible number of study subjects had SBP of more than 160 and less than 120.

DIASTOLIC BLOOD PRESSURE AND ITS DISTRIBUTION

DBP	Total No Of Persons	%
< 80	3	3.1
81 - 90	73	75.3
91 - 100	21	21.6
> 100	0	0
TOTAL	97	100

Table No: 7



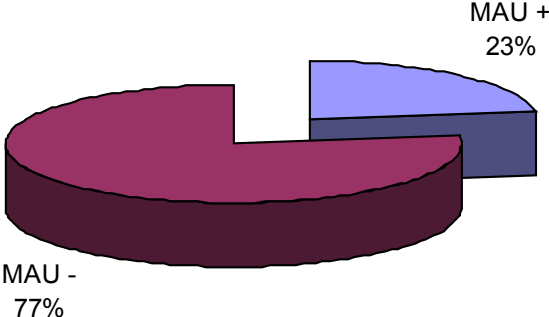
75.3 % of study subjects had diastolic blood pressure between 81 and 90.

INCIDENCE OF MICROALBUMINUREA

MICROALBUMINUREA (MAU)	OUTCOME	%
MAU +	22	22.7
MAU -	75	77.3
TOTAL	97	100

Table No: 8

MAU OUTCOME



■ MAU +
■ MAU -

INCIDENCE OF MICROALBUMINUREA

Out of 97 study subjects who were selected for the study between March 2009 to December 2009, 22.7% of the study subjects had microalbuminuria by immunoturbidity method.

In the Losartan Intervention for Endpoint Reduction (LIFE)^{lx} trial in hypertensive patients with electrocardiographic signs of left ventricular hypertrophy (LVH) the mean age of the 8029 patients was 66 years, 54% were women. Microalbuminuria was found in 23% and macroalbuminuria in 4% of patients.

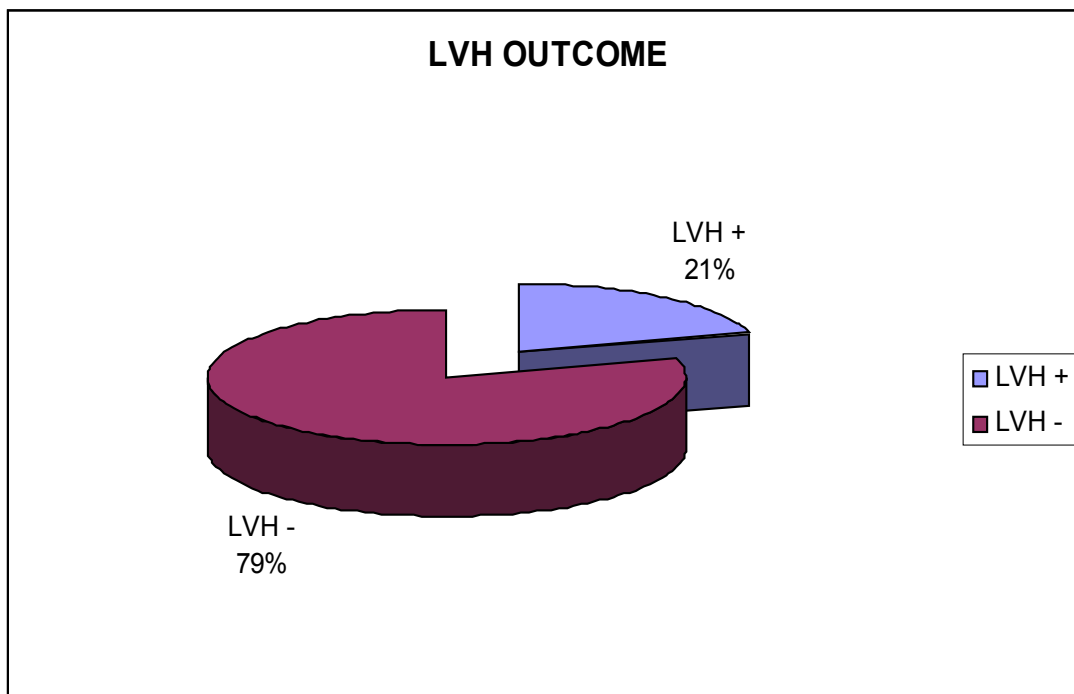
Agewall et al^{lxi} reported an ≈23% prevalence of microalbuminuria in a population of hypertensive patients who were selected as at high risk for cardiovascular disease.

In our study as mentioned above, 22.7% had microalbuminurea.

INCIDENCE OF LEFT VENTRICULAR HYPERTROPHY

LVH	OUTCOME	%
LVH +	20	20.6
LVH -	77	79.4
TOTAL	97	100

Table No: 9



INCIDENCE OF LEFT VENTRICULAR HYPERTROPHY

ECG was taken in all study subjects and it was found that 21 % of the study subjects had left ventricular hypertrophy.

B Hitha, JM Pappachan et al ^{lxii} conducted a cross-sectional cohort study in patients with essential hypertension attending the medical outpatient clinic and those admitted to the medical wards at the Kottayam Medical College, between May 2005 and October 2006. It was found that 26.67 % had microalbuminuria and 10.67 % had ECG evidence of left ventricular hypertrophy.

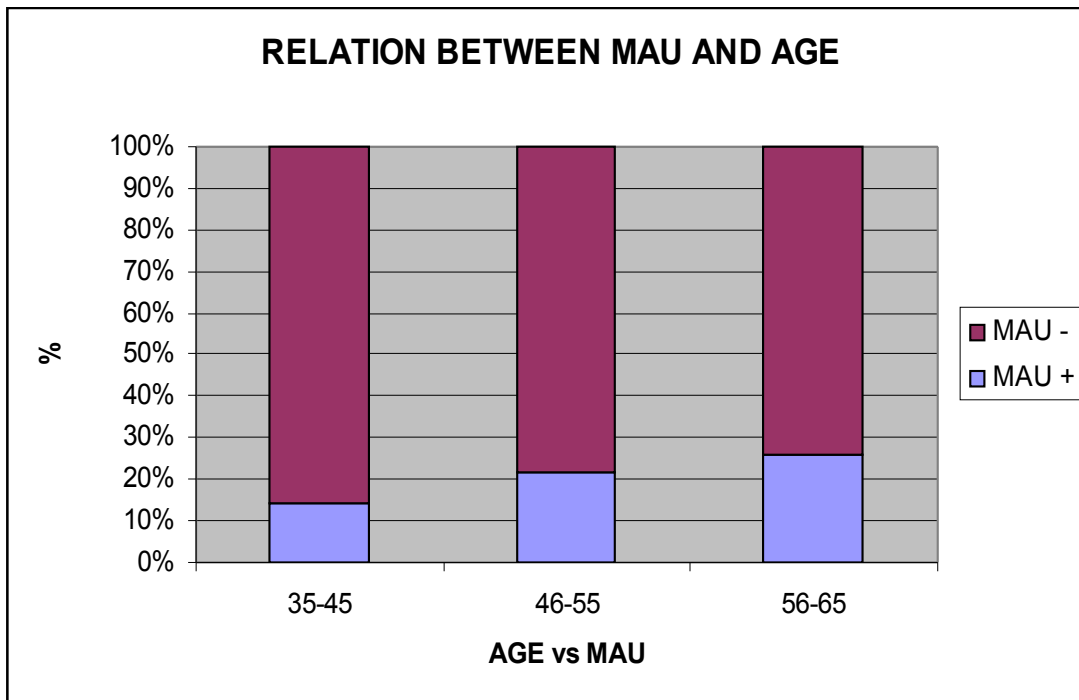
The difference in incidence of left ventricular hypertrophy was due to the difference in ECG methodology and sample size.

RELATION BETWEEN MICROALBUMINUREA AND AGE

Age	Total No Of Persons	MAU +	MAU -	% Of Positivity
35-45	14	2	12	14.3
46-55	36	8	29	22.2
56-65	47	12	34	25.5

Table No: 10

Chi-square value = 6.3 , P value = < 0.05



RELATION BETWEEN MICROALBUMINUREA AND AGE

Majority (25.5 %) of study subjects belong to the age group between 56 to 65 yrs. Only 14 % of the study subjects belong to the age group between 35 to 45 yrs. As the age increases the occurrence of microalbuminuria also increases.

Ricardo Pereira Silva et al ^{lxiii} conducted a study in a group of hypertensive patients (73 individuals) and in a group of patients with coronary artery disease (39 individuals) was determined and compared with a control group (43 individuals). There was a statistically significant correlation between age and the occurrence of microalbuminuria. |

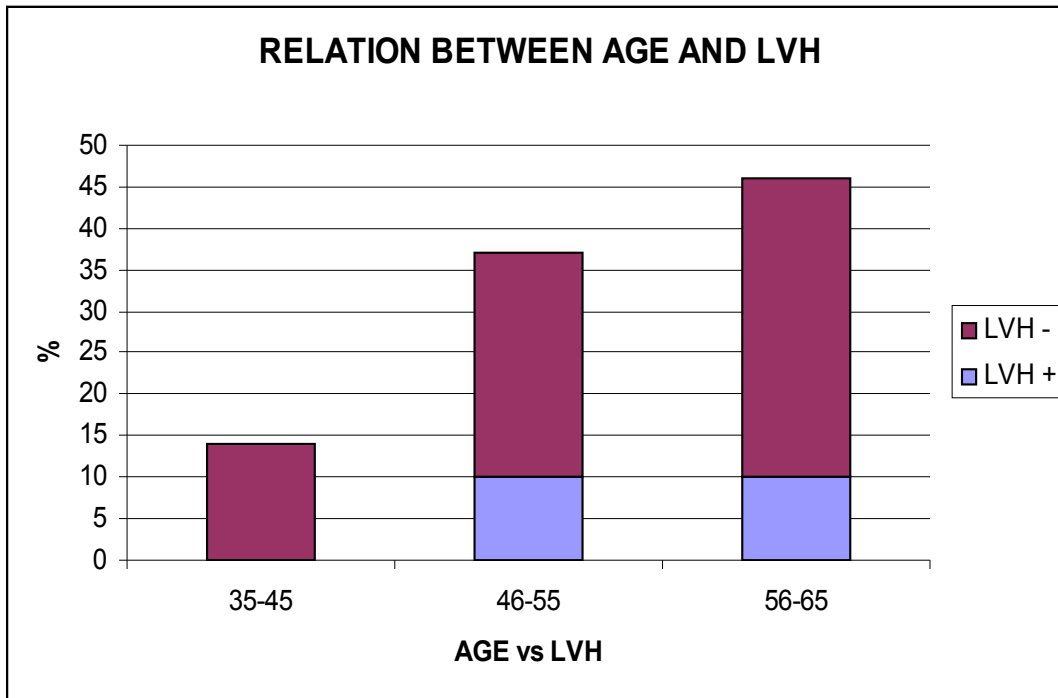
Our study also showed a statistical significant difference ($p < 0.05$) between the age group and the onset of microalbuminuria.

RELATION BETWEEN AGE AND LEFT VENTRICULAR HYPERTROPHY

Age	NO. Of Persons	LVH +	LVH -	% Of Positivity
35-45	14	0	14	0
46-55	37	10	27	27
56-65	46	10	36	21.7

Table No: 11

Chi-square value = 3.6, P value = > 0.05



RELATION BETWEEN AGE AND LEFT VENTRICULAR HYPERTROPHY

27 % of the study subjects who had left ventricular hypertrophy belong to the age group between 46 to 55 and 21.7 % belong to age group between 56 and 65. There is no consistent increase in the incidence of left ventricular hypertrophy with age.

Sybolt O.de vireo, Wilfred F. Heesen et al ^{lxiv} from Department of health sciences, University hospital Groninger: The Netherlands conducted a community based survey program in 1996 in the general population with 277 subjects. It was found that the correlation between age and LV mass in the study was very weak ($p > 0.001$).

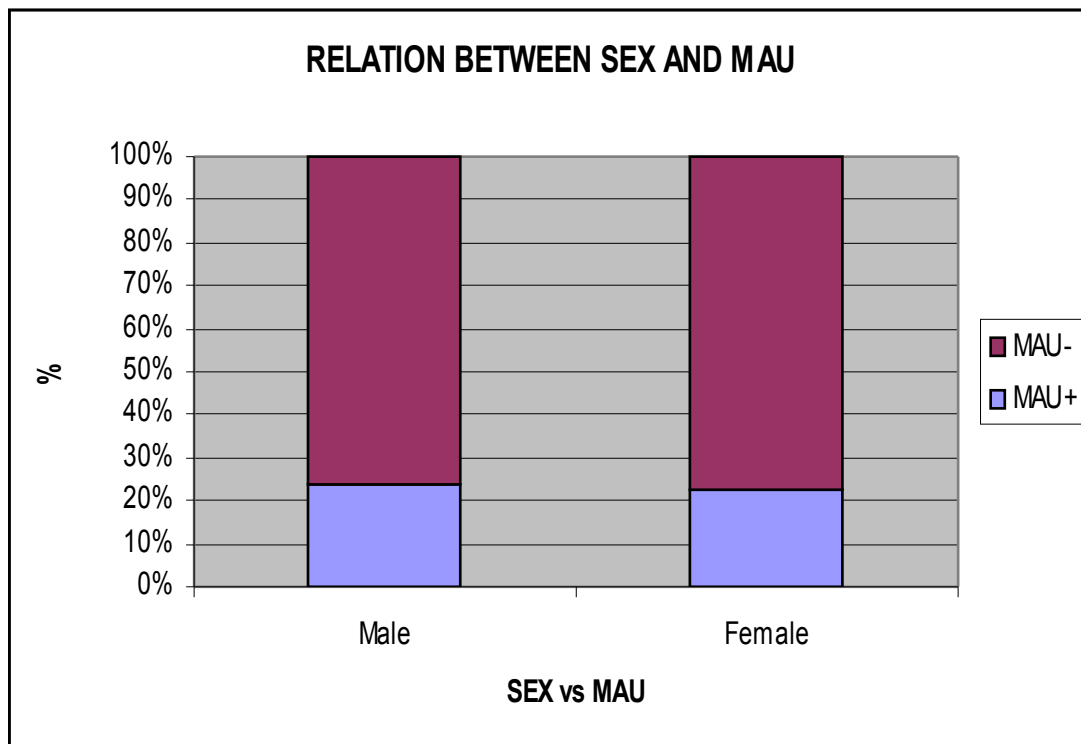
In our study also the correlation between age and LV mass was very weak.

RELATION BETWEEN SEX AND MICROALBUMINUREA

Sex	No Of Male/Female	MAU+	MAU-	% Of Positivity
Male	21	5	16	23.8
Female	76	17	59	22.4

Table No: 12

Chi-square value = 9.3, P value = < 0.01



RELATION BETWEEN SEX AND MICROALBUMINUREA

The above table (No.12) shows a strong correlation between sex and microalbuminuria. The p value was less than 0.01 which signifies a statistical difference between male and female with regard to microalbuminuria.

The Nord-Trøndelag Health Study (HUNT)^{lxv}, Norway, 1995 to 1997 (n=65 258) conducted a 4.3-year follow-up of 2307 men and 3062 women (20 years old) with self-reported treated hypertension. The main outcome measures were relative risk (RR) of all-cause mortality according to increasing albuminuria, defined at different levels of albumin-to-creatinine ratio (ACR). There was a consistent positive association between increasing ACR and all-cause mortality in men. They found a positive association between mortality and increasing number of urine samples with ACR above different cutoff levels, especially in men.

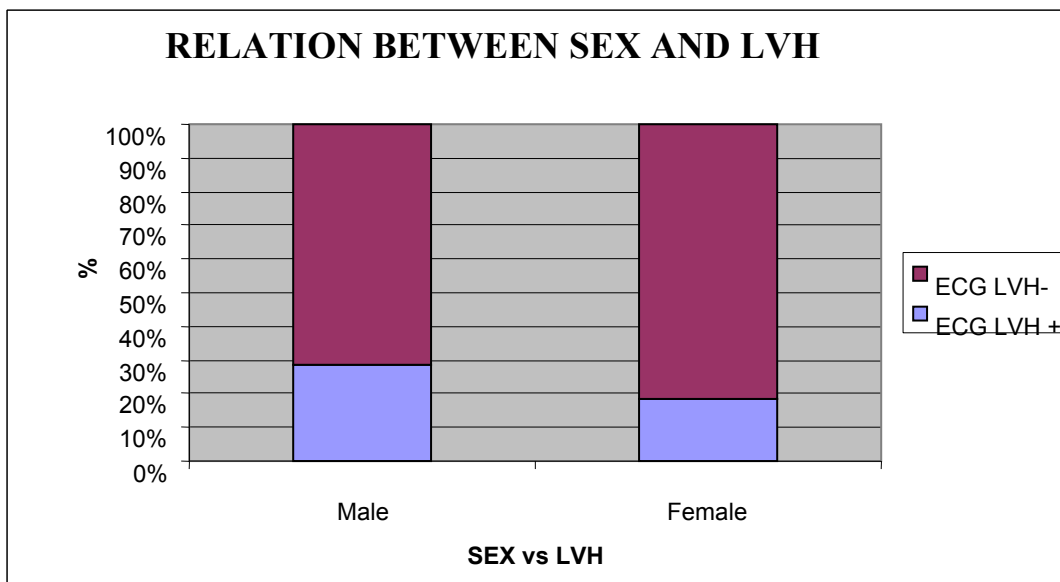
Our study also showed a strong association between sex and the incidence of microalbuminuria.

RELATION BETWEEN SEX AND LEFT VENTRICULAR HYPERTROPHY

Sex	No Of Male/Female	ECG LVH +	ECG LVH-	%
Male	21	6	15	28.6
Female	76	14	62	18.4

Table No: 13

Chi-square value = 6.9, P value = < 0.01



RELATION BETWEEN SEX AND LEFT VENTRICULAR HYPERTROPHY

As per the table (no. 13), out of 21 males 6 had left ventricular hypertrophy (28.6

%) where as it was only 18.4 5 out of 76 females. Regarding the relationship between sex and left ventricular hypertrophy our study showed a very strong statistical difference between sex and left ventricular hypertrophy.

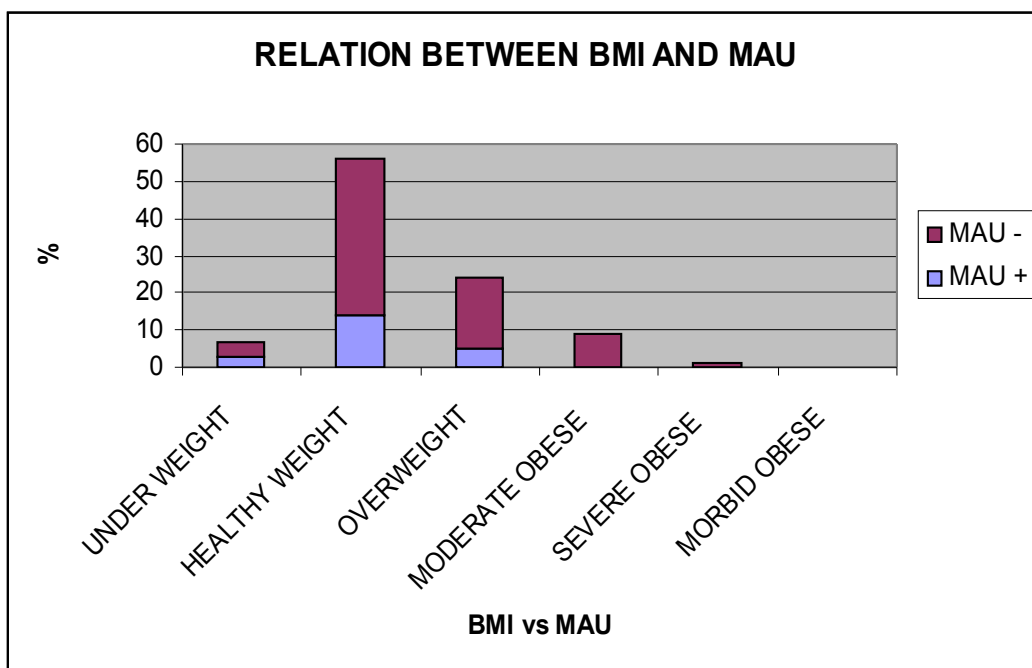
ANTIKAINEN R. et al ^{lxvi} studied 2994 hypertensive patients in whom an electrocardiogram was recorded while not on treatment. It was found that patients with the presence of ECG LVH were more often men.

RELATION BETWEEN BODY MASS INDEX AND MICROALBUMINUREA

BMI	NO. Of Persons	MAU +	MAU -	% Of Positivity
UNDER WEIGHT	7	3	4	42.9
HEALTHY WEIGHT	56	14	42	25
OVERWEIGHT	24	5	19	20.8
MODERATE OBESE	9	0	9	0
SEVERE OBESE	1	0	1	0
MORBID OBESE	0	0	0	0

Table No: 14

Chi-square value = 6.2, P value = > 0.05



RELATION BETWEEN BODY MASS INDEX AND MICROALBUMINUREA

From the table no. 14 it was evident that body mass index has not showed any correlation with microalbuminuria since majority (57.8 %) of the study subjects were with body mass index in healthy weight. Hence there was no statistical significant difference between body mass index and microalbuminuria. Though in general the probability of occurrence of microalbuminuria increases as the body mass index increases.

Ricardo Pereira Silva et al ^{lxvii} conducted a study in a group of hypertensive patients (73 individuals) and in a group of patients with coronary artery disease (39

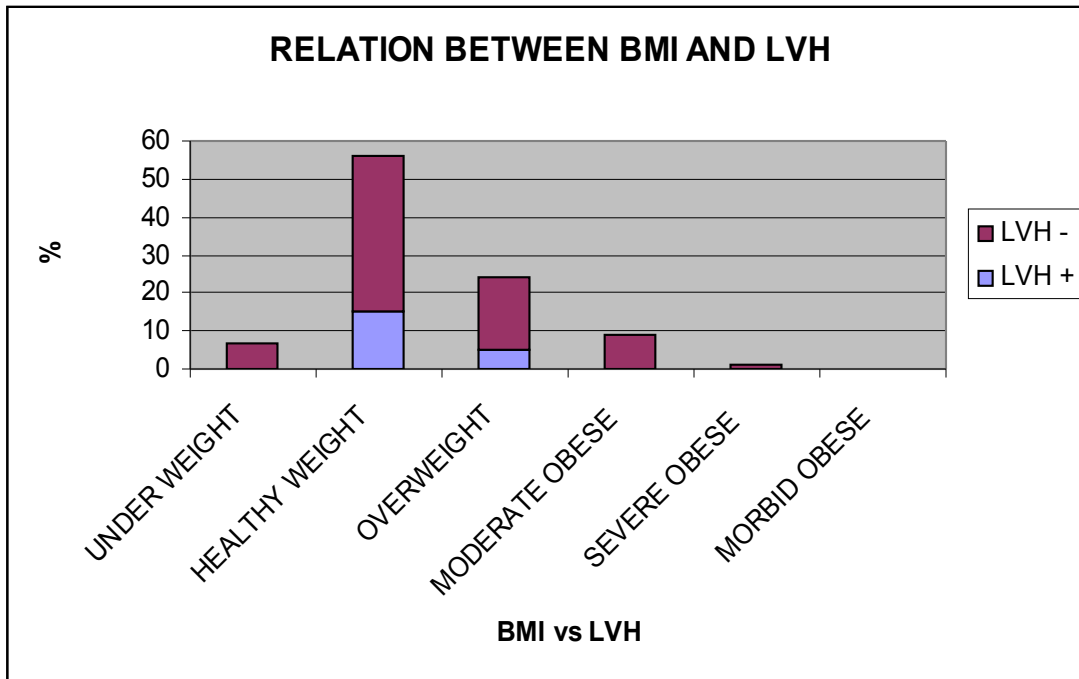
individuals) was determined and compared with a control group (43 individuals). The prevalence of microalbuminuria among obese or overweight patients(13%) was lower than that of the patients with normal weight (17.9%)

RELATION BETWEEN BODY MASS INDEX AND LEFT VENTRICULAR HYPERTROPHY

BMI	NO. Of Persons	LVH +	LVH -	% Of Positivity
UNDER WEIGHT	7	0	7	0
HEALTHY WEIGHT	56	15	41	26.8
OVERWEIGHT	24	5	19	20.8
MODERATE OBESE	9	0	9	0
SEVERE OBESE	1	0	1	0
MORBID OBESE	0	0	0	0

Table No: 15

Chi-square value = 7.1, P value = > 0.05



RELATION BETWEEN BODY MASS INDEX AND LEFT VENTRICULAR HYPERTROPHY

The above table no.15 shows that left ventricular hypertrophy was present only in the subjects who were in healthy weight and over weight. Left ventricular hypertrophy was absent in rest of the subjects. Since majority (57.8 %) of the study subjects as mentioned early were in the healthy weight, the percentage of Positivity for left ventricular hypertrophy was also observed more among them.

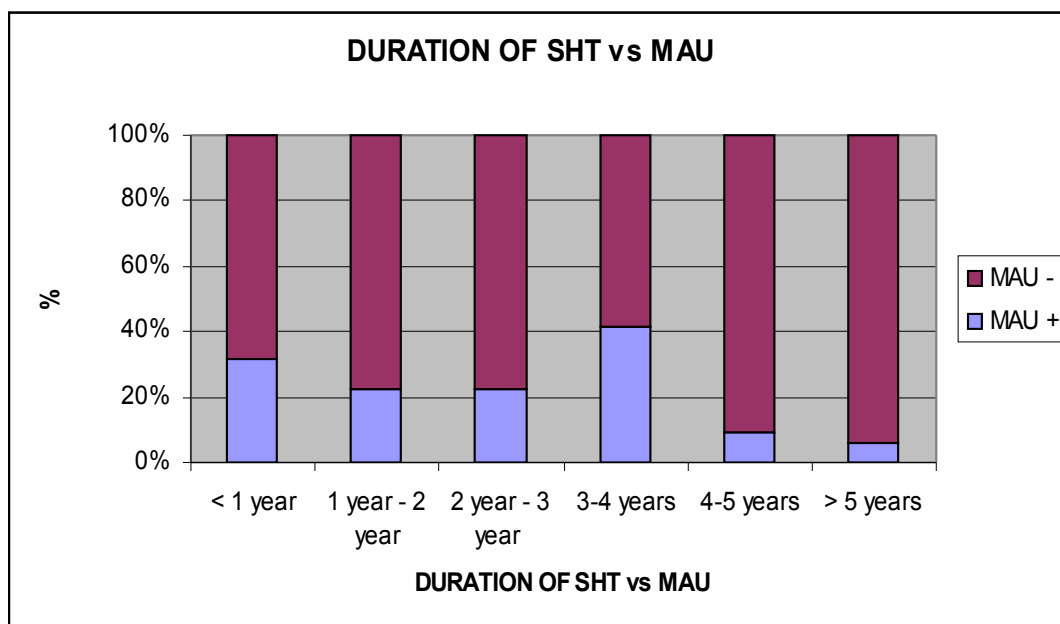
ANTIKAINEN R. et al ^{lxviii} studied 2994 hypertensive patients in 2003 in whom an electrocardiogram was recorded while not on treatment. It was found that systolic (SBP) and diastolic (DBP) blood pressure and pulse pressure were positively related to the increasing ECG voltage, while body mass index (BMI) was inversely related.

RELATION BETWEEN DURATION OF HYPERTENSION AND MICROALBUMINUREA

Duration of SHT	No. Of Persons	MAU +	MAU -	% Positivity
< 1 year	22	7	15	31.8
1 year - 2 year	27	6	21	22.2
2 year - 3 year	9	2	7	22.2
3-4 years	12	5	7	41.6
4-5 years	11	1	10	9.1
> 5 years	16	1	15	6.25

Table No: 16

Chi-square value = 1.3, P value = > 0.05



RELATION BETWEEN DURATION OF HYPERTENSION AND MICROALBUMINUREA

From the above table no. 16 it was evident that there was no statistical significance between the duration of hypertension and the occurrence of microalbuminuria. Only a prospective cohort study can prove the strength of association between the duration of hypertension and the occurrence of microalbuminuria by calculating the relative risk.

Roberto Pontremoli et al ^{lxix} in the Microalbuminuria: A Genoa Investigation on Complications [MAGIC]) Study, the prevalence of microalbuminuria and its relationship with several cardiovascular risk factors and target organ damage were evaluated in a cohort of 787 untreated patients with essential hypertension. Albuminuric patients were more likely to be men and to be characterized by higher blood pressure, body mass index, and uric acid levels and lower HDL cholesterol and HDL cholesterol-to-LDL cholesterol ratio. The log of ACR showed a positive although weak correlation with age and a stronger correlation with BP (DBP and mean BP), but it was unrelated to BMI and the duration of hypertension.

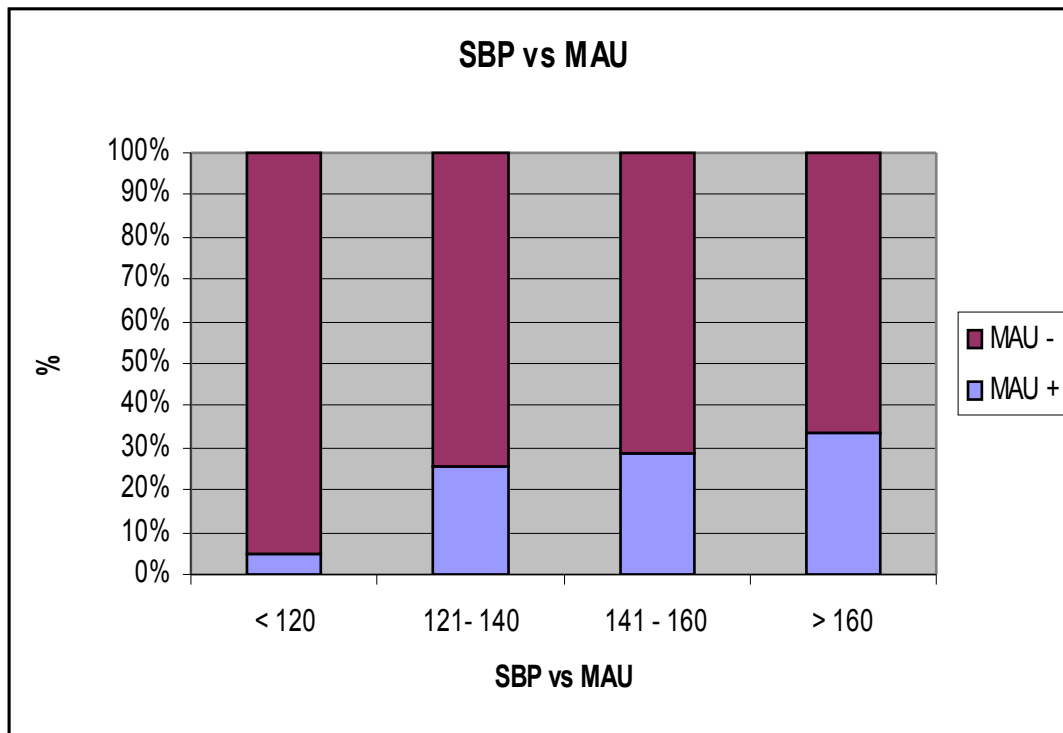
RELATION BETWEEN SYSTOLIC BLOOD PRESSURE AND

MICROALBUMINUREA

SBP	NO. Of Persons	MAU +	MAU -	% Of Positivity
< 120	20	1	19	5
121- 140	43	11	32	25.6
141 - 160	28	8	20	28.6
> 160	6	2	4	33.3

Table No: 17

Chi-square value = 1.7, P value = > 0.05



RELATION BETWEEN SYSTOLIC BLOOD PRESSURE AND MICROALBUMINUREA

From the table no. 17 it was evident that there was no statistical significance between systolic blood pressure and the occurrence of microalbuminuria.

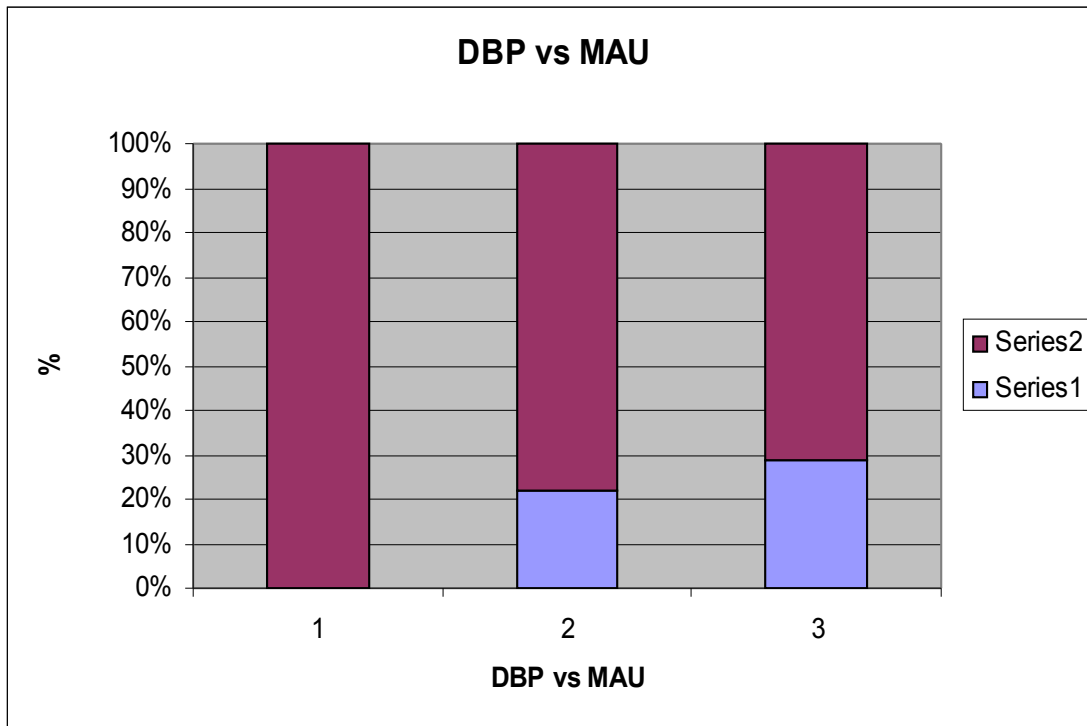
[Massimo Cirillo](#) et al^{lxx} in an Cross-sectional analysis of data in 1998 for 1567 participants in The Gubbio Population Study (677 men and 890 women), found that diastolic blood pressure related directly to microalbuminuria ($P < .001$) when compared to systolic blood pressure.

RELATION BETWEEN DIASTOLIC BLOOD PRESSURE AND MICROALBUMINUREA

DBP	NO. Of Persons	MAU +	MAU -	% Of Positivity
< 80	3	0	3	0
81 - 90	73	16	57	21.9
91 - 100	21	6	15	28.6
> 100	0	0	0	0

Table No: 18

Chi-square value = 5.9, P value = < 0.05



RELATION BETWEEN DIASTOLIC BLOOD PRESSURE AND MICROALBUMINUREA

Our study showed a statistically significant difference between diastolic blood pressure and the occurrence of microalbuminuria. As the diastolic blood pressure increases, the number of percentage of study subjects who were positive for microalbuminuria was also increased.

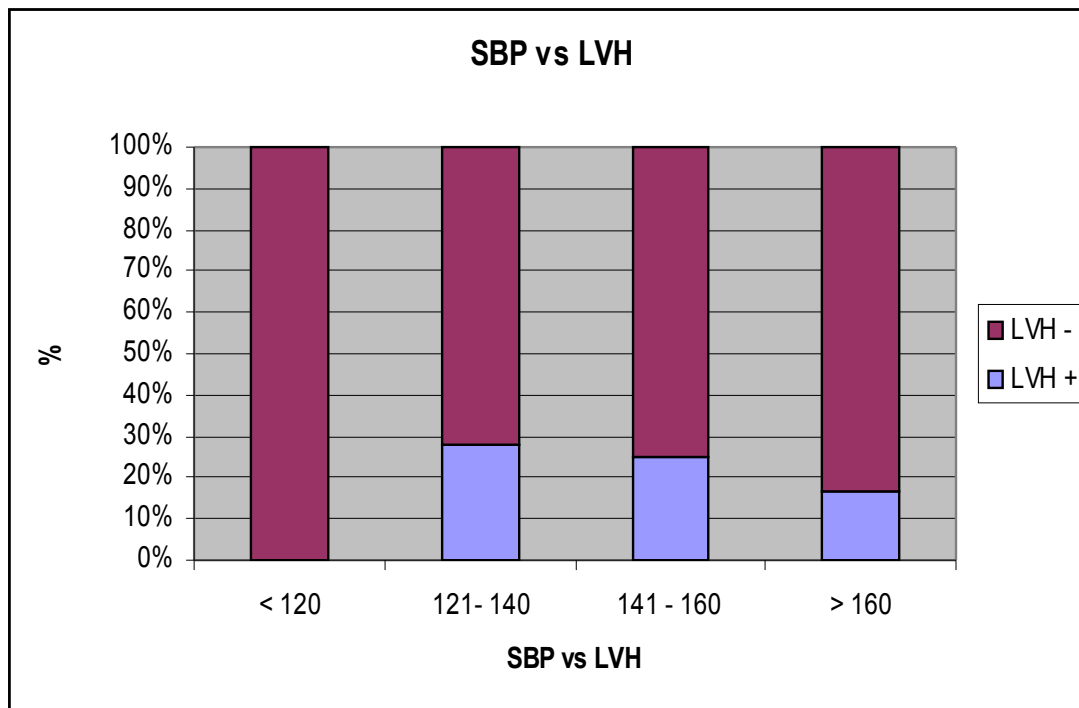
Roberto Pontremoli et al ^{lxxi} conducted a large clinical trial in 1997 with 787 patients (438 men, 349 women) with hypertension found a stronger correlation with diastolic blood pressure and microalbuminuria.

RELATION BETWEEN SYSTOLIC BLOOD PRESSURE AND LEFT VENTRICULAR HYPERTROPHY

SBP	Total No Of Persons	LVH +	LVH -	% Of Positivity
< 120	20	0	20	0
121- 140	43	12	31	27.9
141 - 160	28	7	21	25
> 160	6	1	5	16.6

Table No: 19

Chi-square value = 1.4, P value = > 0.05



RELATION BETWEEN SYSTOLIC BLOOD PRESSURE AND LEFT VENTRICULAR HYPERTROPHY

Out of 20 subjects who had left ventricular hypertrophy, 12 had systolic blood pressure between 121 to 140 and 7 had systolic blood pressure between 140 to 160. By applying chi square test it was not statistically significant.

J.C.N. Mbanya et al^{lxxii} conducted a cross sectional study in 2001 at a referral hospital in the city of Yaoundé (Cameroon) over a six-month period. It was found that diastolic blood pressure was significantly higher in the patients with ventricular hypertrophy, and shortening fraction was negatively correlated with diastolic blood pressure ($r = -0.40$; $p = 0.01$) but not with systolic blood pressure.

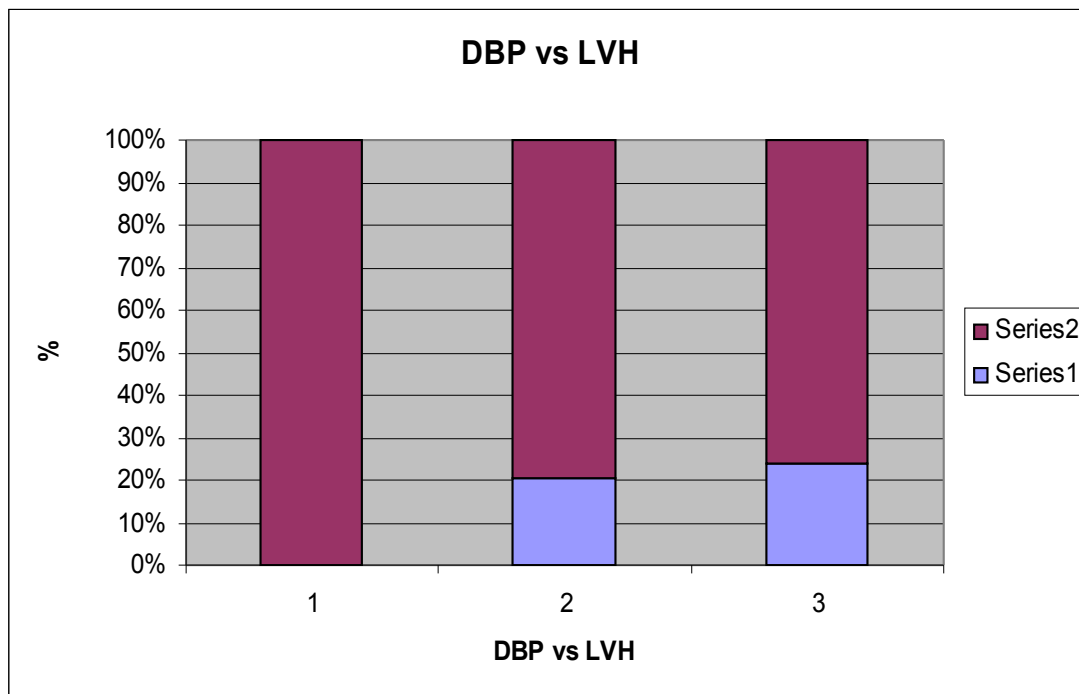
RELATION BETWEEN DIASTOLIC BLOOD PRESSURE AND LEFT

VENTRICULAR HYPERTROPHY

DBP	Total No Of Persons	LVH +	LVH -	% Of Positivity
< 80	3	0	3	0
81 - 90	73	15	58	20.5
91 - 100	21	5	16	23.8
> 100	0	0	0	0

Table No: 21

Chi-square value = 6.1, P value = < 0.05



RELATION BETWEEN DIASTOLIC BLOOD PRESSURE AND LEFT

VENTRICULAR HYPERTROPHY

Out of 21 study subjects of diastolic blood pressure between 91 to 100, 23.8 % had left ventricular hypertrophy were as it was only 20.5 % among the study subjects who had diastolic blood pressure between 81 to 90. There was a constant increase in the left ventricular hypertrophy Positivity with the increase in diastolic blood pressure and it was statistically significant.

J.C.N. Mbanya et al^{lxxiii} conducted a cross sectional study in 2001 at a referral hospital in the city of Yaoundé (Cameroon) over a six-month period. It was found that diastolic blood pressure was significantly higher in the patients with ventricular hypertrophy, and shortening fraction was negatively correlated with diastolic blood pressure ($r = -0.40$; $p = 0.01$) but not with systolic blood pressure.

CONCLUSION

In our study 22.7 % of the study subjects had microalbuminuria and 20.6 % had left ventricular hypertrophy. There was a strong statistical association between microalbuminuria and age, sex and diastolic blood pressure. Similarly the statistical association between left ventricular hypertrophy and diastolic blood pressure was strong. Both microalbuminuria and left ventricular hypertrophy were not statistically associated with body mass index and systolic blood pressure.

PROFORMA

Name :

Age :

Sex :

Address :

Duration Of Hypertension :

History of CAD,CKD,DM :

Treatment :

Blood Pressure :

Height :

Weight :

INVESTIGATIONS

Haemoglobin :

Total & Differential count :

Urine Sugar,Albumin&Deposits.

Blood Sugar(Fasting&Postprandial).

Blood Sugar:

Serum Creatinine:

Microalbuminuria and urine creatinine:

Left ventricular hypertrophy by ECG:

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Ethical committee approval

Ref.No.5028/MEII/2009

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Dated:03.06.2009.

Sub:Medical Education - Chengalpattu Medical College, Chengalpattu - Dr.D.Damodaran, Post Graduate Student in M.D.(General Medicine) - Admitted during the academic year 2007-2008 - Dissertation submitted for the Approval of the Human Ethical Committee Certificate for the study work entitled "A Hospital Based Cross Sectional Study on the Incidence of Micro Albumin Urea and Left Ventricular Hypertrophy among Hypertensive Patients" - Approved - Orders - Issued.

Ref:Application dated:20.02.2009 of the individual.

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Dr.D.Damodaran, Post Graduate Student in M.D.(General Medicine) of this college has submitted a Dissertation for the approval of the Human Ethical Committee Certificate for the study of "A Hospital Based Cross Sectional Study on the Incidence of Micro Albumin Urea and Left Ventricular Hypertrophy among Hypertensive Patients". The study of the above Dissertation work has been approved by the Human Ethical Committee of the Chengalpattu Medical College, Chengalpattu which is forwarded herewith for further course of action.

Further, as per the Regulation of the Tamil Nadu Dr.M.G.R.Medical University, Chennai-32 with regard to submission of 4 Copies of Dissertation with C.D.(4 Copies) for the Post Graduate Degree Course, the Dissertation shall be a bound volume of minimum 50 pages and not exceeding 75 pages of typed matter(Double line spacing and one side only)excluding certification, acknowledgements, annexures and Bibliography". The name of the candidate should not be found anywhere in the dissertation except in the Certificate. The Dissertation should be forwarded through the Unit Chief/H.O.D. as per the colour(LIGHT GREEN)communicated by the University.

Deenathir
D E A N Incharge. 5/6/09

Encl.:Copy of the Dissertation study work.

To
✓ Dr.D.Damodaran, Post Graduate Student in M.D.(General Medicine),
.. through the Head of the Department of General Medicine.

Copy to:

- 1)The Controller of Examinations,
The Tamil Nadu Dr.M.G.R.Medical University, Chennai-32.
- 2)The Head of the Department of General Medicine,
Chengalpattu Medical College, Chengalpattu.
- 3)MEI Section through the Office Superintendent(ME).
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