# A STUDY OF PREVALENCE OF

# MICROALBUMINURIA IN

# NON DIABETIC NON HYPERTENSIVE CAD


**DISSERTATION SUBMITTED FOR M.D.DEGREE**

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**THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY**

**CHENNAI, TAMILNADU**

# CERTIFICATE

This is to certify that this dissertation entitled **"PREVALENCE OF MICROALBUMINURIA IN NON DIABETIC NON HYPERTENSIVE CAD** is a bonafide record of work done by **Dr. KHADHAR MOHAMED SARJUN BASHA**.S under my guidance and supervision in Tirunelveli Medical College Hospital during the period of his Post Graduate Study for M.D.(General Medicine) from 2008 – 2011.

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# DECLARATION

I solemnly declare that the dissertation titled **"Study of prevalence of microalbuminuria in non-diabetic non-hypertensive CAD"** is done by me at Tirunelveli Medical College hospital, Tirunelveli under the guidance and supervision of Prof. Dr.J.Kaniraj Peter, M.D., The dissertation is submitted to The Tamilnadu Dr. M.G.R.Medical University towards the partial fulfilment of requirements for the award of M.D. Degree (Branch I) in General Medicine.

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**CERTIFICATE OF APPROVAL**

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# LIST OF ABBREVATIONS

| | | |
|---|---|---|
| CAD | → | Coronary artery disease |
| CVD | → | Cardiovascular disease |
| CRP | → | C – reactive protein |
| EDRF | → | Endothelial derived relaxing factor |
| ELISA | → | Enzyme linked immunosorbant assay |
| HDL | → | High density lipoprotein |
| IHD | → | Ischemic heart disease |
| LDL | → | Low density lipoprotein |
| TGL | → | Triglycerides |
| MCP-1 | → | Monocyte chemoattractant protein – 1 |
| PDGF | → | Platelet derived growth factor |
| UAE | → | Urine albumin excretion |
| VCAM-1 | → | Vascular cell adhesion molecule-1 |
| TNF | → | Tumour necrosis factor |
| IL-1 | → | Interleukin – 1 |
| RIA | → | Radio immunoassay |
| PGI2 | → | Prostaglandin I2 |
| n | → | Frequency |

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# INTRODUCTION

IHD which has an estimated prevalence of 6–9 % in general population in India may become the leading cause of mortality and morbidity in India by year 2025. Since the pioneering work of Framingham study, many prospective and clinical studies have identified series of independent risk factors for ischemic heart disease among which age, male gender, a positive family history of premature atherosclerotic disease, cigarette smoking, diabetes mellitus, hypertension, dyslipidemia, obesity, physical inactivity are traditional risk factors.

The interest in improving cardiovascular risk assessment, resulting from a better understanding of the pathogenesis of atherosclerosis and identification of new targets for anti-atherosclerotic drug therapy has stimulated the search for novel risk factors. One such novel risk factor is microalbuminuria which has emerged as an independent and robust risk factor.

Microalbuminuria is a marker of widespread vascular damage in diabetic as well as non-diabetic patients. However more and more evidence is accumulating that microalbuminuria is an important cardiovascular risk factor even in general population. Its early detection helps in preventing the progression of cardiac decompensation.

Aggressive treatment of microalbuminuria is associated with improved renal and cardiac functions.

The present study is being conducted to determine the prevalence of microalbuminuria in non-diabetic and non-hypertensive ischemic heart disease patients and its association with other known cardiovascular risk factors.

# AIM OF STUDY

1. To find out the Prevalence of microalbuminuria in non-diabetic and non-hypertensive patients with Coronary heart disease .
2. To find out the association between microalbuminuria and smoking.
3. To find out the association between microalbuminuria and hyperlipidemia.

# REVIEW OF LITERATURE

## IHD

Ischemia refers to a lack of oxygen due to inadequate perfusion of the myocardium, which causes an imbalance between oxygen supply and demand. The most common cause of myocardial ischemia is obstructive atherosclerotic disease of epicardial coronary arteries.

### Pathogenesis of Atherosclerosis:

Marchand introduced the term "Atherosclerosis" describing the association of fatty degeneration and vessel stiffening[1]. This process affects medium and large sized arteries and is characterized by patchy intramural thickening of the sub-intima that encroaches on arterial lumen.

Atherosclerotic plaque has three principal components. The first is cells including smooth muscle cells, macrophage and other leukocytes. The second component is extracellular matrix including collagen, elastic fibers and proteoglycan. The third component is intracellular and extracellular lipid. These components occur in varying proportion and configuration in different lesions

**Initiation of Atherosclerosis:**

**Endothelial Injury:**

The luminal surface of a normal artery is covered with a monolayer of endothelial cells attached to sub endothelial matrix. The endothelial cells also provide the artery with nonthrombogenic surface. Injured endothelial cells appear morphologically different from normal endothelial cells; unlike normal cells, they are typically not aligned in the direction of blood flow and they have fewer intracellular attachments resulting in increased permeability[2]. Injured endothelial cells are also more thrombogenic than the normal endothelial cells because of their diminished production of PGI2 and EDRF-NO[3]. Injured endothelial cell promote vascular smooth muscle cell migration and proliferation by releasing less EDRF-NO and by secreting PDRF and endothelial-1[4]. Finally injured endothelial cell promote the recruitment of macrophage by secreting Monocyte Chemoattractant Protein-1(MCP-1) and by expressing cell surface receptors or selectins to which monocyte can bind[5, 6, 7].

**Role of Inflammation**:

Following endothelial dysfunction, endothelial cells express VCAM-1 which binds monocyte and T-lymphocyte. The monocyte migrates between extracellular matrix to localize in intima and transforms into macrophage and become lipid laden foam cells. Macrophage produces IL-1, TNF and MCP-1, which further cause migration of leukocytes into arterial wall. The lipoproteins that enter into the intima of arteries associate with glycosaminoglycans of arterial extracellular matrix, an interaction that slows egress of these lipid-rich particles from the intima. The lipoprotein undergoes oxidative modification to form oxidized LDL which are preferentially endocytosed by macrophage through macrophage scavenger receptors. Thus the monocyte attachment to endothelium, migration into the intima and maturation to form lipid-laden macrophage represents **key steps in formation of the fatty streak**.

**FIBROTIC LESION FORMATION:**

Fatty streak are converted to fibrotic lesion by forming fibrotic cap composed of smooth muscle cells. The PDGF causes migration of

smooth muscle cells from media into the intima. TGF-BETA potentially stimulates interstitial collagen production by smooth muscle cell thus leading to formation of fibro fatty lesion.

**ADVANCED LESION FORMATION:**

The final stage in the development of an atherosclerotic lesion is the conversion of fibrotic lesion into advanced lesion, a lesion in which a thrombus has formed subsequent to either plaque ulceration or intraplaque hemorrhage.[4, 8,9,10] The relative paucity of smooth muscles in advanced atheroma may result from the predominance of cytostatic mediators such as TGF-ALPHA or INF GAMMA and from smooth muscle cell apoptosis.

**EPIDEMIOLOGY AND PREVENTION OF ATHEROSCLEROSIS:**

Atherosclerosis is a multifactorial disease where age of onset and progression are strongly influenced by inborn and acquired risk factors. Since the pioneering work of the Framingham study, many prospective population and clinical studies have identified a series of independent risk factors for myocardial infarction, stroke and PVD, among which the

pre-existence of atherosclerotic vascular disease, age, male gender, a positive family history of premature atherosclerotic disease, smoking, diabetes mellitus, hypertriglyceridemia, hypercholesterolemia, hypertension and low HDL cholesterol are considered as

**classical risk factors**

In a given individual, the presence of a single risk factor has a low positive predictive value. In contrast, the presence of several moderately expressed risk factors can produce a significant increase in cardiovascular risk.

Therefore at present the most advanced strategy for coronary risk assessment is to combine the information of several risk factors in algorithm or scores. Current international guidelines base their recommendations for the indication of hypolipidemic drug treatment in clinically asymptomatic patients (primary prevention) on the estimation of global risk.

Thousands of cross-sectional case control studies have identified hundreds of clinical, biochemical or genetic markers that showed statistically significant association with coronary heart disease, stroke,

PVD. Most of the associations were either not reproducible in other studies or not independent of clinical risk factors. However some of these emerging risk factors, turned out to be robust and independent. Currently there is an intense discussion whether they should be introduced into routine risk assessment. This specially concerns lipoprotein (a), CRP, fibrinogen, homocysteine and microalbuminuria

**MICROALBUMINURIA:**

A consensus conference in 1995 defined microalbuminuria in individual with diabetes as a abnormal urinary excretion rate of albumin between the range 20 – 200 µg/min or 30 – 299 mg/day.[11] This is still the definition used today and is applicable to all people regardless of associated pathological condition.

**Prevalence of microalbuminuria:**

Type 1 diabetes– 4 - 40%, Type 2 diabetes – 30 - 40%, hypertension – 7- 40%, Non – diabetic – 5 - 7%.

Epidemiologic and experimental date shows that microalbuminuria is associated with an increased risk for all cause and cardiovascular mortality, cardiac abnormality, cardiovascular disease and periodically PVD.

In Heart outcome prevention evaluation (HOPE) study, UAE predicted mortality in patients who were at cardiovascular risk (55 years of age with CVD (or) DM and at least one other cardiovascular risk factor).[12] All cause mortality was 9.4% among patient without microalbuminuria versus 18.2% among those with microalbuminuria [Relative Risk(RR)]2.09% ; 95% confidence interval] . A linear relationship was also observed between microalbuminuria level and cardiovascular events, extending below the traditional microalbuminuria threshold.

The presence of microalbuminuria also seems to predict all cause mortality in the personal population. This was initially shown in Prevention of renal and vascular end stage disease (PREVEND STUDY), in which inhabitant of Gramingam, The Netherland, who were aged 28 - 75 years were sent a questionnaire and a vial to collect an early-morning urine sample for measurement of UAE. A total of 40,548 participants, who were followed for 26 years were included in an analysis of mortality by baseline UAE. A clear positive relationship was observed

between UAE and all cause cardiovascular and non cardiovascular mortality.

In the Steno hypothesis put forward by Deckert et al, albumin leakage into the urine is a reflection of widespread vascular damage. This hypothesis links impaired vascular endothelial function with vascular leakage of albumin. The kidney thus would become a window to the vasculature.

In the non-diabetic American Indians of the strong heart study, the prevalence of coronary heart disease, peripheral vascular disease and stroke was associated with microalbuminuria (ACR between 30 and 300mg/g) independent of age, diabetes, systolic blood pressure, cholesterol and fibrinogen.[51, 52]

Stronger evidence for microalbuminuria as an independent risk marker was produced by Damsgaard et al.[53]who showed prospectively a threefold higher death rate in normoglycemic individuals where albuminuria was equal to or above the median (>7.52 microgram/min).

**Table 1: Clinical studies reporting the risk associated with positive microalbuminuria results**

| No | Study | Microalbuminuria Definition | Population | Risk associated with positive microalbuminuria results (95% CI) |
|---|---|---|---|---|
| **(A) Prospective studies** | | | | |
| 1 | Prospective studies HOPE[17] | ACR >=2 mg/mmol in a First morning spot urine sample | Subjects at high cardiovascular risk >=55 yr with CVD or with diabetes +>=1 CVD risk factor; n = 9043) | All-cause mortality: RR 2.09 (1.84 to 2.38) |
| 2 | PREVEND[18] | UAE 20 to 200 mg/L in an early morning spot urine sample | Residents of Groningen, The Netherlands, 28 to75 yr (n = 40,548) | Cardiovascular death: RR 1.29(1.18 -1.40) Non cardiovascular death: RR 1.12 (1.04 to 1.21) |
| 3 | PREVEND[19] | UAE 30 to 300 mg in a 24-h urine Sample | Residents of Groningen, the Netherlands, 28 to 75yr (n = 7330) | All-cause mortality: HR 3.3 (1.5 to 7.1) for patients with ST-T segment changes with microalbuminuria versus 0.9 (0.4 to 1.9) for ST-T segment changes alone Cardiovascular death: HR 10.4 (2.5 to 43.6) for patients with ST-T segment changes with |

| | | | | |
|---|---|---|---|---|
| | | | | microalbuminuria versus2.7(0.6 to 12.3) for ST-Tsegment changes alone |
| 4 | Hoorn Study[20] | ACR >= mg/mmol in a first morning spot urine sample | Population-based: White individuals, 50 to 75 yr (n = 631) | Cardiovascular death: RR3.22 (1.28 to 8.06) All cause mortality: RR 1.70(0.86 to 3.34) All-cause mortality in patients with hypertension: RR 2.87 (1.22 to 6.33) |
| 5 | HUNT[21] | ACR >=76 mg/mmol (60th percentile) in a first morning urine sample | Non- diabetic, non-hypertensive residents of Nordrondelag Norway, >=20 yr(n=2089) | All-cause mortality: RR 2.3 (1.0 to 5.4) |
| 6 | EPIC-Norfolk[22] | ACR 2.5 to 25 mg/mmol in a random spot urine sample | Residents of Norfolk, UK, 40 to 79 yr (n =20,911) | All-cause mortality: HR1.48 (1.20 to 1.79)Cardiovascular death: HR 2.03 (1.55to 2.67) Fatal stroke: HR 1.58 (1.10 to 3.0)Coronary heart disease death: HR 2.01 (1.40 to 2.90 |
| 7 | EPIC-Norfolk[23] | ACR 2.5 to 25 mg/mmol in a random spot urine sample | Residents of Norfolk, UK, 40 to 79 yrs(n=23,630) | Stroke: HR 1.49 (1.13 to 2.14) |

| | | | | |
|---|---|---|---|---|
| 8 | Danish[24] MONICA | ACR >0.65 mg/mmol (>90thpercentile) in a first-morning spot urine | Population-based: Individuals without IHD, renal failure, UTI, diabetes | Ischemic Heart Disease: RR 2.3 (1.3 to 3.9) |
| 9 | Shibata Study[25] | Positive albumin dipstick test | Residents of Shibata, Japan, >40 yr (n =2651) | Stroke: RR in men 2.5 (1.1 to 5.7) |
| 10 | Portland[26] Study | UAE 20 to 200 mg/L in a first morning spot urine sample) | Older residents of Portland, Oregon, with previous stroke or transient ischemic attack (n =121) | Recurrent stroke: HR 4.9(1.4 to 17.6) |
| 11 | Slowik et al.[27] | UAE 30 to 300 mg in a 24-h urine Sample | Patients admitted within 24 h of *first ischemic stroke (n = 60)* | Mortality: OR 6.0 (1.3 to27.7) |
| **(B) Cross-sectional studies** | | | | |
| 12 | Cross-sectional[28] studies Zander *et al.* | UAE 20 to 200 mg/L in an early morning spot urine sample | Patients with type 2 Diabetes.(n = 1060) | PAD: OR 2.1 (1.4 to 3.2) |
| 13 | PREVEND[29] | UAE 30 to 300 mg in a 24-h urine sample | Non-diabetic residents of Groningen, the Netherlands, 28 to 75yr | Electrocardiographic abnormalities: Infarct patterns: OR 1.61 (1.12 to 2.32) Major ischemia: OR |

| | | | | |
|---|---|---|---|---|
| | | | (*n* = 7579) | 1.43 (1.08 to 1.91) Minor ischemia: OR 1.32 (1.03 to 1.68) |
| 14 | PREVEND[30] | UAE 30 to 300 mg in a 24-h urine sample | Non-diabetic residents of Groningen, the Netherlands, 28 to 75 yr(*n* = 6669) | PAD : OR 0.98(0.68 to 1.41) in multivariate analysis |

**Table 2 : List of the available cross-sectional (C) and prospective (P) studies reporting data about non-diabetic microalbuminuria and/or urinary albumin excretion as a covariate and/or predictor of cardiovascular events**

| No | Author/ref. | Date | N | Design | End-point | Population |
|---|---|---|---|---|---|---|
| 1 | Yudkin[42] | 1988 | 187 | C/P | Major, minor ECG changes, history of MI and angina peripheral vascular disease | Diabetic, glucose-intolerant and non-diabetic subjects |
| 2 | Haffner[43] | 1990 | 316 | C | Self-reported MI | Non-diabetic subjects |
| 3 | Damsgaard[44] | 1990 | 216 | P | Total mortality | Non-diabetic subject |
| 4 | Winocour[45] | 1992 | 447 | C | ECG abnormalities | Diabetic and non-diabetic subjects |
| 5 | Damsgaard[46] | 1992 | 216 | P | Total abnormality | Non-diabetic subjects |
| 6 | Damsgaard[47] | 1993 | 216 | P | Total mortality | Non-diabetic subjects |
| 7 | Gould MI[48] | 1994 | 959 | C | M I, angina, peripheral | Non-diabetic subjects |

| | | | | | | |
|---|---|---|---|---|---|---|
| | | | | | vascular disease | |
| 8 | Howard[49] | 1995 | 4549 | C | Definite MI and ischemic heart disease | Diabetic and non-diabetic American Indians |
| 9 | Kuusisto[50] | 1995 | 1069 | P | Fatal and non-fatal coronary heart disease | Non-diabetic subjects |

**Pathophysiology of microalbuminuria:**

The intimate relationship between low level albumin excretion and vascular permeability makes urinary albumin excretion highly sensitive to the presence of any inflammatory process, including cardiovascular disease. The kidney is ideally placed to amplify any small changes in systemic vascular permeability. The glomeruli receive 25% of cardiac output. Of the 70kg of albumin that pass through the kidneys every 24hours, less than 0.01 % reaches the glomerular ultra filtrate (i.e., less than 7g/24hrs ) and hence enter the renal tubules.[32,33]

Almost all the filtered albumin is reabsorbed by the proximal tubule through a high affinity, low capacity endocytotic mechanism, [34] with only 10-30mg/24hrs appearing in urine. Assuming that 7g of

albumin is filtered every 24hrs; a 1% increase in systemic vascular permeability in response to an inflammatory stimulus would results in an additional 70mg of albumin passing into the filtrate. Since tubular mechanisms for albumin reabsorption are near saturation, urinary albumin excretion would increase from a maximum of 30 to approx 100mg/24hr.

Glomerular permeability to albumin is dependent on endothelial charge selectivity as well as size selectivity. The negative charge conferred on glomerular membrane by its constituent glycoprotein plays a role in restricting the permeability of anionic proteins. Loss of glomerular charge selectivity has been found in both diabetic and non-diabetic population with microalbuminuria.[35, 36]. Similarly, alteration in the fraction of plasma filtered by glomeruli due to changes in blood pressure and intra glomerular pressure regulation may also result in relatively large changes in urinary albumin excretion.

Microalbuminuria may be a marker of generalized vascular disease with arterial endothelial dysfunction being involved in the pathogenesis of atherothrombotic vascular disease.

In an individual with microalbuminuria who does not have diabetes, both endothelial dysfunction and alteration in ECM contribute to increase in vascular permeability and ultimately promote the atherosclerotic process. Defective endothelial permeability permits lipid influx into the vessel wall causing atherosclerotic changes.[37,38,39] An impairment of normal endothelial antithrombotic and vasodilatory properties is a main factor in atherogenesis.[40] Endothelial dysfunction seems to play a key role in non-diabetic glomerulosclerosis and atherosclerosis. Microalbuminuria is also associated with biochemical indices of endothelial dysfunction such as increased VWF and increased platelet adhesiveness. In both non-diabetic and diabetic patients, it was observed that participants with microalbuminuria had higher plasma level of VWF antigen than patient with normal albumin excretion.

Microalbuminuria and cardiovascular disease may be linked not by a common risk factor but rather by a common pathophysiological process. One is that, dysfunction of the vascular endothelium causes both microalbuminuria and cardiovascular disease.[54,55] Generalized endothelial dysfunction now is considered a transducer of atherogenic risk factor

and is thought to play an important role in both the initiation and progression of atherosclerosis. Abnormalities in the endothelial glycocalyx may contribute to microalbuminuria but also have been implicated in pathogenesis of atherosclerosis, thus providing a potential direct link between microalbuminuria and cardiovascular disease.

Microalbuminuria is a marker of widespread vascular damage in diabetic as well as non-diabetic patient. Microalbuminuria is also associated with presence of other well recognized risk factor for CAD. Its early detection helps in preventing the progression of cardiac decompensation. Aggressive treatment of microalbuminuria is also associated with improved renal and cardiac functions.

An unfavorable pattern of lipid abnormality already exists at the stage of microalbuminuria when renal function is still normal. The abnormality consists of elevated total cholesterol and VLDL and reduced HDL.

Another interesting theory is that some individuals are born with varying degree of vascular function within a physiologic range and therefore excrete a variable amount of microalbuminuria. This inherent

variability of the vascular state as determined by urine microalbumin excretion may be associated with susceptibility to subsequent organ damage. This could also explain why microalbuminuria is a predictor of not only CVD but also new-onset hypertension and diabetes. If this proves to be the case, then it may be desirable to identify these individuals to consider early interventional strategies to provide primary prevention.

Microalbuminuria is associated with increased cardiovascular disease as most commonly microalbuminuria reflects pathophysiological process predisposing to atherothrombosis. Atherothrombosis is low grade inflammatory disease of vessel wall characterized by endothelial dysfunction and increased transendothelial passage of leucocytes. This feature could be the pathogenic factor linking microalbuminuria to cardiovascular disease.[56]

**Screening for Microalbuminuria**:

Urinary albumin concentration can be quantified by a number of antibody based assay. These include Radioimmunoassay, Laser neplometry, Immunoturbimetry and ELISA.

Although the gold standard test for microalbuminuria is the RIA, other tests are generally sensitive enough for clinical practices.

Immunoturbidimetric assay of microalbuminuria is depending on the turbidity of a solution when albumin in sample of urine reacts with a spectrophotometer and the absorbency is proportional to the albumin concentration. Recently an assay called HPLC was used to measure unreactive intact albumin that is not recognized by immunologic methods. However, the clinical significance of this method is not fully understood. Whichever method used, the same assay should be used when comparing the albuminuria results over time for a patient. The choice of assay used is largely determined by issues of accuracy, cost & convenience.

One of the most extensively investigated methods to screen for microalbuminuria is the immunometric dipstick micraltest, microbumin test. Because these tests are semi-quantitative, a true co-efficient of variation cannot be determined. In general these albumin reagent strip test are more sensitive than standard dipsticks, but they also have a relatively high rate of false-positive results. For the diagnosis of

microalbuminuria, a 24 hour urine collection is the gold standard. Because of the effort involved, it is not the method of choice for screening. The second best is a timed overnight urine collection. Again because this requires collection of urine over a given time period, this may be acceptable for screening specific groups such as patients with diabetes or hypertension but less feasible for population screening. The next best is a first morning void urine sample. This has advantages over a spot-urine sample because it is always performed at the same time of the day and it is least influenced by hydration status and physical activity of the patient, reducing the variability that is caused by these factors.

The albumin-to-creatinine ratio (ACR) in random or timed urine collection may be more quantitative than a simple dipstick screening procedure but has a number of limitations. For eg:

-Obtaining ACR on morning first void sample may underestimate 24hour protein excretion because of reduction in proteinuria that occurs normally at night.

-The fact that urinary creatinine must be measured in addition to albumin introduces another source of error.

-Urine creatinine concentration is extremely variable, so very different ratios can be obtained in individuals with similar protein excretion rates.

-Moreover creatinine excretion in the urine depends not only on gender but also on age and race.[57, 58]

This may explain why urinary albumin concentration from a spot sample performs equally well for definition of microalbuminuria as albumin-to-creatinine ratio.[59]

**Table 3 :Classification of abnormal urinary albumin excretion**:

| | 24 Urine Albumin (mg/24 h) | Overnight Urine Albumin (microg/min) | Spot Urine | | | |
|---|---|---|---|---|---|---|
| | | | Albumin (mg/L) | Albumin/Creatinine Ratio | | |
| | | | | G* | mmol/g | mg/g |
| Normal | <15 | <10 | <10 | M | <1.25 | <10 |
| | | | | F | <1.75 | <15 |
| High normal | 15 to <30 | 10 to <20 | 10 To<20 | M | 1.25 To <2.5 | 10 To<20 |
| | | | | F | 1.75 To<3.5 | 15 to<30 |
| Microalbuminuria | 30 to<300 | 20 to <200 | 20 To<200 | M | 2.5 to 25 | 20 to<200 |
| | | | | F | 3.5 to 35 | 30 to<300 |
| Macroalbuminuria | >300 | >200 | >200 | M | >25 | >200 |
| | | | | F | >35 | >300 |

G* - Gender

# MATERIAL AND METHODS

The study was carried out in the Department of General Medicine, Tirunelveli medical college hospital, Tirunelveli.

**Study Design**:

Randomized hospital based study.

**Study subjects**:

50 non diabetic and non hypertensive CAD patients were selected from those admitted in General Medicine wards and Cardiology Department during the period March 2010 to November 2010

**Inclusion Criteria**

1. The diagnosis of coronary heart disease was based on 12 lead ECG, cardiac enzyme estimation, echocardiography and rose questionnaire.
2. Normal values of Total cholesterol <200 mg/dl, HDL >40mg/dl [Male] >50mg/dl [Female], TGL ≤150mg/dl, LDL <130 has been taken as the cut-off value for this study.

**Exclusion Criteria**

1. Hypertension defined as JNC VII and patients who were taking anti hypertensive drugs.
2. Diabetes mellitus defined as per ADA criteria
3. Patients with urinary tract infection, congestive cardiac failure, seizures and fever
4. Urine showing - macroalbuminuria.

**Methods of collection of DATA**

This was hospital based study involving 50 patients.

Data collection was by clinical history, examination and investigations such as blood investigations- fasting blood sugar, blood urea, serum creatinine, serum electrolytes, fasting lipid profile, urine albumin and deposits, chest X ray PA view, ECG, Echo are done and reports were analyzed thoroughly.

Microalbuminuria was detected by immunoturbidimetric method in early morning first voided urine.

The reference range of normal microalbuminuria is 0 – 20 mg/l

Early morning first voided urine after discarding initial 10 – 20 ml urine was collected using sterile plastic container and investigation was done within 2 hours of collection of urine.

**Statistical Analysis:**

The DATA is presented as Mean ± 2SD. The limits of significance were calculated using SPSS version 13 software.

**Statistical Software:**

Microsoft Word and Microsoft Excel were used to generate graphs, tables, etc.

## OBSERVATIONS AND RESULTS

**RESULTS:**

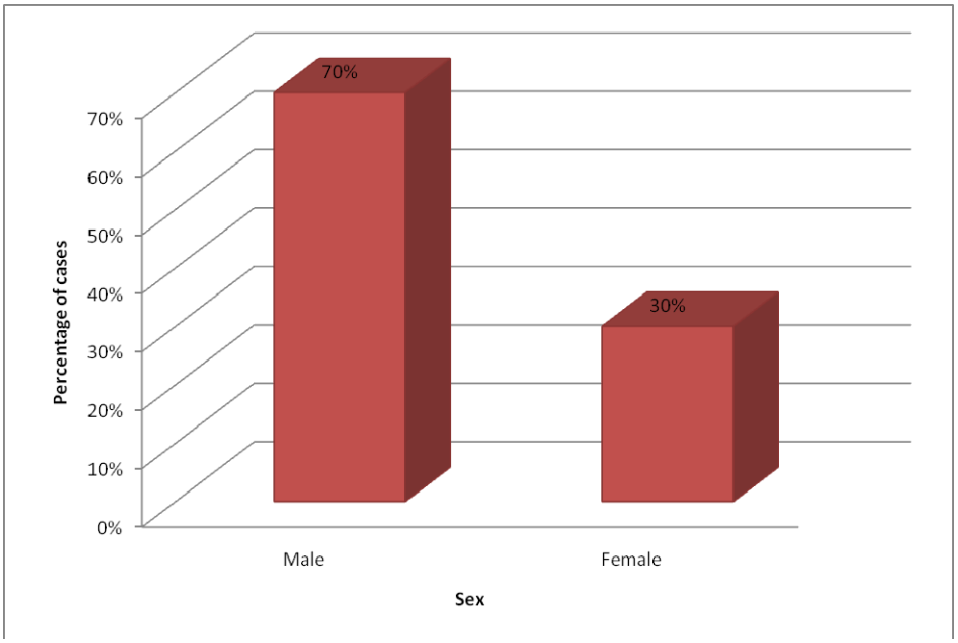
Fifty patients fulfilling the criteria for the study were included. The study was done over a period of nine months. Data was collected as per the proforma attached

**1. Sex distribution:**

In the present study, out of 50 patients, 35 were males and 15 were females.

**Table 4: Sex distribution in study subjects**

| Sex | No. of patients | Percentage |
|---|---|---|
| Male | 35 | 70% |
| Female | 15 | 30% |




M: F ratio = 2.33: 1 in this study

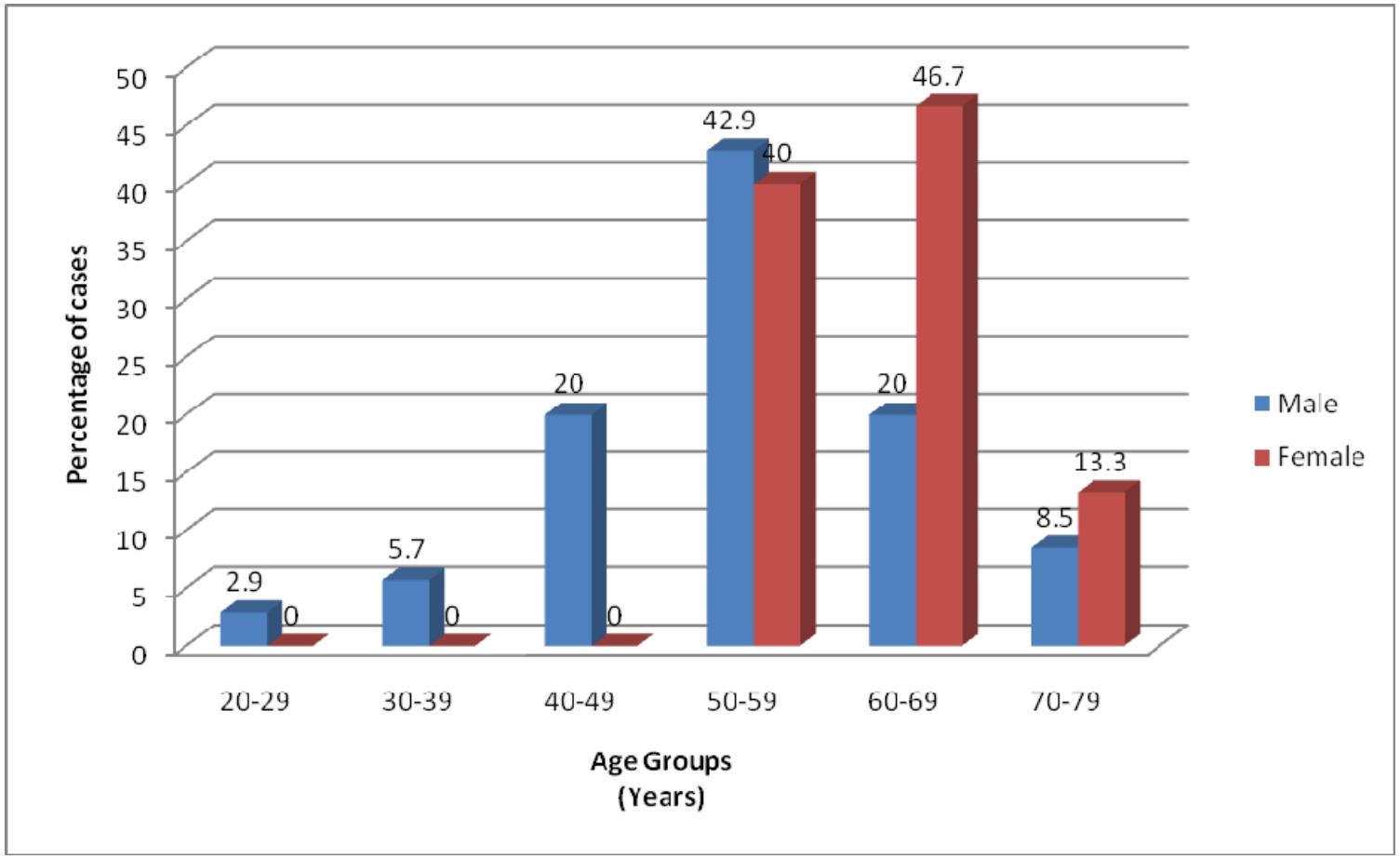
**2. Age and sex wise distribution of the study subjects:**

**Table 5: Age and sex wise distribution of the study subjects**

| Age Groups (Years) | Sex | | | | Total | |
|---|---|---|---|---|---|---|
| | Male | | Female | | | |
| | n | % | n | % | n | % |
| 20-29 | 1 | 2.9 | - | - | 1 | 2 |
| 30-39 | 2 | 5.7 | - | - | 2 | 4 |
| 40-49 | 7 | 20 | - | - | 7 | 14 |
| 50-59 | 15 | 42.9 | 6 | 40.0 | 21 | 42 |
| 60-69 | 7 | 20 | 7 | 46.7 | 14 | 28 |
| 70-79 | 3 | 8.5 | 2 | 13.3 | 5 | 10 |
| Total | 35 | 100 | 15 | 100 | 50 | 100 |
| Mean± SD | 53.8± 11.6 | | 60.5± 6.7 | | 55.8± 10.7 | |
| Significance | t=2.099 and P <0.05 | | | | | |

The mean age of the study population was 55.8 ± 10.7 with the range of 26 – 75years. The median age was 57years. The mean age for males was 53.8±11.6years and the same of females was 60.5 ± 6.7years.The difference of mean was statistically significant (P <0.05).This interpretation reveals that males were affected by CAD earlier than females

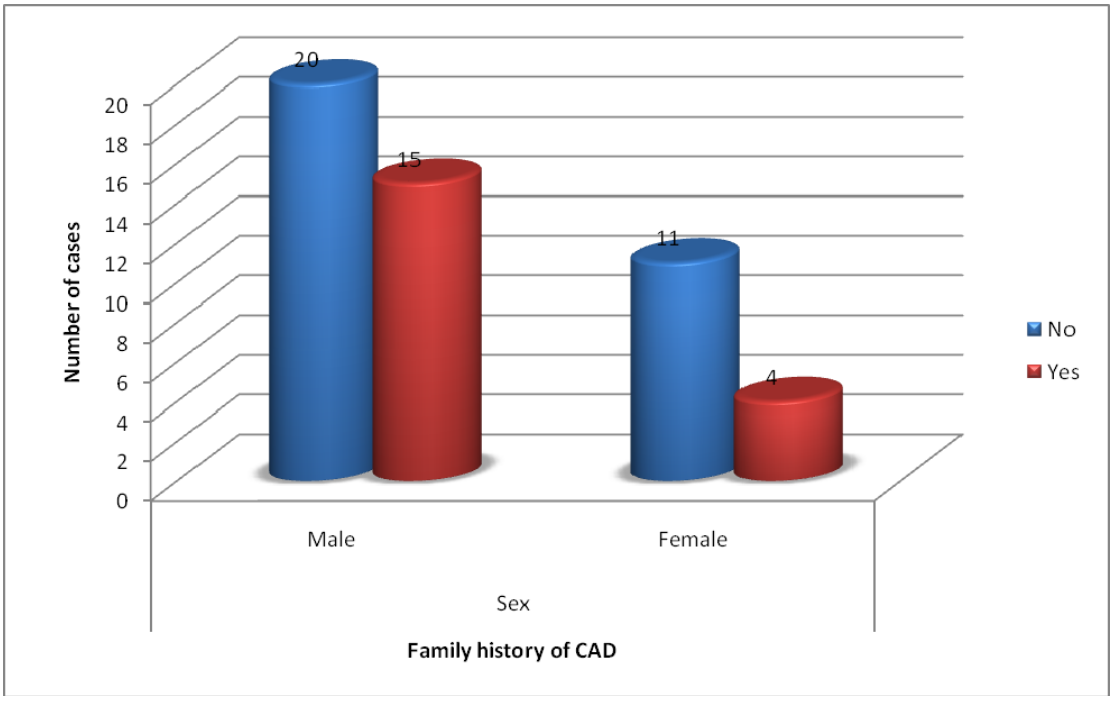
**Age and sex wise distribution of the study subjects**



### 3. Family history of ischemic heart diseases:

**Table 6: Family history of ischemic heart disease in study subjects**

| Family history of IHD | Sex | | Total |
|---|---|---|---|
| | Male (n=35) | Female (n=15) | |
| No | 20 | 11 | 31 |
| Yes | 15 | 4 | 19 |




Family history of IHD was present in 38%of the study subjects.

**4. Sex wise classification of study subjects according to the smoking habit**

**Table 7: Sex wise classification of study subjects according to the smoking habit**

| History | Sex | | | | Total | |
|---|---|---|---|---|---|---|
| | Male (n=35) | | Female (n=15) | | | |
| | n | % | n | % | n | % |
| Smokers | 33 | 94.3 | Nil | Nil | 33 | 66 |
| Non smokers | 2 | 5.7 | 15 | 100 | 17 | 34 |
| Total | 35 | 100 | 15 | 100 | 50 | 100 |

Among the males 94.3% were smokers and 5.7% were non smokers.100% of women were non smokers.

**5.Sex wise classification of study subjects according to family history**

**Table 8: Sex wise classification of study subjects according to family history**

| History | Male | | Female | | Total | |
|---|---|---|---|---|---|---|
| | n | % | n | % | n | % |
| Yes | 15 | 42.8 | 4 | 26.6 | 19 | 38 |
| No | 20 | 57.1 | 11 | 73.3 | 31 | 62 |
| Total | 35 | 100 | 15 | 100 | 50 | 100 |

**6.Physiological ,biochemical profile & microalbuminuria of the study subjects**

**Table 9:Physiological, biochemical profile and microalbuminuria of the study subjects**

| Variables | Mean | SD | Median | Mode |
|---|---|---|---|---|
| Systolic BP | 115.6 | 9.5 | 120 | 110 |
| Diatolic BP | 74.8 | 6.5 | 80 | 80 |
| Fasting blood sugar | 100.0 | 8.6 | 100 | 100 |
| Total CHO | 178.6 | 26.8 | 178.5 | 175 |
| TGL | 144.5 | 51.1 | 137 | 110 |
| HDL | 42.6 | 3.8 | 42.5 | 40 |
| LDL | 123 | 24.8 | 120 | 120 |
| Microalbuminuria | 35.9 | 22.2 | 30 | 30 |

The above table shows description of the physiological measures.The Mean±SD, Median, Mode of each variables were represented. In this study, the major dyslipidemia was increased TGL, increased LDL & decreased HDL

**7.Comparison of smoking habit with the age of study subjects**

**Table 10 :Comparison of smoking habit with the age of study subjects**

| Category | Yes | | | No | | | Difference of mean | t | Degree of freedom | P |
|---|---|---|---|---|---|---|---|---|---|---|
| | n | Mean age | SD | n | Mean age | SD | | | | |
| Smoking habit | 33 | 52.9 | 11.3 | 17 | 61.5 | 6.8 | 8.6 | 2.891 | 48 | <0.01 |

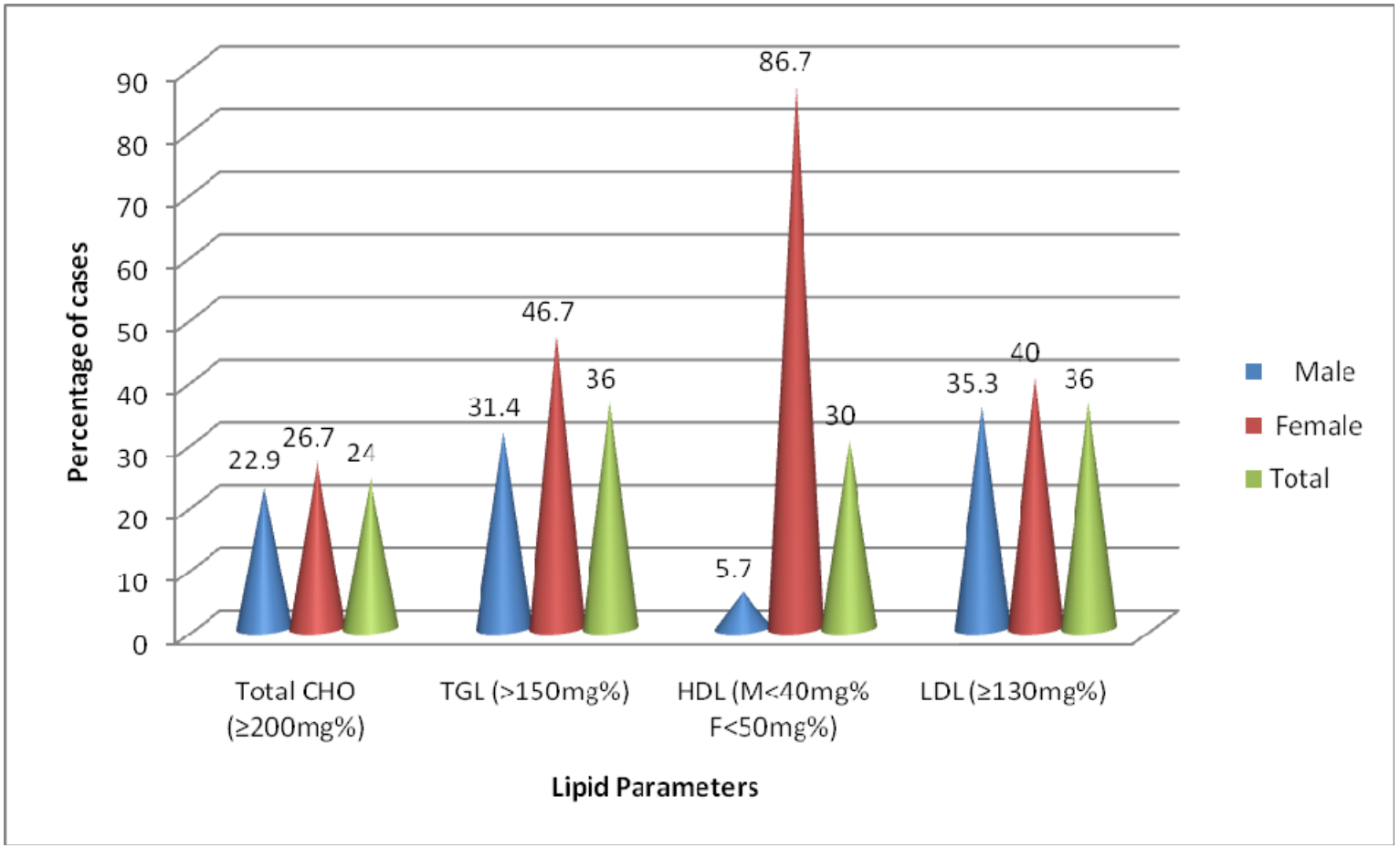
The above table shows the mean age of smokers who were affected by the disease was 52.9±11.3years and the same of the non-smokers was 61.5±6.8 years.The difference between the mean was statistically highly significant (P< 0.01).

**8.Abnormal Lipid Parameters:**

**Table 11 :Abnormal lipid parameters**

| Lipid parameters | Male (n=35) | | Female(n=15) | | Total (n=50) | |
|---|---|---|---|---|---|---|
| | n | % | n | % | n | % |
| Total CHO (≥200mg%) | 8 | 22.9 | 4 | 26.7 | 12 | 24 |
| TGL (>150mg%) | 11 | 31.4 | 7 | 46.7 | 18 | 36 |
| HDL (M<40mg% F<50mg%) | 2 | 5.7 | 13 | 86.7 | 15 | 30 |
| LDL (≥130mg%) | 12 | 35.3 | 6 | 40 | 18 | 36 |

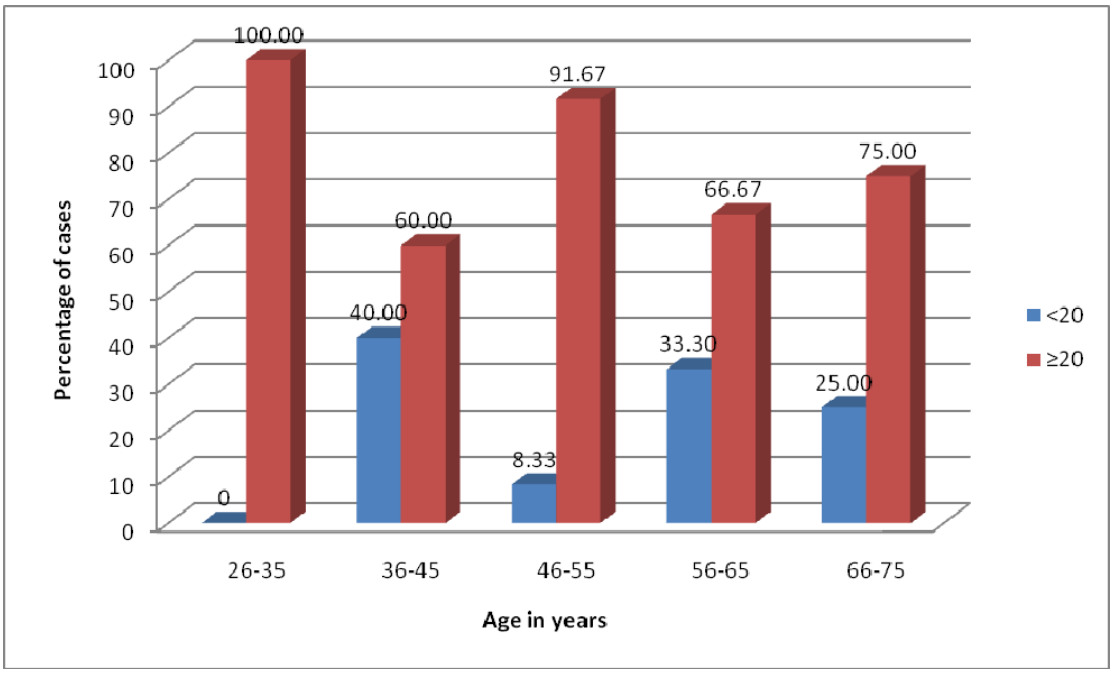
## Abnormal Lipid Parameters



## 9. Distribution of microalbuminuria in various age groups:

**Table 12: Distribution of microalbuminuria in various age groups**

| Age in years | Microalbuminuria (mg/L) | | | |
|---|---|---|---|---|
| | <20 | | ≥20 | |
| | n | % | n | % |
| 26-35 | 0 | - | 4 | 100 |
| 36-45 | 2 | 40 | 3 | 60 |
| 46-55 | 1 | 8.33 | 11 | 91.67 |
| 56-65 | 7 | 33.3 | 14 | 66.67 |
| 66-75 | 2 | 25 | 6 | 75 |



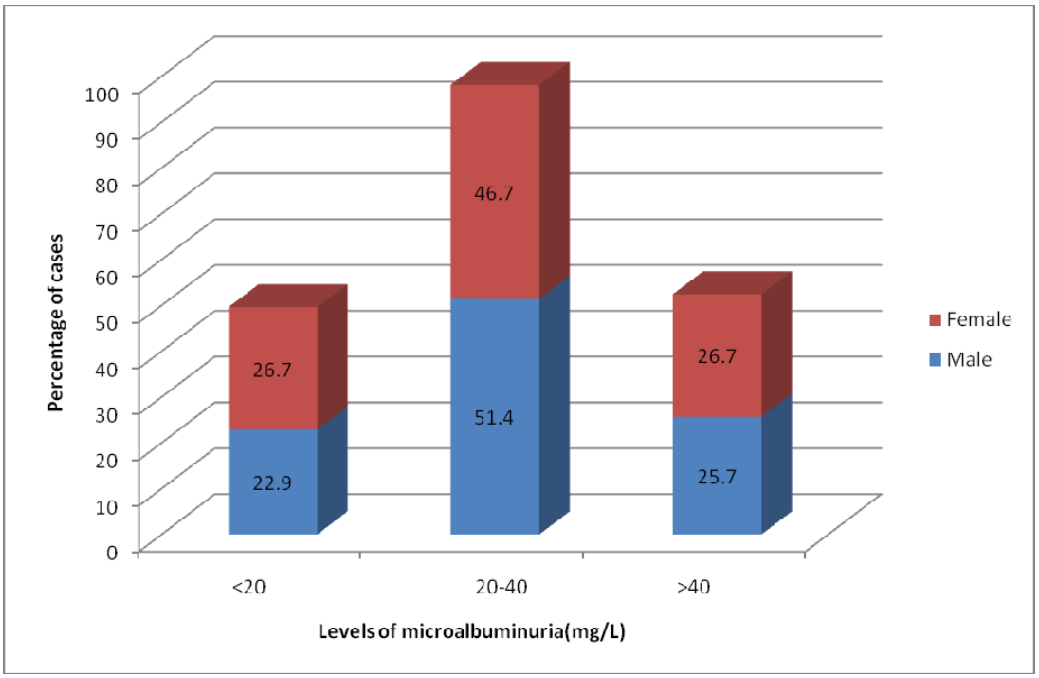
## 10. Level of microalbuminuria (mg/L)

**Table 13: Levels of microalbuminuria (mg/L)**

| Microalbuminuria (mg/L) | Male (n=35) | | Female (n=15) | | Total (n=50) | |
|---|---|---|---|---|---|---|
| | n | % | n | % | n | % |
| <20 | 8 | 22.9 | 4 | 26.7 | 12 | 24% |
| 20-40 | 18 | 51.4 | 7 | 46.7 | 25 | 50% |
| >40 | 9 | 25.7 | 4 | 26.7 | 13 | 26% |

There were 25 males (71.4%) and 12 females (80%) with abnormal microalbuminuria level in the present study.

This shows that patients with microalbuminuria are having a higher risk of developing ischemic heart disease.



## 11.Prevalence of microalbuminuria in non-diabetic non-hypertensive CAD patients:

**Table13: Prevalence of microalbuminuria in non-diabetic non-hypertensive CAD patients**

| Microalbuminuria | CAD Patients | | Significance |
|---|---|---|---|
| | n | % | |
| Normal(<20mg/l) | 12 | 24 | P < 0.001 |
| Abnormal(≥20mg/l) | 38 | 76 | |

The above table shows the prevalence of microalbuminuria among CAD patients as 76% and the same of the normal population is 15%.The difference between the two prevalence rates was statistically significant (P<0.001).

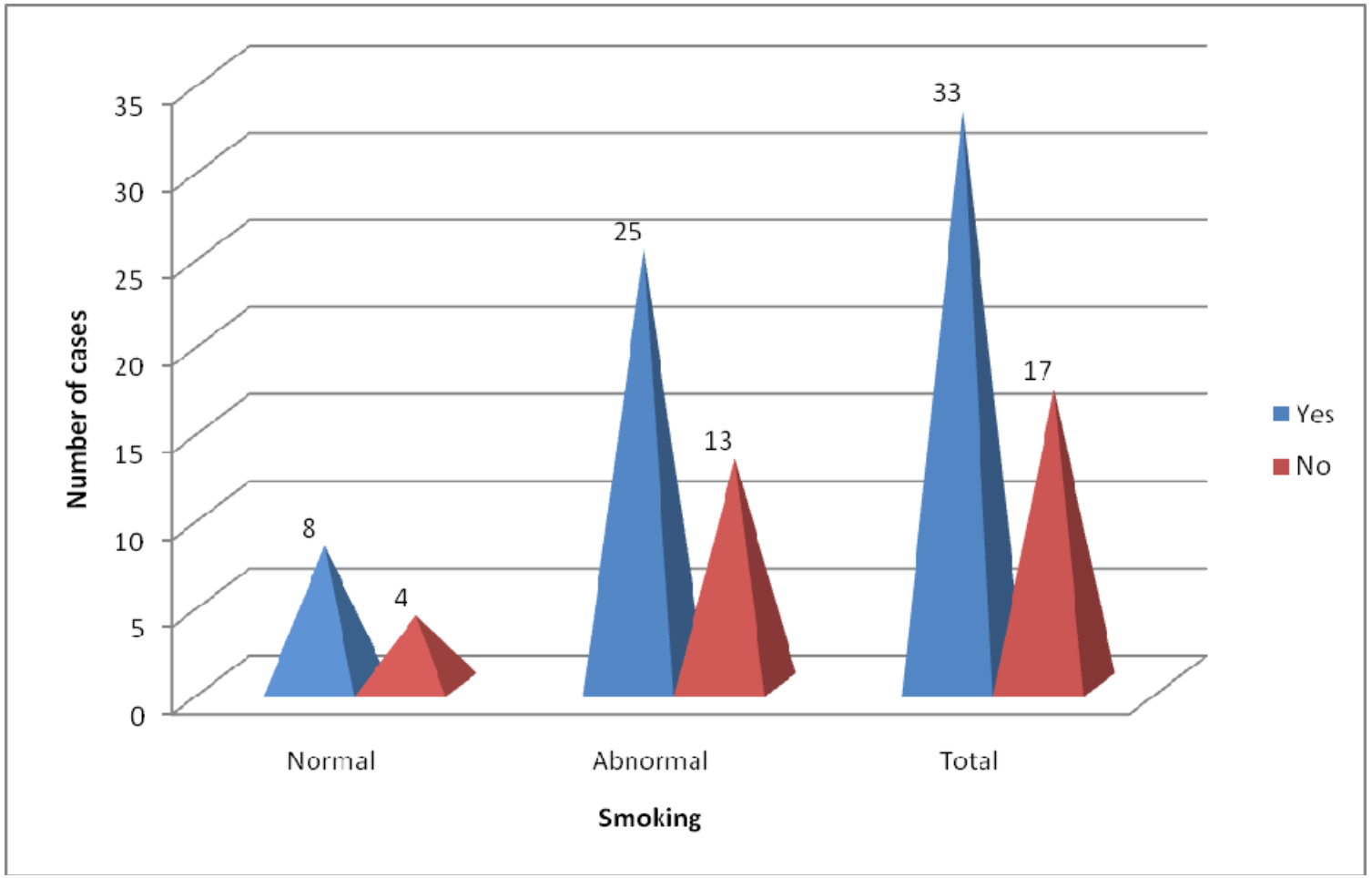
**12. Association between microalbuminuria and smoking habit in the study subjects:**

**Table 14: Association between microalbuminuria and smoking habit in the study subjects**

| Smoking | Microalbuminuria | | | Chi-square | Degree of Freedom | Significance |
|---|---|---|---|---|---|---|
| | Normal (<20mg/l) | Abnormal (≥20 mg/l) | Total | | | |
| Yes | 8 | 25 | 33 | 0.003 | 1 | P>0.05 |
| No | 4 | 13 | 17 | | | |
| Total | 12 | 38 | 50 | | | |

There are 33 patients with history of smoking, out of which 25 had microalbuminuria and in 17 nonsmokers, 13 had microalbuminuria. The observation from the above table shows that there was no association between microalbuminuria and smoking (P>0.05).

**Association between microalbuminuria and smoking habit in the study subjects**

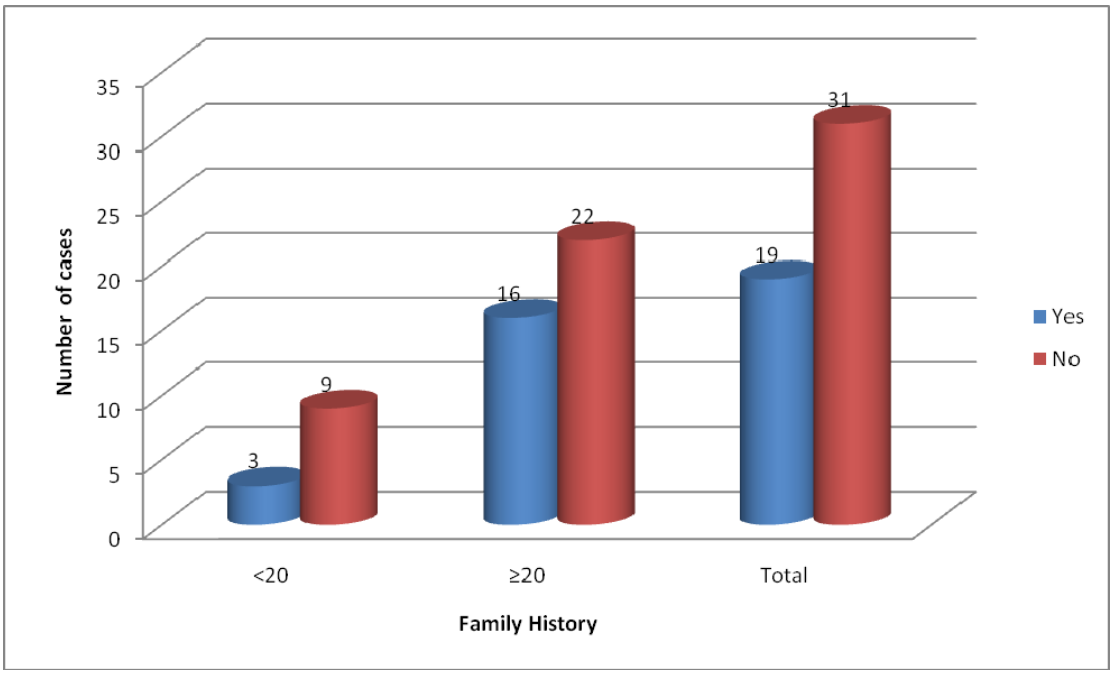


## 13. Association between microalbuminuria and familyhistory of CAD

**Table 14: Association between microalbuminuria with family history of CAD**

| Family history | Microalbuminuria(mg/l) | | | Chi-square | Degree of Freedom | Significance |
|---|---|---|---|---|---|---|
| | <20 | ≥20 | Total | | | |
| Yes | 3 | 16 | 19 | 1.133 | 1 | P>0.05 |
| No | 9 | 22 | 31 | | | |
| Total | 12 | 38 | 50 | | | |

The observation from the above table shows that there was no significant association between microalbuminuria and family history of disease (P>0.05).That means both were independent.



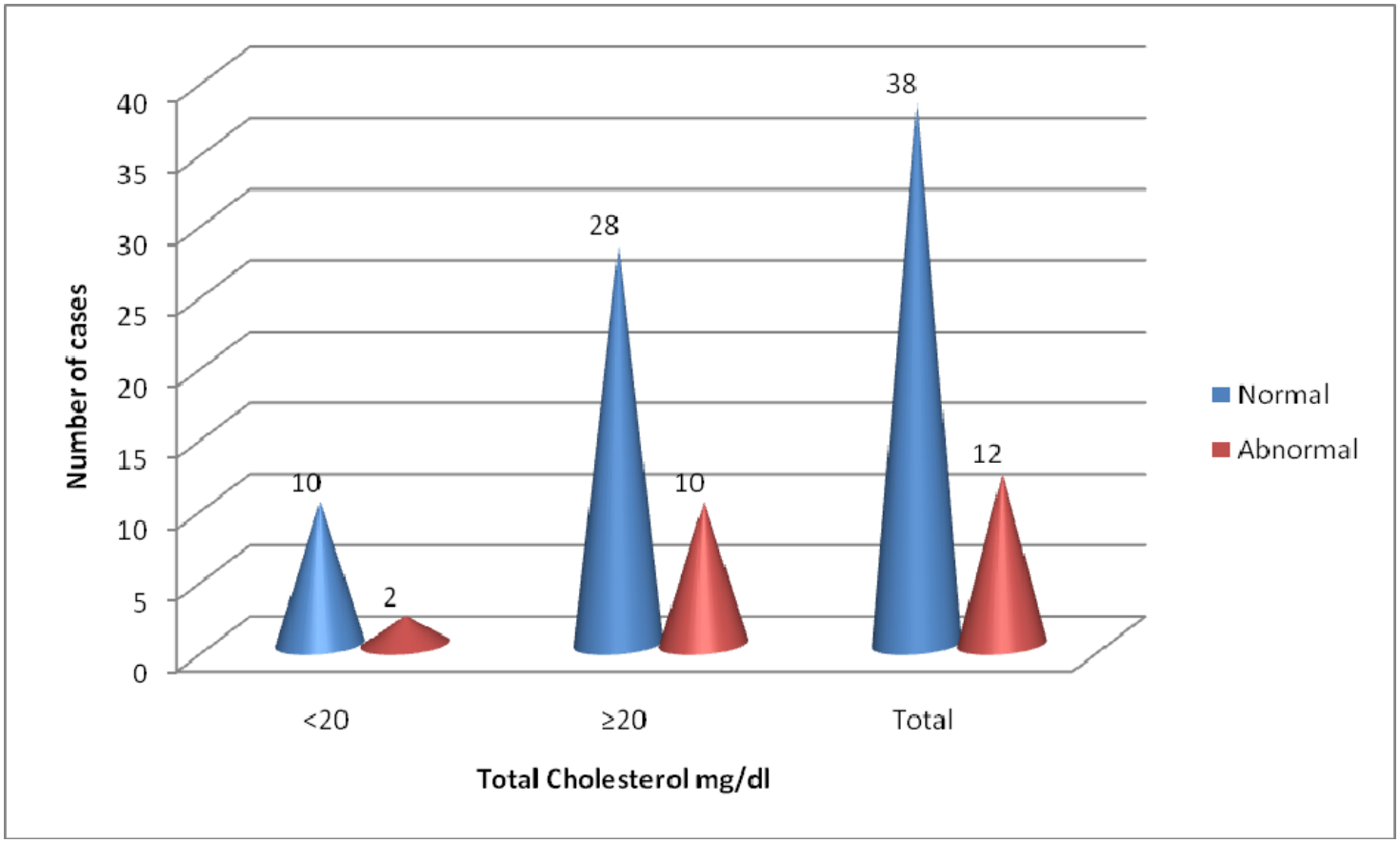
## 14. Association between total cholesterol and microalbuminuria:

**Table15:Association between total cholesterol and microalbuminuria**

| Total Cholesterol (mg%) | Microalbuminuria (mg/l) | | | Chi-square | Degree of freedom | Significance |
|---|---|---|---|---|---|---|
| | <20 | ≥20 | Total | | | |
| Normal (<200) | 10 | 28 | 38 | 0.466 | 1 | P>0.05 |
| Abnormal (≥200) | 2 | 10 | 12 | | | |
| Total | 12 | 38 | 50 | | | |

There are 12 patients with abnormal total Cholesterol, out of which 10 patients (83.33%) had Microalbuminuria and out of 38 patients (73.68%) with normal total cholesterol, 28 had microalbuminuria. The observation from above table shows that there was no significant association between total cholesterol and microalbuminuria (P>0.05)

**Association between total cholesterol and microalbuminuria**

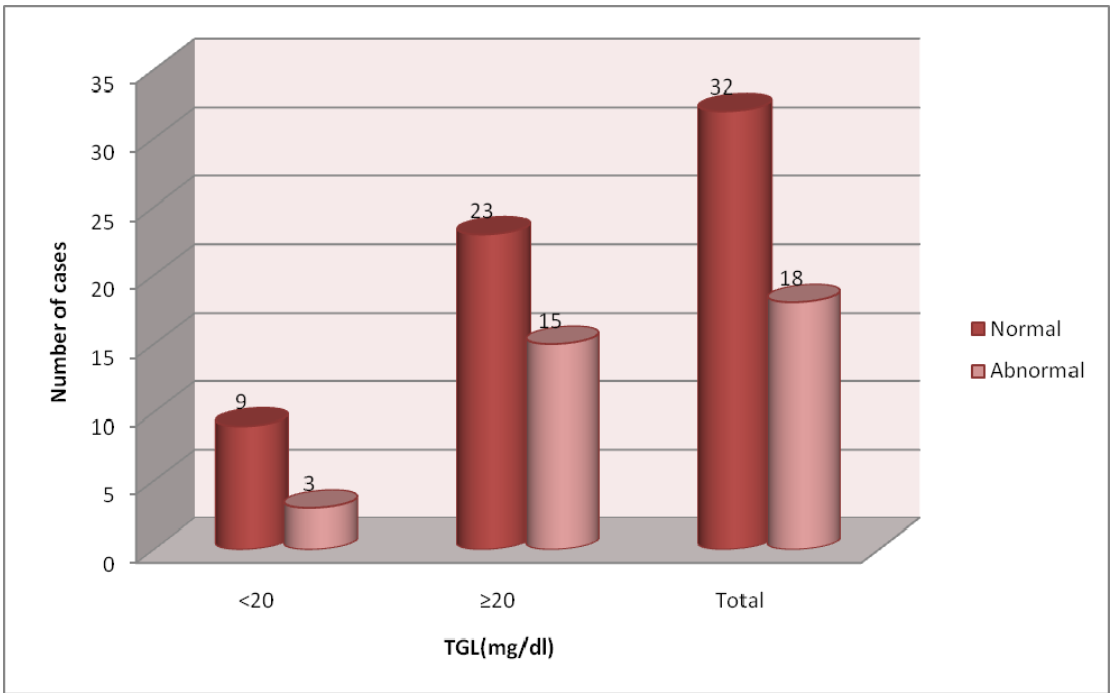


## 15. Association between microalbuminuria and triglycerides (TGL):

**Table 16: Association between microalbuminuria and triglycerides**

| TGL(mg/dl) | Microalbuminuria (mg/l) | | | Chi-square | Degree of freedom | Significance |
|---|---|---|---|---|---|---|
| | <20 | ≥20 | Total | | | |
| Normal(≤150) | 9 | 23 | 32 | 0.829 | 1 | P>0.05 |
| Abnormal(>150) | 3 | 15 | 18 | | | |
| Total | 12 | 38 | 50 | | | |

There are 32 patients with normal TGL, out of which 23 had microalbuminuria and in 18 patients with abnormal TGL, 15 had microalbuminuria. The results of the above table shows that there is no significant association between TGL and microalbuminuria (P>0.05).

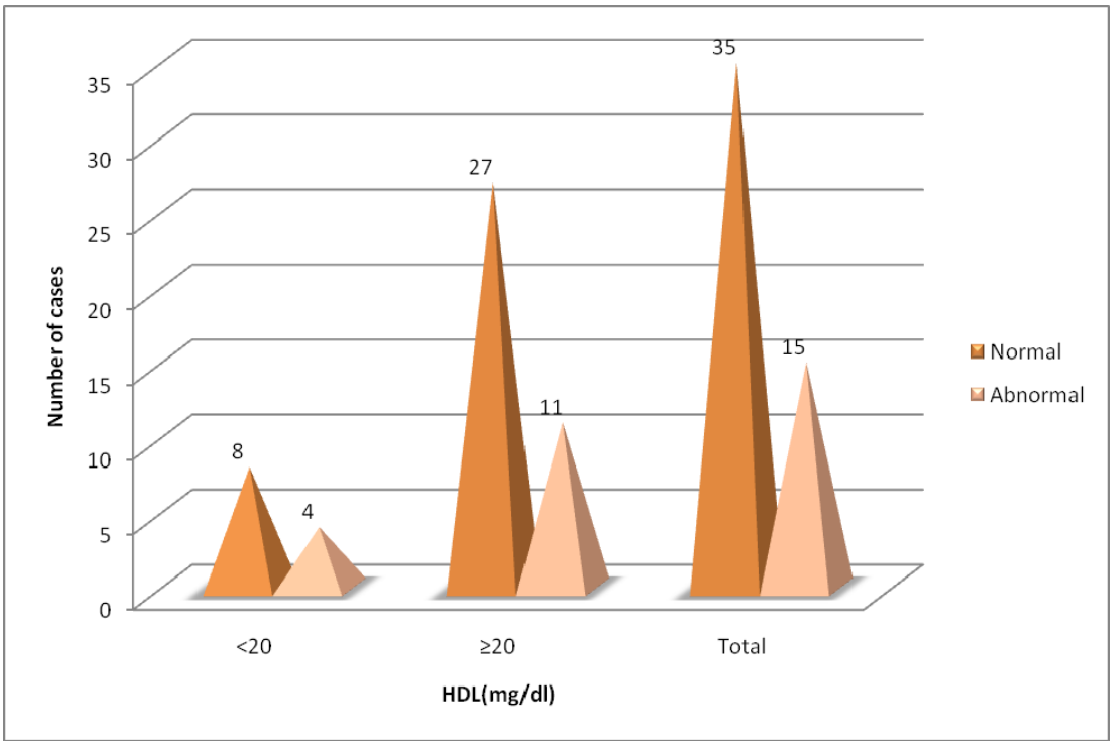


## 16. Association between microalbuminuria and HDL:

**Table 17: Association between microalbuminuria and HDL**

| HDL(mg/dl) | Microalbuminuria (mg/l) | | | Chi-square | Degree of freedom | Significance |
|---|---|---|---|---|---|---|
| | <20 | ≥20 | Total | | | |
| Normal | 8 | 27 | 35 | 0.084 | 1 | P>0.05 |
| Abnormal | 4 | 11 | 15 | | | |
| Total | 12 | 38 | 50 | | | |

There are 15 patients with abnormal HDL, out of which 11 had microalbuminuria and out of 35 patients with normal HDL, 27 had microalbuminuria. The results of the above table shows no statistically significant association between microalbuminuria and HDL (P>0.05)

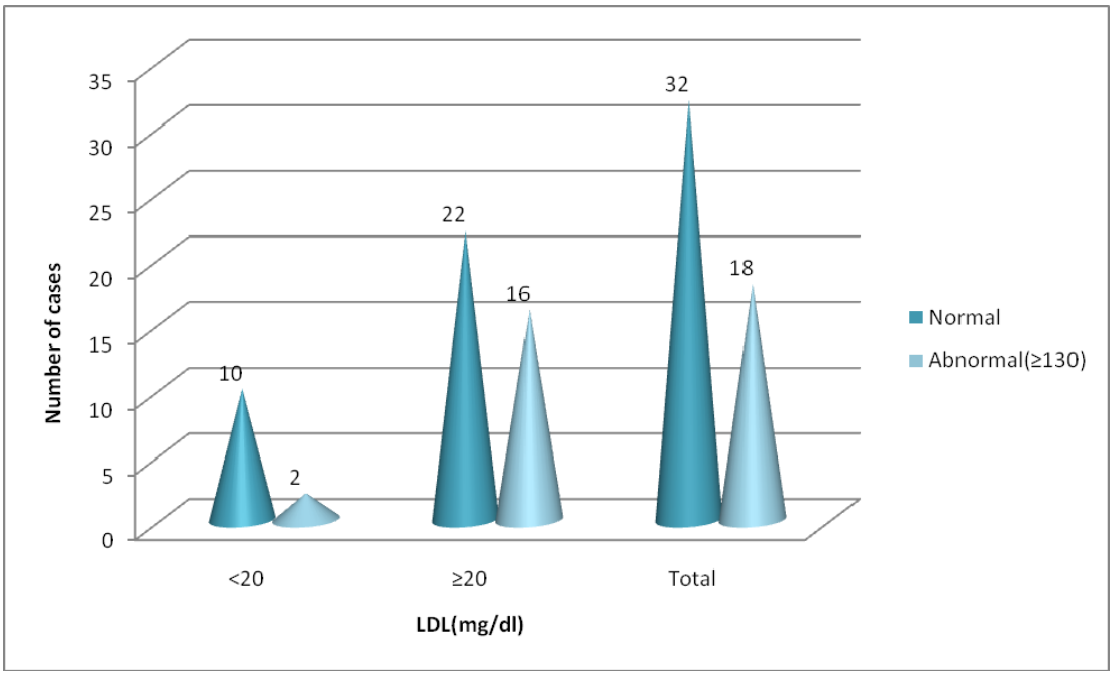


## 17. Association between microalbuminuria and LDL:

**Table 18: Association between microalbuminuria and LDL**

| LDL(mg/dl) | Microalbuminuria (mg/l) | | | Chi -square | Degree of freedom | Significance |
|---|---|---|---|---|---|---|
| | <20 | ≥20 | Total | 2.562 | 1 | P>0.05 |
| Normal | 10 | 22 | 32 | | | |
| Abnormal(≥130) | 2 | 16 | 18 | | | |
| Total | 12 | 38 | 50 | | | |

There are 18 patients with abnormal LDL, out of which 16 patients had microalbuminuria and out of 32 with normal LDL,22 had microalbuminuria. The results of the above table shows no statistically significant association between microalbuminuria and LDL (P>0.05).



# DISCUSSION

Ischemic heart disease will become a major disease burden in India by the year 2015.[60] To target preventive strategies, risk stratification of the population should be effective. There are many reports emanating from the western literature about microalbuminuria as an independent risk factor for development of IHD.

This study had 76% male patients compared to 26% female patients. This is in accordance with males are more prone for IHD than females.

The mean age of study population was 55.8±10.7 years. The median age was 57 years. The mean age of males was 53.8±11.6years and the same of females was 60.5±6.7 years. The significant difference of mean age between the sexes shows that male are affected by coronary heart disease quite earlier than females(Table - 5).All the females were in post-menopausal age group, which shows that sex hormones has a protective effect as far as cardiovascular system is concerned.

Smoking is present in 66% of the study subjects .Among males 94.3% were smokers but none of the females were smokers (Table 7).The

smokers had been affected by the disease quite earlier than the nonsmokers, since the mean age of the smokers was 52.9±11years and the mean age of nonsmokers was 61.5± 6.8 years. The difference between the mean was statistically highly significant (P <0.01).This indicates that smoking is an important risk factor for IHD.**Umesh N khot et al**[62] had found a prevalence of 41.6% in males and 29.5% in females in their study for smoking as a risk factor.

Family history of IHD was present in only 19% of study subjects. **H. C. Hillege et al** had an incidence of 29%.

The mean fasting blood glucose was 100±8.6mg/dl, the mean systolic BP was 115.6±9.5 mmHg, the mean diastolic BP-74.8±6.5mmHg indicating all patients are non-diabetic non-hypertensive.

The mean Total cholesterol was 178±26.8, mean TGL was 144.5±51.1, mean HDL was 42.6±3.8, mean LDL was 123±24.8.The major dyslipidemia in the study subjects were increased TGL (36%), increased LDL (36%) and decreased HDL (30%).

The present study showed that 76% of patients with IHD had microalbuminuria which shows a positive association.

The PREVEND study[62] showed that in a multivariate model adjusted for established cardiovascular risk factor, microalbuminuria was independently associated with infarct pattern (7.1%)(OR-1.61),major ischemia (10.6%)(OR-1.43),minor ischemia(15.1%)(OR-1.32).

In the PREVEND study[62], 32.8% of patients had microalbuminuria and in HOPE study cohort conducted betwen 1994 – 1999[63], 20.4% had microalbuminuria compared to 76% in our study. This was probably because the present study had a cohort of IHD patients in whom microalbuminuria was estimated whereas the studies mentioned above was done on the general population.

The smoking habit was not statistically significantly associated with microalbuminuria (P>0.05).

The lipid profile was not statistically significantly associated with microalbuminuria (P>0.05).

**L.Gomes MB, Da Cruz et al**. showed the incidence of lipid abnormality in >45% of diabetic patients.

**Campus VM, Blanchis et al** showed the incidence of lipid abnormality in essential hypertension as 44 – 48%. In our study the

incidence of hyperlipidemia was below 37%.This shows hyperlipidemia is more prevalent in diabetic and hypertensive patients with CAD.

The present study showed microalbuminuria can be used as an additional cardiovascular risk indicator even in non-diabetic and non-hypertensive individual.

# SUMMARY

- Male to female ratio was 2.33: 1
- Subjects in the age group 50 – 59 years constituted 42% of the study subjects.
- The youngest patient in the study was 26 years
- Family history of IHD was present in 38% of study subjects.
- Smoking history was present in 66% of study subjects. All of them are males (94.3%). Smokers are affected by disease quite earlier than non smokers.
- The abnormal lipid pattern seen was increased triglyceride (36%), increased LDL (36%) and decreased HDL (30%)
- Smoking and hyperlipidemia was not significantly associated with non-diabetic non-hypertensive coronary artery disease patients.
- Microalbuminuria (≥20mg/l) was present in 76% of study subjects with coronary artery disease.

# CONCLUSION

- Among the 50 non-diabetic non-hypertensive CAD patients, 38 patients (76%) had microalbuminuria.
- Microalbuminuria is positively associated with the Ischemic heart disease in non-diabetic non-hypertensive CAD patients and can be regarded as additional risk factor for ischemic heart disease.
- Hence screening for microalbuminuria is a worthwhile public tool for cardiac risk stratification and targeting preventive strategies.

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# PROFORMA

Name: Age:

Sex:

OP/IP Number: Occupation:

Presenting complaints:

Chestpain / breathlessness / palpitation / syncope / others

Past history:

Family history:

Personal history:

General examination:

Vital signs

Examination of cardiovascular system

Examination of respiratory system

Abdominal examination

Examination of Central nervous system

**Investigations:**

Urine – Albumin: Sugar: Deposits:

Early morning first void microalbumin:

Fasting blood sugar: Urea:

Creatinine:

Fasting serum lipid profile:

Total cholesterol: TGL: HDL: LDL:

12 lead ECG, Cardiac enzyme estimation, Echocardiogram.

## MASTER CHART

| S. No | Name | IP/OP No | Age | Sex | Family H/o IHD | Smoking | SBP mmHg | DBP mmHg | FBS mg% | TC mg% | TGL mg% | HDL mg% | LDL mg% | Micro albuminuria mg/l |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Prabhakaran | 36886 | 34 | M | Yes | Yes | 120 | 70 | 110 | 142 | 110 | 39 | 81 | 30 |
| 2 | Samuel | 29323 | 35 | M | No | Yes | 110 | 80 | 110 | 249 | 179 | 40 | 185 | 30 |
| 3 | Krishnan | 34231 | 72 | M | Yes | Yes | 130 | 80 | 105 | 208 | 154 | 41 | 128 | 16 |
| 4 | Subramani | 20126 | 53 | M | Yes | Yes | 120 | 60 | 104 | 186 | 102 | 40 | 125 | 68 |
| 5 | Sivakami | 35255 | 64 | F | No | No | 130 | 70 | 110 | 176 | 141 | 39 | 11 | 7 |
| 6 | Muthammal | 38354 | 58 | F | Yes | No | 120 | 80 | 99 | 240 | 170 | 41 | 184 | 37 |
| 7 | Muthukumaraswamy | 12252 | 74 | M | Yes | Yes | 130 | 80 | 94 | 161 | 119 | 40 | 97 | 24 |
| 8 | Arumugam | 45196 | 67 | M | Yes | Yes | 120 | 80 | 103 | 156 | 116 | 40 | 92 | 30 |
| 9 | Kandaswamy | 25971 | 56 | M | No | Yes | 100 | 70 | 107 | 175 | 120 | 44 | 107 | 18 |
| 10 | Rajammal | 33155 | 59 | F | Yes | No | 110 | 80 | 101 | 217 | 100 | 48 | 149 | 27 |
| 11 | Maharajan | 39684 | 33 | M | No | Yes | 110 | 70 | 110 | 202 | 167 | 49 | 120 | 61 |
| 12 | Pappa | 37904 | 51 | F | No | No | 120 | 70 | 109 | 179 | 111 | 52 | 104 | 64 |
| 13 | Sankaran | 30325 | 65 | M | Yes | Yes | 130 | 80 | 98 | 184 | 101 | 45 | 120 | 82 |
| 14 | Mani | 29240 | 47 | M | No | Yes | 110 | 70 | 94 | 178 | 187 | 48 | 92 | 30 |
| 15 | Rajaram | 16355 | 61 | M | No | Yes | 100 | 80 | 100 | 180 | 110 | 48 | 124 | 16 |
| 16 | Sankar | 40424 | 26 | M | No | Yes | 100 | 70 | 110 | 160 | 120 | 40 | 120 | 30 |
| 17 | Eswaran | 13244 | 58 | M | Yes | Yes | 120 | 70 | 104 | 188 | 114 | 40 | 138 | 70 |
| 18 | Peer mohamed | 23355 | 50 | M | Yes | Yes | 110 | 80 | 108 | 154 | 114 | 42 | 98 | 29 |
| 19 | Gandhi | 16490 | 55 | M | No | Yes | 130 | 70 | 100 | 110 | 115 | 40 | 120 | 30 |
| 20 | Sornathammal | 14207 | 57 | F | No | No | 110 | 80 | 98 | 190 | 441 | 30 | 130 | 28 |
| 21 | Vembu | 37585 | 69 | M | Yes | No | 120 | 60 | 104 | 175 | 146 | 45 | 120 | 49 |
| 22 | Chellammal | 50064 | 62 | F | No | No | 130 | 80 | 110 | 185 | 192 | 46 | 100 | 25 |
| 23 | Lakshmanan | 23172 | 62 | M | No | Yes | 100 | 70 | 108 | 180 | 110 | 44 | 194 | 60 |
| 24 | Ganapathy | 39322 | 50 | F | No | No | 100 | 80 | 104 | 210 | 164 | 44 | 138 | 24 |
| 25 | Eruthayamani | 49597 | 69 | M | Yes | No | 110 | 60 | 100 | 200 | 160 | 44 | 170 | 28 |
| 26 | Pandaram | 30345 | 58 | M | No | Yes | 120 | 80 | 96 | 142 | 110 | 42 | 90 | 15 |
| 27 | Perumal | 17680 | 40 | M | No | Yes | 110 | 70 | 102 | 180 | 110 | 42 | 130 | 29 |
| 28 | Sedhu | 29196 | 50 | M | Yes | Yes | 120 | 80 | 82 | 114 | 120 | 40 | 94 | 34 |
| 29 | Shanmugam | 31654 | 56 | M | No | Yes | 110 | 80 | 90 | 142 | 110 | 42 | 90 | 15 |
| 30 | Chithra | 44433 | 53 | F | No | No | 110 | 70 | 112 | 170 | 100 | 45 | 120 | 29 |
| 31 | Abdul kadar | 30434 | 50 | M | No | Yes | 110 | 80 | 84 | 204 | 170 | 44 | 132 | 38 |
| 32 | Petchiammal | 17362 | 61 | F | No | No | 130 | 80 | 100 | 176 | 141 | 35 | 114 | 18 |
| 33 | Sankarammal | 50847 | 63 | F | No | No | 120 | 70 | 99 | 200 | 168 | 43 | 140 | 44 |
| 34 | Sheik abdul kadar | 16010 | 72 | M | Yes | Yes | 120 | 80 | 98 | 184 | 192 | 44 | 108 | 30 |
| 35 | Muthukrishnan | 22052 | 40 | M | No | Yes | 130 | 80 | 90 | 150 | 14 | 40 | 110 | 18 |
| 36 | Paul raj | 13068 | 54 | M | Yes | yes | 110 | 80 | 80 | 160 | 120 | 44 | 108 | 29 |
| 37 | Damodaran | 31083 | 42 | M | No | Yes | 120 | 70 | 88 | 180 | 134 | 43 | 127 | 61 |
| 38 | Ramachandran | 25995 | 48 | M | No | Yes | 100 | 70 | 98 | 210 | 165 | 42 | 170 | 19 |
| 39 | Srinivasan | 22180 | 54 | M | Yes | Yes | 110 | 80 | 100 | 210 | 174 | 4 | 134 | 30 |
| 40 | Sankarapandian | 14734 | 40 | M | No | Yes | 120 | 70 | 104 | 175 | 140 | 38 | 120 | 17 |
| 41 | Alagammal | 46006 | 62 | F | No | No | 110 | 80 | 90 | 185 | 192 | 44 | 110 | 43 |
| 42 | Chelladurai | 27895 | 42 | M | No | Yes | 120 | 80 | 80 | 180 | 153 | 40 | 130 | 35 |
| 43 | soosaimuthu | 13704 | 60 | M | No | Yes | 130 | 70 | 88 | 210 | 174 | 43 | 134 | 30 |
| 44 | Ramiah | 32672 | 56 | M | Yes | Yes | 120 | 60 | 90 | 154 | 140 | 43 | 136 | 28 |
| 45 | Petchiammal | 17669 | 71 | F | Yes | No | 110 | 80 | 100 | 178 | 110 | 50 | 110 | 14 |
| 46 | Seenivasan | 40455 | 58 | M | No | Yes | 100 | 80 | 99 | 160 | 120 | 42 | 110 | 44 |
| 47 | Muthammal | 50487 | 75 | F | No | No | 120 | 80 | 102 | 175 | 146 | 40 | 112 | 60 |
| 48 | Maagani | 31661 | 62 | F | Yes | No | 110 | 80 | 112 | 158 | 164 | 39 | 134 | 15 |
| 49 | Saraswathy | 45854 | 60 | F | No | No | 110 | 70 | 104 | 170 | 150 | 46 | 108 | 20 |
| 50 | Arumugam | 16902 | 57 | M | No | Yes | 120 | 80 | 114 | 170 | 134 | 45 | 130 | 58 |

## KEY TO MASTER CHART

I P →Inpatients

O P →Out patients

H/0 →History

SBP →Systolic blood pressure

DBP →Diastolic blood pressure

FBS →Fasting blood sugar

TC →Total cholesterol

TGL →Triglycerides

HDL →High density lipoprotein

LDL →Low density lipoprotein

IHD →Ischemic heart disease

S.No →Serial number