ESTIMATION OF PROPORTION OF MICROALBUMINURIA AMONG NON DIABETIC PATIENTS WITH ACUTE CORONARY SYNDROME

DISSERTATION SUBMITTED TO THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY, CHENNAI

In partial fulfilment of the requirements for the degree of

M .D BRANCH-I
(GENERAL MEDICINE)
Register No :200120104008

DEPARTMENT OF GENERAL MEDICINE

TIRUNELVELI MEDICAL COLLEGE HOSPITAL

TIRUNELVELI – 627011

MAY -2023

CERTIFICATE BY THE DEAN

I hereby certify that this dissertation entitled " **ESTIMATION OF PROPORTION OF MICROALBUMINURIA AMONG NON DIABETIC PATIENTS WITH ACUTE CORONARY SYNDROME"** is a record of work done by **Dr. KIRAN RAJ A** in the Department of General Medicine,Tirunelveli Medical College, Tirunelveli, during her postgraduate degree course period from 2020 – 2023.This work has not formed the basis for previous award of any degree.

Prof.Dr.M.RAVICHANDRAN,MD.,
The DEAN
Tirunelveli Medical College
Tirunelveli

Date :
Place: TIRUNELVELI

DEAN
VELI MEDICAL COLLEGE
VELI - 11

CERTIFICATE BY HEAD OF THE DEPARTMENT

This is to certify that the dissertation entitled **"ESTIMATION OF PROPORTION OF MICROALBUMINURIA AMONG NON DIABETIC PATIENTS WITH ACUTE CORONARY SYNDROME "**submitted by **Dr KIRAN RAJ A** , to the Tamilnadu Dr.M.G.RMedical University,Chennai,in partial fulfillment of the requirement for the award of M.D Degree Branch- I (General Medicine) is a bona-fide research work carried out by his under direct supervision and guidance.

Date: 17/12/22
Place :TIRUNELVELI

Prof.Dr.S.ALAGESAN,MD.DM
Professor and Head of the Department,
Department of General Medicine ,
Tirunelveli Medical College ,
Tirunelveli

Dr. S. Alagesan, MD.,
Reg. No. 43658
Prof. & HOD of Medicine
Tirunelveli Medical College Hospital,
Tirunelveli.

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation entitled **"ESTIMATION OF PROPORTION OF MICROALBUMINURIA AMONG NON DIABETIC PATIENTS WITH ACUTE CORONARY SYNDROME "**submitted by **Dr KIRAN RAJ A** , to the Tamilnadu Dr.M.G.R Medical University,Chennai,in partial fulfillment of the requirement for the award of M.D Degree Branch- I (General Medicine) is a bona-fide research work carried out by his under direct supervision and guidance.

Date: 17/12/22
Place:Tirunelveli

Prof.Dr.R.PERIYASAMY,M.D
Professor of medicine
Unit Chief,
Department of General Medicine,
Tirunelveli Medical College
Tirunelveli.

Dr. R. Periyasamy MD.,
Reg.No. 51232
Professor of Medicine
Tirunelveli Medical College
Tirunelveli

DECLARATION

I solemnly declare that the dissertation entitled **"ESTIMATION OF PROPORTION OF MICROALBUMINURIA AMONG NON DIABETIC PATIENTS WITH ACUTE CORONARY SYNDROME "** is done by me at Tirunelveli MedicalCollege and Hospital, Tirunelveli under the guidance and supervision of Prof. Dr. S.ALAGESAN, M.D, D.M.,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.

Date: 17/12/2022

Place:TIRUNELVELI

Dr KIRAN RAJ

Register No :20012010400

Postgraduate student,

M.D General Medicine,

Tirunelveli Medical College,

Tirunelveli

ACKNOWLEDGEMENT

I wish to express my heartfelt gratitude to our Dean **Prof. Dr. M. Ravichandran,MD** Tirunelveli Medical College for allowing me to do the study in this institution.

I would like to express my humble thanks to our Professor & Head of the Department Prof.**Dr. S. Alagesan M.D.,D.M.,** Department of General Medicine, Tirunelveli Medical College Hospital, whose valuable guidance and constant help have gone a long way in the preparation of this dissertation.

I express my sincere thanks to my renowned teacher and my guide Chief **Dr.R.Periasamy,M.D.** Professor, Department of General Medicine, Tirunelveli Medical College for his guidance, valuable suggestions and constant encouragement throughout the study.

I am greatly obliged to **Dr.K.S.Dakshinamoorthy M.D.,** & **Dr.E.Sheik Mohammed Raja M.D.,** Assistant Professors, Dept.of General Medicine for their valuable suggestions in preparing this dissertation.

CERTIFICATE - II

This is to certify that this dissertation work entitled **"ESTIMATION OF PROPORTION OF MICROALBUMINURIA AMONG NON DIABETIC PATIENTS WITH ACUTE CORONARY SYNDROME"** of the candidate **Dr.KIRAN RAJ A** with registration Number for the award of **M.D.** Degree in the branch of **GENERAL MEDICINE (I)**. I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion page and result shows **9** percentage of plagiarism in the dissertation

Guide & Supervisor
sign with Seal

Dr. R. Periyasamy MD.,
Reg.No. 51232
Professor of Medicine
Tirunelveli Medical College
Tirunelveli

TIRUNELVELI MEDICAL COLLEGE

INSTITUTIONAL RESEARCH ETHICS COMMITTEE
TIRUNELVELI STATE OF TAMILNADU, SOUTH INDIA PIN 627011
91-462 2572733-EXT 91-462 2572944 91-462-2579789 91-462 2572611 91
online@tvmc.ac.in, tirec@tvmc.ac.in www.tvmc.ac.in

CERTIFICATE OF REGISTRATION & APPROVAL OF THE TIREC

REF NO: 2109/ GM/2021

PROTOCOL TITLE: ESTIMATION OF PROPORTION OF MICROALBUMINURIA AMONG NON-DIABETIC PATIENTS WITH ACUTE CORONARY SYNDROME
PRINCIPAL INVESTIGATOR: DR. KIRAN RAJ MBBS
DESIGNATION OF PRINCIPAL INVESTIGATOR: RESIDENT
DEPARTMENT & INSTITUTION: GENERAL MEDICINE, TIRUNELVELI MEDICAL COLLEGE, TIRUNELVELI

Dear DR. KIRAN RAJ MBBS, Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during The IEC meeting held on ***06.07.2021.***

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1.	TIREC Application Form	☐
2.	Study Protocol	☐
3.	Department Research Committee Approval	☐
4.	Patient Information Document and Consent Form in English and Vernacular Language	☐
5.	Investigator's Brochure	☐
6.	Proposed Methods for Patient Accrual Proposed	☐
7.	Curriculum Vitae of The Principal Investigator	☐
8.	Insurance /Compensation Policy	☐
9.	Investigator's Agreement with Sponsor	☐
10.	Investigator's Undertaking	☐
11.	DCGI/DGFT approval	☐
12.	Clinical Trial Agreement (CTA)	☐
13.	Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA)	☐
14.	Clinical Trials Registry India (CTRI) Registration	☐

THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

1. The approval is valid for a period of 2 year/s or duration of project whichever is later
2. The date of commencement of study should be informed
3. A written request should be submitted 3weeks before for renewal / extension of The validity
4. An annual status report should be submitted.
5. The TIREC will monitor the study
6. At The time of PI's retirement/leaving the institute, The study responsibility should be transferred to a person cleared by HOD
7. The PI should report to TIREC within 7 days of the occurrence of the SAE. If the SAE is Death, the Bioethics Cell should receive the SAE reporting form within 24 hours of the occurrence.
8. In the events of any protocol amendments, TIREC must be informed and the amendments should be highlighted in clear terms as follows:
   a. The exact alteration/amendment should be specified and indicated where the amendment occurred in the original project. (Page no. Clause no. etc.)
   b. The PI must comment how proposed amendment will affect the ongoing trial. Alteration in the budgetary status, staff requirement should be clearly indicated and the revised budget form should be submitted.
   c. If the amendments require a change in the consent form, the copy of revised Consent Form should be submitted to Ethics Committee for approval. If the amendment demands a re-look at the toxicity or side effects to patients, the same should be documented.
   d. If there are any amendments in the trial design, these must be incorporated in the protocol, and other study documents. These revised documents should be submitted for approval of the IEC, only then can they be implemented.
   e. Approval for amendment changes must be obtained prior to implementation of changes.
   f. The amendment is unlikely to be approved by the IEC unless all the above information is provided.
   g. Any deviation/violation/waiver in the protocol must be informed.

STANDS APPROVED UNDER SEAL

TIRUNELVELI MEDICAL COLLEGE

318m

Dr.K.Sivantaraman, MD
Member Secretary, TIREC
Tirunelveli Medical College, Tirunelveli – 627011
State of Tamilnadu, South India

Ouriginal by Turnitin

Document Information

Analyzed document	THESIS (1) (2).docx (D153604664)
Submitted	2022-12-16 10:11:00
Submitted by	Kiran raj
Submitter email	annaduraikiran47@gmail.com
Similarity	9%
Analysis address	annaduraikiran47.tnmg@analysis.urkund.com

Sources included in the report

SA **The Tamil Nadu Dr. M.G.R. Medical University / DISSERTATION PLAG.pdf** 5
Document DISSERTATION PLAG.pdf (D151916688)
Submitted by: ranjivr170795@gmail.com
Receiver: ranjivr170795.tnmg@analysis.urkund.com

SA **Final Thesis for plagiarism check.docx** 2
Document Final Thesis for plagiarism check.docx (D30571896)

SA **The Tamil Nadu Dr. M.G.R. Medical University / maha thesis 2022 final.docx** 3
Document maha thesis 2022 final.docx (D152896228)
Submitted by: maha260296@gmail.com
Receiver: maha260296.tnmg@analysis.urkund.com

ABBREVIATIONS

ACE	**Angiontensin Converting Enzyme**
ACR	**Albumin Creatinine Ratio**
ARBs	**Angiontensin Receptor Blockers**
CAD	**Coronary Artery Disease**
CRP	**C reactive protein**
CVD	**Cardiovascular Disease**
EDRF	**Endothelium Derived Releasing Factor**
ELISA	**Enzyme Linked ImmunosorbentAssay**
HDL	**High Density Lipoprotein**
IHD	**Ischemic Heart Disease**
IL-1	**BETAInterleukin 1 beta**
LDL	**Low Density Lipoprotein**
MA	**Microalbuminuria**
NO	**Nitric Oxide**
PVD	**Peripheral Vascular Disease**
RIA	**Radio Immunoassay**
TMT	**Thread Mill Testing**
TNF	**Tumor Necrosis Factor**
UAE	**Urinary Albumin Excretion**
VCAM- 1	**Vascular Cell Adhesion Molecule**

LIST OF DIAGRAMS

LIST OF TABLE

ABSTRACT

BACKGROUND AND OBJECTIVE

In diabetics microalbuminuria have been an established indicator for micro and macrovascular pathology but there is increasing evidence that microalbuminuria may be an important indicator for future chance of developing ischemic heart disease. This study was done to establish a relationship between microalbuminuria and coronary arterial disease in non diabetics

METHODOLOGY

137 non diabetic patients with coronary arterial disease who fulfill the criteria were randomly selected for the study, were evaluated for traditional risk factors and microalbuminuria in Tirunelveli medical College.

INTRODUCTION

In the general population of India, the estimated prevalence of Coronary artery disease is 6-9%, and may become the leading cause of morbidity and mortality in our country. Since the pioneering work of Framingham trials many cohort based and cross sectional studies have identified series of independent risk factors for coronary arterial disease among which age, male gender, previous family history of young ischemic heart disease, cigarette smoking, diabetes mellitus, hypertension,dyslipedemia, physical inactivity, obesity are taken as colloquial risk determinants .

The interest in improving cardiovascular risk assessment stemmed from better knowledge of pathophysiology of atherosclerosis and identification of new targets for anti atherosclerotic drug therapy, have 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 has been a historically accepted marker of widespread vascular damage in diabetics as well as non diabetic patients ,however there has been more evidence pointing out to the fact, that microalbuminuria is a significant cardiac risk factor even in general population, so the early detection of microalbuminuria helps to prevent the progression of cardiac decompensation. Inclusion of microalbuminuria to the existing panel of risk factors have enabled risk stratification and is also beneficial owing to its easy determination.

The present study have been conducted to determine the Microalbuminuria prevalence among non diabetic patients with coronary arterial disease drawn from

the south indian population admitted to the Tirunelveli medical College, Tirunelveli and outpatients attending the Ops of the department of general medicine of tirunelveli medical College, Tirunelveli.

AIM OF STUDY

- To estimate the prevalence of Microalbuminuria in non diabetics having evidence of coronary arterial disease.
- To study the relationship between microalbuminuria and ischemic heart disease in the subject.

REVIEW OF LITERATURE

ISCHEMIC HEART DISEASE

Ischemia refers to a deficiency of oxygen as a result of impaired blood supply to the myocardium which causes a mismatch between the oxygen demand and supply. The most common cause of myocardial ischemia is atherosclerotic obstructive disease of epicardial coronary arteries.

ATHEROSCLEROSIS PATHOGENESIS

The term atherosclerosis which was introduced by Marchand who described the association between the fatty degeneration and vessel stiffening [1.] The phenomenon affects large sized medium and small vascular vessels and is characterized by discrete intramural subintimal sclerosis which encroaches on the lumen of the arteries. The characteristic lesion of atherosclerotic plaque has three major parts.

Of which the first is Cellular part which includes smooth muscle cells macrophage and other leukocytes, The second component is extracellular matrix including collagen elastic fibres and proteoglycan, The third part is lipd layer in intracellular region which accumulates inside the macrophages. As a result of which macrophages changes to foam cells.

The Atherosclerotic lesions are due to inflammatory reactions and subsequent massive release kf cytokines, smooth myocytes multiplication, extracellular matrix in the cells and accumulation of lipids, macrophages and fibroblast.

THE FORERUNNER OF ATHEROGENESIS

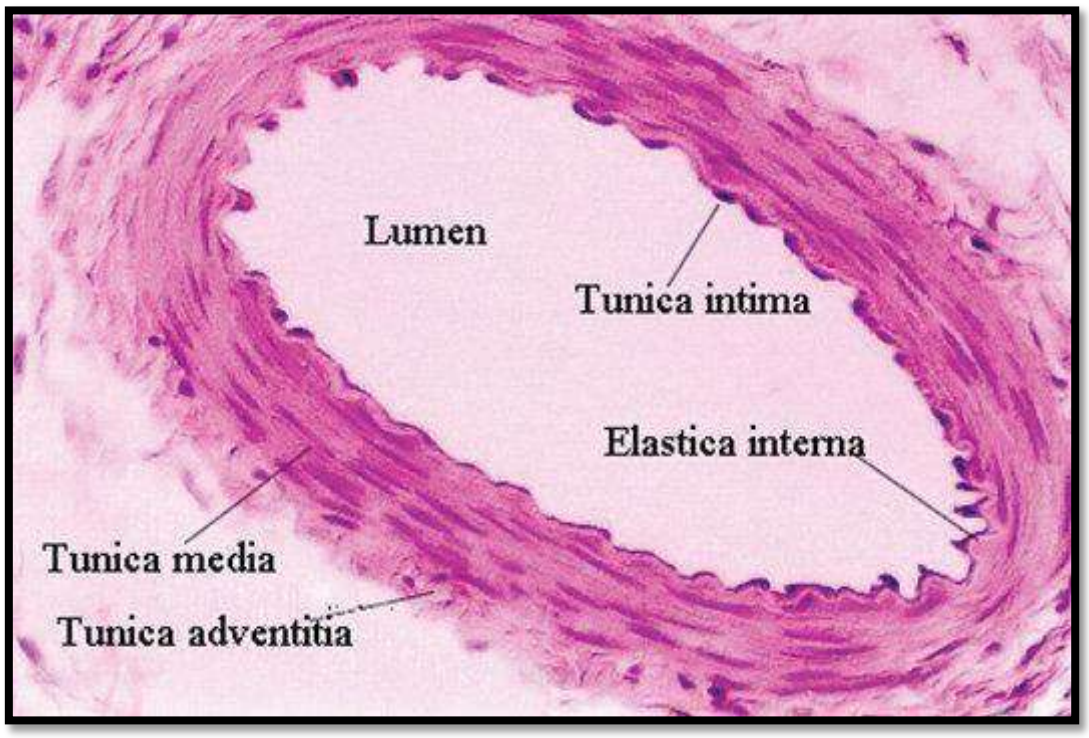
ENDOTHELIAL INJURY

The luminal surface of normal artery has a covering made of endothelium monolayer which adheres to the subendothelial matrix.

The cells of endothelium also provide the artery with non occlusive layer, platelet aggregation will be retarded by the presence of negative charge which endothelium posses and endothelial secretions of inhibitors prostacyclin and also endothelial relaxation factor known as EDRF NO. PGI2 along with EDRF NO secreted by endothelial cells have relaxation effect on smooth muscle in Tunica media [2] . The Endothelial cells produces chemicals which will antagonize smooth muscle cell proliferation and transfer the cytokines like chondroitin sulphate, heparin sulphate and EDRF NO. Finally endothelial cells will protect the vessel with lumen layer to which macrophage lymphocytes platelet not possible to stick.

The endothelial cells which has injured appears morphologically different from that of normally appearing endothelial cells, unlike normal cells they are not aligned in the direction of blood flow and have few intracellular attachment result in increased permeability. The Endothelial cells which are injured are also more thrombotic than the normal endothelial cells because of the diminished production of PGI2 and EDRF NO . The injured endothelial cells promotes the vascular smooth muscle cell migration by releasing the less amount of EDRF NO and by secreting PDRF and endothelial -1 . Finally the injured endothelial cell will promote the recruitment of macrophage by secreting MCP 1 and by expressing the cell surface receptors or selectins to which monocyte can bind [3].

HISTOLOGY OF NORMAL MUSCULAR ARTERY



The above diagram shows outer tunica adventitia middle tunica media and inner endothelial layer (tunica intima) with internal elastic lamina.

ROLE OF INFLAMMATION

The slowly progressing atherosclerotic process is obvious, in the early times by the disruption in the endothelial function. Atherogenesis is started as a cellular later of endothelium which over express adhering molecules as a response to the turbulent blood flow in the presence of unfavorable lipid profile. The VCAM- 1 expression on endothelium was an important process in the development of Atherogenesis[4].

The enhanced level of cellular adherence and the coexisting endothelial dysfunction will result in the aggregation of inflammatory chemicals cytokine release and aggregation of lipid molecules into atherogenic plaque.

Now, it have been generally shown that the early progress of atheroclerosis will be controlled predominantly as a result of inflammatory cascade. VCAM -1 attachment aggrevates the transfer of macrophages (monocytes), T-lymphocytes. Subsequently when the injired endothelial cells will secrete monocyte chemoattracting protein by neutroohils amplifies inflammatory cascade with recruitment of further cells stimulating polymorphs in the tunica media layer resulting in transfer and further growth of smooth muscle cells. As a response to the signs produced inside the early plaque of atheroma monocytes attached to the endothelial layer and subsequently gets transferred to intimal layer and basement membrane by expressing enzymes like regionally generated matrix metalliproteinase enzymes which will destroy the connective tissue network [5].

The monocytes which has been transferred will secrete further harmones start travelling through the endothelial surface into the layer of tunica media.this process is still aggrevated by the local secretion of monocyte colony stimulating factor which will result in macrophage proliferation. This local stimulation of monocytes macrophages

leading ro the cytokine mediated process of atherosclerosis and the oxidative damage of low density lipoprotein.

Many inflammatory mediators are known ro affect the plaque development in atherosclerosis. CD40L which was elaborated inside the plaque was known to hike the production of tissue activating factor (it means it will enhances the possibility of thrombosis) in Atherosclerotic plaque due to atherosclerosis, anti CD40L neutralize the evolution of the present lesion of atherosclerosis in the trial models in animals [6,7.]

Inflammatory cytokines produced by myocytes within the plaque due to atherosclerosis include but will not be confined to, interleukin 1 beta, tumor necrosis factor alpha and beta, MCP -1, IL-18, GM-CSF and CD40 .

The effect of the cytokine is huge and include cellular matrix proliferation, mitogenesis, new vessel generation and foam cell growth at streaks are the most often first recognisable plaque lesion usually seen first in the surroundings of vessel branch sites where there will he an abnormal haemodynamic shearing stress which will result in capillary damage. Fat streaks are mainly flat lesion seen in tunica intima comprised of smooth myocytes macrophages which contain lipid droplets which gives the tissue foam like morphology [8].

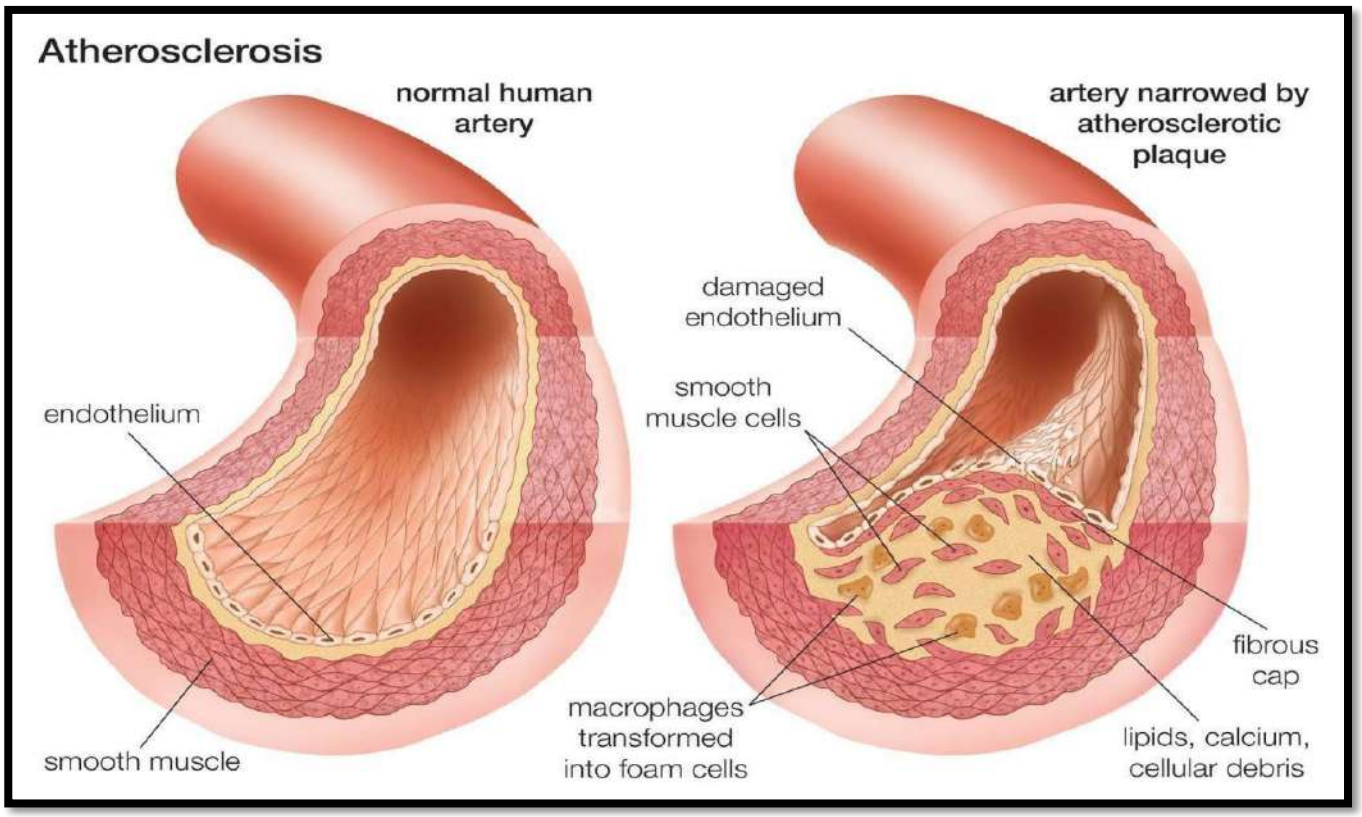
Initiators of this insult macroscopic type 1 lesion may be seen among infants. Fatty streaks normally seen at places where capillary insult has aggravated the capillary leakge enough to let the low density lipoprotein and other large molecules to permeate in the endothelium.

Below the endothelium. LDL has big favor for amino sugars and becomes sequestrated. LDL go through the series of changes which will cause the production of modified variety of LDL[.9 .]

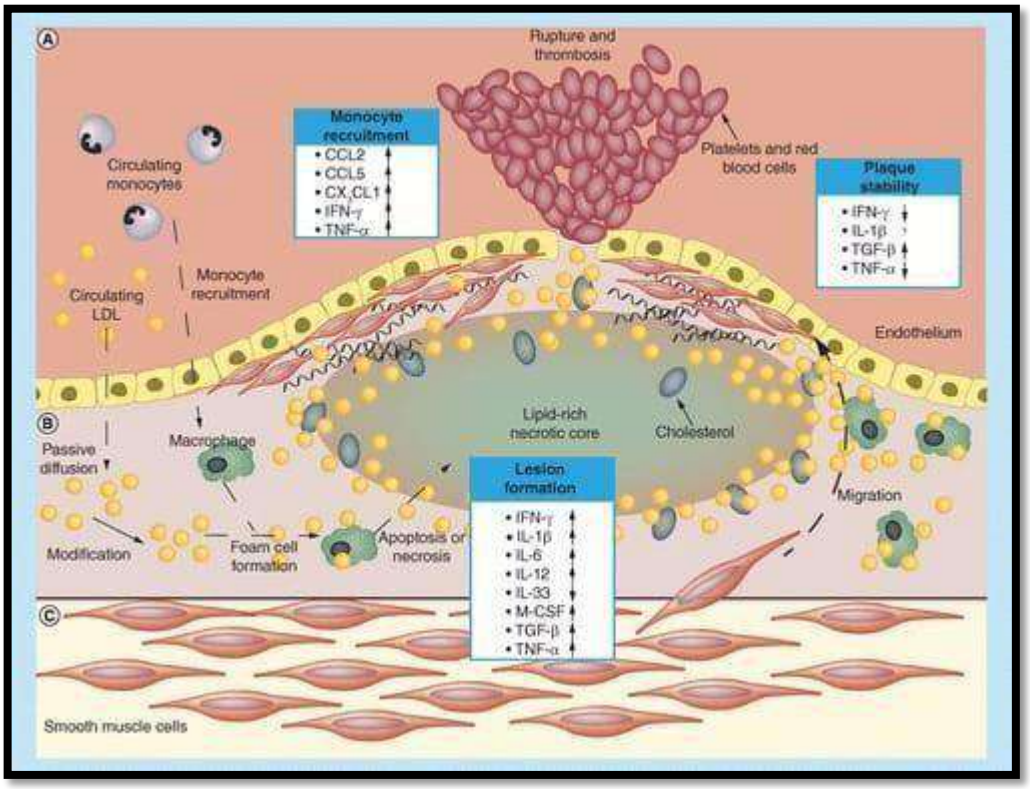
This alterations in LDL is important for the formation of Atherosclerotic plaque. The chemical attractant molecules with monocytes, chemoattractant protein released by injured capillary cells will transfer the circulating macrophages into the subendothelium where they will be transformed into macrophages. This monocyte transfer will be controlled by aggregation molecule, capillary mediated leukocyte sticking and chemoattractant molecules. Vessel wall adherence will be expressed in injured endothelium the new LDL prevent the escape of macrophages from the site . Finally the change of LDL help the tissue to get large amount of fat laden cells. Thus the monocyte attachment to the endothelium, migration into the tunica intima and maturation to form lipid laden macrophages represents the key steps in the formation of fatty streaks. Fibrotic lesion formation

Fatty streaks are converted to fibrotic lesion are the next step in the pathogenesis of atherosclerosis. The fibrous type of injury composed to fibrous cap made up of myocytes which are transferred from underneath endothelium and tunica media. The remaining components of fibrous cap consists of non functional endothelium present on the lumen of fibrous cap, monocytes, macrophages, platelet and Tcells. Underneath the fibrous cap, fibrous injury is made up of foam cells of macrophage and smooth muscle cells. One of the initial event causing change of streak to fibrotic lesion comprises of focal destruction of endothelium which cause the lipid streak. This is caused by shear stress exerted on the non functioning tissues from derangement of the vessel wall and from free radicals produced in the body, free radicals and products of lipid peroxidation produced by underlying foam cells.

CROSS SECTION IMAGE ILLUSTRATING ATHEROSCLEROTIC PLAQUE OCCLUDING THE ARTERIAL VESSEL.



MOLECULAR MEDIATORS INVOLVED IN THE FORMATION OF ATHEROSCLEROTIC PLAQUE.



SPECIMEN OF ARTERY DAMAGED BY PROGRESSIVE ATHEROSCLEROSIS

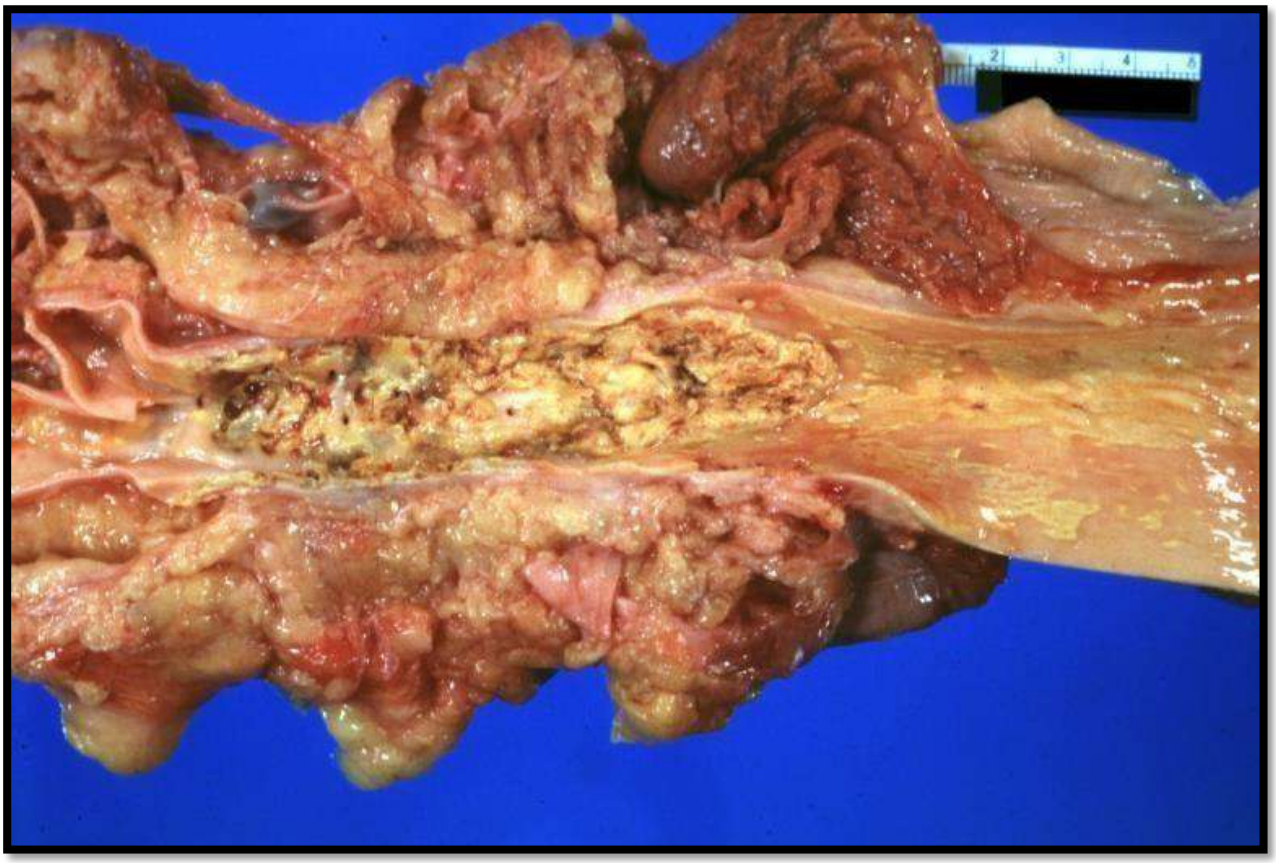
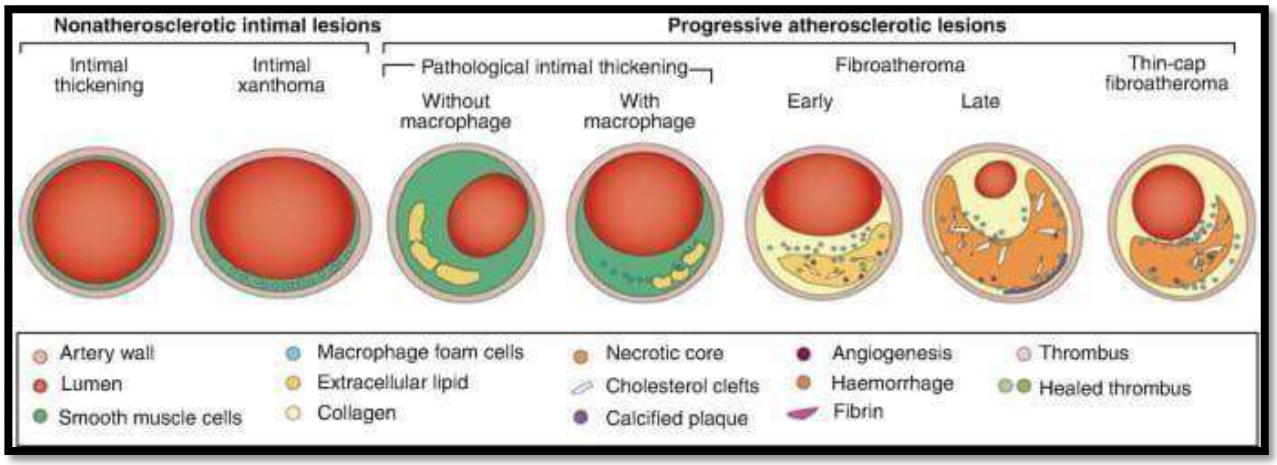
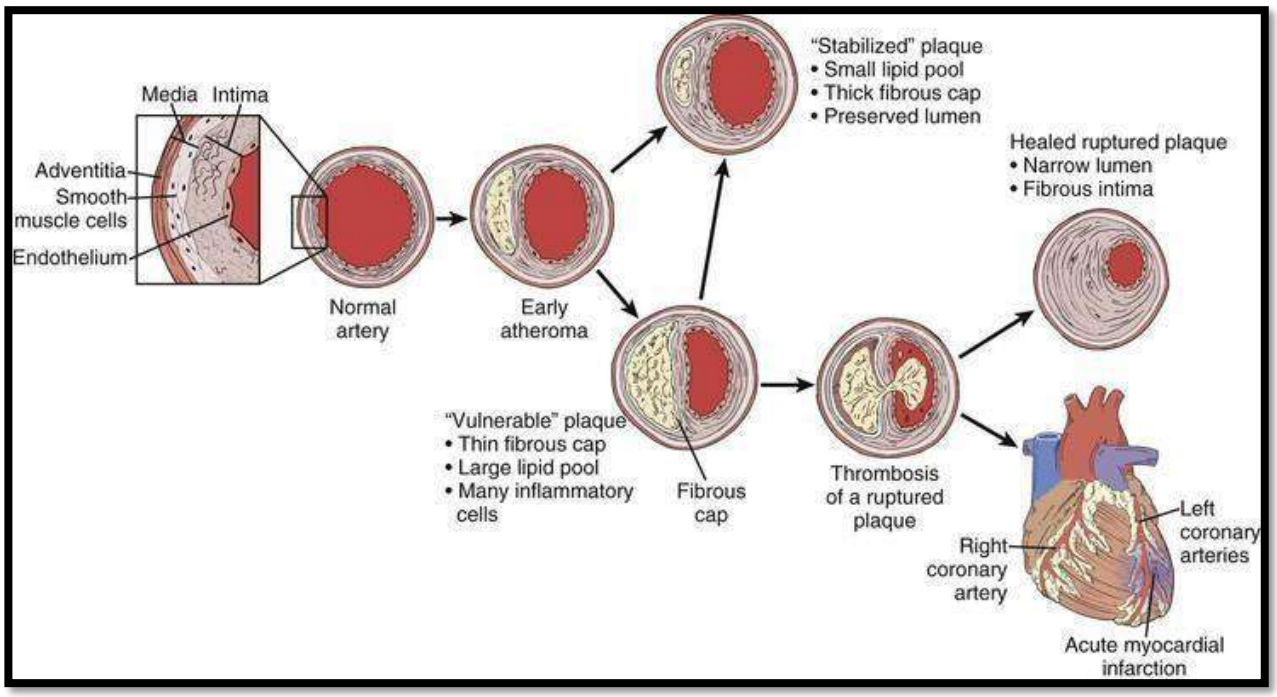


ILLUSTRATION OF PROGRESSIVE PLAQUE LESION AND ARTERIAL LUMEN NARROWING.





FINAL STAGES OF PLAQUE DEVELOPMENT

The final stage in the development of Atherosclerotic plaque is the conversion of fibrotic lesion into advanced lesion. A lesion in which a thrombus has formed subsequent either due to plaque ulceration or intraplaque haemorrhage. The factors causing plaque fracture composed of turbulence and mechanical shearing stress bleeding into plaque because of break in the small arteries and increased vessel lumen strain on the fibrous tip of plaque due to the Presence of pre existing lipid pool along with production of extracellular tissue destroying enzymes by macrophages which are present inside the lesion [10].

THE ROLE PERFORMED BY LIPIDS

The fat and lipid hypothesis which was proposed as the cause of atherosclerotic plaque has been drastically changed over the period of past twenty years. It has been accepted that internalisation and aggregation of lipid starts in response to the initial changes in the endothelial vessel wall pathology which was once seen as a initial part of atherosclerosis.

Till now the lipid accumulation is needed for the growth and further development of the definite plaque lipid deposit begin initially with the LDL movement from bloodstream into blood vessels once it reaches inside the tunica media layer, three different types of fate can occur to LDL, it can return back to blood flow (it is a feature of plaque regression and it is process which will be helped by some fat decreasing measures), it can also be oxidised (with the help of free radicals or as a result of direct activity of polymorphs), or it may be endocytosed by macrophages and then it is converted into foam cells. Peroxidised LDL can be notoriously atherosclerotic and has affinity towards macrophages and platelets. Monocyte system which includes

macrophages and monocytes will affach to the LDL which was present inside the tuncia intima through a group of new receptors known as scavenger receptors will identify the LDL molecule after it is peroxidised. The intake of peroxidised LDL will make the monkcytes less active hence this will promote the aggregation of fat filled cells in the tunica intima. The lipid laden foam cells will regain the activeness and releases more variety of chemokines and inflammatory cells. Result of this aggregation includes transfer and aggregation of smooth muscle cells to release more locally acting chemokines. Further LDL destruction and peroxidation, movement and transfer of extra macrophages and monocytes (foam cells with further destruction and damage to the endothelial cell function.)

Few plaques will proliferate inside the plaque,probably in response to the local antigenic cascade.the potential significance of angiogenesis in the pathophysiology of atherosclerosis is illustrated in experiments which shows that the antiangiogeneic treatment will decrease the atheromatous lesion progress in placebo control trial in atherosclerosis prone mice

Two types of destruction are seen which are superficial and deep injury .the first type injury will results in patchy

EP of focal endothelial destruction which futher grow and leads to the development of mural and even adhesive thrombosis.Plaques which is filled with collagen tissue lined by lipid laden foam cells leads to superficial damage.[11]

Deep vessel damage is seen by a split or shear which extends from the lumen of plaque into the substance of plaque. This type of damage which occurs in plaques has a big fat rich pool,exposing blood and it’s blood components to the highly thrombotic substance that makes up the plaque.

Activation of thrombus will affect the course of atherogenesis, in presence of conditions like MI and unstable angina caused due to the release of chemokines like von willebrand tissue factor leading to controlled platelet adhesion aggregation activation .

PREVENTION AND EPIDEMIOLOGY OF ATHEROSCLEROSIS

Atherosclerosis is a multifactorial disease where age of onset and progression are greatly influenced by inborn and acquired risk factors. Since the pioneering work of Framingham study various prospective trials, clinical and non clinical studies have identified a group of independent risk factors for myocardial infarction, stroke and peripheral vascular disease among which pre existence of atherosclerotic vascular disease, age, male sex, a positive family history of premature atherosclerotic lesion, history of smoking, diabetes mellitus, dyslipidemia and low serum HDL cholesterol have been considered as classical risk factors [12].

A single risk determinant does not carry very much significance but a combination of various risk factors carry great weight. Therefore at present the most advanced method for assessing the risk of coronary heart disease is to combine the information of various risk factors in algorithm or score. This method will be taken as the best method to assess whether a person will have ischemic insult to heart within next decade. Many algorithms have been formed worldwide but the best out of them are FRAMINGHAM guidelines from USA and Germany and PROCAM trials. [14,15] ·Current international guidelines bass their recommendations for the starting hypolipidemic drug treatment in a clinically asymptomatic patient on the estimation of global risk.

Even with the best screening strategy, the chance of detecting the people with the low risk of coronary illness will require cost intensive screening of big population but in

that situation the presence of high false positive results will lead to the wrong treatment to the general public. Thousands of cross sectional, case control studies have shown that hundreds of clinical, biochemical and genetic factors that shows statistically significant association with ischemic heart disease, stroke, PVD. Most of this association aren't reproducible in other studies or not an independent clinical risk factors. However some of these emerging risk factors appears to be genuinely independent and not dependent on classical risk factors. Currently there is an immense discussion about their utility in the routine risk assessment. Of all these, this specially concerns lipoprotein A, C reactive protein, homocysteine, fibrinogen and microalbuminuria[13].

C REACTIVE PROTEIN

CRP levels more than 1mg/L is associated with mild risk while a level above 3mg/L have been associated with significant risk of coronary arterial disease. Both types of inflammation can affect the CRP levels and it is usual to find levels more than 10mg/L and the patient is still in good shape [16..]

LIPOPROTEIN A

Most of the laboratories take a standard value of 30ng/mL above which the risk of ischemic heart disease is to be suspected in spite the patient should have high genetic predisposition for blood lipoprotein A level. It will cause renal impairment and over excretion of proteins but doesn't have much intra individual variation. It will result in high serum lipoprotein A, in other terms lipoprotein A level aren't of much significance, but prevalence of variability in apolipoprotein A ultimately will determine the patient's susceptibility to cardiac illness.

FIBRINOGEN

Fibrinogen test have not been standardised internationally like CRP and lipoprotein A. Citrate is needed to formulate the specimen which makes it tedious. Fibrinogen is an acute phase marker so it is useless in patients having acute and chronic inflammation.

MICROALBUMINURIA :

DEFINITION:

A Consensus conference in 1995 described microalbuminuria as abnormal in people with impaired glycemic metabolism as an abnormal excretion of albumin between the range of 20-200 mcg/min or 30-299 mg/day. This definition is still used today and is applicable to all people regardless of associated pathological conditions [17.] .

After the research of VIBERTI and colleagues , the word microalbuminuria has come into usage but till now there is no demarcated values for inference and is bound to get alterted on the basis of results. [14, 18]

All the public screening and survey data has given the fact that microalbuminuria has been closely associated to increased risk for generalised morbidity and mortality, stroke and probably implicated in pathogenesis of peripheral vascular disease.

As a result of seven year follow up study of apparently healthy subjects (whom do not have comorbid factors like hypertension diabetes, and cardiovascular disease) the elicited inference strongly suggested a linear relationship between patients with UAE, not developing cardiac pathology, it is also applicable in cohort subjects having excretion of albumin just below the minimum cut off for microalbuminuria.

In the heart outcome prevention evaluation (HOPE) study, UAE predicted mortality in patients who are at cardiovascular risk (55 years of age with CVD or Diabetes mellitus and atleast one other cardiovascular risk determinant) . The causes of mortality due to all causes was 9.4% among patients without microalbuminuria vs 18.2% among those with microalbuminuria (relative risk 2.09% , 95% confidence interval). A direct association was also observed between microalbuminuria level and cardiovascular events which extend below the traditional microalbuminuria threshold. The incidence of microalbuminuria also seems to predict all cause of death in the general population. This was initially depicted in Prevention of Renal and Vascular end stage disease (PREVEND study), where natives of city of Groningen, Netherlands in the age group between 29-77 years where they were given with a verbal question bank and provided a bottle to collect an early morning urine sample for measurement of urinary albumin excretion [19]. A total of 40,458 Candidates who were followed for 2.8 years were included in the analysis of mortality by baseline UAE . A clear positive relationship was observed between Urinary albumin excretion and all causes of death due to cardiovascular and non cardiovascular cases. In the STENO hypothesis put forward by DECKERT et al, albumin excreted into the urine is a reflection of widespread vascular damage. This hypothesis states that impaired vascular endothelial function with vascular permeability of albumin . The strong heart TRIAL shows relationship of microalbuminuria and abnormal echocardiographic parameters, both right and left ventricle systolic and diastolic work assesment in a group of 1578 native Americans with impaired glucose tolerance [20].

Table 1: Clinical studies pertaining to the risks associated with positive microalbuminuria result.

A. PROSPECTIVE STUDIES

No.	Study	Microalbuminuria definition	Population	Risk associated with positive microalbuminuria result.(95% CI)
1	Prospective Studies HOPE	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	UAE 30 to 300 mg in a 24-h urine Sample	Residents of Groningen, the Nederland, 28 to 75 yr (n = 7330)	All-cause mortality: HR 3.3 (1.5 to 7.1) for patients with ST-T segment changes 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 _ microalbuminuria versus 2.7(0.6 to 12.3) for ST-T segment changes alone
3	PREVEND	UAE 20 to 200 mg/L in	Residents of Gronngen, the	Cardiovascular death:
		an early morning spot urine sample	Nederland, 28 to 75 yr (n = 40,548)	RR 1.29 (1.18 to 1.40) Non cardiovascular death: RR 1.12 (1.04 to 1.21)

B. CROSS SECTIONAL STUDIES:

No.	Study	Microalbuminuria definition	Population	Risk associated with positive microalbuminuria result. (95%CI)
1	Hoorn Study	ACR >= mg/mmol in a first morning spot urine sample	Population-based: White individuals, 50 to 75 yr (n = 631)	Cardiovascular death: RR 3.22 (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)
2	Danish MONICA	ACR >0.65 mg/mmol (>90thpercentile) in a firstmorning spot urine sample	Population-based: Individuals without Ischemic Heart Disease, renal disease, urinary tract infection, or diabetes (n =2085))	Ischemic Heart Disease: RR 2.3 (1.3 to 3.9)
3	Shibata Study	Positive albumin dipstick test	Residents of Shibata, Japan, >40 yr (n = 2651)	Stroke: RR in men 2.5 (1.1 to 5.7)
4	Slowik et al.	UAE 30 to 300 mg in a 24-h urine Sample	Patients admitted within 24 h of A first ischemic stroke (n = 60)	Mortality: OR 6.0 (1.3 to 27.7)
5	HUNT	ACR >=76 mg/mmol (_60th percentile) in a first morning spot urine sample	Nondiabetic, nonhypertensive residents of Nord-røndelag,Norway, >=20yr (n =2089)	All-cause mortality: RR 2.3 (1.0 to 5.4)
6	Third Copenhagen City Heart Study	UAE >.8 _g/min (> 3 rd quartile) in a timed overnight urine sample	Residents of Copenhagen, Denmark, 30 to 70 yr, without coronary heart disease	All-cause mortality: RR 1.9 (1.5 to 2.4) Coronary heart disease: RR 2.0 (1.4 to 3.0)

7	Cross-sectional 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)
8	EPIC-Norfolk	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: HR 1.48 (1.20 to 1. 79)Cardiovascular death: HR 2.03 (1.55 to 2.67) Fatal stroke: HR 1.58 (1.10 to 3.0)Coronary heart disease death: HR 2.01 (1.40 to 2.90)
9	Earle et al.	UAE 20 to 200 _g/min in a timed overnight urine sample	Patients with type 1 diabetes and without CVD	Silent myocardial ischemia: OR 6.3 (1.2 to 37.8)
10	PREVEND	UAE 30 to 300 mg in a 24-h urine sample	Nondiabetic residents of Groningen, (, 28 to 75 yr(n = 7579)	Electrocardiographic abnormalities: Infarct patterns: OR 1.61 (1.12 to 2.32) Major ischemia: OR 1.43 (1.08 to 1.91) Minor ischemia: OR 1.32 (1.03 to 1.68)
11	Portland 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)
12	EPIC-Norfolk	ACR 2.5 to 25 mg/mmol	Residents of Norfolk, UK, 40 to 79 yr (n _=23,630)	Stroke: HR 1.49 (1.13 to 2.14)
		in a random spot urine sample		

Through a low capacity phagocytic mechanism, the proximal tubule reabsorbs the majority of the filtered albumin. The amount of albumin in urine will be between 15 and 28 mg every 24 hours. A unit increase in the generalised vascular permissiveness of system in response to an insult or an injury would result in an additional 75 mg of albumin passing past the filters, if we assume that 8 grammes of albumin are processed daily. Urinary albumin excretion will increase from a range of 30 to about 120 mg in 24 hours as a result of the fact that the tubular reabsorption of albumin's corrective mechanisms have already been totally exhausted.[21] Both the charge and size selectivity found on the endothelium are related to the nephronal permissiveness to albumin. The glycoproteins that make up the nephronal membranes will give them a negative charge as a result of their role in preventing anion loss.

Additionally, alterations in the glomerular blood pressure regulation that affect how much plasma is processed by the nephrons will result in rather large fluctuations in the amount of albumin excretion. With arterial endothelial dysfunction playing a role in the aetiology of atherothrombotic vascular disease, microalbuminuria may be a sign of widespread vascular pathology.

There is currently no agreement on the precise mechanism through which MA promotes the development of atheroma and ensuing degeneration.

However, it is now believed that patients with or without diabetes and co-existing hypertension have different pathways of vascular damage due to MA.[22]

The endothelial dysfunction and alterations in cellular participation in MA patients who do not have diabetes will increase vascular leakage, which in turn speeds up the degenerative process. Lipid inflow into the tunica media and intima of vessels due to aberrant endothelial leakage in atherosclerosis results in degenerative alterations. Since hypertension is the only known cause of non-diabetic vascular or renal illness, it is

currently believed that MA won't be directly linked to these diseases' development. We can connect to the association that the albumin moiety doesn't have a glycated state thanks to this uncertainty. The endothelium aids in the creation of various crucial cardiac and renal proteins as well as cellular matrix proteins. Atheromas are mostly brought on by an imbalance between normal endothelial function and a lack of homeostasis between vasodilatation and antithrombosis. Therefore, it has been believed that impaired endothelium permeability may be the cause of MA in the general population.[23]

The endothelial dysfunction and alterations in cellular participation in MA patients who do not have diabetes will increase vascular leakage, which in turn speeds up the degenerative process. Lipid inflow into the tunica media and intima of vessels due to aberrant endothelial leakage in atherosclerosis results in degenerative alterations. Since hypertension is the only known cause of non-diabetic vascular or renal illness, it is currently believed that MA won't be directly linked to these diseases' development. We can connect to the association that the albumin moiety doesn't have a glycated state thanks to this uncertainty. The endothelium aids in the creation of various crucial cardiac and renal proteins as well as cellular matrix proteins. Atheromas are mostly brought on by an imbalance between normal endothelial function and a lack of homeostasis between vasodilatation and antithrombosis. Therefore, it has been believed that impaired endothelium permeability may be the cause of MA in the general population.[23]

Endothelial dysfunction is not a discrete process, but a number of studies and observations indicate that it may share a pathophysiology with both micro and macro vascular morbidities. When it comes to atherosclerosis from non-diabetic sources, endothelial dysfunction will be crucial. The intima's altered permissiveness allows lipoproteins (oxidised LDL) to enter major blood arteries and contribute to the formation of plaques. The impairment of insulin action is caused by the prevention of insulin-aided

skeletal myocyte vasodilation, which interferes with insulin-associated glucose absorption, and the accentuation of vascular leakage along with a diminished responsiveness of beta-receptor.

Microalbuminaria has been linked to biochemical markers of endothelial dysfunction such as increased von Willebrand factor and enhanced platelet aggregation. It was found that individuals with microalbuminuria had higher plasma levels of vWF antigen than those with normal albumin excretion, both in diabetic patients and non-diabetic subjects with essential hypertension. An rise in serum levels of angiotensin II, PA), and a thrombotic profile composed of plasminogen activator and inhibitor-1 (PAI-1) and new platelets are further biochemical indicators of endothelial dysfunction.[24] Therefore, it can be inferred that endothelial dysfunction contributes significantly to the development of atherosclerosis,MA-assisted insulin sensitivity, and glomerulosclerosis in non-diabetics. MA has been linked to enhanced cardiac mortality and morbidity in non-diabetics, but it is also an early signal of target end organ damage associated with CVD. According to Aggerwall and colleagues, patients with MA had a considerably greater incidence of ischemic heart disease, CVA, and peripheral artery disorders.

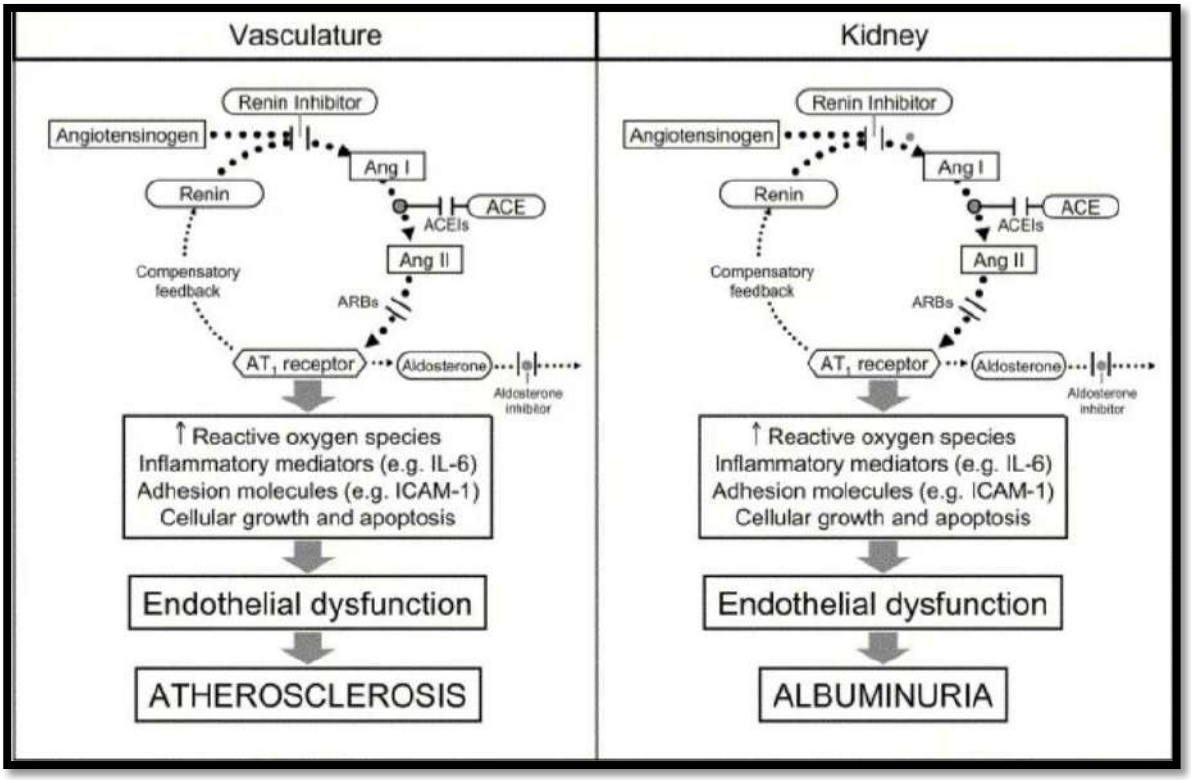
The endothelial dysfunction and alterations in cellular participation in MA patients who do not have diabetes will increase vascular leakage, which in turn speeds up the degenerative process. Lipid inflow into the tunica media and intima of vessels due to aberrant endothelial leakage in atherosclerosis results in degenerative alterations. Since hypertension is the only known cause of non-diabetic vascular or renal illness, it is currently believed that MA won't be directly linked to these diseases' development. We can connect to the association that the albumin moiety doesn't have a glycated state thanks to this uncertainty. The endothelium aids in the creation of various crucial cardiac and renal proteins as well as cellular matrix proteins. Atheromas are mostly brought on

by an imbalance between normal endothelial function and a lack of homeostasis between vasodilatation and antithrombosis. Therefore, it has been believed that impaired endothelium permeability may be the cause of MA in the general population.[23]

Endothelial dysfunction is not a discrete process, but a number of studies and observations indicate that it may share a pathophysiology with both micro and macro vascular morbidities. When it comes to atherosclerosis from non-diabetic sources, endothelial dysfunction will be crucial. The intima's altered permissiveness allows lipoproteins (oxidised LDL) to enter major blood arteries and contribute to the formation of plaques. The impairment of insulin action is caused by the prevention of insulin-aided skeletal myocyte vasodilation, which interferes with insulin-associated glucose absorption, and the accentuation of vascular leakage along with a diminished responsiveness of beta-receptor.

Microalbuminaria has been linked to biochemical markers of endothelial dysfunction such as increased von Willebrand factor and enhanced platelet aggregation. It was found that individuals with microalbuminuria had higher plasma levels of vWF antigen than those with normal albumin excretion, both in diabetic patients and non-diabetic subjects with essential hypertension. An rise in serum levels of angiotensin II, PA), and a thrombotic profile composed of plasminogen activator and inhibitor-1 (PAI-1) and new platelets are further biochemical indicators of endothelial dysfunction.[24] Therefore, it can be inferred that endothelial dysfunction contributes significantly to the development of atherosclerosis,MA-assisted insulin sensitivity, and glomerulosclerosis in non-diabetics. MA has been linked to enhanced cardiac mortality and morbidity in non-diabetics, but it is also an early signal of target end organ damage associated with CVD. According to Aggerwall and colleagues, patients with MA had a considerably greater incidence of ischemic heart disease, CVA, and peripheral artery disorders.

Illustration showing vicious cycle by which microalbuminuria causes endothelial injury and end organ damage



Microalbuminuria and its relation with endothelial injury

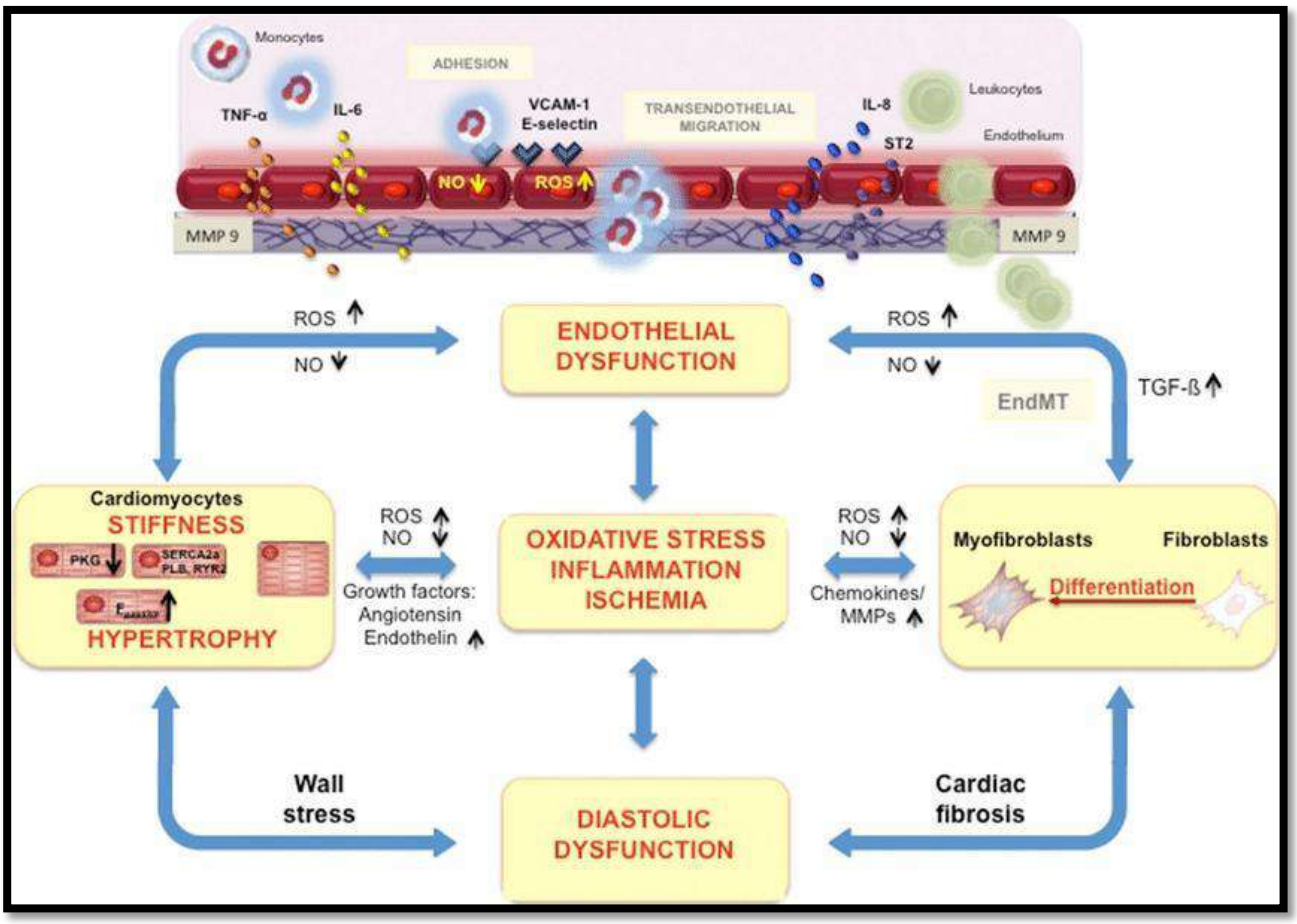


Table 2:Pathologic steps in relation with MA

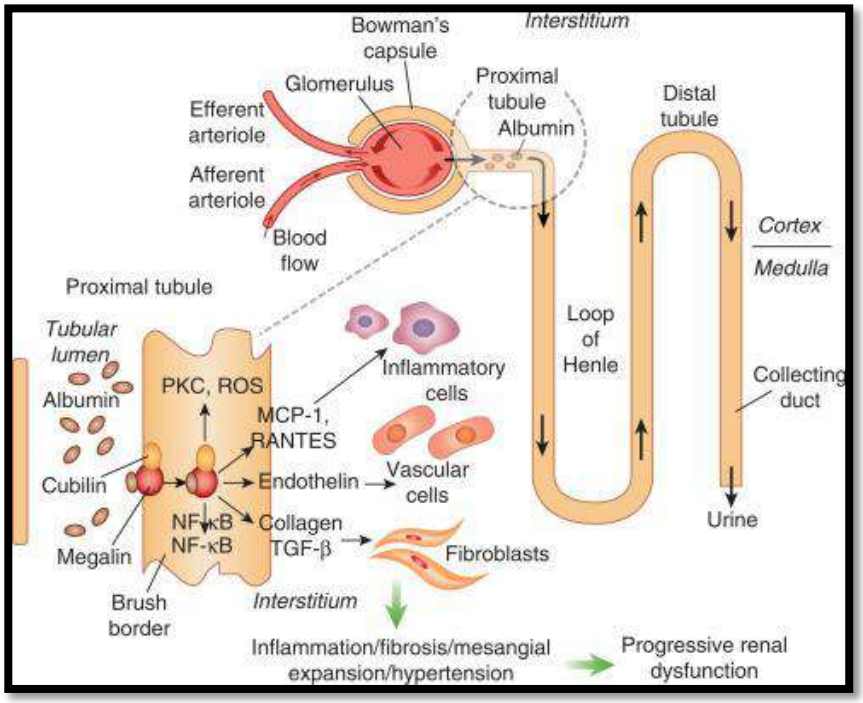
LOCAL STEPS
1.Increased intraglomerular capillary oncotic Pressure
2.More prominent transport of albumin mediated via the pores in glomerulus

SYSTEMIC STEPS
1.Activation of inflammatory mediators cascade
2.Increased leakage of intracapillary albumin
3.Malfunction of endothelial vessels

Table 3: Factors influencing the MA development

1.Increased BP (Both systolic and diastolic)
2.Insulin resistance (syndrome X)
3.High body mass index
4.Dyslipidemia
5.Increased sensitivity to salt
6.Malfunctioning of endothelium
7.Old age
8.History of cigarettes smoking and beedi

Multiple effects of Microalbuminuria on renal systems



The degree of morbidity has a direct relationship with MA. These two conditions are ischemia and reperfusion, respectively. Additionally, MA has been observed in cases of acute myocardial infarction and peripheral artery disease, and it has a direct correlation to the severity of the infarct or the degree of claudication. the STENO hypothesis put forth by Deckard and associates. Urinary albumin leakage is an indication of widespread vascular injury[25]. Thus, the nephron is considered to be a crucial component of the vascular system. There may be other approaches to establish a connection between micro albuminuria and ischemic heart disease in place of the theories of endothelial dysfunction and chronic persistent inflammation. But the book has a lot of contradictions.

Although it is true that persistent inflammation can lead to endothelial dysfunction, some studies have used markers like TNF- IL-1 and IL6, which suggest that persistent inflammation is linked to the development of microalbuminuria and is associated with a higher risk of atherosclerotic vessel damage. However, according to a different set of studies, despite their connections to microalbuminuria, endothelial dysfunction, and persistent and low-grade inflammation, these factors are also thought to be independent risk factors for cardiac mortality.[26] Numerous case-control, cohort, prospective trials, and cross-sectional studies revealed relationships between microalbuminuria and a number of cardiac risk factors, including age, a history of hypertension, diabetes, smoking, the prevalence of obesity, and an abnormal lipid profile. These only partially explain the relationship between microalbuminuria and kidney disease.

Therefore, there is a chance that endothelium function and inflammation will contribute to both microalbuminuria and ischemic heart disease. However, another theory is that most people have varying degrees of vessel derangement within the normal range and, as a result, excrete varying amounts of micro albumin. Urine microalbuminuria

elimination can be used to evaluate the status of the vessel wall's current vagarisms, which may be associated to the possibility of additional end organ injury. This explains why micro albuminuria is a reliable indicator of ischemic heart disease, as well as of recent-onset systemic hypertension, glycemic dysfunction, and hypertension.

This situation, therefore it is advised to mark these susceptibility to assess early primary prevention. In such a scenario, the renal system may serve as an approximate measure of BP; that is, goal BP where normal urinary albumin excretion is also present. In terms of therapy, stabilising albumin elimination along with strong sugar control and LDL reduction will be a good sign of prognosis.[27]

In the Islington Diabetes trials, Yudkkin et al. conducted a cohort analysis of MA (24-hour daytime UAE > 30 microgram/min) with evaluation of cardiovascular and peripheral artery disease risk in a cohort of 189 people (58 with diabetes or impaired glucose tolerance). After a median follow-up of 3.7 years, Yudkkin et alcohort .'s study showed that MA had an independent relationship with the prevalence of all-cause morbidity and mortality [7/19 (34.12%)] vs. [3/148 (2.03%)] in 167 patients.[33]

In the recently completed PREVEND experiment, 7589 people without a history of diabetes who received MA (30-300 mg/24 h) showed a consistent and independent relationship with electrocardiographic prevalence of either infarction or the presence of ischemia.[29] In addition, the HOPE experiment showed that MA (MA: ACR 3mg/mmol) plays a significant and independent function as a predictor among 5709 people without diabetes who have well-established vascular pathology[29]. Regardless of age, blood pressure, glycemic impairment, dyslipidemia, or serum fibrinogen, the presence of ischemic cardiac disease, CVA, and peripheral arterial disease in American Indians who

were non-diabetics in the Strong Heart trials was associated with micro albuminuria prevalence (ACR - 32 and 320 mg/g).[39]

In 1079 non-diabetic Kuopo community members who were prospectively followed up for 3.5 years, the incidence of death from cardiac and coronary diseases doubled. This was in response to an ACR of > 3.32 mg/mmol. The relationship was lessened when other confounding factors, such as hypertension, were adjusted for. In their groundbreaking study, Damsgaard et al. found that microalbuminuria is linked to three times greater mortality.[30,31]

When compared to individuals below (43/109 vs. 9/108), albuminuria was equivalent to or significantly higher in patients with normal blood sugar levels (>7.62 micro g/min). It's possible that there is a pathophysiologic link between microalbuminuria and ischemic heart disease that is more than just coincidental. Despite the fact that various abnormalities in the vascular wall's physiology predispose to microalbuminuria and ischemic heart disease Atherosclerosis is thought to be caused by endothelial dysfunction, which is also thought to play a big part in how the disease will progress. Consequently, a link between microalbuminuria and extensive endothelial dysfunction may exist, which, if so, would explain why microalbuminuria is a reliable indicator of ischemic heart disease.

As determined by blood levels of chemokine agents like tissue- plasminogen type activator, vascular cell, and soluble variety of E- selectin as well as by endothelium-dependent dilatation of blood vessels as a response to augmentation in flow or increase in cholinergic chemicals, microalbuminuria in both types of diabetes typically is associated with endothelial malfunction and, and EDRF NO synthesis and its availability and acceptability.

A short survey revealed that microalbuminuria was associated with impaired endothelial vascular responsiveness. It was observed in the brachial renal arteries and other abdominal arteries, but little is known about any experiments that have been conducted, for example, in coronary vascular circulation. [32] There have been very few studies on the amount of endothelium dysfunction in non-diabetic patients with micro albuminuria, but it has been hypothesised that endothelium dysfunction exists in diabetes and regulates hemostasis, the fibrinolysis system, polymorph adhesion, EDRF NO Productions, and accessibility.

For instance, a recent large-scale population study with 647 participants (median age 69 years; 249 subjects with normal blood sugar, 139 subjects with impaired glycemic metabolism, and 261 subjects with a history of type 2 diabetes) found that endothelial NO production, as determined by ultrasonically calculated brachial artery endothelial flow-dependent, flow-determined dilation, was abnormal in people with diabetes. [34]These findings emphasise that, regardless of the presence or absence of glycemic impairment, increased endothelial EDRFNO production plays a part in the relationship between microalbuminuria and all types of cardiovascular morbidity and mortality risk. Additionally, multiple studies have emphasised how endothelial dysfunction precedes and predicts the onset of microalbuminuria in both diabetic and non-diabetic patients.

As a result, it is tempting to claim that endothelial dysfunction in microalbuminuria explains why the condition has always existed. a reliable and persistent indicator of an elevated risk of atherothrombosis. In the past, it was thought that the structure of the glomerulus' basement membrane and the architecture of the slit diaphragm were the main determinants of glomerular permissivity. The glomerular endothelium has a considerable active role in determining albumin penetration, according

to recent investigations. For determining glomerular permeability and ion selectivity, the negatively charged glycocalyx that covers the endothelium fenestrations is especially important. [35] Microalbuminuria will be influenced by changes in the outer glycocalix endothelium layer, although this has been linked to the pathophysiology of atherosclerosis.

Recent studies have provided new evidence supporting the Steno theory, which holds that increased albumin leakage from glomerulus vascular disease results from a systemic flow-dependent intravascular loss of albumin. This, in turn, predominately increases the presence of atheromatous lipoprotein plaque molecules in the arterial tunica media.[36]

At this time, endothelial dysfunction and chronic inflammation are recognised as important first events in the phenomena of atherosclerosis. Endothelial dysfunction is caused by long-term low-grade deterioration, and both of these conditions are strongly linked to long-term persistent vascular inflammation, which may be detected by monitoring C-reactive protein levels in both diabetic and non-diabetic patients. When compared to recognised risk variables, microalbuminuria has been linked to a tendency to cardiac risk that is independent. In the future, microalbuminuria may prove to be a valuable tool for more accurate and sophisticated cardiac risk assessment. The specific mechanism through which microalbuminuria is associated to the chance of developing heart pathology is unknown, but it is clear that rigorous monitoring of cardiovascular risk factors is important when the two conditions coexist.

At the moment, it is unclear if microalbuminuria causes atheroma or vice versa. There is some speculation that a common risk factor may be the root of the relationship between microalbuminuria and heart disease and illness, although there is no conclusive

evidence to support this. Microalbuminuria and heart disease may be related by a similar pathophysiology, such as endothelial dysfunction and persistent, chronic inflammation. To advance our understanding in this area, a lot more experiments are undoubtedly required.[37]

MONITORING, SCREENING DIAGANOSIS AND TREATMENT

ALBUMINURIA

BASIC PUBLIC PERSPECTIVE

All healthcare professionals should pay more attention to the early diagnosis and ongoing management of patients with microalbuminuria because it is becoming increasingly known that treating microalbuminuria in patients without glycemic impairment will be a cost-effective alternative to preventing cardiovascular disease. Numerous antibody assays are currently employed to evaluate lower concentrations of both qualitative and quantitative urine albumin. Nephelometry and ELISA are some of these. RIA,immuno turbidimetry[38] The radio immunoassay has been described as the gold standard diagnostic technique for microalbuminuria; many other tests performed in conventional labs are often very sensitive for everyday clinical use. The quantitative liquid phase immunoprecipitation has formed the foundation for immuno turbidimetric testing for microalbuminuria.

An sample of patient urine and a reaction buffer are combined with antibodies against human albumin. Urine albumin and antibodies combine to form an agglutination reaction, which raises the turbidity of the mixture. Using a clinical chemistry analysis at a wavelength of Ca 405 nm, turbidity is assessed.

A unique method for measuring both immunoreactive and immunounreactive albumin was recently developed. A considerable number of patients who were previously not conspicuously albuminuric were discovered to have protein excretion in the microalbuminuric range[40]. Previously, only one kind could be examined with this type of test. However, it is now unknown whether the patients who were screened using the novel method are at the same risk. But doing so is wise. compare the amount of

albuminuria in new to old samples. a very long time Even though it is not the method of choice, it has been considered that the gold standard test for micro albuminaria is 24-hour urine sample screening due to the effort needed. But in reality, there are many challenges with this.

Timed urine collected overnight is the second-best option. Because urine must be collected over a certain period of time, it can only be used for high-risk screening of certain groups, such as individuals with diabetes or hypertension. It has not yet been practicable to screen the entire public. Urinary sample taken in the morning is another choice. This has certain advantages over a spot urine sample because it is typically performed at the same time every day and is less affected by the patient's state and whether or not they are physically active, which will reduce the variance brought on by the aforementioned two determinants.[41]

When a subject visits a doctor in a real clinic, a spot-urinary sample is taken, or the sample is taken in a primary health centre. The elimination of albumin in unit time must be determined per minute or as UEA in a 24 hour sample in order to measure albuminuria (as in regarding timed night samples). It is preferable to evaluate the spot albumin for untimed collection: ratio of creatinine The introduction of the albumin-to-creatinine ratio poses a new difficulty because it calls for the use of changing. Creatinine elimination is reliant on age and race rather than gender in abnormal outcomes for both men and women. For the measurement of microalbuminuria in terms of albumin creatinine ratio, urine albumin testing from a spot sample is preferable.[42]

If a patient has positive microalbuminuria, he should ideally undergo repeated screenings. A test should ideally be repeated two or three times. If the best of the three numbers is positive, it is regarded as positive.[43,44]Recently, it has been hypothesised that

micro albuminaria may emerge before overt glycemic impairment and systemic hypertension. It has also been proposed that microalbuminaria could be an indication of insulin resistance. Additionally, research has demonstrated a direct correlation between the quantitative excretion of microalbuminuria and the overt expression of metabolic syndrome X. All of these makes us question the use of using micro albuminaria as a diagnostic and screening tool for a variety of non-communicable diseases as opposed to viewing it as an unimportant bystander.[45]

METHODOLOGY

Study Design

Randomised hospital based study.

Study subjects

Hundred and thirty seven (137)non-diabetic subjects with Ischemic Heart Disease attending the outpatient clinic or admitted as inpatients in the Department of Medicine and cardiology wards.

Inclusion Criteria

The diagnosis of Ischemic Heart Disease was based on the 12 lead ECG and exercise thread mill testing (in two subjects), cardiac enzyme estimation and the Rose questionnaire. Also echocardiogram was done to asses ejection fraction

Exclusion Criteria

1. Diabetic patients by ADA criteria (2004).

2. Congestive cardiac failure as presentation.

3. Urine showing

- Macro albuminuria (dipstick positive albuminuria)

- RBCs > 50/µl

- Leucocytes > 75/µl

4. Female patients with vaginal discharge

Method of collection of data

137 participants were included in this retrospective investigation conducted at a hospital. Clinical history, examinations, and investigations were used to acquire the data. These information was entered in accordance with the proforma that is an annexure. Patients who were initially self-reported, infarct pattern, major or mild ischemia by ECG or TMT positive for inducible ischemia, were included in the study. Estimates of cardiac enzymes were made. The ejection fraction was assessed using echo cardiography. The patients received a container for collecting urine during a 24-hour period, which was then sent for immunological turbidimetry assessment of the microalbuminuria level. The outcome was given as x mg/day of albumin.

Statistical Analysis

The data is presented as Mean ± SD. The limit of significance was calculated using SPSS Version 15 software

Statistical Software

Both MS word and excel of windows 7 ultimate were extensively utilized for drawing graphs, tables, etc. Statistical software namely ANOVA & SPSS Version 16 was used for the analysis of data.

Algorithm for assessment of microalbuminuria in diabetics and hypertension

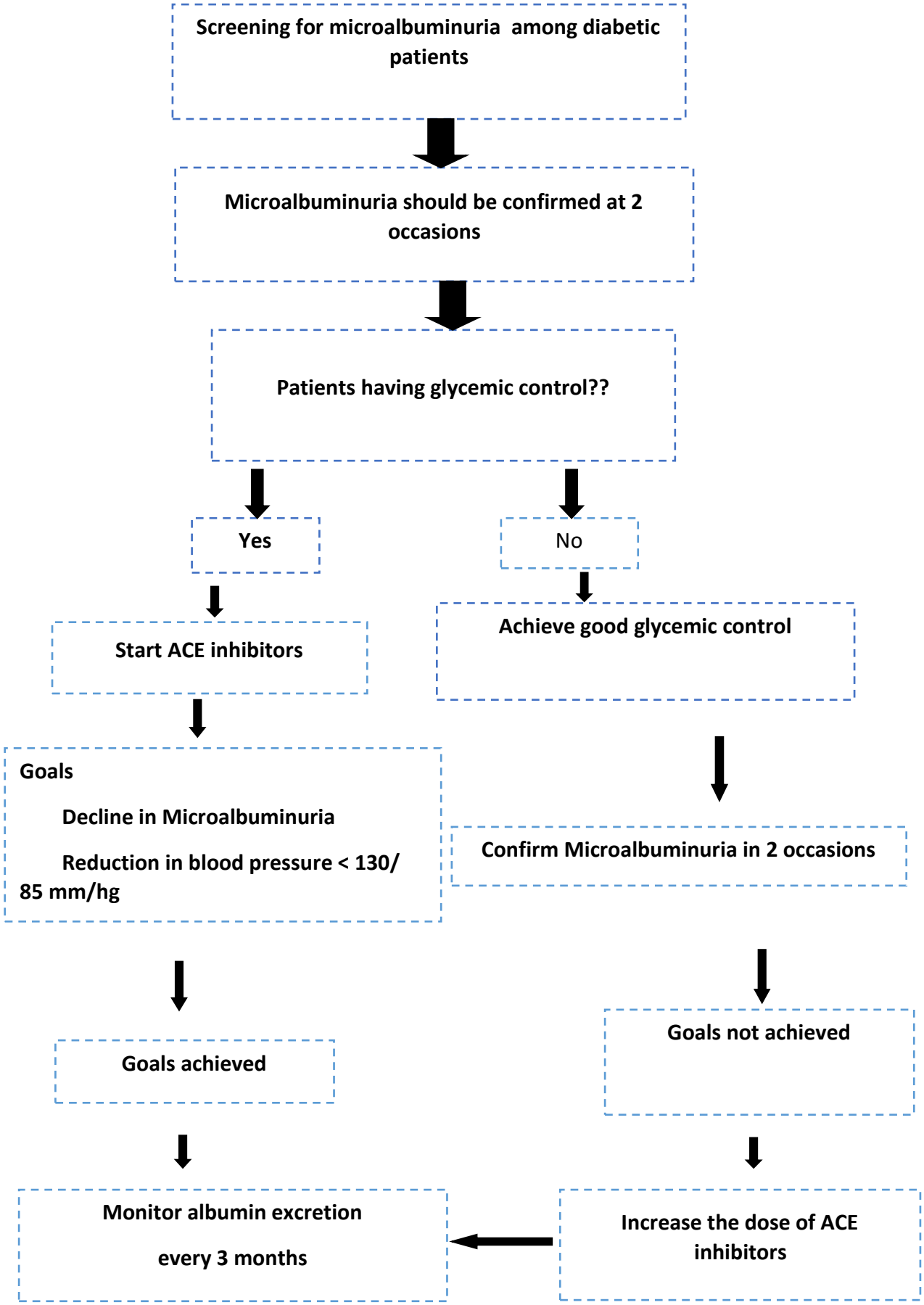


Table 5:Classification of abnormal urinary albumin excretion

	24 hr urine albumin (mg/24 hr)	Overnight Urine albumin (microgram/min)	Spot urine			
			Albumin (mg/L)	Alb/Creatinine proportion		
				gender	mg/mmol	mg/g
Normal	<15	<10	<10	male	<1.30	<11
				female	<1.77	<16
High limit	16 to <30	11 to <21	11 to <21	M	1.25 to <2.5	10 to <20
				F	1.76 to <3.5	15 to <29
Macroalbuminuria	>300	>200	>200	M	>25	>200
				F	>35	>300
Microalbuminuria	32 to <299	22 to <100	22 to <220	M	2.6 to 26	22 to <200
				F	3.6 to 36	30 to <300

Table 4:List comparing horizontal (C) and cohort (P) trials showing microalbuminuria in non diabetics And urinary albumin excretion which is used as predictor of cardiac pathology

No	Author/ref.	Year	No	Design	End point	Population
1	Yudkin	1988	187	P	ECG changes Incidence of MI,PVD and Angina	Diabetes, IGT Non Diabetic patients
2	Damsgaard	1999	217	C	Deaths in total	Normal subjects
3	Damsgaard	1992	218	C	ECG abnornality	Normal patients
4	Haffner	1990	316	P	MI	Normal subjects
5	Ljungman	1996	120	P	PVD	HTN s and normal people
6	Kuusisto	1997	1079	C	CAHD Fatal and non fatal	Normal patients
7	Howard	2002	4569	P	Definite MI	Diabetic Americans
8	Bigazzi	1998	92	P	MI	Normotensives
9	Jensen	1997	2613	P	MI	Both groups
10	Pontremoli	2002	53	C	Subclinical carotid atherosclerosis	Hyper and normotensive subjects
11	Jensen	2003	1254	P	Previous CAHD	Hypertensives
12	Beamer	1999	121	P	Stroke ,MI	Diabetics and normal patients
13	Pedrinelli	2000	136	C	Carotid ischemia	Uncomplicated hypertensive men
14	Jager	1999	631	P	All CVS Mortality	Both groups
15	Gerstein	2001	5545	P	All CAHD mortality	Non diabetic with cardiovascular disease
16	Gerstein	2000	5708	C	Peripheral vascular disease	Nondiabetic with cardiovascular disease
17	Roest	2001	1118	P	MI,angina	Postmenopausal women
18	Gould	2001	957	P	MI,PVD	Normal patients
19	Mykkanen	2004	1441	C	Subliminal carotid atherosclerosis	991 non diabetics ,450 diabetics
20	Fabsitz	2001	4276	C	Ankle/Brachial index less than 0.9	American diabetics

RESULTS

Table 1. Distribution of age group

Age group	Frequency	Percent
<35	12	8.8%
36-45	28	20.4%
46-55	24	17.5%
56-65	47	34.3%
>66	26	19.0%
Total	137	100.0%

In the present study, most of the patients were aged 56-64 (34.3%), age <35 was 12 (8.8%), age 36-45 was 28 (20.4%), age 46-55 was 24 (17.5%), and >66 was 26 (19%).

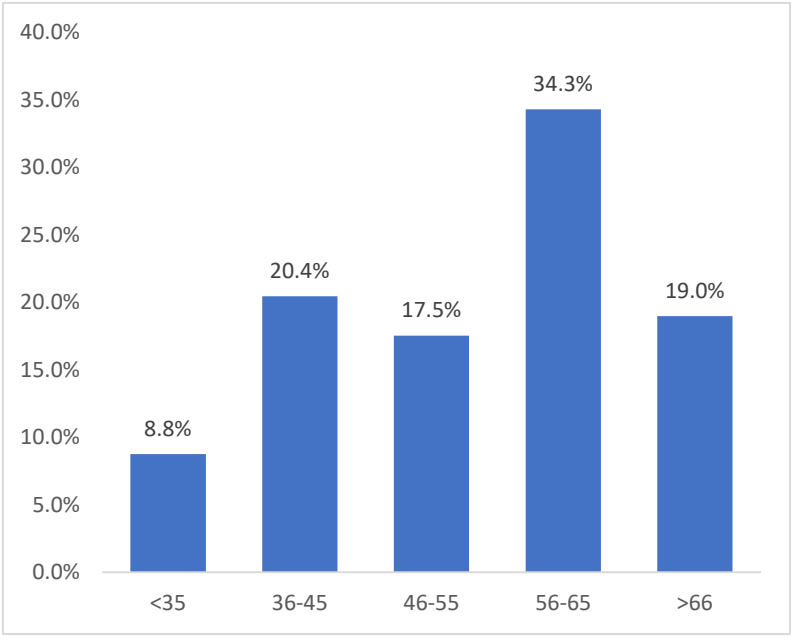


Figure 1. Distribution of age group

Table 2. Distribution of gender

Sex	Frequency	Percent
F	51	37.2%
M	86	62.8%
Total	137	100.0%

In the present study, out of 137, 86 (62.8%) were male, and 51 (37.2%) were female.

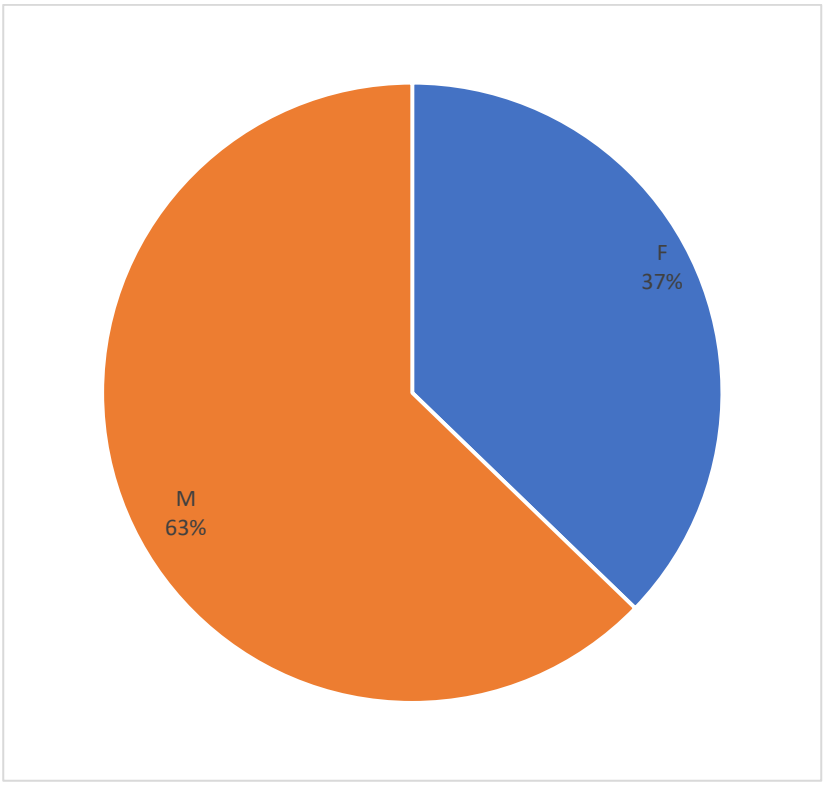


Figure 2. Distribution of gender

Table 3. Distribution of family history of ischemic heart disease

Family History of IHD	Frequency	Percent
NO	74	54.0%
YES	63	46.0%
Total	137	100.0%

Among 137 patients, 63 (46%) had a history of ischemic heart disease, and 74 (54%) had no history of IHD.

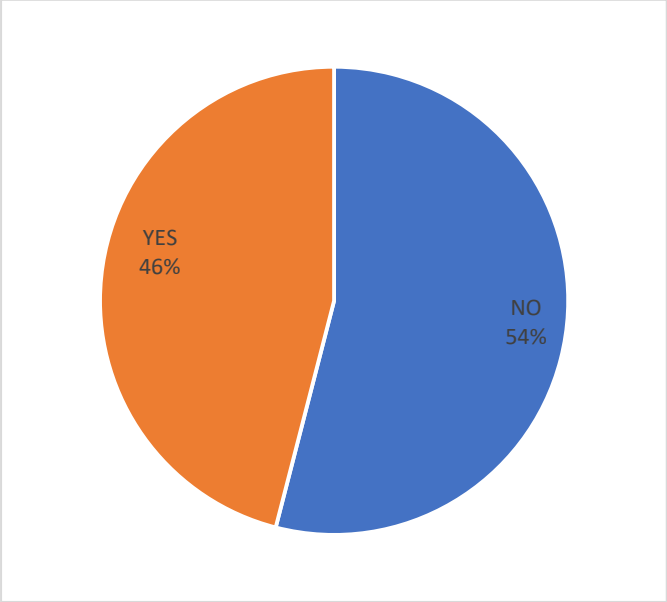


Figure 3. Distribution of family history of ischemic heart disease

Table 4. Distribution of smoking

Smoking	Frequency	Percent
NO	67	48.9%
YES	70	51.1%
Total	137	100.0%

Among 137 patients, 70 (51.1%) patients were regular smoking habits, and 67 (48.9%) were no smokers.

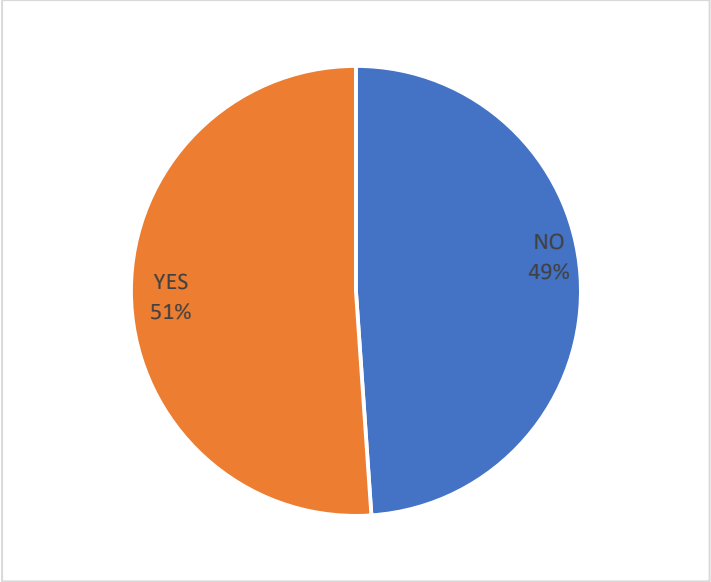


Figure 4. Distribution of smoking

Table 5. Distribution of BMI

BMI	Frequency	Percent
Normal weight	19	13.9%
Overweight	109	79.6%
Obese	9	6.6%
Total	137	100.0%

In BMI, most of the patients were overweight 109 (79.6%), patients with normal weight were 19 (13.9%), and obese were 9 (6.6%).

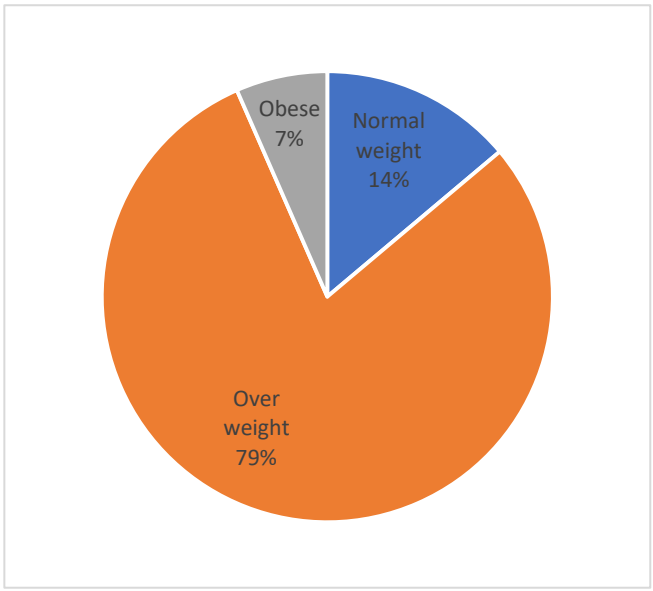


Figure 5. Distribution of BMI

Table 6. Distribution of infarcts

INFARCT	Frequency	Percent
AMI	74	54.0%
IWMI	10	7.3%
NO	53	38.7%
Total	137	100.0%

In the present study, out of 137, acute myocardial infarction was observed in 74 (54%) patients, and IWM was in 10 (7.3%) patients.

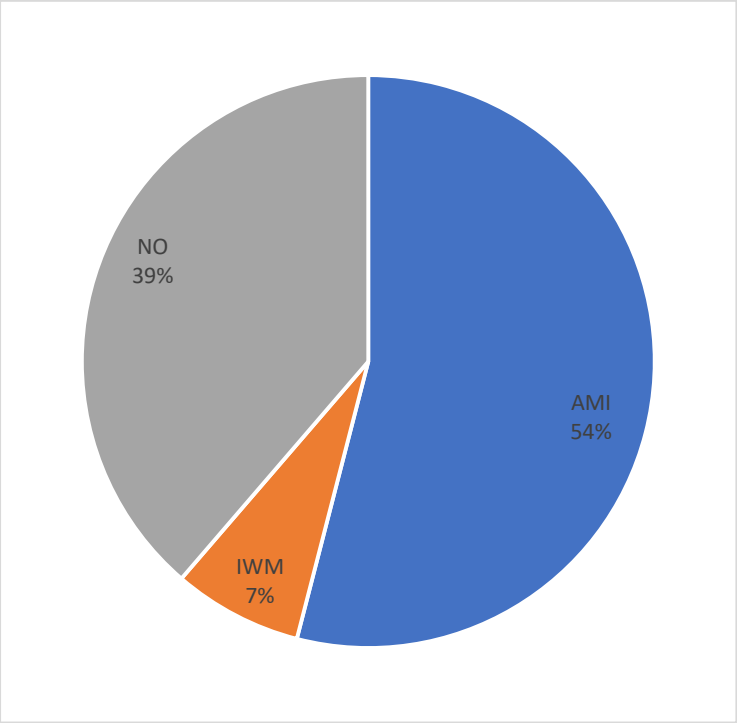


Figure 6. Distribution of infarcts

Table 7. Distribution of ischemia

ISCHEMIA	Frequency	Percent
NO	83	60.6%
YES	54	39.4%
Total	137	100.0%

Among 137 patients, 54 (39.4%) had ischemia, and 83 (60%) had no ischemia.

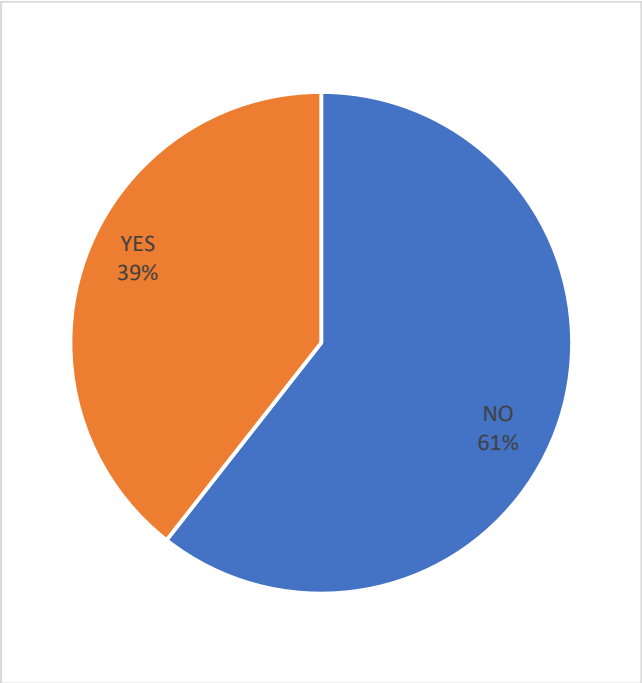


Figure 7. Distribution of ischemia

Table 8. Distribution of troponin T

TROP T	Frequency	Percent
NEGATIVE	29	21.2%
POSITIVE	108	78.8%
Total	137	100.0%

Among 137 patients, 108 (78.8%) tested positive for TROP T, and 29 (21.2%) patients were tested negative for TROP T

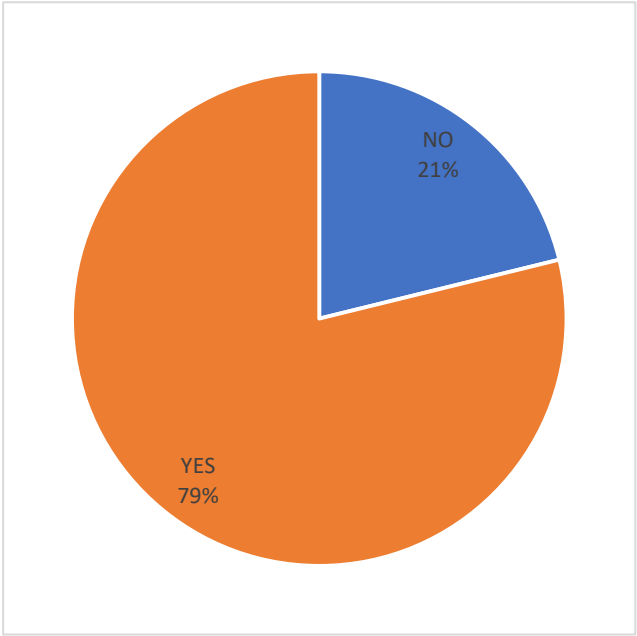


Figure 8. Distribution of Troponin T

Table 9. Distribution of total cholesterol

TC	Frequency	Percent
<200	24	17.5%
>200	113	82.5%
Total	137	100.0%

In the present study, >200 total cholesterol was observed in 113 (82.5%) of patients, and <200 total cholesterol was in 24 (17.5%) of patients.

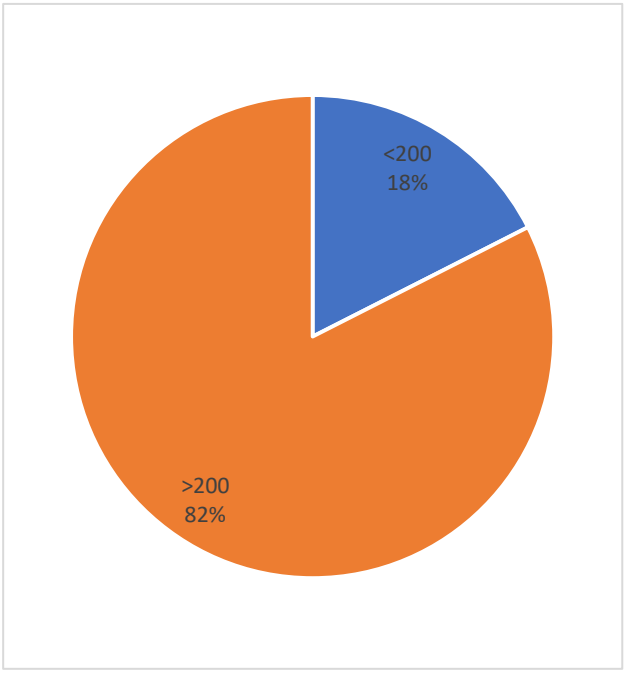


Figure 9. Distribution of total cholesterol

Table 10. Distribution of triglycerides

TG	Frequency	Percent
<150	132	96.4%
>150	5	3.6%
Total	137	100.0%

In the present study, <150 triglycerides were observed in 132 (96.4%) of patients, and >150 triglycerides were in only 5 (3.6%) of patients

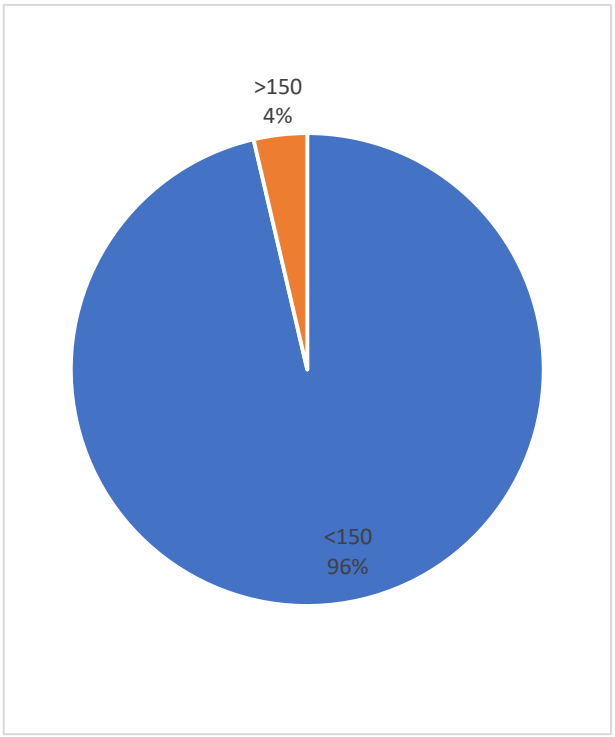


Figure 10. Distribution of triglycerides

Table 11. Distribution of HDL

HDL	Frequency	Percent
<40	43	31.4%
>40	94	68.6%
Total	137	100.0%

In the present study, >40 HDL were observed in 94 (68.6%) of patients, and <40 HDL were in 43 (31.4%) of patients.

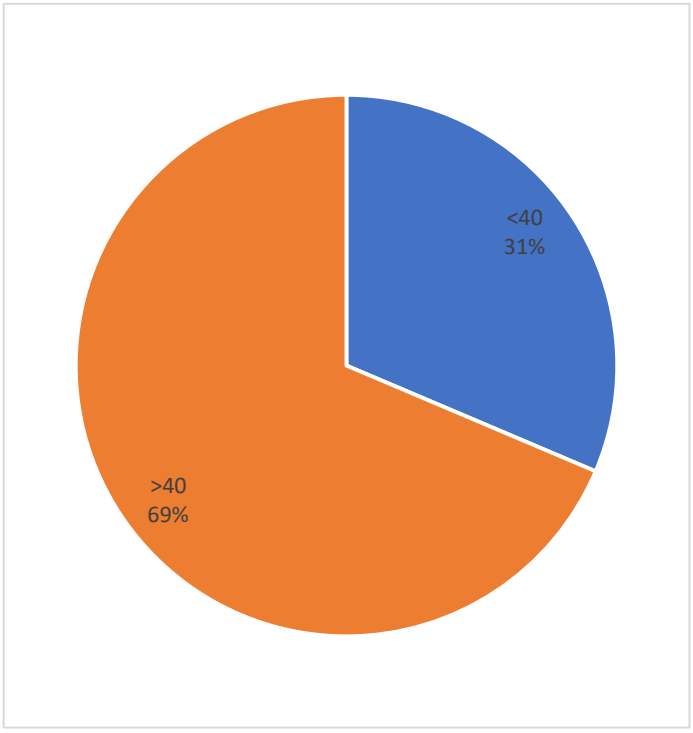


Figure 11. Distribution of HDL

Table 12. Distribution of LDL

LDL	Frequency	Percent
<150	129	94.2%
>150	8	5.8%
Total	137	100.0%

In the present study, <150 LDL were observed in 129 (94.2%) of patients, and >150 LDL were in only 8 (5.8%) of patients.

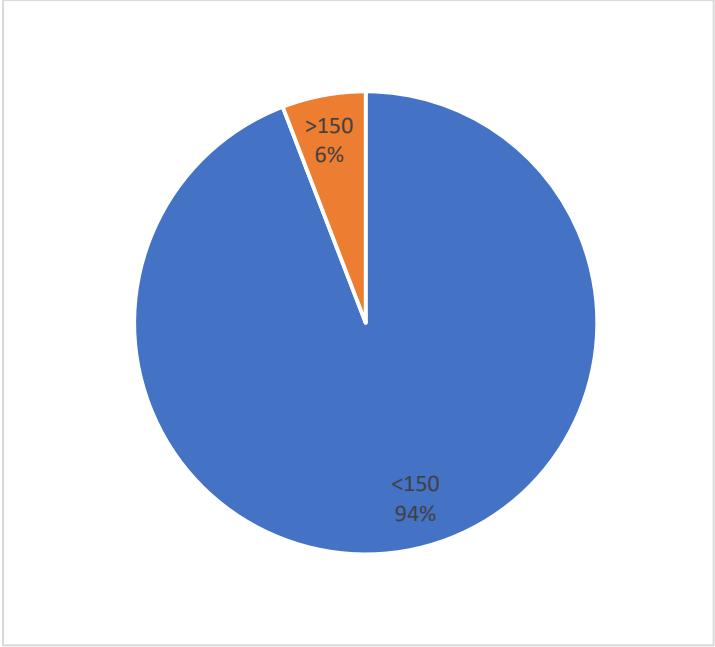


Figure 12. Distribution of LDL

Table 13. Distribution of microalbuminuria

MA	Frequency	Percent
<30	32	23.4%
30-50	61	44.5%
>50	44	32.1%
Total	137	100.0%

In the present study, 30-50 microalbuminuria was observed in most of the patients, 61 (44.5%), >50 microalbuminuria was in 44 (32.1%), and <30 microalbuminuria was in 32 (23.4%).

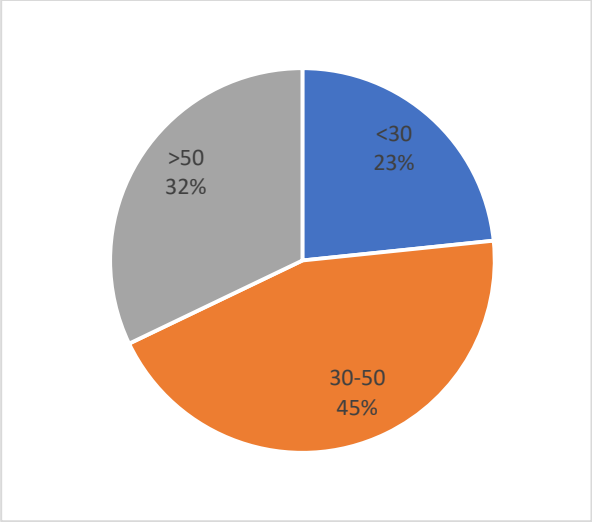


Figure 13. Distribution of microalbuminuria

Table 14. Crosstabulation of microalbuminuria with infarcts

			INFARCT			Total	P value
			AMI	IWM	NO		
MA	<30	Count	17	3	12	32	0.001
		% within MA	53.1%	9.4%	37.5%	100.0%	
	30-50	Count	30	2	29	61	
		% within MA	49.2%	3.3%	47.5%	100.0%	
	>50	Count	27	5	12	44	
		% within MA	61.4%	11.4%	27.3%	100.0%	
Total		Count	74	10	53	137	
		% within MA	54.0%	7.3%	38.7%	100.0%	

There is significant difference in microalbuminuria with infarcts ($p<0.001$).

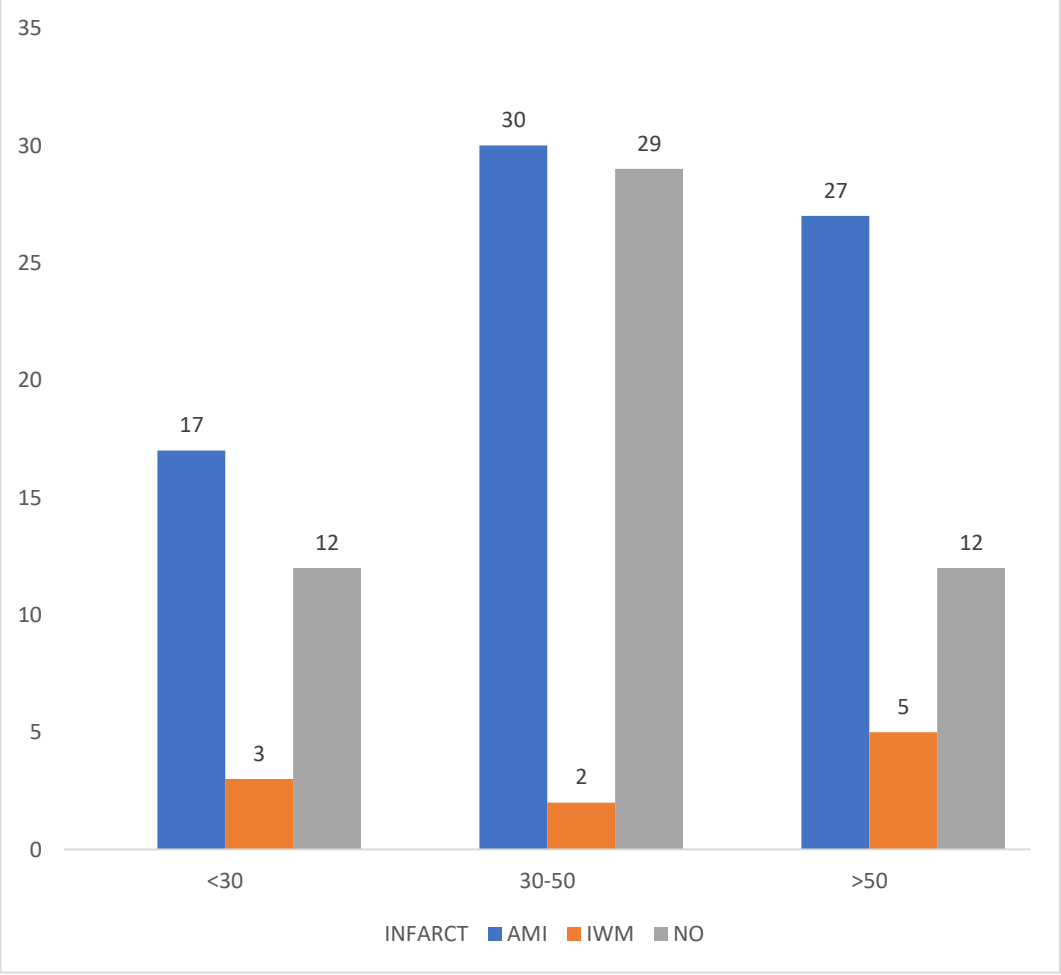


Figure 14. Distribution of microalbuminuria with infarcts

Table 15. Crosstabulation of microalbuminuria with ischemia

			ISCHEMIA		Total	P value
			NO	YES		
MA	<30	Count	19	13	32	0.001
		% within MA	59.4%	40.6%	100.0%	
	30-50	Count	32	29	61	
		% within MA	52.5%	47.5%	100.0%	
	>50	Count	32	12	44	
		% within MA	72.7%	27.3%	100.0%	
Total		Count	83	54	137	
		% within MA	60.6%	39.4%	100.0%	

There is significant difference in microalbuminuria with ischemia ($p<0.001$).

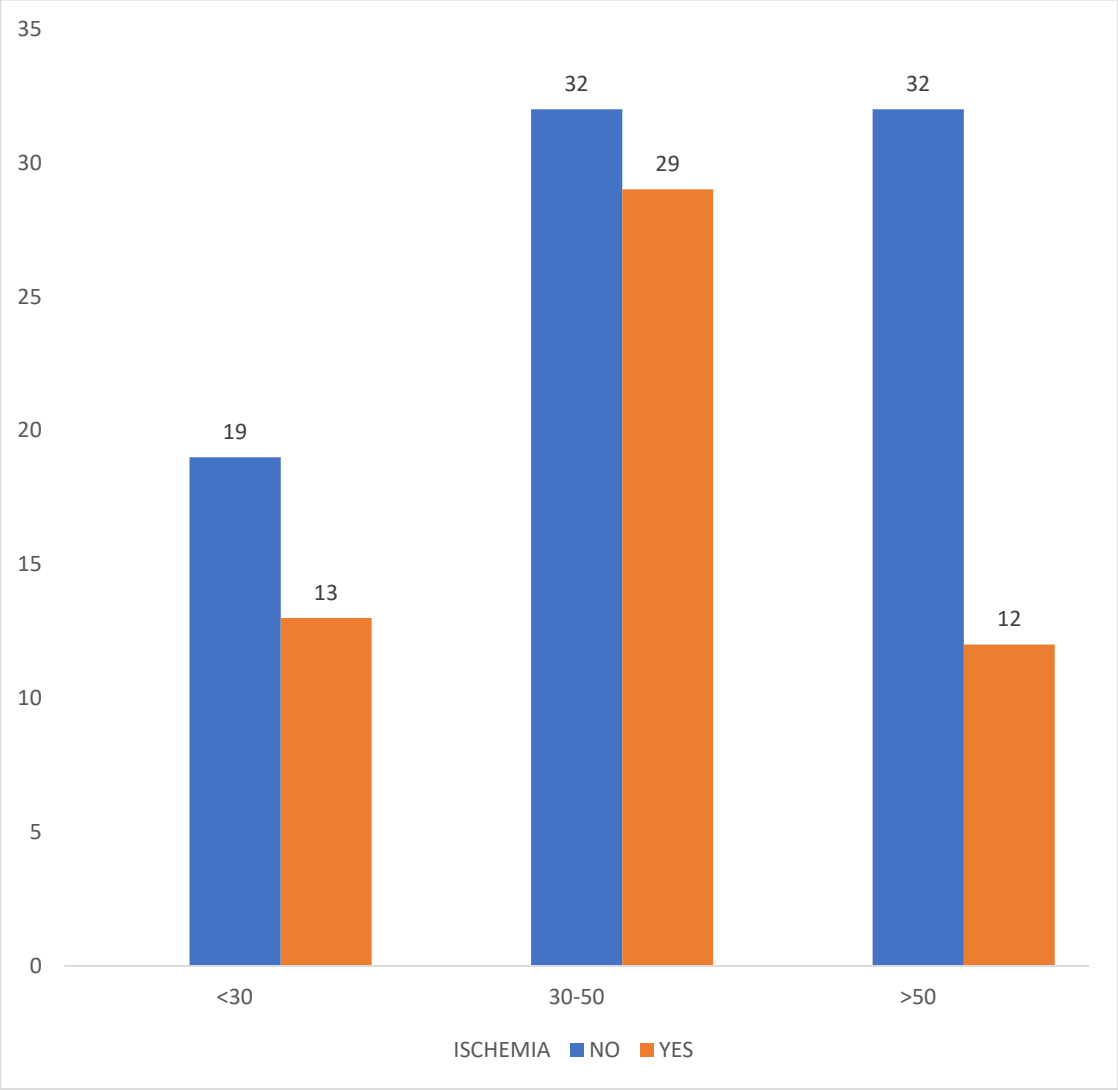


Figure 15. Distribution of microalbuminuria with ischemia

Table 16. Crosstabulation of microalbuminuria with troponin T

			TROP T		Total	P value
			NO	YES		
MA	<30	Count	8	24	32	0.332
		% within MA	25.0%	75.0%	100.0%	
	30-50	Count	15	46	61	
		% within MA	24.6%	75.4%	100.0%	
	>50	Count	6	38	44	
		% within MA	13.6%	86.4%	100.0%	
Total		Count	29	108	137	
		% within MA	21.2%	78.8%	100.0%	

There is no significant difference in microalbuminuria with troponin T (p=0.33)

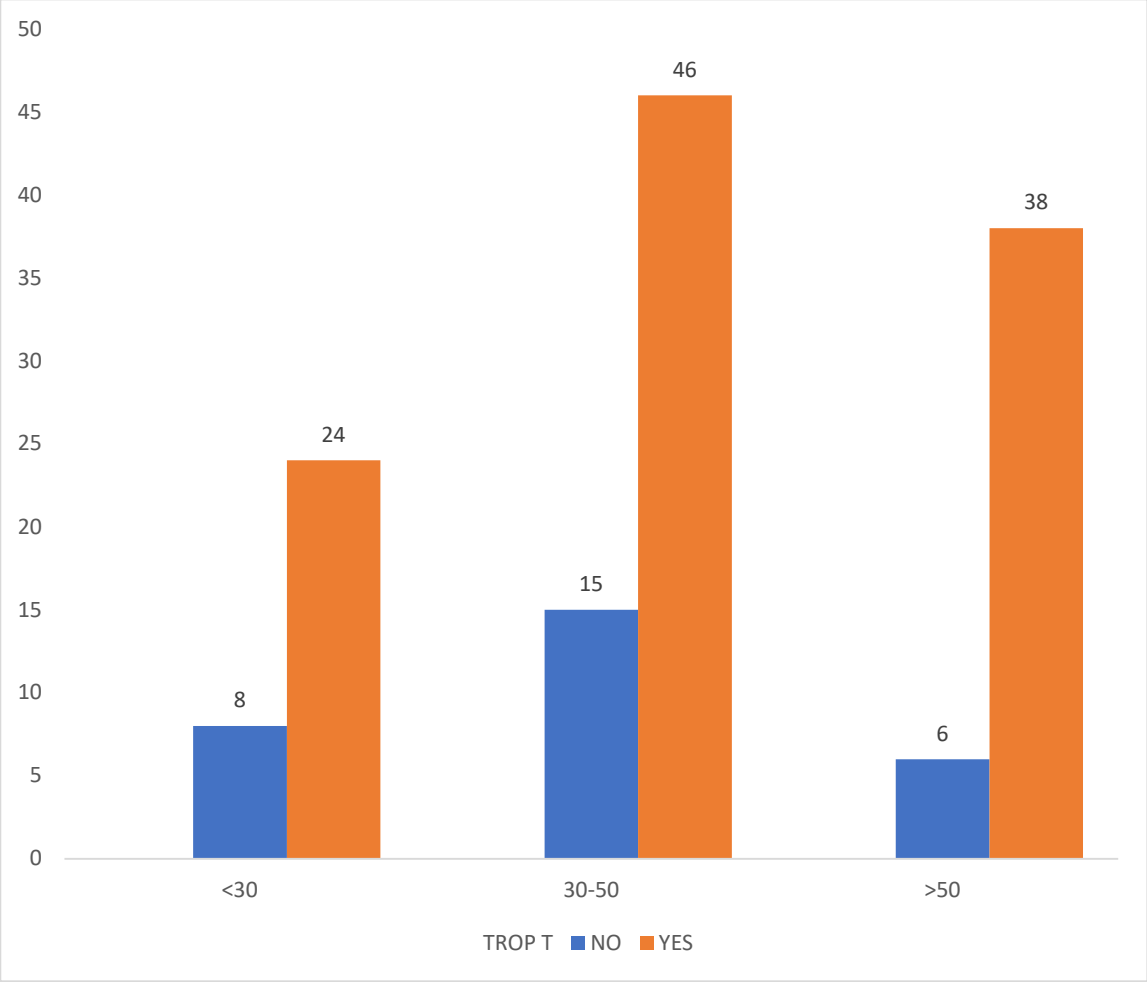


Figure 16. Distribution of microalbuminuria with troponin T

Table 17. Correlation of microalbuminuria with Ejection fraction %

		Ejection fraction%
MA	Pearson Correlation	0.047
	P value	0.586

There is no correlation in microalbuminuria withEjection fraction% (r=0.047, p=0.586).

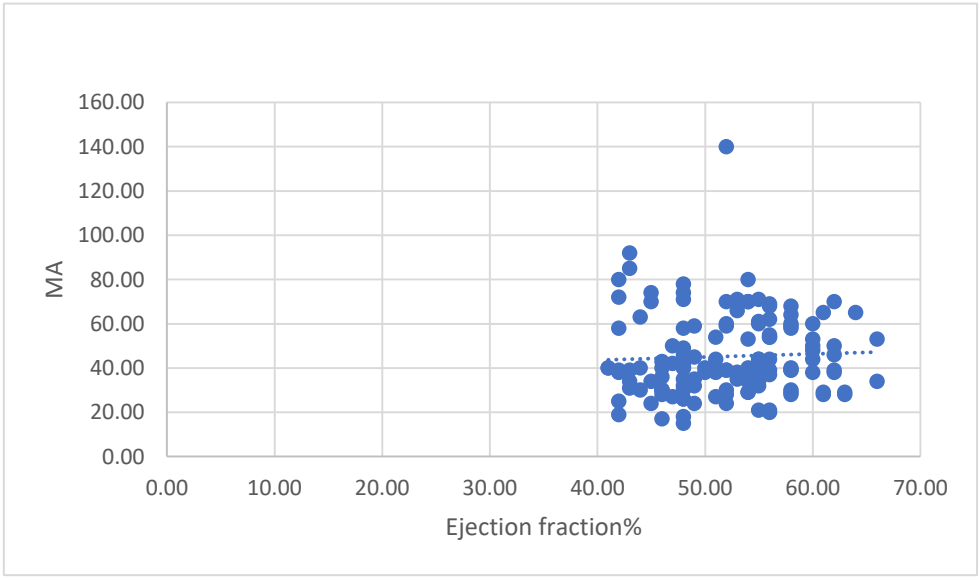


Figure 17. Correlation of microalbuminuria with Ejection fraction %

Table 18. Cross-tabulation of age groups between microalbuminuria

			MA			Total	P value
			<30	30-50	>50		
Age group	<35	Count	3	5	4	12	0.112
		% withinthe Age group	25.0%	41.7%	33.3%	100.0%	
	36-45	Count	3	12	13	28	
		% withinthe Age group	10.7%	42.9%	46.4%	100.0%	
	46-55	Count	5	13	6	24	
		% withinthe Age group	20.8%	54.2%	25.0%	100.0%	
	56-65	Count	17	21	9	47	
		% withinthe Age group	36.2%	44.7%	19.1%	100.0%	
	>66	Count	4	10	12	26	
		% withinthe Age group	15.4%	38.5%	46.2%	100.0%	
Total		Count	32	61	44	137	
		% withinthe Age group	23.4%	44.5%	32.1%	100.0%	

Comparing age groups between microalbuminuria shows patients aged 56-65 years, 30-50 microalbuminuria in 21 (44.7%) patients, and <30 microalbuminuria in 17 (36.2%) patients.Patients aged 36-45 years>50 microalbuminuria in 13 (46.4%) patients and 30-50 microalbuminuria in 12 (42.9%) patients.Patients aged >66 years >50 microalbuminuria in 12 (46.2%) patients and 30-50 microalbuminuria in 10 (38.5%) patients.The results show no significant difference in the age group between microalbuminuria (p=0.112).

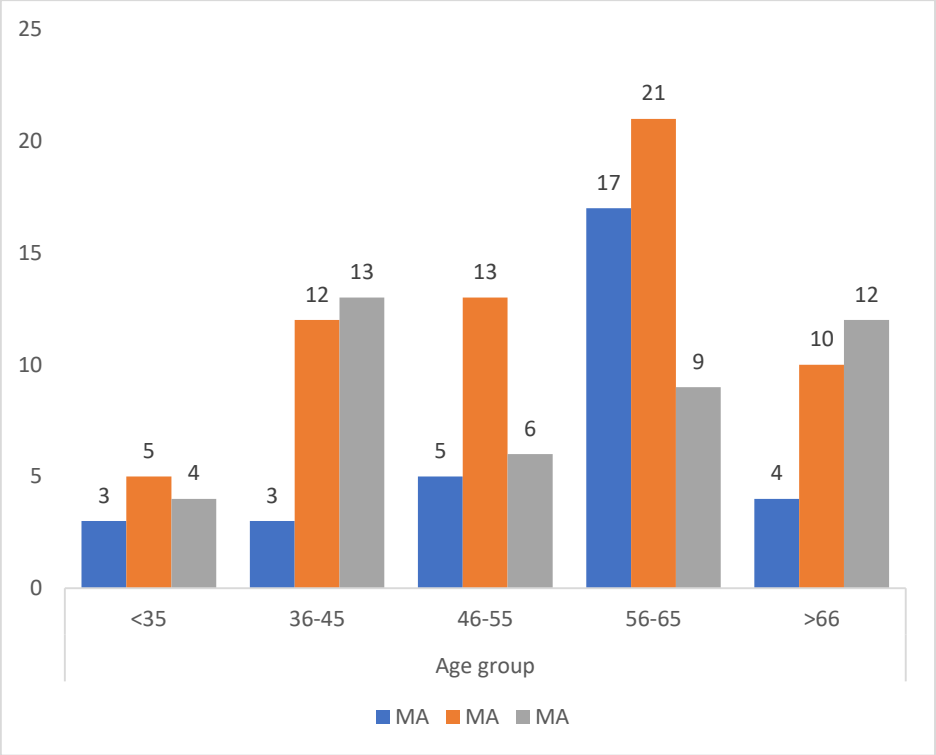


Figure 18. Distribution of age groups between microalbuminuria

Table 19. Cross-tabulation of gender between microalbuminuria

			MA			Total	P value
			<30	30-50	>50		
Sex	F	Count	10	25	16	51	0.647
		% within Sex	19.6%	49.0%	31.4%	100.0%	
	M	Count	22	36	28	86	
		% within Sex	25.6%	41.9%	32.6%	100.0%	
Total		Count	32	61	44	137	
		% within Sex	23.4%	44.5%	32.1%	100.0%	

Comparing gender between microalbuminuria shows male patients with 30-50 microalbuminuria in 36 (41.9%) patients, >50 microalbuminuria in 28 (32.6%), and <30 microalbuminuria in 22 (25.6%) patients.Female patients with 30-50 microalbuminuria in 25 (49%) patients, >50 microalbuminuria in 16 (31.4%), and <30 microalbuminuria in 10 (19.6%) patients.The results show no significant difference in gender between microalbuminuria (p=0.647).

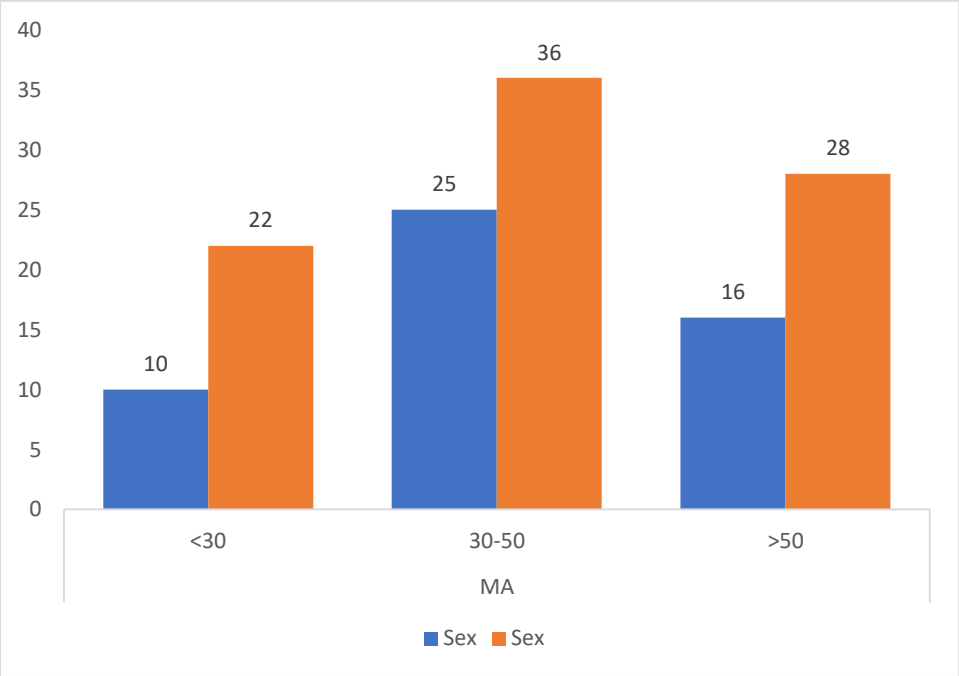


Figure 19. Distribution of gender between microalbuminuria

Table 20. Cross-tabulation of family history of IHD between microalbuminuria

			MA			Total	P value
			<30	30-50	>50		
Family History of IHD	NO	Count	21	28	25	74	0.174
		% within Family History of IHD	28.4 %	37.8 %	33.8%	100.0%	
	YES	Count	11	33	19	63	
		% within Family History of IHD	17.5 %	52.4 %	30.2%	100.0%	
Total		Count	32	61	44	137	
		% within Family History of IHD	23.4 %	44.5 %	32.1%	100.0%	

Comparing the family history of IHD with microalbuminuria showspatients who had a family history of IHD with 30-50 microalbuminuria in 33 (52.4%) patients and >50 microalbuminuria in 19 (30.2%) patients.Patients who had a no family history of IHD with 30-50 microalbuminuria in 28 (37.8%) patients and >50 microalbuminuria in 25 (33.8%) patients.The results show no significant difference in the family history of IHD between microalbuminuria (p=0.174)

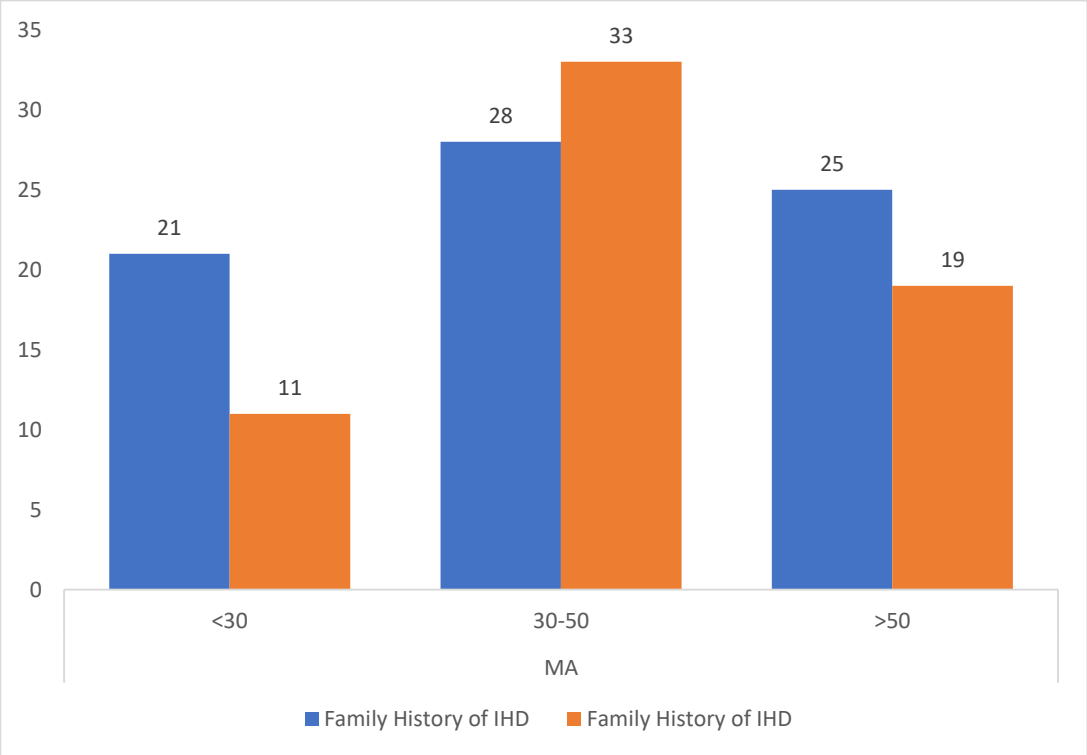


Figure 20. Distribution of family history of IHD between microalbuminuria

Table 21. Cross-tabulation of smoking between microalbuminuria

			MA			Total	P value
			<30	30-50	>50		
Smoking	NO	Count	11	35	21	67	0.106
		% within Smoking	16.4%	52.2%	31.3%	100.0%	
	YES	Count	21	26	23	70	
		% within Smoking	30.0%	37.1%	32.9%	100.0%	
Total		Count	32	61	44	137	
		% within Smoking	23.4%	44.5%	32.1%	100.0%	

Comparing smoking with microalbuminuria shows patients who have smoking with 30-50 microalbuminuria in 26 (37.1%) patients,>50 microalbuminuria in 23 (32.9%) patients, and<30 microalbuminuria in 21 (30%) patients.Patients who have notsmoked with 30-50 microalbuminuria in 35 (52.2%) patients, >50 microalbuminuria in 21 (31.3%) patients, and <30 microalbuminuria in 11 (16.4%) patients.The results show no significant difference in smoking between microalbuminuria (p=0.106)

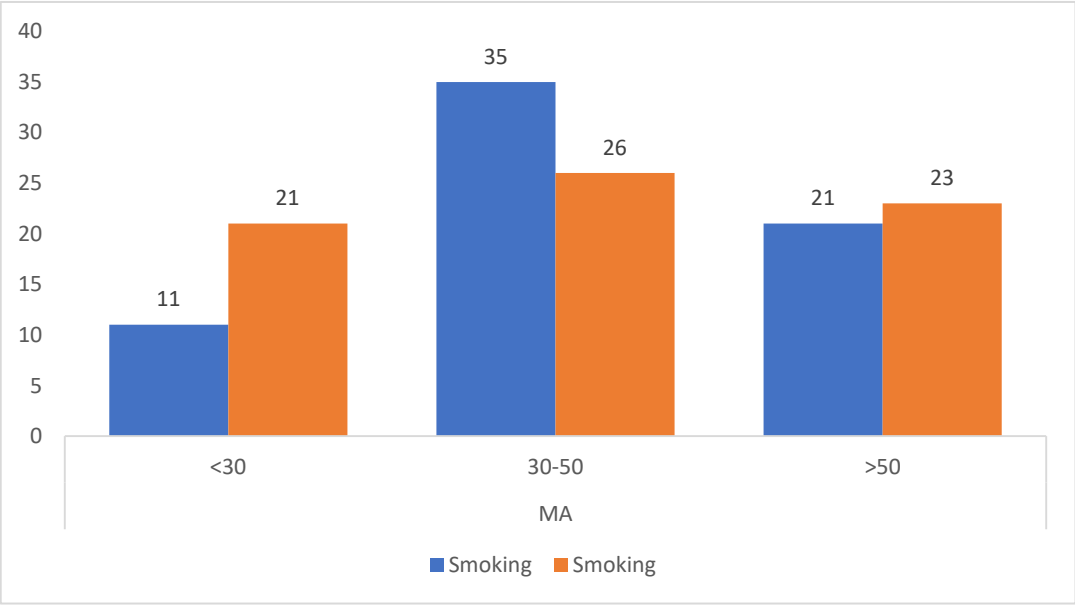


Figure 21. Distribution of smoking between microalbuminuria

Table 22. Cross-tabulation of BMI between microalbuminuria

			MA			Total	P value
			<30	30-50	>50		
BMI	Normal weight	Count	4	9	6	19	0.592
		% within BMI	21.1%	47.4%	31.6%	100.0%	
	Overweight	Count	26	50	33	109	
		% within BMI	23.9%	45.9%	30.3%	100.0%	
	Obese	Count	2	2	5	9	
		% within BMI	22.2%	22.2%	55.6%	100.0%	
Total		Count	32	61	44	137	
		% within BMI	23.4%	44.5%	32.1%	100.0%	

Comparing BMI with microalbuminuria shows patients who are overweight with 30-50 microalbuminuria in 50 (45.9%) patients, >50 microalbuminuria in 33 (30.3%) patients, and <30 microalbuminuria in 26 (23.9%) patients.The results show no significant difference in BMI between microalbuminuria (p=0.592).

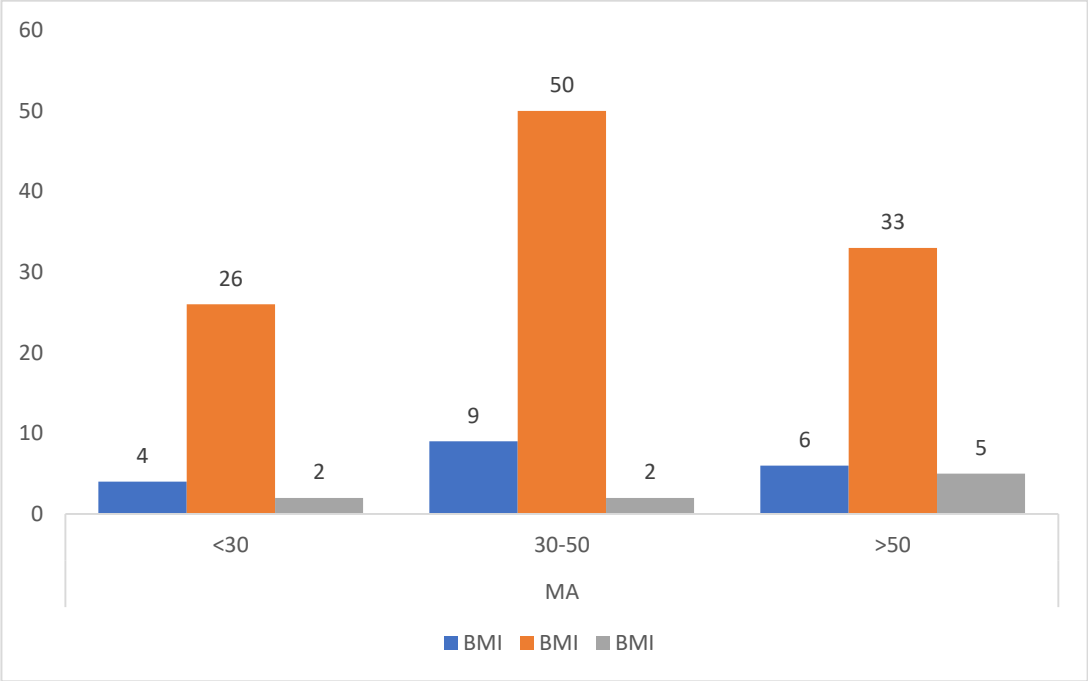


Figure 22. Distribution of BMI between microalbuminuria

Table 23. Cross-tabulation of FBS between microalbuminuria

			MA			Total	P value
			<30	30-50	>50		
FBS	<100	Count	15	42	27	84	0.118
		% within FBS	17.9%	50.0%	32.1%	100.0%	
	>100	Count	17	19	17	53	
		% within FBS	32.1%	35.8%	32.1%	100.0%	
Total		Count	32	61	44	137	
		% within FBS	23.4%	44.5%	32.1%	100.0%	

Comparing FBS with microalbuminuria shows patients who have <100 FBS with 30-50 microalbuminuria in 42 (50%) patients, >50 microalbuminuria in 27 (32.1%) patientsand <30 microalbuminuria in 15 (17.9%) patients. Patients who have >100 FBS with 30-50 microalbuminuria in 19 (35.8%) patients, >50 microalbuminuria in 17 (32.1%) patients and <30 microalbuminuria in 17 (32.1%) patients.The results show no significant difference in FBS between microalbuminuria (p=0.118).

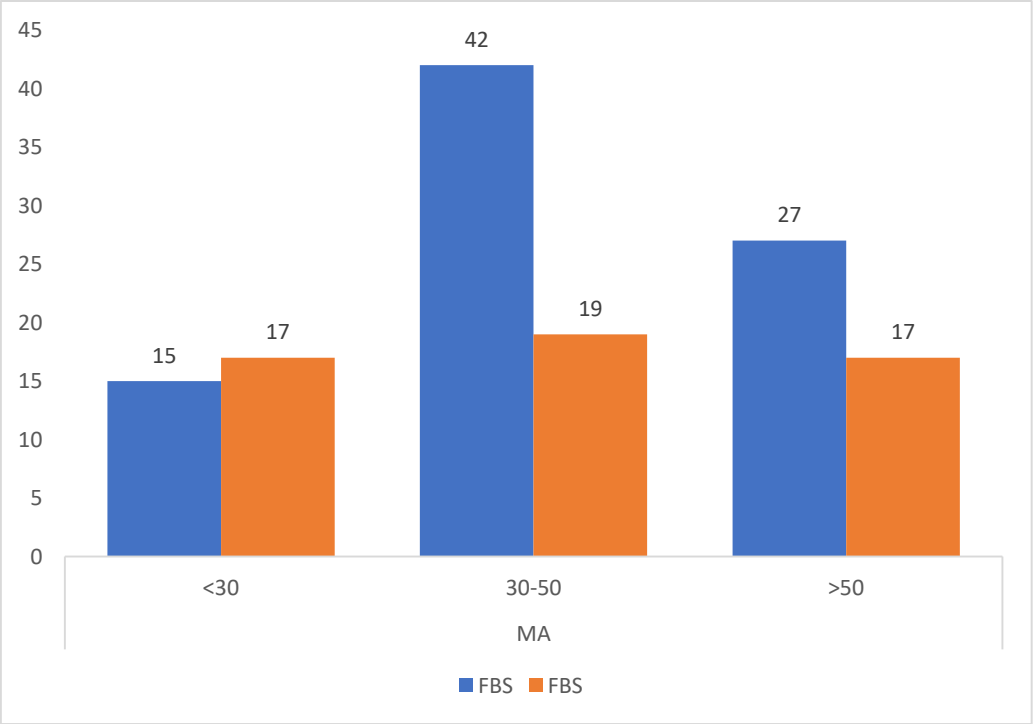


Figure 23. Distribution of FBS between microalbuminuria

Table 24. Cross-tabulation of PPBS between microalbuminuria

			MA			Total	P value
			<30	30-50	>50		
PPBS	<140	Count	16	45	29	90	0.072
		% within PPBS	17.8%	50.0%	32.2%	100.0%	
	>140	Count	16	16	15	47	
		% within PPBS	34.0%	34.0%	31.9%	100.0%	
Total		Count	32	61	44	137	
		% within PPBS	23.4%	44.5%	32.1%	100.0%	

Comparing PPBS with microalbuminuria shows patients who have <140 PPBS with 30-50 microalbuminuria in 42 (50%) patients, >50 microalbuminuria in 29 (32.2%) patients and <30 microalbuminuria in 16 (17.8%) patients. Patients who have >140 PPBS with 30-50 microalbuminuria in 16 (34%) patients, >50 microalbuminuria in 15 (31.9%) patients and <30 microalbuminuria in 16 (34%) patients. The results show no significant difference in PPBS between microalbuminuria (p=0.072).

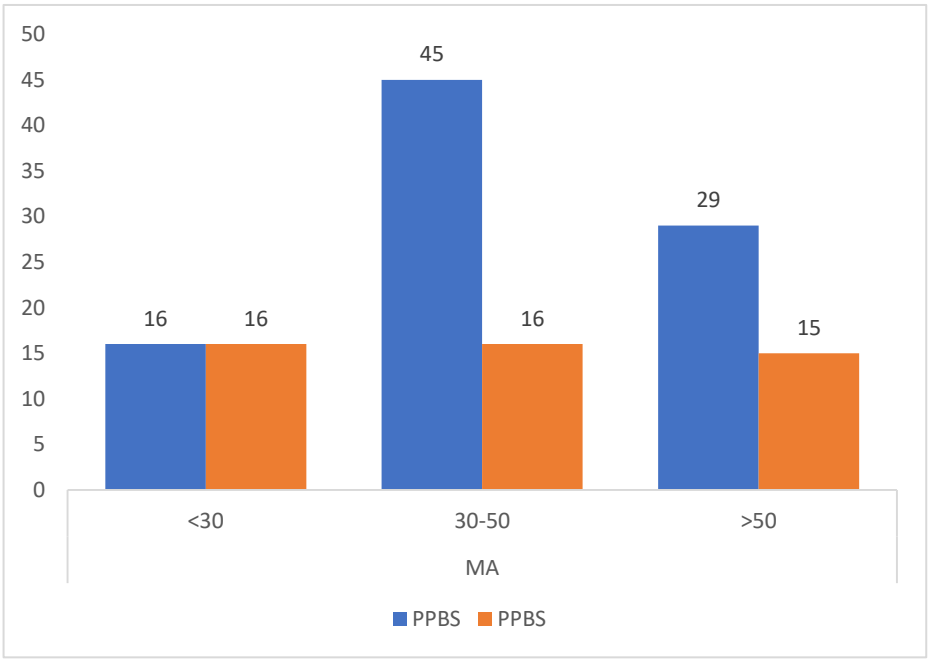


Figure 24. Distribution of PPBS between microalbuminuria

Table 25. Cross-tabulation of TC between microalbuminuria

			MA			Total	P value
			<30	30-50	>50		
TC	<200	Count	7	16	1	24	0.005
		% within TC	29.2%	66.7%	4.2%	100.0%	
	>200	Count	25	45	43	113	
		% within TC	22.1%	39.8%	38.1%	100.0%	
Total		Count	32	61	44	137	
		% within TC	23.4%	44.5%	32.1%	100.0%	

Comparing TC with microalbuminuria shows patients who have >200 TC with 30-50 microalbuminuria in 45 (39.8%) patients, >50 microalbuminuria in 43 (38.1%) patients and <30 microalbuminuria in 25 (22.1%) patients. Patients who have <200 TC with 30-50 microalbuminuria in 16 (66.7%) patients, >50 microalbuminuria in 1 (4.2%) patient and <30 microalbuminuria in 7 (29.2%) patients. The results show a significant difference in TC between microalbuminuria (p=0.005).

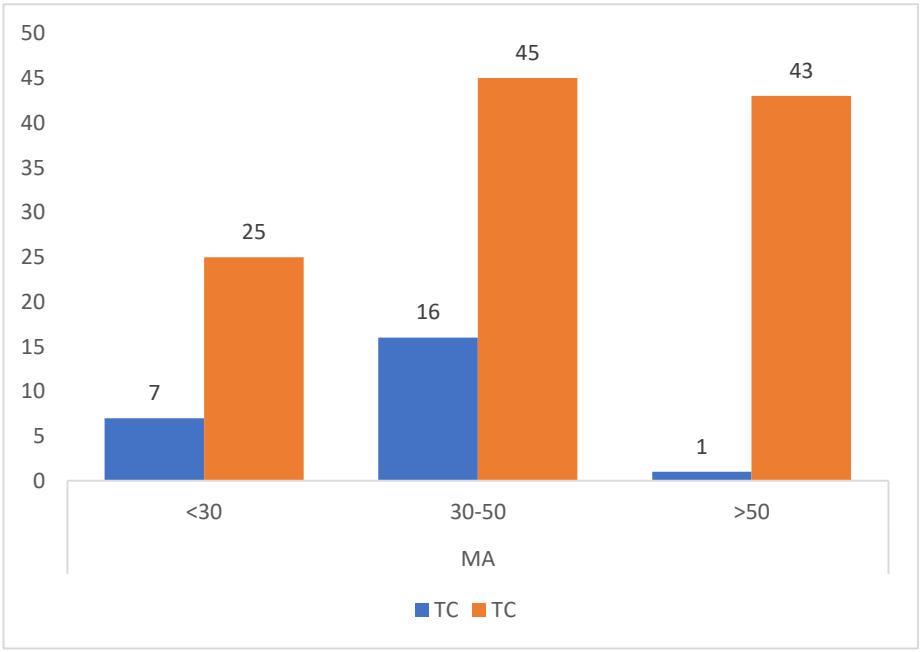
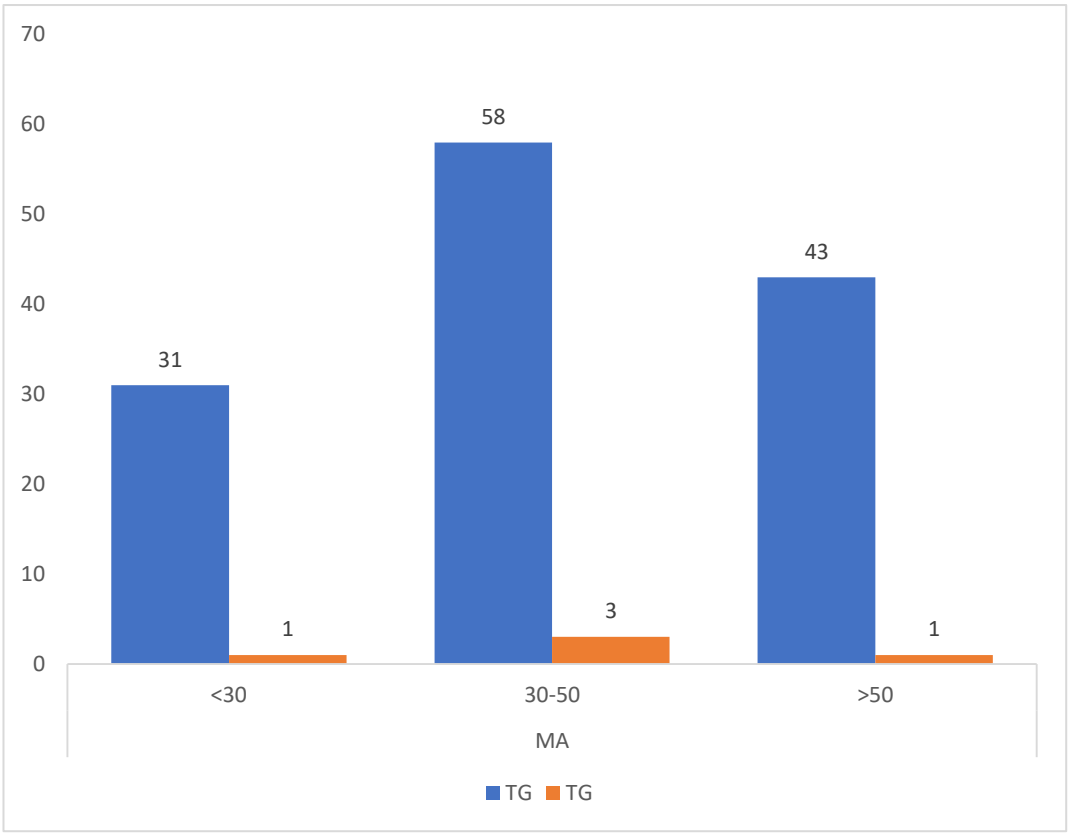


Figure 25. Distribution of TC between microalbuminuria

Table 26. Cross-tabulation of TG between microalbuminuria

			MA			Total	P value
			<30	30-50	>50		
TG	<150	Count	31	58	43	132	0.763
		% within TG	23.5%	43.9%	32.6%	100.0%	
	>150	Count	1	3	1	5	
		% within TG	20.0%	60.0%	20.0%	100.0%	
Total		Count	32	61	44	137	
		% within TG	23.4%	44.5%	32.1%	100.0%	

Comparing TG with microalbuminuria shows patients who have <150 TG with 30-50 microalbuminuria in 58 (43.9%) patients, >50 microalbuminuria in 43 (32.6%) patients and <30 microalbuminuria in 31 (23.5%) patients. The results show no significant difference in TG between microalbuminuria (p=0.072).



26. Distribution of TG between microalbuminuria

Table 27. Cross-tabulation of HDL between microalbuminuria

			MA			Total	P value
			<30	30-50	>50		
HDL	<40	Count	11	22	10	43	0.319
		% within HDL	25.6%	51.2%	23.3%	100.0%	
	>40	Count	21	39	34	94	
		% within HDL	22.3%	41.5%	36.2%	100.0%	
Total		Count	32	61	44	137	
		% within HDL	23.4%	44.5%	32.1%	100.0%	

Comparing HDL with microalbuminuria shows patients who have >40HDL with 30-50 microalbuminuria in 39 (41.5%) patients, >50 microalbuminuria in 34 (36.2%) patients and <30 microalbuminuria in 21 (22.3%) patients. The results show no significant difference in HDL between microalbuminuria (p=0.319).

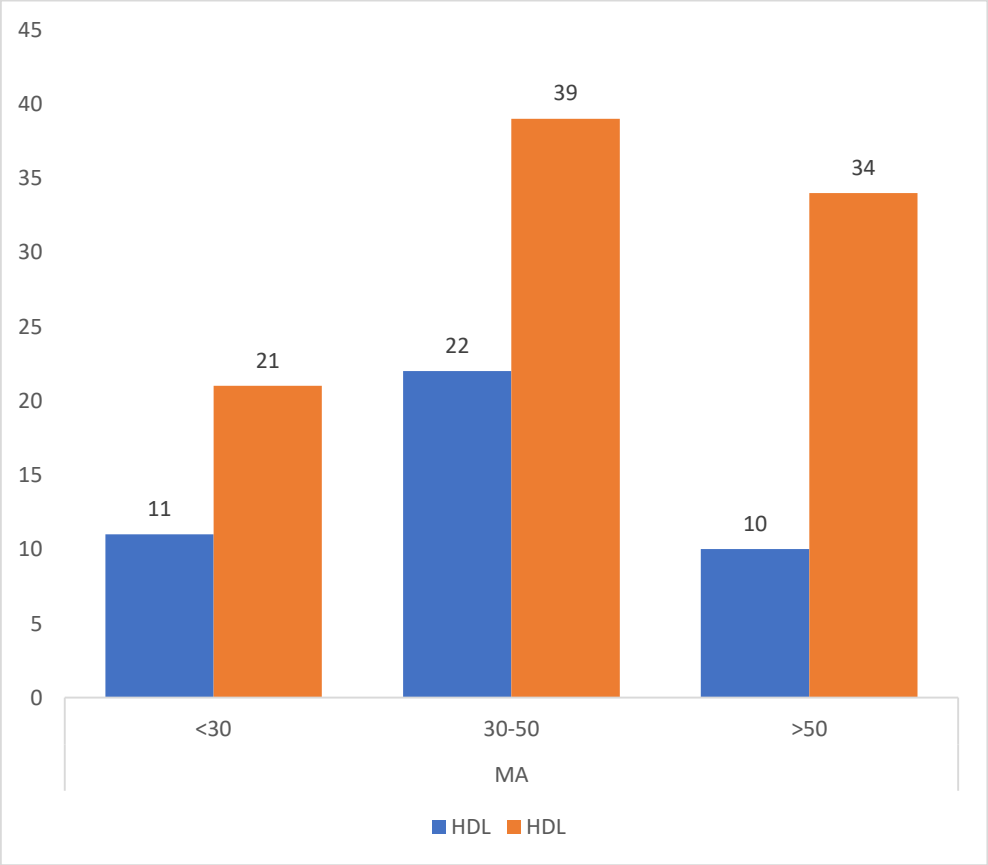


Figure 27. Distribution of HDL between microalbuminuria

Table 28. Cross-tabulation of LDL between microalbuminuria

			MA			Total	P value
			<30	30-50	>50		
LDL	<150	Count	31	57	41	129	0.755
		% within LDL	24.0%	44.2%	31.8%	100.0%	
	>150	Count	1	4	3	8	
		% within LDL	12.5%	50.0%	37.5%	100.0%	
Total		Count	32	61	44	137	
		% within LDL	23.4%	44.5%	32.1%	100.0%	

Comparing LDL with microalbuminuria shows patients who have <150 LDL with 30-50 microalbuminuria in 57 (44.2%) patients, >50 microalbuminuria in 41 (31.8%) patients and <30 microalbuminuria in 31 (24%) patients. The results show no significant difference in LDL between microalbuminuria (p=0.755)

\

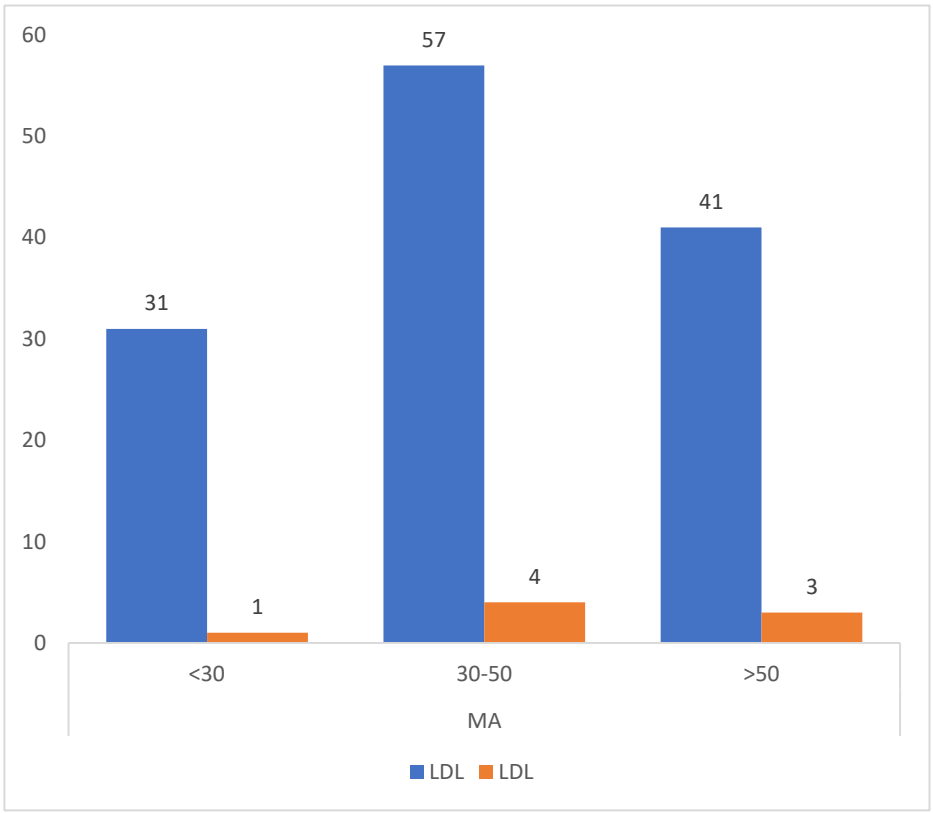


Figure 28. Distribution of LDL between microalbuminuria

Table 29. Cross-tabulation of ACS between microalbuminuria

			MA		Total	P value
			Normal	Abnormal		
ACS	ISCHEMIA	Count	13	41	54	0.001
		% within ACS	24.1%	75.9%	100.0%	
	MI	Count	19	64	83	
		% within ACS	22.9%	77.1%	100.0%	
Total		Count	32	105	137	
		% within ACS	23.4%	76.6%	100.0%	

Comparing acute coronary syndrome with microalbuminuria shows ischemia patients with abnormal microalbuminuria in 41 (75.9%) patients and normal microalbuminuria in 13 (24.1%) patients.Myocardial infarctionpatients with abnormal microalbuminuria in 64 (77.1%) and normal microalbuminuria in 19 (22.9%) patients.The results show significant difference in acute coronary syndrome between microalbuminuria (p=0.001).

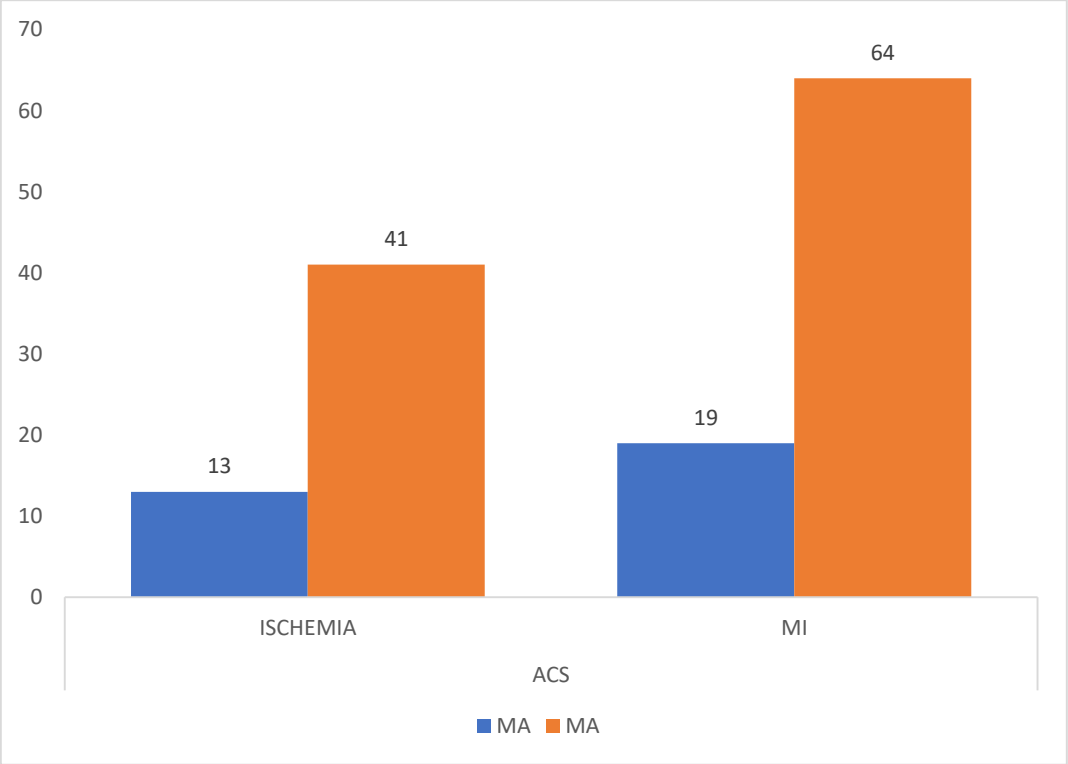


Figure 29. Distribution of ACS between microalbuminuria

TABLE NO : 30 PREVALENCE OF MICROALBUMINURIA IN NON-DIABETIC CAD PATIENTS.

Microalbuminuria	CAD patients		Significance
	n	**%**	
Normal (<30 mg/L)	32	23%	
Abnormal (≥20mg/L)	105	77%	p < 0.001

The above table shows the prevalence of microalbuminuria among CAD patients as 77%. The difference between the two prevalence rate was statistically significant (p<0.001).

DISCUSSION

By 2018, Ischemic Heart Illness will account for a significant portion of India's disease burden. Risk categorization of the population should be successful in focusing preventive strategies. There are several studies regarding micro albuminuria coming from western literature, and many nations already view it as a separate risk factor for the onset of ischemic heart disease.[46]

Before, microalbuminuria was thought to be a sign of endothelial dysfunction in people with diabetes mellitus. However, numerous investigations have revealed that microalbuminuria is now a reliable sign of generalised vascular dysfunction in people who are not diabetic.[47,48]

The purpose of this study was to determine whether IHD and MA in non-diabetic participants are related.

IHD was identified in the current study using ECG alterations. ECG has been demonstrated to be a useful screening technique for IHD in the general population by de Bruyne MC et al.[49]

In this study, 74% of the participants were men and 26% were women. This is consistent with the understanding that men are more likely than women to have ischemic heart disease. Additionally, more men were involved in the EPIC NORFLOK study than women, which is consistent with my research. [41]

The study group's average age in my study was 55.68 ± 11.10 years. For women, it was 59.92 ± 6.42 years. The fact that all of the ladies were post-menopausal indicates that sex hormones have a reducing effect on cardiovascular risk. This is

consistent with Roeste and Banga et alfinding .'s that non-diabetic postmenopausal group urine albumin excretion is considerably higher than premenopausal group.[50,51]

Smoking habit was present in 72% of the study participants, suggesting that smoking excessively may be a significant risk factor for IHD. In their study, Umesh N. Khot et al. discovered a prevalence of smoking as a risk factor of 41.6% in men and 29.5% in women.[51,59]

The majority of the study group had a BMI more than 25 kg/m2. This prevalence was substantially greater than what Singh R.B. et al. found (27.2% in urban areas and 11.0% in rural areas). In the current study, 58% of patients (51.4% of men and 76.9% of women) had hypertension. This prevalence is greater than that discovered by Fabitz et al. in their STRONG HEART STUDY (38.4% in males and 55.9% in females), but it exhibits a comparable trend.[58,59]

The current study found a favourable relationship between microalbuminuria and ischemic heart disease, with 76% of patients having it. The PREVEND study has shown that the presence of microalbuminuria was independently associated with infarct pattern (7.2%), main type of ischemia (10.8%), and minor types of ischemia (15.3%) in a multivariate adjusted scenario while taking in comparison existing risk determinants (OR-1.33).[54,55,56]

In the HOPE (HEART OUTCOMES AND PREVENTION AND EVALUATION survey, which was conducted between the years of 1998 and 2003, the prevalence of micro albuminuria was estimated to be in 15% of a cohort of individuals.[52,53,54]

A survey found that, compared to 13.8% of people without microalbuminuria, 20.6% of those had a greater risk of coronary artery disease, myocardial infarction, and stroke.

In the PREVEND trial, 20.8% of the people with ischemic cardiac disease had microalbuminuria, compared to 76% in this study, and 32.4% of the patients with ischemic coronary disease were documented here.[55,56]

This was likely due to the fact that the current study included a cohort of IHD patients, whereas the studies listed above were conducted on the general population.

A survey found that, compared to 13.8% of people without microalbuminuria, 20.6% of those had a greater risk of coronary artery disease, myocardial infarction, and stroke.[57,60]

SUMMARY

In the present study,

1. Male to female ratio was 1.69:1
2. Subjects in the age group 56-65 years constituted 34. 3% of the study group.
3. The youngest patient in the study was 26 years old.
4. 46% of patients had a family history of Ischemic Heart Disease.
5. 51.1% of patients had regular smoking habits
6. 79.6% of patients in the study were overweight and 9% were obese
7. 54% of patients in the study group had AMI and 7.3% patients had IWMI and 38.7% suffered from ischemia
8. 78.8 % of the patients had Trop T positivity.
9. 82.5% of patients had total cholesterol more than 200 mg/dl
10. 31.4% of patients had HDL less than 40 mg/dl
11. 76.6% of patients had microalbuminuria in the study group.
12. 77.9% of patients with total cholesterol more than 200mg/dL is found to have microalbuminuria more than 30mg/dL. There is a significant association between total cholesterol and microalbuminuria.
13. 76.6% of acute coronary syndrome patients found to have microalbuminuria more than 30mg/day . Microalbuminuria >30mg/day was present in 75.9% of the patients with ischemic pattern on ECG, 77.1% of patients with infarct pattern on ECG. So 76.6% of all cases of ischaemic heart disease patient. p value <0.001

CONCLUSION

76.6% of the 137 non-diabetic IHD patients studied had microalbuminuria. Thus, in non-diabetic people, micro albuminuria is strongly correlated with ischemic heart disease.

It could be considered a significant new risk factor for ischemic heart disease.

Therefore, in the future, vigorous screening of the general population, especially in the young age group, may be a valuable public tool for assessing cardiac risk and focusing on preventive measures

BIBLIOGRAPHY

1. Aschoff L. Introduction. In: Cowdry EV, ed. Arteriosclerosis: A Survey of the Problem. New York: Macmillan; 1933:1.

2. Faggiotto A, Ross A, Hanker L. Studies of hypercholesterolemia in the nonhuman primate. I. Changes that lead to fatty streak formation. Arteriosclerosis 1984;4:323-340

3. Groves PH, Lewis MJ, Cheadle HA, Penny WJ. SIN-i reduces platelet adhesion and platelet thrombus formation in a porcine model of balloon angioplasty.Circulation 1993:87:590-597.

4. Garg UC, Hassid A. Nitric oxide-generating vasodilatons and 8- bromo-cyclic guanosine monophosphate inhibit mitogenesis and proliferation of cultured rat vascular smooth muscle cells. J Clin Invest 1989;83:1774-1 777

5. Ross R. The pathogenesis of atherosclerosis: a perspective for the 1990s. Nature 1993:362:801-809

6. Brinkman JW, Bakker SJL, : C. Kidney Int Suppl 92: S69–S75, 2004.

7. Fabsitz RR, Sidawy AN, Go O et al. Prevalence of peripheral arterial disease and associated risk factors in American Indians: the Strong Heart Study. Am J Epidemiol 1999; 149: 330–38.

8. Haraldsson B, Sorensson J: Why do we not all have proteinuria? An update of our current understanding of the glomerular barrier. News Physiol Sci 19: 7–10, 2004

9. Levey AS, Eckardt KU, A, Coresh J Definition and classification of chronic kidney disease: A global position statement from Kidney Disease: Improving Global Outcome (KDIGO). Kidney Int 67: 2089–2100, 2005

10. Mattix HJShaykevich S, Curhan G: Use of the albumin/creatinine ratio to detect microalbuminuria: Implications of sex and race. J Am Soc Nephrol 13: 1034–1039,2002

11. Verhave JC, Hillege HL, , de Jong PE: How to measure the prevalence of microalbuminuria Gansevoort RT, Verhave JC, Hillege HL, ; the PREVEND Study Group: screening based on spot morning urine samples to detect subjects with microalbuminuria . Kidney Int Suppl 94: S28–S35, 2005.

12. Deekert T, Feldt : Rasmussen B, Bore Johnsen K. Jensen T, Kofoed Enevolsen A : Microalbuminuria reflects widespread vascular damage. The Steno Hypothesis in Diabetalogia 1989; 32 : 219-226.

13. Stehouwer CDA, Nauta JJP, Zeldenhurst GC : Urinary albumin, cardiovascular disease and endothelial dysfunction. Lancet 1992; 340 : 329-323.

14. Coller A, Rumley AG, Leach JP, Lowc GDO, Small M : Free radical activity and hemostatic factors in NIDDM patients with and without Microalbuminuria. Diabetes 1992; 41 : 909-13.

15. Redon J et al : Ambulatory blood pressure and MAL in essential hypertension. J. Hypertension 1994 Aug 12 (8) : 947-53.

16. Mimarm A ; Miicroalbuminuria in essential hypertension. Clin. Exp. Hypertension 1997; 19 : 753-67.

17. Cirillo M, Stellato D, Laurnzi M. Panarrelli W, Zanchetti A, De Santo NG : Pulse pressure and isolated systolic hypertension : Association with microalbuminuria. The GUBBIO study Collaboration Research Group. Kidney Int. 2000 Sep : 58 (5) : 1211-8.

18.Bennett PH, Haffner S, Kasiske BL. Screening and management of microalbuminuria in patients with diabetes mellitus: recommendations to the Scientific Advisory Board of the National Kidney Foundation from an ad hoc committee of the Council on Diabetes mellitus of the National Kidney Foundation. Am J Kidney Dis 1995;25:107-12.

19.Gerstein HC, Mann JF, Yi Q, Zinman B, Dinneen SF, Hoogwerf B, Halle JP,Young J, Rashkow A, Joyce C, Nawaz S, Yusuf S: Albuminuria and risk of cardiovascular events, death, and heart failure in diabetic and nondiabetic individuals. JAMA 286: 421–426, 2001

20.Brantsma AH, Bakker SJ, Hillege HL, de Zeeuw D, de Jong PE, Gansevoort RT: Urinary albumin excretion and its relation with C54 reactive protein and the metabolic syndrome in the prediction of type 2 diabetes. Diabetes Care 28:2525– 2530, 2005

21.Romundstad S, Holmen J, Kvenild K, Hallan H, Ellekjaer H: Microalbuminuria and all-cause mortality in 2,089 apparently healthy individuals: A 4.4-year followup study.The Nord-Trondelag Health Study (HUNT), Norway. Am J Kidney Dis 42: 466–473, 2003

22.Yuyun MF, Khaw KT, Luben R, Welch A, Bingham S, Day NE, Wareham NJ: Microalbuminuria independently predicts all-cause and cardiovascular mortality in a British population: The European Prospective Investigation into Cancer in Norfolk (EPIC-Norfolk) population study. Int J Epidemiol 33: 189–198, 2004

23.Stary HC, Bleakley A, Glagov 5, et al. A definition of initial, fatty streak, and intermediate lesions of atherosclerosis: Council on Arteriosclerosis, American Heart Association. 1997**;** 14:840-856

24. Witztum J, Steinberg D. Role of oxidized low density lipoprotein in athenogenesis. J Clin Invest 2001:88:1785-1792

25. Adams DH, Schoen FJ. Contemporary concepts in atherosclerosis pathology. In: White RA, Ed. Atherosclerosis 2003

26. Fuster V. , Ambrose JA, Chesebro JH. Atherosclerotic plaque rupture and thrombosis: evolving concepts. Circulation EJC 2003

27. Kolodgie FD, Gold HK, , et al. Intraplaque hemorrhage and progression of coronary atheroma. N Engl J Med. 2003;349(24):2316–2325.

28. Yudkin JS, Forrest RD, and Jackson C. Microalbuminuria as a predictor of vascular disease in non diabetic subjects. Lancet 1988. 11 : 530-33.

29. Microalbuminuria is independently associated with ischemic electrocardiographic abnormalities in large non-diabetic population. PREVEND (Prevention of Renal and Vascular Endstage Disease) TRIAL. European Heart Journal 2000, 21 : 1922-27.

30. Gerstein HC, Mann JF, Pongue J, Yusuf S. Prevalence and determinants of microalbuminuria in high risk diabetic and non diabetic patients in HOPE study. Diabetes Care 2000 April, 23 : 35- 39. Suppl B.

31. Felat – Rasmussen B, Nephrological Department, Rigs Hospital, Copenhagen, Denmark. Microalbuminuria, endothelial dysfunction and cardiovascular risk. Diabetes M etab 2000 July, 26 Suppl. 4 : 64-6.

32. Gosling P. Microalbuminuria a marker of systemic disease. British Journal of Hospital medicine, 1995, 54 : 285-90.

33.Wachtell K, Palmieri V, Olsen MH, Bella JN, Aalto T, Dahlof B, Gerdts E, Wright JT Jr, Papademetriou V, Mogensen CE, Borch- Johnsen K, Ibsen H, Devereux RB: Urine albumin/creatinine ratio and echocardiographic left ventricular structure and function in hypertensive patients with electrocardiographic left ventricular hypertrophy: The LIFE study. Losartan Intervention for Endpoint Reduction. Am Heart J 143: 319–326, 2002.

34.De Wardener HE. The Kidney. Edinburgh: Churchill Livingstone, 1985: 49-50.

35. Deen WM, Myers BD, Brenner BM. The glomer-ular barrier to acromolecules: theoretical and experimental considerations. In: Brenner BM, Stein JA, eds. Nephrotic Syndrome. New York: Churchill Livingstone, 1982: 8-9

36.Park CH, Maack T. Albumin absorption and catabolism by isolated perfused convoluted tubules ofthe rabbit. J Clin Invest 1984; 73: 767-77

37.Gosling P, Beevers DG. Urinary albumin excretion in the general population. Clin Sci 1989; 76: 39-42.

38. Pedrinelli R, DellOmo et al. Dissociation between microalbuminuria and common carotid thickness in essential hypertensive men. J Hum Hypertens 2000; 14: 831–36.

39. Roest M, Banga JD, et al. Excessive urinary albumin levels are associated with future cardiovascular mortality in postmenopausal women. Circulation 2001; 103: 3057–61

40. Gerstein HC, Mann JF, Yi Q et al. Albuminuria and risk of cardiovascular events, in diabetic and nondiabetic individuals. JAMA 2001; 25: 421–26.

40. Stehouwer CDA, Schalkwijk : International Textbook of Diabetes, 3rd ed., edited by DeFronzo RA, 2004, pp 1409–1423

41. Stehouwer CDA. Endothelial dysfunction in diabetic nephropathy: State of the art and potential significance for non-diabetic renal disease [Editorial]. Nephrol Dial Transplant 19: 778–781, 2004

42. Mongenson CE, Christensed CK. Predicting diabetic nephropathy in IDDM. New England Journal of Medicine, 1984-311 : 89.

43. Yip J. Mattok MB, Morocutti A, Sethi M, Treviasa R. Insulin REsistance in IDDM with microalbuminuria. Lancet 1993, 342, 883-87.

44. Mimarn A, Microalbuminuria in essential hypertension. Clini Exp Hypertension 1997, 19 : 753-67.

45. Bigazzi R, Bianchi S. Microalbuminuria as a marker of cardiovascular and renal disease in essential hypertension Nephrology Dialysis Transplant 1995, 10 (suppl 6) : 10 14.

46. Microalbuminuria is associated with carotid intimal medial thickening and coronary Artery disease in Diabetics, JAPI 2002, 50 : 1124-1129.

47. Cummings MH,Watts GF, Tavakolian A, Fitzgerald A. Prevalence of micro albuminuria, lipoprotein(a), and coronary artery disease in the lipid clinic. Journal Clinic Pathology 1996 Jan, 49(1) : 19-23.

48..Howard BV, Lee ET, Cowan LD et al. Coronary heart disease prevalence and its relation to risk factors in American Indians. The Strong Heart Study. Am J Epidemiol 1995; 142: 254–68

49.Fabsitz RR, Sidawy AN, Go O et al. Prevalence of peripheral arterial disease and associated risk factors in American Indians: the Strong Heart Study. Am J Epidemiol 1999; 149: 330–38.

50..Damsgaard EM, Froland A, Jorgensen OD, Mogensen CE. Microalbuminuria as predictor of increased mortality in elderly people. BMJ 1990; 300: 297–300.

51.Stehouwer CDA, Schalkwijk CG: Endothelial function and dysfunction. In: International Textbook of Diabetes, 3rd ed., edited by DeFronzo RA, Ferrannini E, Keen H, Zimmet P, Chichester, Wiley, 2004, pp 1409–1423

52.Stehouwer CDA. Endothelial dysfunction in diabetic nephropathy: State of the art and potential significance for non-diabetic renal disease [Editorial]. Nephrol Dial Transplant 19: 778–781, 2004

53. Stehouwer CD, Smulders YM: Microalbuminuria and risk for cardiovascular disease: Analysis of potential mechanisms 2006

54. Jager A, van H, V, Kostense PJ,: C-reactive protein and soluble vascular cell adhesion molecule-1 are associated with elevated urinary albumin excretion 598, 2002

55. Yudkin JS, Forrest RD, Jackson CA. Islington Diabetes Survey. Lancet 1988; ii:530–33

56. Howard BV, Lee ET, Cowan LD et al. Coronary heart disease prevalence . The Strong Heart Study 2000

58. Kuusisto Laakso M. Hyperinsulinemic microalbuminuria. A new risk indicator for coronary heart disease. Circulation 2005; 91: 831–37.

59. Damsgaard EM, Froland A, Jorgensen OD, Mongensen CE, Microalbuminuria as a predictor of increased mortality in elderly people. British Medical Journal 1990, 300 : 297-300.

60. Bright R. Cases and Observation illustrative of renal disease accompanied with the secretion of albuminous urine. Guy's Hosptial Report, 1836 : 10-338-400.

61. Kimmelsteil P, Wilson C. Intercapillary lesions in the glomeruli in the kidney. AM J Pathology, 1936, 12 : 83-97.

62. Stehouwer CD, Naceta JJ, Zeldeurest GC, Donker AJ, Ottolander GJ, Urinary albumin excretion, cardiovascular disease and endothelial dysfunction in NIDDM. Lancet 1992, 3 : 340 : 319-23.

நோயாளிகளுக்கு அறிவிப்பு மற்றும் ஒப்புதல் படிவம்
(மருத்துவ ஆய்வில் பங்கேற்பத்திற்கு)

ஆய்வு செய்யப்படும் தலைப்பு:
பங்கு பெறுவரின் பெயர்:
பங்கு பெறுவரின் வயது:

		பங்கு பெறுவர் இதனை ✓ குறிக்கவும்
1.	நான் மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்களை படித்து புரிந்து கொண்டேன். என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டுள்ளது என அறிந்து கொண்டேன்.	☐
2.	நான் இவ்வாய்வில் தன்னிச்சையாக தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும், எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.	☐
3.	இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்து மேலும் ஆய்வு மேற்கள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.	☐
4.	இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக் கொள்ள மறுக்க மாட்டேன்.	☐
5.	இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன் எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின் படி நடந்து கொள்வதுடன், ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ, அல்லது எதிர்பாராத, வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாலோ உடனே இதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்றேன்.	☐

பங்கேற்பவரின் கையொப்பம் / ..... இடம் .....
கட்டைவிரல் ரேகை
பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....
ஆய்வாளரின் கையொப்பம் /..... இடம் .....
ஆய்வாளரின் பெயர் .....
மையம் .....
கல்வியறிவு இல்லாதவற்கு (கைரேகை வைத்தவர்களுக்கு) இது அவசியம் தேவை
சாட்சியின் கையொப்பம் /..... இடம் .....
பெயர் மற்றும் விலாசம் .....

CONSENT FORM

Format for Informed Consent Form for Parent / Guardian of the Subjects

Informed Consent form to participate in a research study

Study Title:

Study Number: _____

Subject's Initials: _____ **Subject's Name:** _____

Date of Birth / Age: _____

(i) I confirm that I have read and understood the information sheet dated _____ for the above study and have had the opportunity to ask questions. [ ]

(ii) I understand that my son / daughter's participation in the study is voluntary and that he/she is free to withdraw at any time, without giving any reason, without his/her medical care or legal rights being affected. [ ]

(iii) I understand that the Ethics Committee and the regulatory authorities will not need my permission to look at my son / daughter's health records both in respect of the current study and any further research that may be conducted in relation to it, even if he/she withdraws from the trial. I agree to this access. However, I understand that my son / daughter identity will not be revealed in any information released to third parties or published. [ ]

(iv) I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s). [ ]

(v) I agree for the participation of my son/daughter in the above study. [ ]

Signature (or Thumb impression) of the Subject's parent /Legally Acceptable Guardian
Date: ____/____/_____
Signatory's Name: _____
Signature: _____
Or
Representative: _____
Date: ____/____/_____
Signatory's Name: _____

Signature or thumb impression of the Witness: _____
Date: ____/____/_____
Name & Address of the Witness: _____

PROFORMA

CASE NO:

A.PATIENT PARTICULARS:

Name
Age
Sex
Occupation
OP/Number IP/Number

1. Chest pain
    - Location
    - Duration
    - Type
    - Radiation
    - Aggravating and relieving factors
    - Associated symptoms

2. Palpitations
    - Onset
    - Duration
    - Type
    - Aggravating or relieving factors3.

3.Syncope

- Onset
- Duration
- Aggravating factors
- Associated symptoms (palpitations, seizures)

4. Dyspnoea

- Onset
- Progression
- Duration
- Paroxysmal nocturnal dyspnea
- Aggravating and relieving factors

5. Easy fatiguability

B. OTHER PRESENTING COMPLAINTS

C. PAST HISTORY

- Ischemic Heart Disease'
- Congestive cardiac failure
- Stroke
- Treatment for hypertension/bronchial asthma/Diabetes mellitus

D. FAMILY HISTORY

- Diabetes mellitus
- Ischemic Heart Disease
- Stroke
- Hypertension

E. PERSONAL HISTORY

Diet- Vegetarian /Mixed

Smoking (cigarette/beedis) - Yes/No

Alcohol - Yes/No

MENSTRUAL HISTORY:

If menopausal _____ years after menopause

F. EXAMINATION

1. Height
2. Weight
3. BMI
4. Pulse
5. Blood pressure
6. General Examination
7. Cardiovascular system
8. Other systems: a. RS b. GIT c. CNS

KEY TO MASTER CHART

Sl.No	~	Serial Number
IHD	~	Ischemic heart disease
Ht	~	Height
Wt	~	Weight
BMI	~	Body Mass Index (Weight(kg)/Height (m2))
SBP	~	Systolic Blood Pressure
DBP	~	Diastolic Blood Pressure
FBS	~	Fasting Blood Sugar
PPBS	~	Post Prandial Blood Sugar
TC	~	Total Cholesterol
TG	~	Triglycerides
HDL	~	High Density Lipoproteins
LDL	~	Low Density Lipoproteins
MA	~	Microalbuminuria
+	~	Present
-	~	Not present

SL.NO	NAME	Age in years	Sex	Family History of IHD	Smoking	HT	WT	BMI	SBP	DBP	FBS	PPBS	TC	TG	HDL	LDL	MA	INFARCT	ISCHEMIA	TROP T	Ejection fraction%
1	Raja	66	M	NO	YES	170	74	25.6	160	80	99	150	184	114	42	98	39	AMI	-	+	43
2	Ramesh	48	M	YES	YES	166	70	25.4	130	80	100	140	202	167	51	120	71	AMI	-	+	53
3	Suraj	58	M	YES	YES	174	80	26.4	150	90	100	138	202	114	40	138	80	IWMI	-	+	42
4	Surya	53	M	YES	YES	172	74	25	140	80	88	112	156	116	40	98	40	AMI	-	+	44
5	Durai	64	M	NO	YES	164	68	25.3	140	90	100	130	184	188	44	108	40	AMI	-	+	50
6	Muthu	60	M	NO	YES	164	67	24.9	140	90	103	154	174	110	50	110	24	-	YES	+	49
7	Mary	58	F	NO	NO	158	64	25.6	140	90	84	115	176	141	34	123	17	IWMI	-	+	46
8	Ganapathy	62	M	YES	YES	168	72	25.5	140	80	102	140	170	120	40	120	40	-	YES	-	46
9	Arun	48	M	NO	YES	166	74	26.9	140	90	98	144	210	185	42	170	29	-	YES	+	54
10	Mani	71	M	NO	NO	164	70	26	140	100	104	160	150	124	40	110	28	AMI	-	+	63
11	Mariyaal	59	F	YES	NO	150	68	30.2	110	80	101	140	217	100	48	149	27	-	YES	+	51
12	Ahammad	58	M	NO	YES	154	60	25.3	10	80	96	128	176	141	35	114	15	IWMI	-	+	48
13	Manoj	54	M	YES	YES	166	74	26.9	110	80	80	110	160	120	44	108	29	-	YES	+	54
14	Abisha	60	F	NO	NO	154	60	25.3	110	70	104	144	170	150	46	108	20	-	YES	+	56
15	Arjun	26	M	NO	YES	168	70	24.8	100	70	110	154	160	120	40	120	30	-	YES	-	44
16	Suhail	34	M	YES	YES	172	78	26.4	120	70	110	142	142	110	39	81	30	AMI	-	+	48
17	Jeya	63	F	NO	NO	154	60	25.3	120	70	99	112	200	168	43	140	44	-	YES	+	56
18	Soosai	40	M	NO	YES	169	75	26.3	130	80	90	130	142	110	42	130	29	AMI	-	+	58
19	Chellam	57	F	NO	NO	150	68	30.2	110	80	98	132	190	441	30	130	28	AMI	-	-	52
20	Mathan	33	M	NO	YES	168	75	26.6	110	70	110	154	202	167	49	120	61	AMI	-	+	55
21	Sudalai	55	M	NO	YES	158	60	24	130	70	100	138	110	115	40	120	30	IWMI	-	-	46
22	Sheela	51	F	NO	NO	154	68	28.7	120	70	109	134	179	111	52	109	64	AMI	-	-	58
23	Nataraj	50	M	YES	YES	172	74	25	120	80	82	108	114	120	40	94	34	-	YES	+	45
24	Moorthy	67	M	YES	YES	168	69	24.5	120	80	103	140	156	116	40	92	30	AMI	-	+	52
25	Ganesh	50	M	NO	YES	168	75	26.6	140	80	88	108	192	110	48	124	60	AMI	-	+	60
26	Ammal	57	F	NO	NO	15	66	28.6	140	100	100	134	200	160	43	140	54	-	YES	+	56
27	Raman	34	M	YES	YES	168	75	26.6	130	80	96	140	186	102	40	128	78	AMI	-	+	48

28	Raadha	60	F	NO	NO	154	62	26.1	160	100	100	148	169	154	38	120	53	AMI	-	+	54
29	Pachaimuthu	60	M	NO	YES	157	68	27.6	140	90	110	170	148	124	46	108	30	AMI	-	-	46
30	Arumugam	60	M	NO	YES	174	80	26.4	150	90	115	160	190	119	40	140	46	-	Yes	+	48
31	Ragu	35	M	NO	YES	172	78	26.4	140	90	102	144	208	154	48	128	26	AMI	-	+	48
32	Chellapa	65	M	NO	YES	174	76	25.1	150	80	98	130	175	142	44	107	28	AMI	-	+	46
33	Valli	54	F	YES	NO	154	68	28.7	150	80	90	110	210	164	44	133	34	AMI	-	-	55
34	Vadivu	68	F	NO	NO	150	68	30.2	150	80	98	120	140	441	30	130	38	-	YES	+	53
35	Murugan	54	M	YES	YES	162	69	26.3	140	80	104	142	142	110	42	90	24	-	YES	+	45
36	Saravanan	42	M	NO	YES	169	75	26.3	130	80	94	138	170	134	43	127	71	AMI	-	+	55
37	Sundar	55	M	YES	YES	165	69	25.3	140	80	90	146	142	120	39	81	40	-	YES	-	58
38	Rajesh	31	M	YES	YES	168	74	26.2	130	80	80	110	151	119	40	94	34	AMI	-	+	54
39	Mala	50	F	NO	NO	145	60	28.5	140	100	104	140	170	100	45	120	39	IWMI	-	+	51
40	Lingam	70	M	NO	YES	160	64	25	160	90	82	110	249	179	40	185	140	AMI	-	+	52
41	Shanthi	75	F	NO	NO	154	74	31.2	160	90	100	120	179	121	52	104	74	AMI	-	+	45
42	Raani	61	F	NO	NO	154	60	25.3	150	90	112	181	217	98	48	149	37	-	YES	+	56

43	Senthil	42	M	NO	YES	169	75	26.3	120	80	88	124	170	134	43	117	71	-	YES	-	48
44	Selvamani	66	F	NO	NO	150	62	27.6	160	90	110	158	176	141	36	114	34	AMI	-	+	43
45	Aathimoolam	56	M	YES	YES	166	72	26.1	150	90	80	110	168	187	48	92	40	-	YES	+	41
46	Laxman	26	M	NO	YES	168	70	24.8	130	90	90	110	184	121	45	120	92	AMI	-	+	43
47	Jegan	58	M	YES	YES	174	84	27.7	160	100	110	150	170	135	45	130	68	AMI	-	-	58
48	Raam	54	M	YES	YES	158	60	24	150	90	107	140	210	164	43	134	40	AMI	-	+	48
49	Mahesh	60	M	NO	YES	165	70	25.7	140	90	98	135	144	120	40	98	44	AMI	-	+	51
50	Maarimuthu	69	M	NO	YES	166	65	23.6	150	90	114	150	180	102	44	154	70	AMI	-	+	54
51	Mathavan	49	M	NO	YES	166	71	25.8	140	90	108	150	180	110	42	120	39	AMI	-	-	42
52	Jamuna	60	F	NO	NO	150	68	30.2	130	90	104	150	175	136	45	102	59	AMI	-	-	52
53	Raamadhas	75	M	NO	YES	163	68	25.6	160	90	104	134	165	146	40	112	70	-	YES	+	62
54	Parvathy	56	F	YES	NO	156	58	23.8	150	80	98	120	190	431	30	130	38	-	YES	+	53
55	Maruthu	40	M	YES	YES	170	78	27	130	90	110	154	160	120	44	118	39	AMI	-	-	62
56	Paandi	62	M	YES	YES	166	74	26.9	170	100	108	154	204	170	44	133	48	AMI	-	-	60
57	Beema	54	M	YES	YES	158	60	24	150	90	107	140	210	174	43	134	40	AMI	-	+	48
58	Babu	70	M	YES	YES	158	62	24.8	150	100	99	142	160	120	42	110	55	-	YES	+	56
59	Ambujam	50	F	NO	NO	150	64	28.4	150	100	109	176	185	193	44	110	53	-	YES	+	66
60	Ravi	40	M	YES	YES	168	74	26.2	130	90	104	144	200	160	44	170	38	-	YES	+	42

61	Nallathangam	60	F	NO	NO	154	60	25.3	110	70	104	142	170	151	48	110	21	-	YES	+	56
62	Subramanian	73	M	YES	YES	164	70	26	120	80	98	125	178	190	45	102	29	-	YES	+	61
63	Sujatha	61	F	NO	YES	150	64	28.4	130	80	100	138	173	138	35	115	21	-	YES	+	55
64	Sheeba	60	F	NO	NO	154	62	26.1	130	80	110	144	175	187	40	105	24	AMI	-	+	52
65	Seenivasan	58	M	NO	YES	162	69	26.3	120	80	96	119	129	108	44	98	19	-	YES	-	42
66	Chandran	42	M	YES	NO	166	70	25.4	120	70	84	108	174	130	44	127	63	-	YES	+	44
67	Muthaiyaah	57	M	NO	YES	166	72	26.1	120	90	104	140	180	129	40	124	58	IWMI	-	+	48
68	Sundaram	56	M	YES	YES	174	80	26.4	110	60	90	115	164	130	43	136	28	AMI	-	+	61
69	Muthumani	62	F	NO	NO	154	62	26.1	120	70	94	120	179	149	45	118	30	-	YES	-	46
70	Anbu	39	M	YES	NO	166	70	25.4	110	70	97	132	198	158	45	110	41	AMI	-	-	48
71	Sheela	38	F	YES	NO	168	75	26.6	130	90	91	110	178	114	40	130	39	-	YES	+	58
72	Vadivel	60	M	YES	YES	162	69	26.3	140	90	100	140	165	110	42	117	27	AMI	-	-	47
73	Anjali	44	F	YES	NO	160	64	25	140	100	106	144	104	124	34	119	35	IWMI	-	+	53
74	Arivarasan	45	M	YES	NO	166	74	26.9	120	80	98	126	112	156	40	156	40	-	YES	+	54
75	Sudherson	36	M	NO	NO	165	69	25.3	110	70	88	126	170	118	39	119	66	AMI	-	+	53
76	Shoba	69	F	YES	NO	145	60	28.5	140	90	104	140	165	104	60	106	70	AMI	-	+	45

77	Shenbagam	58	F	YES	NO	158	64	25.6	130	90	86	114	156	134	30	150	32	AMI	-	+	48
78	Shanmugam	71	M	YES	YES	152	66	28.6	110	70	94	134	184	154	43	140	54	-	YES	+	56
79	Paulraj	65	M	NO	NO	162	69	26.3	130	70	107	129	149	142	40	96	39	-	YES	+	55
80	Peter	58	M	NO	NO	157	68	27.6	140	90	110	142	180	114	40	136	42	AMI	-	+	48
81	Parimalam	40	F	YES	NO	154	62	26.1	120	70	100	134	190	164	41	139	58	-	YES	+	58
82	Sasi	39	F	NO	NO	154	68	28.7	140	90	99	124	180	244	32	128	45	AMI	-	+	49
83	Alex	40	M	YES	YES	170	74	25.6	140	90	110	144	175	140	48	121	28	AMI	-	+	58
84	Michael	65	M	NO	YES	158	69	24.5	130	90	94	130	150	138	46	129	38	AMI	-	+	50
85	Selvi	60	F	NO	NO	150	60	26.7	140	90	90	134	115	180	45	108	35	-	YES	+	48
86	Maaran	64	M	NO	YES	167	74	26.5	150	90	108	158	164	130	38	135	25	AMI	-	+	42
87	Vel	34	M	YES	YES	150	70	31.1	140	90	110	130	190	100	44	120	65	AMI	-	+	64
88	Petchiammal	68	F	NO	NO	150	65	28.9	130	80	98	132	240	180	45	170	50	-	YES	-	47
89	Nanthan	48	M	YES	YES	170	85	29.4	120	90	102	138	200	150	52	125	65	AMI	-	+	61
90	Uma	56	F	NO	NO	155	60	25	130	80	100	134	160	170	50	100	40	-	YES	+	41
91	Vijay	40	M	YES	YES	160	70	27.3	140	90	88	142	180	150	40	106	42	-	YES	+	47

92	Muthamil	62	M	YES	YES	172	85	28.7	150	80	90	138	198	180	48	125	50	AMI	-	+	60
93	Nanthini	58	F	NO	NO	160	65	25.4	120	80	88	128	180	145	42	118	31	AMI	-	+	43
94	Sahana	26	F	NO	NO	155	55	22.9	120	80	84	128	170	100	40	110	44	AMI	-	+	55
95	Yogesh	69	M	NO	YES	170	90	31.1	140	80	102	142	202	105	42	150	80	AMI	-	+	54
96	Yasmin	66	M	YES	YES	160	75	29.3	150	90	101	140	150	120	39	101	43	-	YES	+	48
97	Durga	54	F	YES	NO	158	68	27.2	130	90	88	130	212	184	52	135	32	AMI	-	-	55
98	Duraiarasan	52	M	YES	YES	164	67	24.9	120	80	98	128	156	112	54	112	35	-	YES	+	49
99	Chandran	60	M	NO	YES	174	76	25.1	130	90	90	110	170	108	50	125	30	AMI	-	+	58
100	Senthil	58	M	NO	NO	160	64	25	140	80	100	156	140	115	45	118	46	-	YES	+	62
101	Thangam	76	M	YES	YES	169	75	26.3	130	90	89	140	190	142	43	136	62	AMI	-	+	56
102	Fathima	69	F	NO	NO	162	69	26.3	140	80	98	132	143	112	44	132	43	-	YES	-	46
103	Selvam	55	M	YES	NO	166	74	26.9	130	80	84	134	141	119	42	99	38	-	YES	+	51
104	Issac	35	M	NO	NO	172	74	25	110	70	84	108	158	126	40	114	44	AMI	-	+	55
105	Harish	38	M	YES	YES	168	75	26.6	130	90	98	135	164	114	44	130	32	AMI	-	+	49

106	Mariyammal	62	F	NO	NO	166	72	26.1	120	70	98	139	218	102	54	134	36	-	YES	+	46
107	Mohan	58	M	YES	YES	159	68	26.9	120	80	88	112	190	112	46	120	36	AMI	-	+	54
108	Muthumaari	45	F	YES	NO	155	63	26.1	130	80	96	136	168	144	40	110	60	-	YES	+	52
109	Vijaya	36	F	NO	NO	164	70	26	140	90	100	138	178	119	43	140	72	IWMI	-	+	42
110	Sarawathi	65	F	NO	NO	154	60	25.3	130	70	100	148	188	145	45	120	53	AMI	-	+	60
111	Duraiswamy	40	M	NO	NO	166	70	25.4	150	90	104	139	112	180	48	149	39	-	YES	+	58
112	Annamalai	68	M	YES	YES	172	73	24.7	120	80	95	136	190	124	43	127	69	AMI	-	+	56
113	Saroja	43	F	NO	NO	154	62	26.1	150	90	114	150	180	144	44	136	58	IWMI	-	+	42
114	Muthuswamy	66	M	YES	YES	168	75	26.6	170	100	110	148	202	180	46	134	50	AMI	-	+	62
115	Palanimuthu	39	M	NO	NO	172	74	25	140	90	88	120	188	108	44	126	85	AMI	-	+	43
116	Arunthathi	47	F	NO	NO	158	60	24	130	80	96	120	208	154	46	148	40	AMI	-	-	55
117	Ananthi	33	F	YES	NO	160	69	26.9	140	80	92	114	195	179	48	100	39	-	YES	+	52
118	Ilavarasan	70	M	YES	NO	172	78	26.4	120	70	99	143	196	114	40	135	68	AMI	-	+	56
119	Sundari	43	F	NO	NO	168	75	26.6	140	90	104	148	185	156	49	109	59	AMI	-	-	58

120	Moorthy	67	M	NO	YES	168	72	25.5	120	80	110	149	159	117	42	94	39	AMI	-	-	56
121	Latha	29	F	NO	NO	152	66	28.6	130	90	80	112	168	119	44	98	34	-	YES	-	66
122	Manjula	39	F	YES	NO	154	62	26.1	140	80	102	144	192	150	45	129	26	AMI	YES	-	48
123	Muthammal	47	F	NO	NO	152	60	26	120	80	101	141	175	104	49	108	18	AMI	-	+	48
124	Vembu	42	F	YES	NO	160	72	28.1	110	80	96	130	149	115	42	125	35	-	YES	+	54
125	Ganapathy	57	M	YES	YES	176	85	27.4	120	60	102	139	17	134	46	129	59	-	YES	+	49
126	Paapa	61	F	YES	NO	150	50	22.2	140	90	99	129	247	168	48	175	39	AMI	-	+	52
127	Sedhu	46	M	NO	NO	160	55	21.5	150	80	98	132	198	441	30	130	60	-	YES	-	58
128	Abdul	53	M	YES	NO	168	72	25.5	120	70	99	122	186	104	44	128	38	AMI	-	+	62
129	Peer Mohammad	69	M	YES	YES	159	68	26.9	130	80	108	140	190	110	46	100	49	AMI	-	+	48
130	Chitra	40	F	NO	NO	153	63	26.9	110	60	100	134	200	169	44	120	60	-	YES	+	55
131	Ramachandran	66	M	YES	NO	172	62	21	130	80	94	133	240	180	45	119	38	-	YES	-	60
132	Muthalaghu	42	F	YES	NO	156	74	30.4	120	90	96	141	187	100	43	126	70	AMI	-	+	52
133	Maangani	36	F	NO	NO	162	80	30.5	130	80	90	146	140	119	40	81	44	-	YES	-	60

134	Madaswamy	72	M	YES	YES	162	75	28.6	140	80	104	149	179	149	46	103	54	AMI	-	+	51
135	Nagendran	63	M	YES	YES	170	80	27.7	130	90	99	137	188	116	48	136	74	IWMI	-	+	48
136	George	68	M	YES	NO	165	70	25.7	140	100	101	158	159	146	49	110	29	AMI	-	+	63
137	Yasodhai	39	F	NO	NO	155	51	21.2	130	90	112	150	190	116	46	156	70	AMI	-	+	54