“MICROALBUMINURIA IN NON DIABETIC ACUTE CORONARY SYNDROME PATIENTS”

By

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Dissertation submitted to BLDE University, Vijayapur

In partial fulfillment of the requirements for the award of the degree of

DOCTOR OF MEDICINE

IN

GENERAL MEDICINE

Under the guidance of

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Affectionately Dedicated to

My Beloved Parents

Mr. Jayaram

and

Mrs.Jayalakshmi

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Dr. SHANKAR KUMAR TALIKOTA

LIST OF ABBREVIATIONS USED

DD	:	Duration of disease
HTN	:	Hypertension
MI	:	Myocardial infarction / Angina
PH	:	Pulmonary hypertension
ACS	:	Acute Coronary Syndrom
AER	:	Albumin Excretion Rate
GFR	:	Glomerular Filtration Rate
HDL	:	High Density Lipoproteins
IHD	:	Ischemic Heart Disease
LDL	:	Low Density Lipoprotein
PVD	:	Peripheral Vascular Disease
VLDL	:	Very Low Density Lipoprotein
SIG	:	Significant
CVD	:	Cardiovascular disease
CAD	:	Coronary Artery Disease
VSMC	:	Vascular Smooth Muscle Cell
STEMI	:	ST-segment Elevation Myocardial Infarction
NSTEMI	:	Non–ST-segment Elevation Myocardial Infarction
UA	:	Unstable Angina

VHD	:	Valvular heart disease
EF	:	Ejection fraction
RWMA	:	Regional wall motion abnormality
PCI	:	Percutaneous Coronary Intervention
IMT	:	Intima-Media Thickness
AP	:	Angina Pectoris
TC	:	Total leucocyte Count
Sr	:	Serum
D	:	Days
H	:	Hours
P	:	Present
Ab	:	Absent
CP	:	Chest pain
JVP	:	Jugular venous pressure
Mrm	:	Murmur
V	:	Vegetarian
NV	:	Non vegetarian

ABSTRACT

Background :

Atherosclerosis remains the leading cause of death and premature disability. Coronary diseases Aute coronary Syndrome(ACS) is a common complication and this is associated with more than 2.5 million hospitalizations world wide each year, Endothelial dysfunction seems to play a key role in non-diabetic glomerulosclerosis and atherosclerosis. Increased permeability of the endothelium allows atherosclerotic lipoprotein particles (oxidized LDL and others) to penetrate into the vessel wall and promote the development of atherosclerotic plaques be associated with higher prevalence of coronary artery disease , microalbuminuria has been suggested as a risk factor indicator for cardiovascular events.

Objective :

To estimate microalbuminuria in non-diabetic patients with Acute Coronary Syndrome And assess the relationship between the two

Methodology :

All patients age >18yrs, both sexes diagnosed as acute coronary syndrome based on history and relevant investigations and are admitted in ICU in BLDEU'S Shri B.M PATIL Medical college hospital and research centre Vijayapur. microalbuminuria was measured at admission and compared with standard normal mean value.

Results :

This study was conducted on 60 patients, of the study group 70.0% were male and 30.0% were female. The age ranged from 30 to 85 years of age. The mean age of the

group was 55.5 ±13.19 SD. The known risk factors of ACS were studied and correlated, 37.2 % of all patients were smokers, 31% were tobacco chewers, 24.7 % had diabetes mellitus, 31.8% were hypertensive and 8 % had family history of ACS. The mean microalbuminuria value in mg/dl for STEMI was 35 ± 0.30 SD, for NSTEMI it was 21± 1.6 and for unstable angina it was 22 ± 1.0 SD. The mean microalbuminuria in patients with ACS was 44.6 ± 3.2 SD mg/dl compared to microalbuminuria levels of 30mg/l in normal population ($p<0.0001$).

Conclusion :

This study showed an correlation of microalbuminuria with ACS. This reinforces the fact that microalbuminuria acts as emerging potential risk factor marker.

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INTRODUCTION

MICROALBUMINURIA :

Microalbuminuria is defined as an increased urinary albumin excretion which is detectable only by sensitive immunoassay (1) (expressed either by time or with reference to creatinine concentration), it has been used for many years as a predictor of develop nephropathy in diabetic patients. (2) Recently it has been suggested that micro albuminuria may be a risk factor for the development of cardiovascular disease in the non-diabetic patients and may therefore have role in screening programmes .

ALTERNATIVE MARKERS OF CARDIOVASCULAR RISK :

There is a need for a marker that will more precisely identify those at higher risk. Most of the markers investigated have failed to find general application. For example the significance of lipoprotein (a) remains uncertain and of the ten largest prospective studies , only six concluded that it was an independent risk factor . (4) Less controversy surrounds raised plasma fibrinogen concentration as an independent risk factor, as indicated by early Framingham data and confirmed in subsequent large-scale studies.(5,6) Its failure to be widely adopted is partly due to the lack of a fibrinogen –reducing therapy, although there is evidence that some lipid-lowering agents have fibrinogen –lowering capability.(7) Denesh et al. have recently reviewed the clinical and epidemiological evidence linking chronic infection, either bacterial (helicobacter pylori, Chlamydia pneumonia) or viral (cytomegalovirus), with coronary artery disease.(8) despite over 50 studies, the association remains dubious.

Against this background, microalbuminuria has been suggested as a risk factor indicator for cardiovascular events.

Microalbuminuria is used clinically to monitor diabetic nephropathy, but it is also know to be a non- specific marker of inflammation, as both systemic and local (9-11) and appears to be useful as a predictor of outcome in several clinical situation.(12-16)

An increase in urinary albumin excertion is indicative of increased glomerular permeability. The normal urinary albumin excertion of albumin is less than 30 mg/day. Albuminuria normally refers to greater than 300mg/day of albuminuria, with the term Microalbuminuria being used to describe smaller degrees of albuminuria.

Microalbuminuria is defined as urinary albumin excretion rate of more than 20mg/l and less than or equal to 200mg/l .(18) or 30 to 300mg/day in a 24hr collection.(19).

Endothelial dysfunction seems to play a key role in non-diabetic glomerulosclerosis and atherosclerosis. Increased permeability of the endothelium allows atherosclerotic lipoprotein particles (oxidized LDL and others) to penetrate into the vessel wall and promote the development of atherosclerotic plaques .

Microalbuminuria is thought to reflect the glomerular component of a systemic capillary leak (20) is fundamental to the pathogenesis of multiple organ failure (21). In the healthy kidney over 99% of filtered albumin is recollected by mechanisms that are close to saturation. A small increase in glomerular vascular permeability resolves in an increase in filtered albumin presented to the renal tubules. This cannot be reabsorbed and results in large increases in urinary albumin.

This amplification resolves in sudden rise in albumin excertion after the occurance of systemic inflammation, maximum microalbuminuria find up to 2 days before than the other markers of systemic inflammation such as C reactive protein (22).

ISCHAEMIC HEART DISEASE:

EPIDEMIOLOGY

World scenario :

Coronary heart disease is a worldwide health epidemic.[28] Aute coronary Syndrome(ACS) is a common complication and this is associated with more than 2.5 million hospitalizations worldwide each year. In the United states, for example, A conservative estimate for the number of discharges with ACS from hospital data in 2006 was 13,65000 unique for ACS.[29]

Indian scenario :

India has the highest number of cases ACS in the world. Treatment and outcomes of Acute Coronary Syndromes in India (CREATE) in order data base has provided contemporary data on 20,468 patients with ACS from 89 centers from 10 regions and 50 cities in India and found higher 30 day mortality than developed countries.[30] Cardiovascular risk factors for ACS are on the rise in people of Indian origin and ACS is now the leading cause of death.[6,7,8,9,10]

Coronary arterial vasoconstriction[46]

Vasoconstriction causing dynamic obstruction of coronary arterial flow may result from spasm of the epicardial coronary arteries (Prinzmetal angina)—constriction of small intramural, muscular coronary arteries resulting in increased coronary vascular resistance. This constriction may result from vasoconstrictors released by platelets, endothelial dysfunction or adrenergic stimuli (e.g., the “fight-or-flight” response, cold,

cocaine, or amphetamines). More than one of these mechanisms may be present simultaneously.

Imbalance between the supply and demand of the myocardium for oxygen[46,52]

In normal conditions, for any given level of a demand for oxygen, the myocardium will control the supply of oxygen-rich blood to prevent underperfusion of myocytes and the subsequent development of ischemia and infarction. The major determinants of myocardial oxygen demand (MVO_2) are heart rate, myocardial contractility and myocardial wall tension (stress).

By reducing the lumen of the coronary arteries, atherosclerosis limits appropriate increases in perfusion when the demand for flow is augmented, as occurs during exertion or excitement. When the luminal reduction is severe, myocardial perfusion in the basal state is reduced.

Myocardial ischemia also can occur if myocardial oxygen demands are markedly increased and particularly when coronary blood flow may be limited, as occurs in severe left ventricular hypertrophy due to aortic stenosis. The latter can present with angina that is indistinguishable from that caused by coronary atherosclerosis largely owing to subendocardial ischemia. A reduction in the oxygen-carrying capacity of the blood as in severe anemia or in the presence of carboxyhemoglobin, the hypotension rarely causes myocardial ischemia by itself but may lower the threshold for ischemia in patients with moderate coronary obstruction.

When an increase in myocardial O2 demand (e.g., tachycardia, fever, thyrotoxicosis) occurs in a patient with fixed narrowing of an epicardial coronary artery, secondary non ST elevation-acute coronary syndrme(NSTE-ACS) may develop.

Not infrequently, two or more causes of ischemia coexist in a patient, such as an increase in oxygen demand due to left ventricular hypertrophy secondary to hypertension and a reduction in oxygen supply secondary to coronary atherosclerosis and anaemia. Abnormal constriction or failure of normal dilation of the coronary resistance vessels also can cause ischemia. When it causes angina, this condition is referred to as microvascular angin

Ischemic refers to not having enough of oxygen due to inadequate supply, which results from an imbalance between oxygen supply and demand. The most common cause of myocardial ischaemia is atherosclerotic plaque of epicardial coronary arteries. This condition causes more mortality and morbidity, it leads to greater economic costs than any other illness in the developed world.

AIMS AND OBJECTIVES

OBJECTIVE OF STUDY:

1. To estimate microalbuminuria in non-diabetic patients with Acute Coronary Syndrome And assess the relationship between the two.

REVIEW OF LITERATURE

REVIEW OF LITERTURE

PATHOPHYSIOLOGY OF MICROALBUMINURIA :

The close relationship between low-level albumin excretion and vascular permeability makes urinary albumin excretion more sensitive to the presence of any inflammatory process, including cardiovascular disease. The kidney is ideally placed to amplify any small changes in systemic vascular permeability. The glomeruli receive 25% of the cardiac output of the 70kg of albumin that pass through the kidneys every 24hr, less than 0.01% reaches the glomerular ultrafiltrate (ie. Less than 7g/24hr) and hence enters the renal tubules.(23,24)

Almost all filtered albumin is reabsorbed by the proximal tubule via a high-affinity, low capacity endocytotic mechanism, (25) with only 10-30mg/24hr appearing in the urine. presuming that 7g of albumin is filtered every 24hr, a 1% increase in systemic vascular permeability in response to an inflammatory stimulus would result in an additional 70mg of albumin passing into the filtrate. Since tubular mechanisms for albumin reabsortion are near saturation, urinary albumin excretion would increase from maximum of 30 to approximately 100mg/24hr.

Glomerular permeability to albumin is dependent on endothelial charge selectivity as well as size selectivity. The negative charge conferred on the glomerular membrane by its constituent glycoproteins play a role in restricing the permeability of anionic proteins . Loss of glomerular charge selectivity has been found in both diabetic and non-diabetic population with microalbuminuria.(26,27)

Pathophysiological processes associated with microalbuminuria.

Local process:

1. Increased intraglomerular capillary pressure
2. Increased shunting of albumin through glomerular

membrane pores.

Systemic process:

1. Activation of inflammatory mediators
2. Increased transcapillary escape rate of albumin
3. Vascular endothelial dysfunction.

Endothelial dysfunction seems to play a key role in non-diabetic glomerulosclerosis and atherosclerosis. Increased permeability of the endothelium allows atherosclerotic lipoprotein particles (oxidized LDL and others) to penetrate into the vessel wall and promote the development of atherosclerotic plaques .

The sub clinical increases in urinary albumin excretion , which define microalbuminuria, are probably glomerular in origin. Microalbuminuria is not accompanied by the changes in excretion of β2-microglobulin, a low molecular weight protein with an Einstein stokes radius of approximately 1.6nm, which is freely filtered across the glomerular capillary barrier and taken up by proximal tubular cells. its excretion reflects the degree of tubular uptake of the filtered protein (Peterson et al .1969, wibell 1974). The consistency of β2 microglobulin excretion rate in the face of augmented albumin excretion rate , whether at rest or after exercise, suggests that the excess albuminuria is not the result of a change in tubular reabsorption of protein, but is more likely to derive from increased glomerular leakage.

(Feldt –Rasmussen et al .1986)

The glomerular capillary blood- urine-barrier can be regarded functionally as amembrane perforated by pores of an average size of 5.5nm and uniformly coated with a negative electrical charge (pappenhemimer et al.1951; Brenner et al. 1978; Venkatachalam and Renke 1978; Deen and Satvat 1981; Myers et al. 1982). Therefore both the size and charge of circulating molecule, as well as the set of haemodynamic forces operating across the capillary wall, will determine the passage of protein across the glomerular membrane (Viberti et al .1983; Viberti and Keen 1984). These early increases are likely to be the consequence of alterations in glomerular haemodynamics. An increased transglomerular pressure gradient would result in a greater concentration of protein within the glomerular capillary wall, and would provide a driving force for protein diffusion in to the bowman's space in humans.

The absolute urinary clearance of neutral dextran is increased over a wide range of molecular weights in parallel with elevation in the glomerular filtration rate (Parving et al. 1979).

As microalbuminuria becomes persistent and increases in degree, the selectivity index, i.e. the ratio of the clearances of IgG to the clearance of albumin, starts to decline, reaching its lowest values when albumin excretion is approximately 90µg/min or more . this is due to a disproportionate increase in the filtration of albumin compared with that of IgG, and it marks a new stage of selective glomerular leakage of polyanionic albumin (Viberti et al 1983 ; Vibertiand Keen 1984). Experiments with clearance of neutral dextran have shown that medium size pores are unchanged at this stage (Morgenen 1971; Myers et al.1982). A probable reason for the increased glomerular filtration of albumin is a loss of fixed negative electrical charge on the membrane (Westberg and Michael 1973;

Parthasarathy and Spiro 1982; Schober et al .1982; Winetz et al.1982) this would permit increased permeation of anionic albumin , but would have little influence on IgG, a neutral molecule, the filtration of which is regulated by pore radius or number and by glomerular pressure and flow.

The mechanism of this transition from low to high levels of microalbuminuria is unknown, but it may result from a combination of haemodynamic abnormalities and the cumulative metabolic derangement of synthesis of the electronegative membrane glycosialoprotein proteoglycans (Hostetter et al .1982; Kanwar et al. 1983; Vibeti and Keen 1984; Brenner 1985). However recent studies suggest that preferential filtration on the basis of charge discrimination due to loss of glomerular polyanion may not be required to explain the facilitated clearance of anionic proteins .

Permeation of both large and medium –sized molecules through a non-size-discriminatory shunt pathway could entirely account for the observed renal clearances of albumin, IgG, and dextran probe molecules (Nakamura and Myers 1988).(28)

The glomercular capillary wall (GCW) provides a barrier to filtration of large macromolecules. The GCW has 3 components. The glomercular basement membrane (GBM), epithelial cells, and endothelial cells. The barrier to filtration is provided by two mechanisms size selectivity and charge selectivity.

Size selectivity is a feature of the size of the pores in the components of the barrier. The endothelial cells have fenestrations with an approximate radius of 40 nm and as such, do not provide an effective barrier to albumin, which has a radius of 3.6nm. the GBM has pores with a radius of 4 nm. Between the foot processes of epithelial cells, a

thin membrane are the same size as those in the GBM. The GBM and the slit diaphragms are therefore, the major components of size selectivity.

Charge selectivity is provided by negatively charged anions, such as heparin sulphate proteoglycan, which repel negatively charged molecules as albumin.

These anions are present on both endothelial cells and the GBM, which thus provide charge selectivity. The negative charge on the GBM also may be important for adhesion of epithelial cells, such that loss of the negative charge may results in disruption of the epithelial cell barrier, which in turn contributes to increased permeability to macromolecules such as albumin (due to loss of size selectivity).

When damage to the basement membrane or components of the glomerular epithelial cell occur often the first manifestation is the appearance of plasma proteins in the urine . Because albumin is the major circulating protein in plasma and is relatively close in size to that of the size of the selectivity barrier, its appearance in the urine is the most sensitive indicator of damage or disruption of the glomerular filtration barrier.(29,30,31,32).

PATHOPHYSIOLOGY OF IHD :

Epicardial coronary arteries are a main site of atherosclerotic disease. The major risk factors for atherosclerosis [high plasma low density lipoprotein [LDL], low plasma high density lipoprotein [HDL], smoking, hypertension, and diabetes mellitus] are thought to alter the normal functions of vascular endothelium. These functions are local control of vascular tone, maintenance of an anticoagulant surface, and defences against inflammatory cells. The loss of these defences leads to inappropriate constriction, luminal clot formation, and abnormal interaction with blood monocytes and platelets. These sequences leads to sub-intimal collections of fat and debris (i.e. atherosclerotic plaques) which develop at irregular rates in different artery sites of epicardial coronary tree and lead ultimately to segmental reductions in cross-sectional area (stenosis). The relationship between pulsatile flow and luminal stenosis is complex but experiments have shown that when a stenosis decreased the cross sectional area by approximately 75%, a full range of increase in flow to meet increased myocardial demand is not possible.

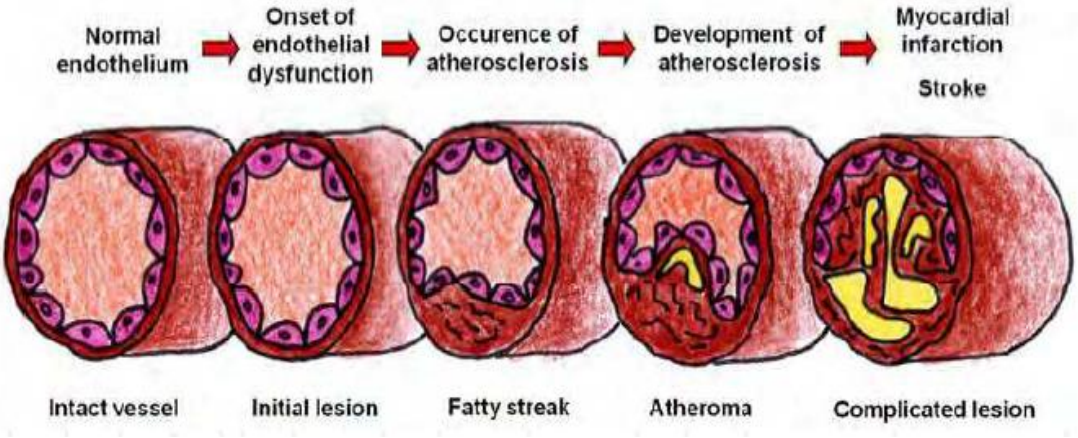
When the luminal area is decreased by more than 80%, blood flow at rest may be reduced , and further small decrease in stenotic orifice can reduce coronary flow dramatically and cause myocardial ischemia.

Segmental atherosclerotic narrowing of epicardial coronary arteries is caused most commonly by plaque , which is subject to narrowing, haemorrhage, thrombosis. Any of these conditions can temporarily worsen this obstruction leading to decreased coronary blood flow, and cause clinical manifestations of myocardial ischemia.

The location of obstruction will affect the quantity of myocardium which is ischemic thus decide the severity of clinical findings, in severe coronary narrowing and myocardial ischemia are regularly accompanied by the development of collateral vessels, mostly when the narrowing develops gradually. Well development such vessels can by themselves provide sufficient blood flow to sustain the viability of the myocardium at rest but not during conditions of increased demand.

Once stenosis of a proximal epicardial artery has decreased the cross-sectional area by more than approximately 70% , the distal resistance vessels [when they function normally] dilate to decrease vascular resistance and maintain coronary flow. A pressure gradient develops across the proximal stenosis, and post stenotic pressure is decreased . When resistant vessels are greatly dilated, and myocardial blood flow becomes supports on the pressure in the coronary artery distal to the obstruction.

In these conditions ischemia in the region perfused by the stenotic artery can be aggrevated by increases in myocardial oxygen demand caused by physical activity, emotional stress, and or tachycardia. Changes in the area of the stenosed coronary artery due to physiologic vasomotion, loss of endothelial control of dilation, pathologic constriction, or small platelet plugs can all upset the critical balance between oxygen supply and demand and thus aggrevates myocardial ischemia.



Steps involved in atherosclerotic progression

Ischemic heart disease(IHD) is a condition in which there is an inadequate supply of blood and oxygen to a portion of the myocardium; it typically occurs when there is an imbalance between myocardial oxygen supply and demand. The most common cause of myocardial ischemia is atherosclerotic disease of an epicardial coronary artery (or arteries) sufficient to cause a regional reduction in myocardial blood flow and inadequate perfusion of the myocardium supplied by the involved coronary artery.[118]

Ischemic heart disease may be manifested clinically as either chronic stable angina or an acute coronary syndrome (ACS).

Features that help differentiate ACS from stable angina are (1) onset of symptoms at rest (or with minimal exertion) and lasting longer than 10 minutes unless treated promptly; (2) severe oppressive pressure or chest discomfort; and (3) an accelerating pattern of symptoms that develop more frequently, occur with greater severity, or awaken the patient from sleep.[46] ACS can be subdivided into ST-segment elevation myocardial

infarction (STEMI), non–ST-segment elevation myocardial infarction (NSTEMI), or unstable angina (UA).

Because NSTEMI and Unstable angina are indistinguishable at initial evaluation and the entity of UA is receding as the sensitivity of biomarkers of myocardial injury increases, they are often described together as NSTE-ACS, symptoms alone do not suffice to distinguish the three types of ACS from one another. Patients without persistent (>20 minutes) ST-segment elevation in two or more contiguous leads but with biomarker evidence of myocardial necrosis are classified as having NSTEMI, whereas in patients without such evidence of myocardial necrosis, UA is diagnosed—a condition generally carrying a better prognosis.[46]

THERAPEUTIC GOALS AND APPROACHES FOR ACS[53,54]

Initial assessment:

Consider the diagnosis in patients with chest discomfort, shortness of breath, or other suggestive symptoms. Women with above 60yrs and patients with diabetes may have "atypical" presentations.

Geeting 12 lead ECG with in 10 minutes of arrival; repeat after every 10 to 15 minutes if initial ECG non diagnostic but clinical suspicion remains high (initial ECG often not diagnostic).

1. STEMI: ST segment elevation ≥1 mm (0.1 mV) in two anatomically continous leads or ≥2 mm (0.2 mV) in leads V2 and V3, or new LBBB and presentation consistent with ACS.

2. Non-STEMI or unstable angina: ST segment depressions or deep T wave inversions without Q waves or possibly no ECG changes.

Geeting emergency cardiology consultation for ACS patients with cardiogenic shock, left heart failure, or sustained ventricular tachyarrhythmia.

Initial interventions:

Evaluate and stabilize airway, breathing and circulation in casualty.

Attach cardiac and oxygen saturation monitors; provide supplemental oxygen as needed to maintain O2 saturation >90 percent, establish IV access.

Treat sustained ventricular arrhythmia rapidly according to ACLS protocols.

Give aspirin 325 mg (non-enteric coated), to be chewed and swallowed (unless aortic dissection is being considered). If oral administration not feasible, give as rectal suppository.

Give three sublingual nitroglycerin tablets (0.4 mg) one at a time, spaced five minutes apart, If patient has persistent chest discomfort, hypertension, or signs of heart failure and there is no sign of hemodynamic compromise (eg, right ventricular infarction) and no use of phosphodiesterase inhibitors (eg, for erectile dysfunction).

Give morphine sulfate (2 to 4 mg slow IV push every 5 to 15 minutes) for unacceptable persistent discomfort or anxiety related to myocardial ischemia.

Start with 80 mg of atorvastatin as early as possible, and preferably before PCI, in patients not on statin. If patient is taking a low to moderate intensity statin, switch to atorvastatin 80 mg.

Acute management STEMI:

Select reperfusion strategy: Primary percutaneous coronary intervention (PCI) strongly preferred, especially for patients with cardiogenic shock, heart failure, late presentation, or contraindications to fibrinolysis. For patients with symptoms of >12 hours, fibrinolytic therapy is not indicated, but emergent PCI may be considered, particularly for patients with evidence of ongoing ischemia or those at high risk of death.

If symptoms present with less than 12 hours and no contraindications then Treat with fibrinolysis if PCI unavailable within 120 minutes of first medical contact.

Give oral antiplatelet therapy (in addition to aspirin) to all patients:

1. Patients treated with fibrinolytic therapy: Give clopidogrel loading dose 300 mg if age of patient 75 years or less; if age more than 75 years give 75 mg.
2. Patients treated with primary percutaneous coronary intervention: Give ticagrelor loading dose of 180 mg or prasugrel loading dose of 60 mg (if no contraindications: prior stroke or TIA, or relative contraindications for prasugrel such as those age 75 years or older, weight less than 60 kg).

For patients at high risk of bleeding or those for whom prasugrel or ticagrelor cannot be used, we give clopidogrel 600 mg.

Give anticoagulant therapy to all patients: enoxaparin or Unfractionated Heparin.

Acute management of unstable angina or non-STEMI:

Give antiplatelet therapy (in addition to aspirin) to all patients:

1. Patients not treated with an invasive approach: Give ticagrelor loading dose 180mg (2tab 90mg). For these patients who are at very high risk (eg, recurrent ischemic discomfort, ECG changes, or hemodynamic instability) consider adding a GP IIb/IIIa inhibitor (either eptifibatide or tirofiban).

2. For patients treated with an invasive approach: Give clopidogrel loading dose of 300 mg at presentation. And Prasugrel loading dose of 60 mg may be used as an alternative if given after diagnostic coronary angiography.

For patients age 75 years or older, weight less than 60 kg or past stroke or transient ischemic attack, ticagrelor or clopidogrel are preferred. Clopidogrel may be given in a dose of 300 to 600 mg. For patients who are at high risk for bleeding due to prior hemorrhagic stroke, ongoing bleeding, bleeding diathesis, or clinically relevant anemia or thrombocytopenia, clopidogrel 300 to 600 mg is an option but Give anticoagulant therapy in all patients.

Other supporting studies :

In the **COPENHAGEN City Heart Study**, the authors found that otherwise Healthy individuals urinary albumin excretion level >90th percentile[>7mg/min] were characterised by increased blood pressure and lower plasma concentrations of apolipoprotien A-1 and HDL cholesterol.(33) several studies have shown that an association between small amount increased urinary albumin excretion and

cardiovascular risk factors, even in the general population [34,35,36,37,38,39,40] Moreover, they had generalised transvascular leakage for albumin. (41) These observation suggests that in an individuals with small amount increased urinary albumin excretion may be as a increased risk for subsequent development of ischemic heart disease (IHD).

The pathogenic mechanisms which leading to increased risk are still unknown but microalbuminuria has been suggested as a marker of endothelial disfunction and hypermeability to macromolecules, (42,43) which occurs early in atherogenesis(44).

In this population based study, the authors have shown that a urinary A/C ratio in the upper 10% range is independently associated with an increased risk of developing fatal or non fatal IHD. This result confirms the previous observations in patients with diabetes mellitus (45,46) and shows that microalbuminuria is a effective marker for an increased risk of IHD in non-diabetic population also. Three previous studies have shown similar findings(47,48,49).

The study of **Yudkin et al** in 1988 (47) showed that microalbuminuria (using the definition of microalbuminuria from diabetology) (50) was associated with a 24 times increased mortality rate. This high level of urinary albumin excretion, of 30mg/24hrs or more, is however, rare in non-diabetic population (51,52,53) and therefore of limited clinical relevance

Damsgaard et al in 1990 find out that urinary albumin excretion exceeding only 7.5µg/min was associated with an increased all the causes of mortality (48)

None of these studies are reported a direct relationship with the development of cardiovascular diseases, as had been found in patients with diabetes meallitus. This was however done as a case–control study of women conducted by **Gorgels et al** (49) they observed a 1.6 folds increased risk of IHD associated with an A/C ratio>0.4mg/mmol.

Gerstin HC, Mann JFE, Yi O et al; albuminuria is a risk of cardiovascular events, Have said that seeing for albuminuria may find people who are at high risk of cardiovascular (CV) events, according to an analysis of data from the heart outcomes prevention evaluation study that was published in the journal of the American medical association.

Microalbuminuria is a risk factor for Cardiovascular events, but the realationship between amount of albuminuria and CV risk is unclear**. Mc master university researchers** and others measured urine albumin/creatinine ratio(ACR) at baseline in individuals age 55 or more; and in 5,545 had a history of Cardiovascular disease and 3,498 had a diabetes and with atleast one CV risk factor.

During a median flow up to 4.5 years, researchers find the relationship of ACR to CV events (myocardial infractions, stroke, or CV death), all-cause of hospitalization for congestive heart failure. Microalbuminuria was defined as an ACR of 2 mg/mmol or more and was found in 32.6% of those with a diabetes and 14.8% of those without. Baseline microalbuminuria increased the adjusted relative risk for major CV events by 1.83-fold, all-cause mortality by 2.09-fold, and hospitalization for heart failure by 3.23-fold. The continuous, graded relationship between baseline ACR and the risk of CV outcomes and mortality extended as low as 0.22 mg/mmol. For every 0.4-mg/mmol

increase in ACR, the risk of CV event increased by 5.9% and for all causes death by 6.8%.

According to the authors, albuminuria is a strongly independent continuous risk factor for future Cardivascular events, CHF, and all this cause mortality in middle-age with a dipstick-negative individuals or without diabetes and are at high risk for CV disease.(54)

Mann and colleagues also found that microalbuminuria was an independent risk factor for cardiovascular diseases , and the relative risk of 1.59 (95% CI, 1.37 to 1.84) associated with microalbuminuria was numerically higher than the relative risk of 1.40 (CI , 1.16 to 1.69) associated with elevated creatinine concentration (55). The relation between microalbuminuria and cardiovascular diseases is more firmly established, and microalbuminuria is arguably a more sensitive marker of renal damage than serum creatinine.

Several other studies have found that microalbumnuria, proteinuria, or both predict cardiovascular disease in patients with diabetes , hypertension or both (56-58). It is plausible that intrarenal vascular disease cause glomerular and tubular damages that in turn cause microalbuminuria . if this is true, the same risk factors that lead to extrarenal cardiovascular disease could contribute to the pathogenesisof intrarenal vascular diseases and microalbuminuria . in any case screening for microalbuminuria could help to identify patients who are at high risk of cardiovascular diseases.

Jensen et al and other studies (59,60,61,62,63) strongly indicate that microalbuminuria is thightly correlated with the development of atherosclerosis. If the urinary albumin excretion begins to increase late in the atherosclerotic process, microalbuminuria may be a marker of prevalent subclinical atherosclerosis, as suggested from other cross-sectional studies, (64,65,66) and hence may be a predictor of clinical cardiovascular symptoms. If the urinary albumin excretion is already increased early in atherogenesis, as suggested by jager et al (67) microalbuminuria may reflect an endothelial dysfunction and perhaps an augmented atherogenic susceptibility to other factors, including arterial hypertension.

Sloving this problem would require a longitudinal cohort study with repeated measurements of urinary albumin excretion and severity of atherosclerosis, eg, ultrasonic assesments of the intima- media thickness and flow mediated vasodilation of arteries (68,69) . previous studies have demonstrated that microalbuminuria reflects a renal and systemic transvascular albumin leakage (70-73) that is perhaps due to low vessel wall content of heparin sulphate (74-77).

In animal models, it has been exhibited as that transvascular leakage of albumin and lipoproteins are thightly correlated (78,79) and that both are increased In atherosclerosis (80) and in the atherosclerosis prone sites of arteries. (42)

M.K Garg , J.S. Saini – Microalbuminuria – An evolving concept journal Diab .Assoc. India 1998; vol 38; 29-35 . Recently several studies have shown that Microalbumnuria predicts vascular diseases in the non diabetic population and suggested it to be more universal marker of early death from cardiovascular disease in humans.

Studies by yudkin and Nelson et al observed 11 and 3 fold increase in mortality in non diabetic, proteinuric individuals.

The relationship between minimal loss of albumin by kidney and its effect on cardiovascular system is as yet not entirely understood. Various workers have speculated mechanisms by which this association may be explained.

1. Vascular Hyperpermeability- Steno group have hypothesized that microalbumnuria is caused by the loss of positive charges on glomerular basement membrane permiting leakage of albumin and similar changes occur in blood vessels elsewhere that allow atherosclerotic lipoprotein particles to penetrate into vascular hyperpermeable state.
2. Lipid Abnormaility- it has been proposed that patients with microalbuminuria have abnormalities in lipd metabolism that is increased Lp(a), which exposes the individual to high risk of cardiovascular diseases.
3. Endothelial dysfunction- Microalbuminuria has been shown to be realted to disturbances in coagulation and endothelial function. A strong relationship between plasma von willebrand factor level (a method of endothelial dysfunction), UAE and cardiovascular complications has been reported.
4. Syndrome X – In non diabetic individuals and those pre disposed to diabetes , Microalbuminuria is closely associated with multiple cardiovascular risk factors.

Studies indicate a common linkage between Microalbuminuria , syndrome x and insulin resistance.

MATERIAL AND METHODS

MATERIALS AND METHODS

SOURCE OF DATA:

ACS patients are admitted in to _____with Acute coronary syndrome (ACS), November 2015 to July 2017.

METHOD OF COLLECTION OF DATA :

A total of 60 patients 42 were male and 18 in the age range group of 30 to 60 years were selected on the basis of simple random sampling method , Information was gathered in a pre–tested proforma .

The clinical history , clinical findings and laboratory findings were analyzed and recorded in the proforma , patients were screened for systemic manifestations, renal function tests, lipid profile, urine tests, sugar, complete Haemogram .

Early morning urine samples were collected from patients in a sterile bottle without using any preservatives . samples were tested for microalbuminuria by Turbidometric Immunoassay .

ACS was diagnosed on the basis of ECG findings and rasied cardiac enzymes and trop-t positive , patients included were those who presented with STEMI and NSTMI and UNSTABLE ANGINA.

INCLUSION CRITERIA:

ACS patients who are positive for microalbuminuria by above mentioned method Turbidometric Immunoassay.

1. Age > 18 yrs
2. Non diabetic patients with ACS diagnosis confirmed

EXCLUSION CRITERIA:

ACS patients who had a past history of

1. Patients with Diabetes Mellitus
2. Urine showing
   - Macroalbuminuria
   - RBCs
   - Leucocytes

3.Patients on ACE Inhibitors

MICROALBUMINURIA ASSAY

Sample preparation : Though random urine specimen can be used, preferably first morning urine specimen should be collected in clean dry plastic containers free from detergents and even traces of proteins. Specimen should be tested immediately preferably within 12 hours of collection. Specimen can be stored up to 2 days at 2- 8°C provided they are not contaminated. Specimen should be free from particulate matter.

PRINCIPLE : A turbidimetric immunoassay for the detection of albumin in urine and is based on the principle of agglutination reaction. The test specimen is mixed with the activation buffer (R1) and Turbodyne MA anti-human antibody solution (R2) and allowed to react. Presence of albumin in the test specimen forms an insoluble complex producing a turbidity, which is measured at wavelength ~ 650 nm.

ASSAY PROCEDURE : The assay procedure is based on the immunoprecipitation reaction in liquid phase developed by tulip diagnostics, the assay was performed according to the instruction manual supplied by the company . Turbid or particulate urine specimen must be clarified by centrifugation at 2000 rpm for 10 minutes.

REAGENT :

R1: Turbodyne MA Activation Buffer

R2: Turbodyne MA anti-human albumin Reagent

TEST PROCEDURE:

1. Bring reagent and sample to room temperature before use.
2. Insert the Turbodyne MA smart card in the card reader slot of the Turbodyne SC as described in the user manual.
3. The instrument will indicate to place cuvette with R1 + sample in the reading chamber.
4. Take a disposable Turbodyne cuvette (provided in the kit) and add 250 µl R1 using fresh clean disposable micropipette tips; Then add 20µl sample and incubate the cuvette for 3-5 minutes.
5. Place the cuvette with R1 + sample in the Turbodyne SC reading chamber.
6. Press “Testing”. The instrument will mix the sample and then indicate to add R2.
7. Pipette 100µl R2 reagent with the Turbodyne SC Electronic Pipette to the cuvette with R1+sample.
8. The reaction will start and the counter will start in the display. Results will be displayed on completion of reaction.

RESULTS

In the **60** patients with Acute Coronary Syndrome (ACS) studied **70.0%** were male and **30.0 %** females. Males are more cases than female

Table 1: Distribution of cases according to Sex

SEX	**N**	**%**
Male	42	70
Female	18	30
Total	60	100

M/F ratio	**2.3**

Figure 1: Distribution of cases according to Sex

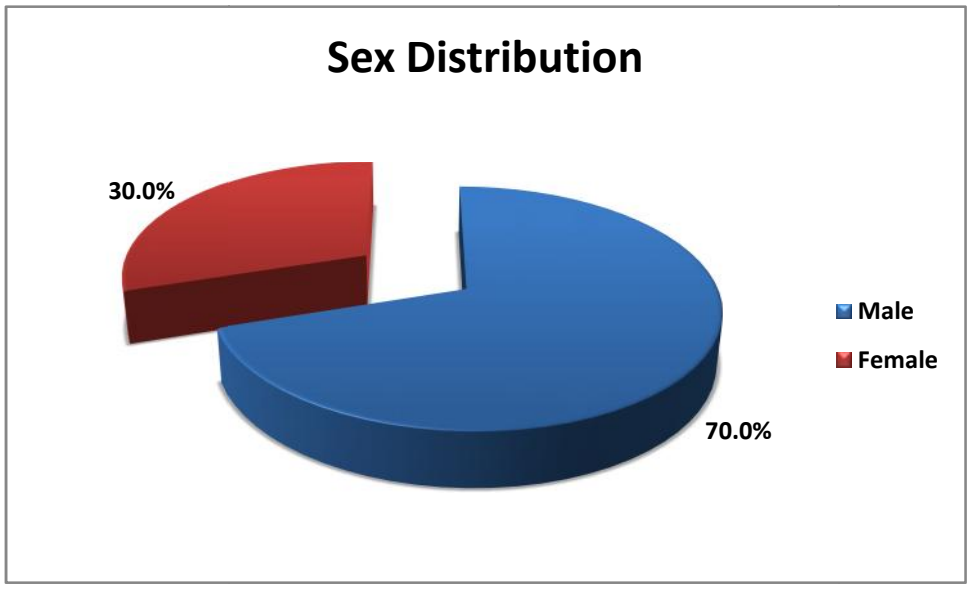
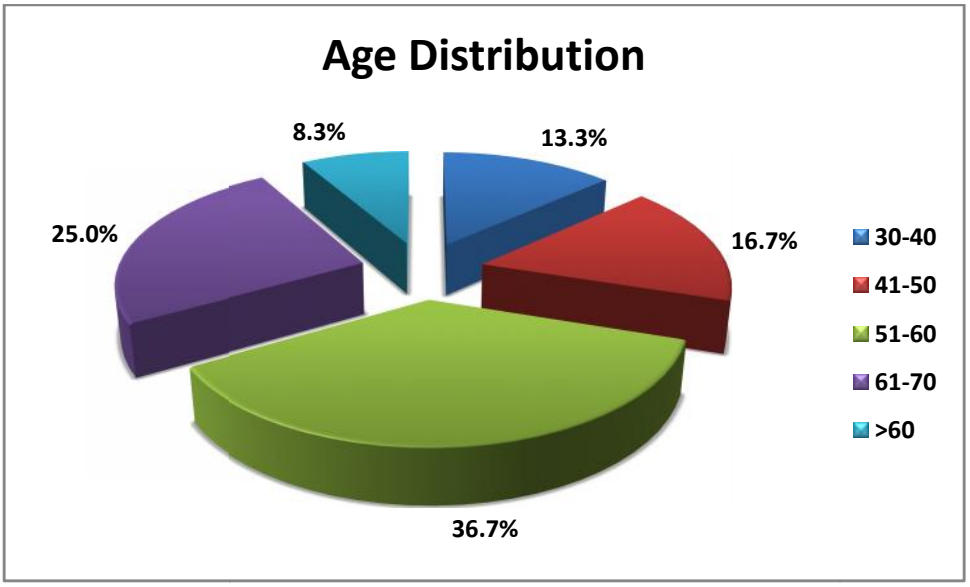


Table 2: Distribution of cases according to Age

Age(yrs)	N	%
30-40	8	13.3
41-50	10	16.7
51-60	22	36.7
61-70	15	25
>60	5	8.3

Figure 2: Distribution of cases according to Age

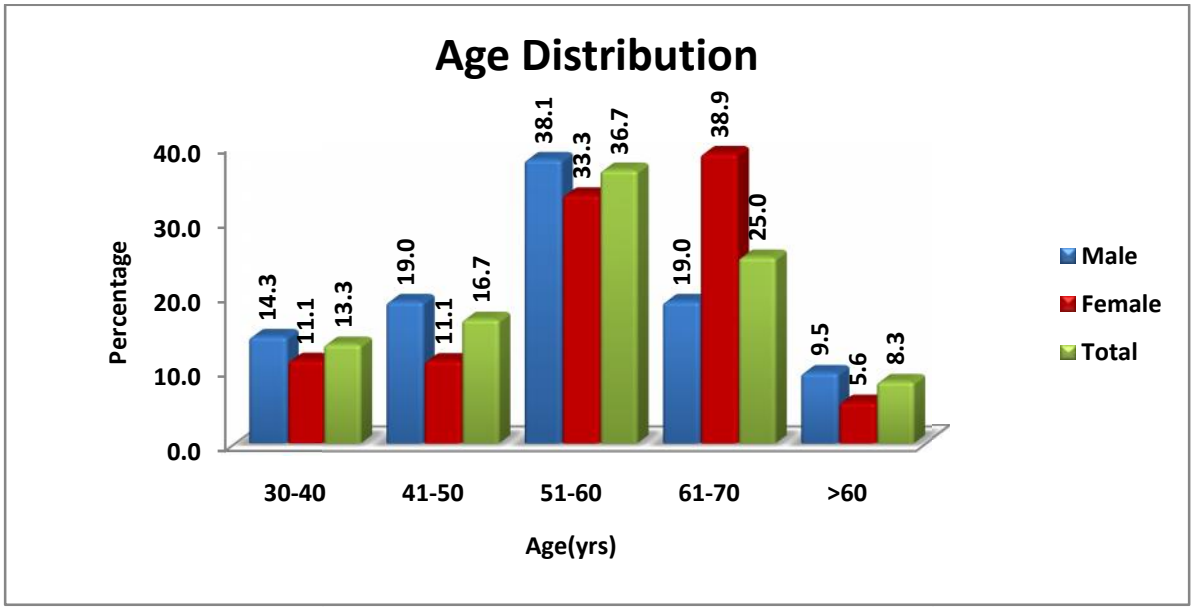


The age ranged from **30 to 85** years of age. The **mean** age of the group was **55.5 ± 13.19** and the **SD** is **12.6 ± 2.2** The patients in the age group of **51 to 60** years had the highest incidence of ACS

Table 3 : Association of Age with Sex

Age(yrs)	Male		Female		Total		p value
	N	%	N	%	N	%	
30-40	6	14.3	2	11.1	8	13.3	0.58
41-50	8	19.0	2	11.1	10	16.7	
51-60	16	38.1	6	33.3	22	36.7	
61-70	8	19.0	7	38.9	15	25.0	
>60	4	9.5	1	5.6	5	8.3	
Total	42	100.0	18	100.0	60	100.0	

Figure 3: Association of Age with Sex



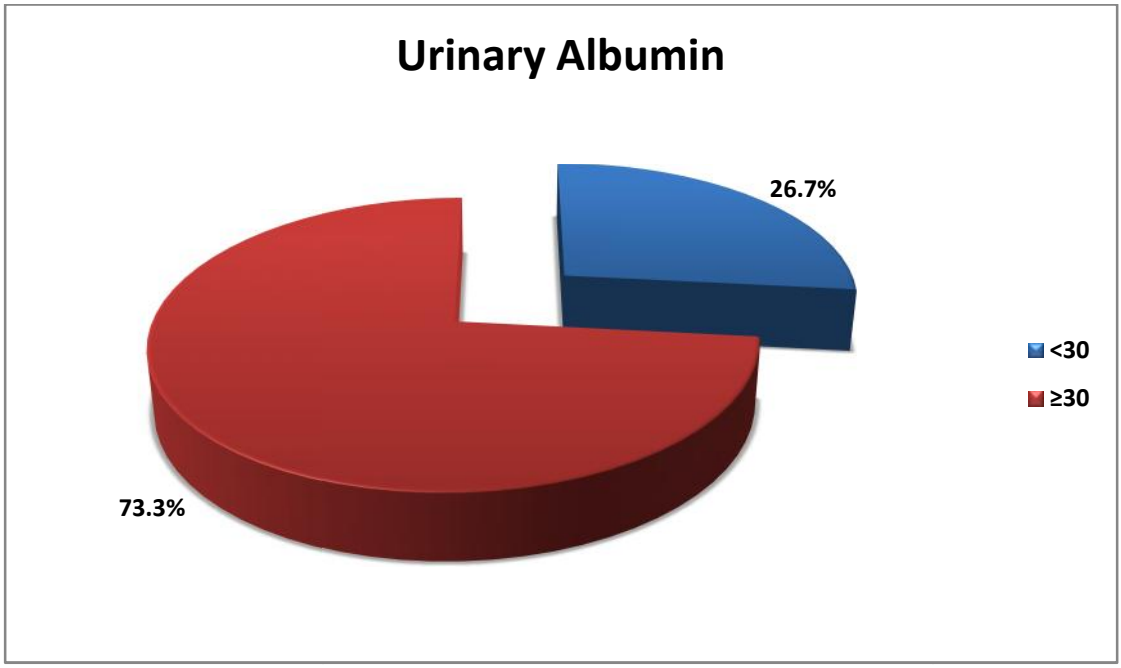
The **mean** age of the study group was **55.7(12.6)** among the males and **58.6 (10.4)** among the females.

There was no statistically significant difference between the two group of the study population.

Table 4 : Distribution of cases according to Urinary Albumin

Urinary Albumin	N	%
<30	16	26.7
≥30	44	73.3
Total	60	100

Figure 4 : Distribution of cases according to Urinary Albumin



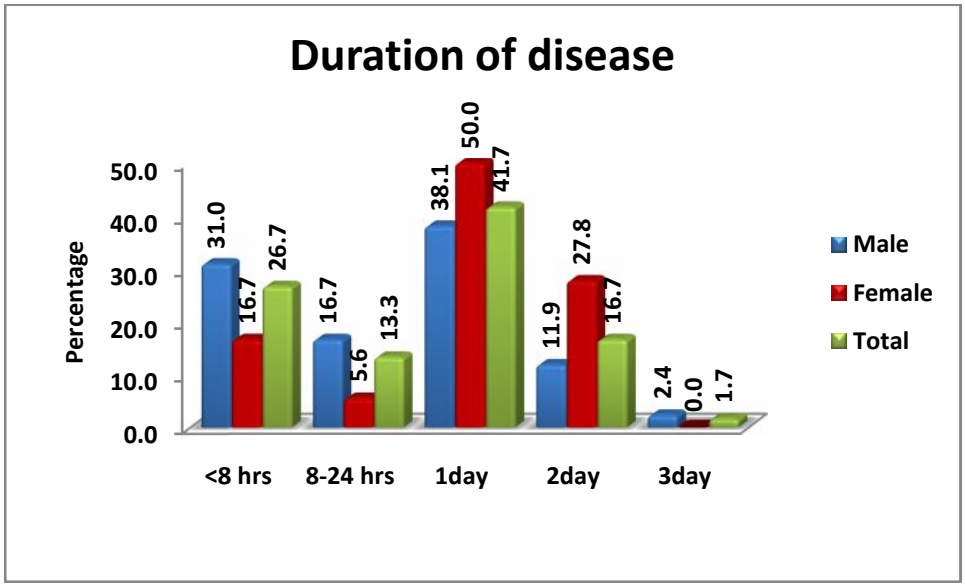
In Urinary albumin the **mean** is **44.6±3.2** and the **SD** is **22.1 ± 2.1**

There is statistically significance, less urinary albumin of ACS than standard normal mean urinary albumin value (0.5) ($p<0.0001$).

Table 5 : Association of Duration of disease with Sex

Duration of disease	Male		Female		Total		p value
	N	%	N	%	N	%	
<8 hrs	13	31.0	3	16.7	16	26.7	0.717
8-24 hrs	7	16.7	1	5.6	8	13.3	
1day	16	38.1	9	50.0	25	41.7	
2day	5	11.9	5	27.8	10	16.7	
3day	1	2.4	0	0.0	1	1.7	
Total	42	100.0	18	100.0	60	100.0	

Figure 5 : Association of Duration of disease with Sex

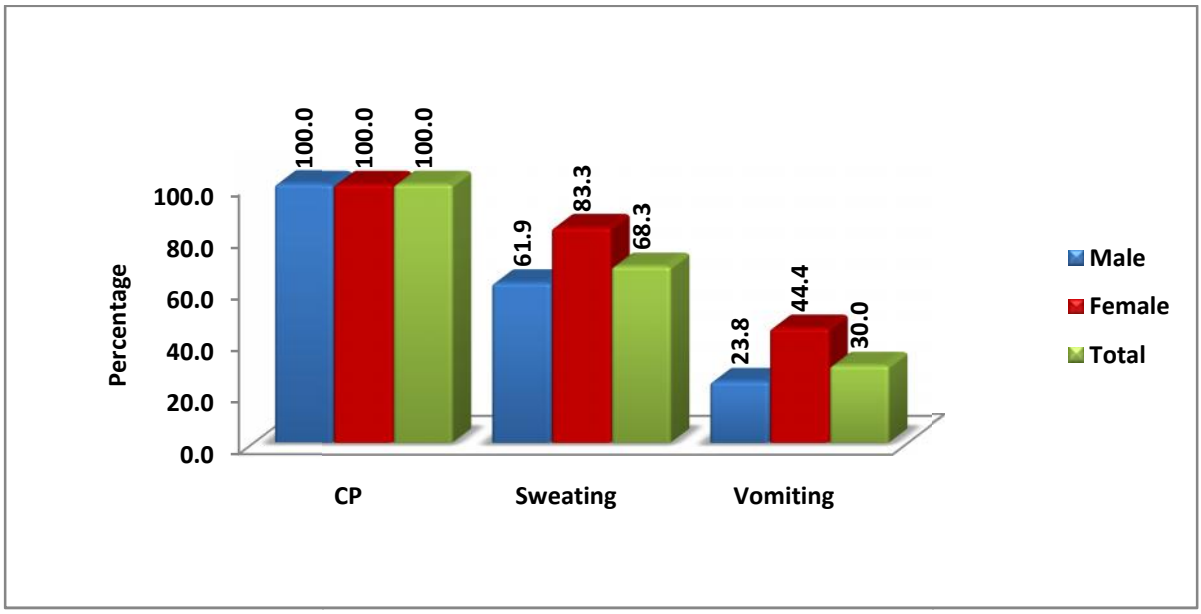


The **mean** duration of disease was 1day

Table 6 : Association of Symptoms with Sex

Symptoms	Male		Female		Total		p value
	N	%	N	%	N	%	
CP	42	100.0	18	100.0	60	100.0	-
Sweating	26	61.9	15	83.3	41	68.3	0.102
Vomiting	10	23.8	8	44.4	18	30.0	0.11

Figure 6: Association of Symptoms with Sex

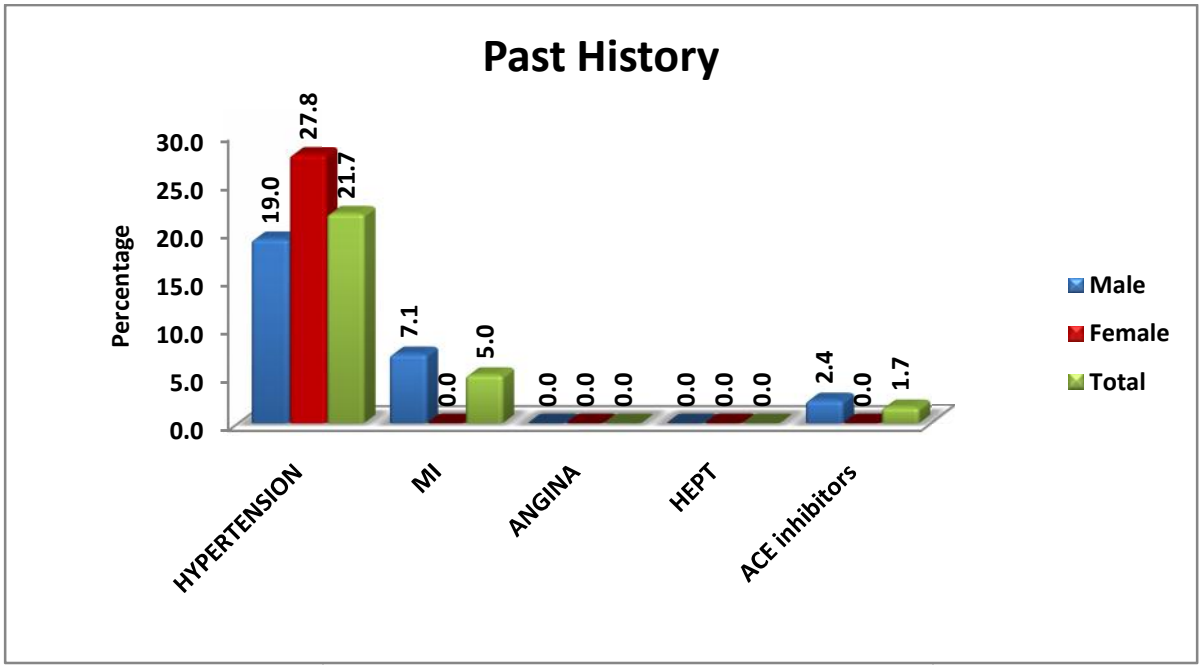


The patients experienced chest pain on presentation both males and females **is 100%** and sweating in females is higher then the males that is **83%** in females and **61.9 %** in males and vomiting presented is **30%** in that males are **23.8%** and females are **44.4% .**

Table 7 : Association of Past History with Sex

Past History	Male		Female		Total		p value
	N	%	N	%	N	%	
HYPERTENSION	8	19.0	5	27.8	13	21.7	0.452
MI	3	7.1	0	0.0	3	5.0	0.245
ANGINA	0	0.0	0	0.0	0	0.0	-
HEPT	0	0.0	0	0.0	0	0.0	-
ACE inhibitors	1	2.4	0	0.0	1	1.7	0.7

Figure 7 : Association of Past History with Sex



The patients presented with past history of hypertension is higher in females and with other and that males are **19%** and females are **27.8%**

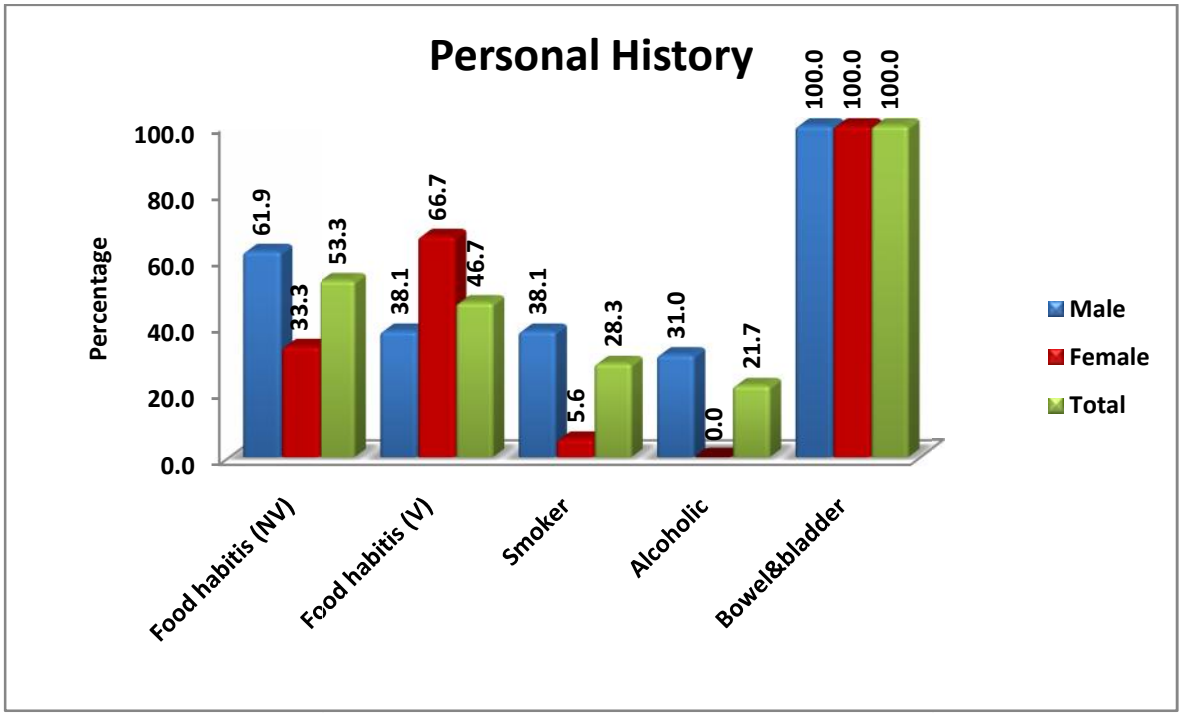
7.1% males patients are presented with past history of MI

Table 8 : Association of Personal History with Sex

Personal History	Male		Female		Total		p value
	N	%	N	%	N	%	
Food habitis (NV)	26	61.9	6	33.3	32	53.3	0.042*
Food habitis (V)	16	38.1	12	66.7	28	46.7	
Smoker	16	38.1	1	5.6	17	28.3	0.01*
Alcoholic	13	31.0	0	0.0	13	21.7	<0.001*
Bowel&bladder	42	100.0	18	100.0	60	100.0	-

Note: *means significant at 5% level of significance (p<0.05)

Figure 8 : Association of Personal History with Sex

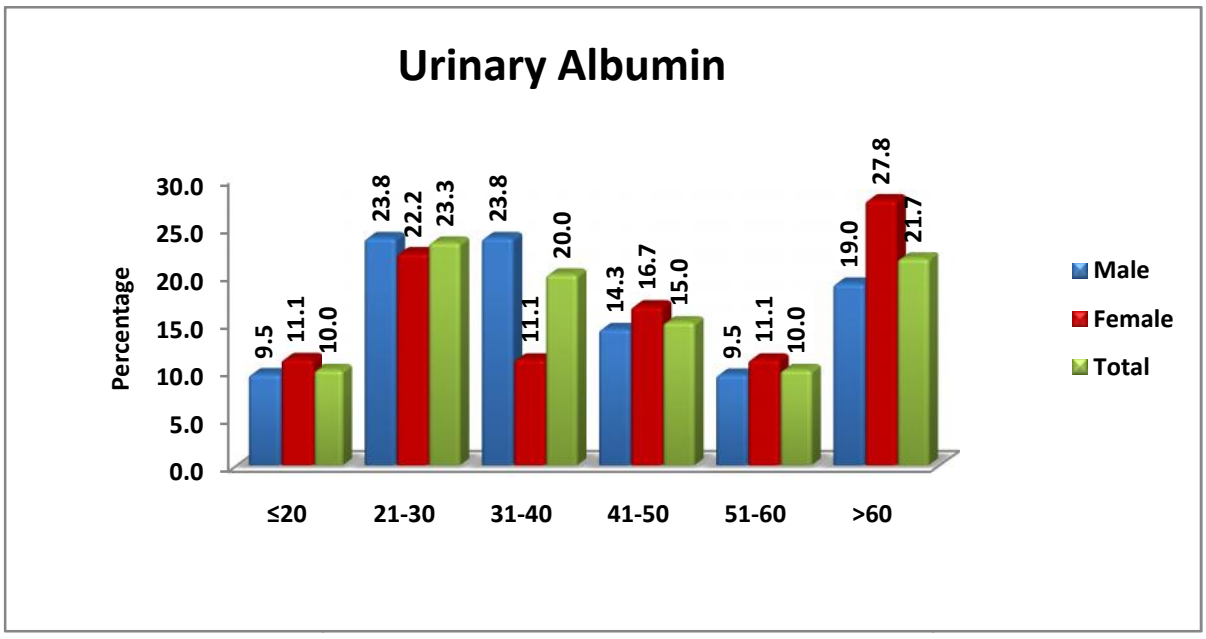


Microalbuminuria positive ACS patients**, 61.9%** of patients are **non vegetarians** and **33.3 %** are **vegetarians** there is significant stastical difference.

Table 9 : Association of Urinary Albumin with Sex

Urinary Albumin	Male		Female		Total		p value
	N	%	N	%	N	%	
≤20	4	9.5	2	11.1	6	10.0	
21-30	10	23.8	4	22.2	14	23.3	
31-40	10	23.8	2	11.1	12	20.0	
41-50	6	14.3	3	16.7	9	15.0	0.903
51-60	4	9.5	2	11.1	6	10.0	
>60	8	19.0	5	27.8	13	21.7	
Total	42	100.0	18	100.0	60	100.0	

Figure 9 : Association of Urinary Albumin with Sex

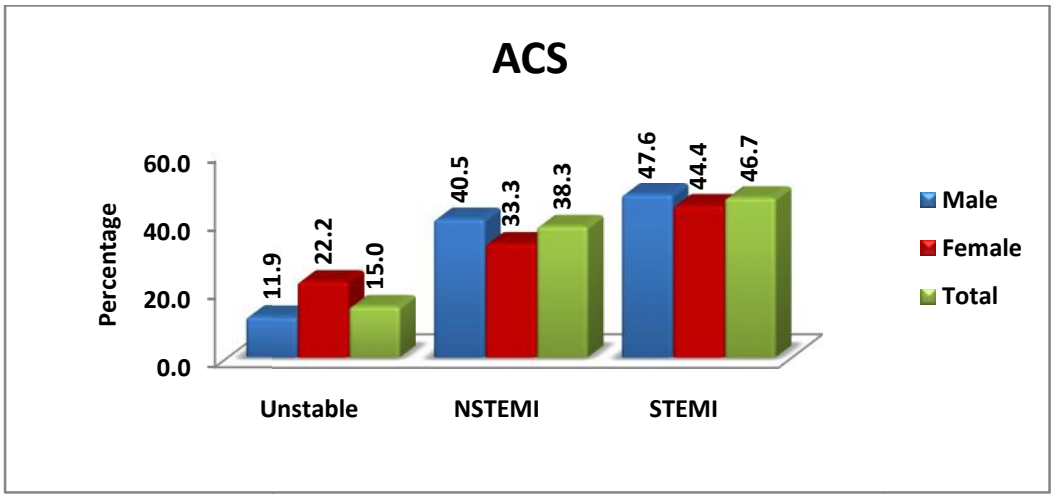


Urinary Albumin with sex is **42** cases are **males** the **mean** is **43.5** and **SD** is **21.3** in **females** the **mean** is **47.4** and **SD** is **24.3** and the **P valve> 0.535 .**

Table 10 : Association of ACS with Sex

ACS	Male		Female		Total		p value
	N	%	N	%	N	%	
Unstable	5	11.9	4	22.2	9	15.0	0.58
NSTEMI	17	40.5	6	33.3	23	38.3	
STEMI	20	47.6	8	44.4	28	46.7	
Total	42	100.0	18	100.0	60	100.0	

Figure 10 : Association of ACS with Sex



The known risk factors of ACS like smoking status,tobacco chewing, diabetes mellitus, family history of ACS and hypertension were studied and correlated. **37.2 %** of all ACS patients were smokers, **31%** chewed tobacco, **24.7 %** had diabetes mellitus, **8 %** had family history of ACS and **31.8%** were hypertensive.

Hypertension had statistically significant correlation with ACS. All risk factors were more associated with **STEMI** compared to **unstable angina** or **NSTEMI.**

Table 11 : Relation of risk factors with components of ACS

Risk factors	ACS								p value
	Unstable		NSTEMI		STEMI		Total		
	N	%	N	%	N	%	N	%	
CP	9	100.0	23	100.0	28	100.0	60	100.0	-
Sweating	6	66.7	12	52.2	23	82.1	41	68.3	0.072
Vomiting	2	22.2	7	30.4	9	32.1	18	30.0	0.851
HYPERTENSION	4	44.4	3	13.0	6	21.4	13	21.7	0.153
MI	1	11.1	2	8.7	0	0.0	3	5.0	0.241
ANGINA	0	0.0	0	0.0	0	0.0	0	0.0	-
HEPT	0	0.0	0	0.0	0	0.0	0	0.0	-
ACE inhibitors	1	11.1	0	0.0	0	0.0	1	1.7	0.056
Food habitis (NV)	3	33.3	12	52.2	17	60.7	32	53.3	0.355
Food habitis (V)	6	66.7	11	47.8	11	39.3	28	46.7	
Smoker	2	22.2	5	21.7	10	35.7	17	28.3	0.494
Alcoholic	0	0.0	10	43.5	3	10.7	13	21.7	0.004*
Total	9	100.0	23	100.0	28	100.0	60	100.0	

Note: *means significant at 5% level of significant

Figure 11 : Relation of risk factors with components of ACS

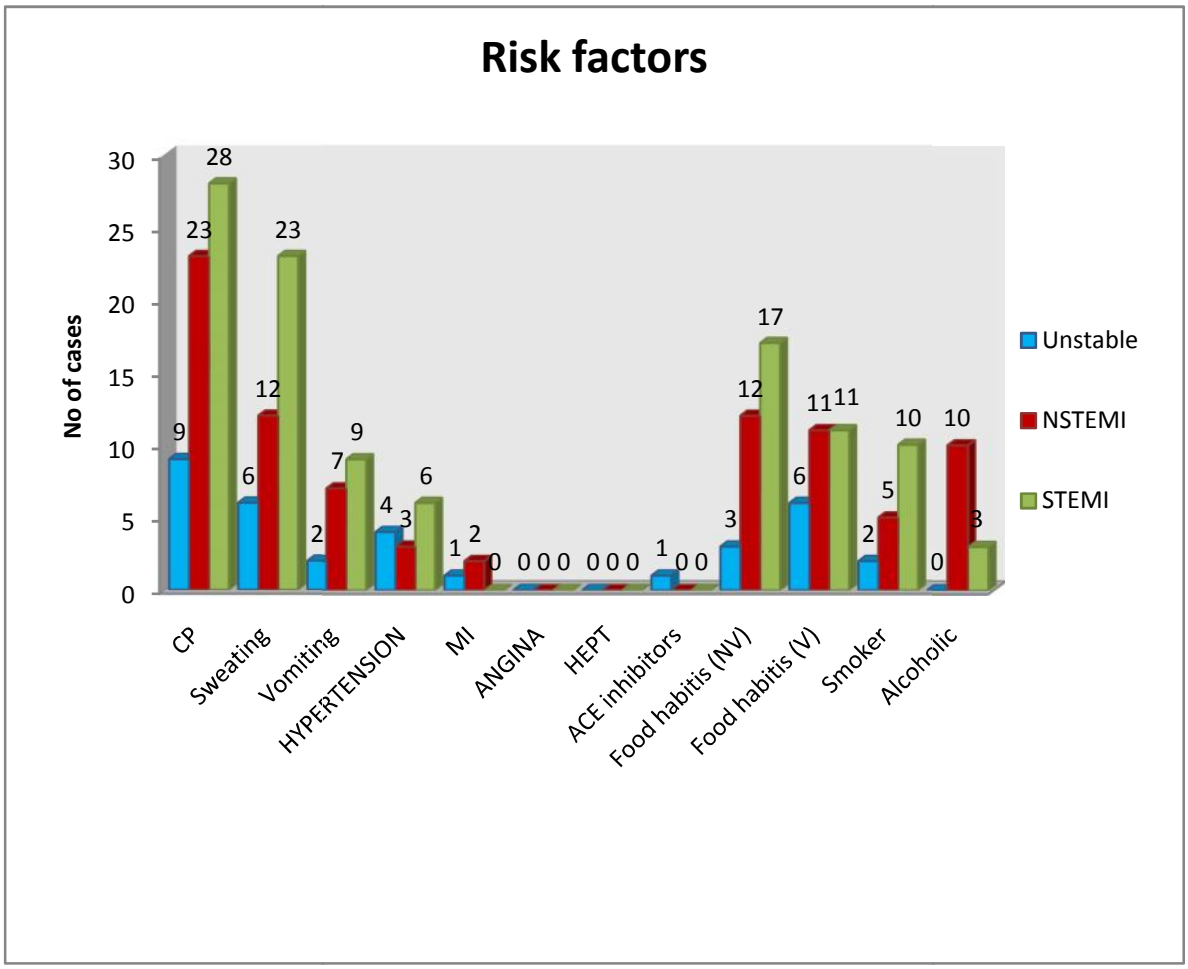


Table 12 : Association of CPK MB with Sex

CPK MB	Male		Female		Total		p value
	N	%	N	%	N	%	
≤80	31	73.8	16	88.9	47	78.3	0.194
>80	11	26.2	2	11.1	13	21.7	
Total	42	100.0	18	100.0	60	100.0	

Figure 12 : Association of CPK MB with Sex

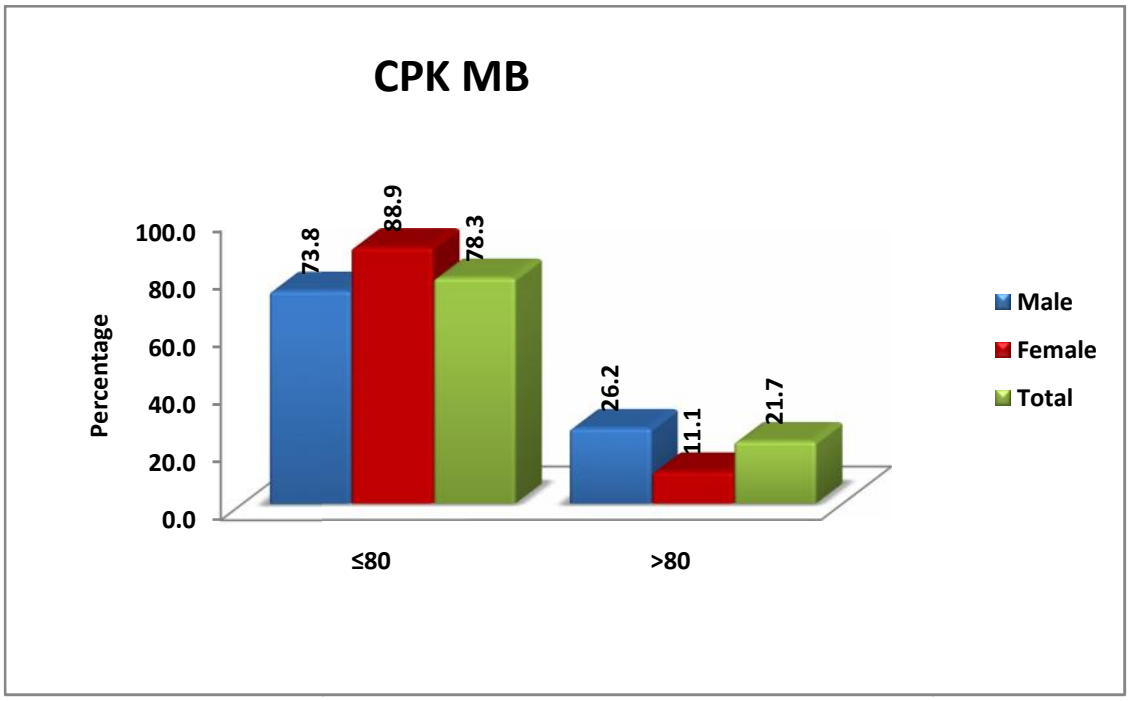
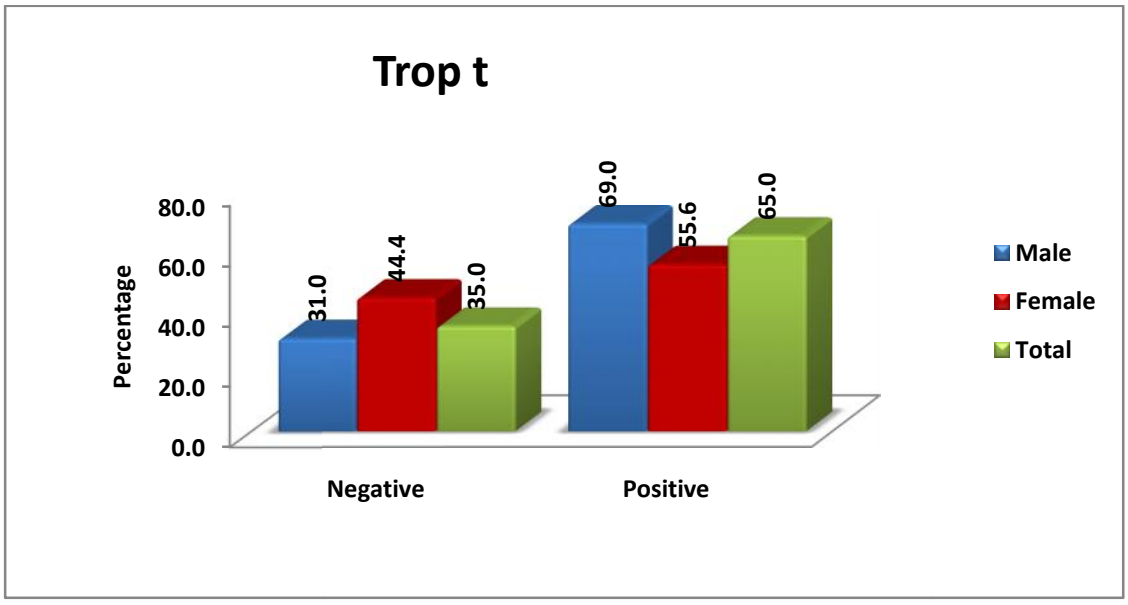


Table 13 : Association of Trop t with Sex

Trop t	Male		Female		Total		p value
	N	%	N	%	N	%	
Negative	13	31.0	8	44.4	21	35.0	0.315
Positive	29	69.0	10	55.6	39	65.0	
Total	42	100.0	18	100.0	60	100.0	

Figure 13 : Association of Trop t with Sex

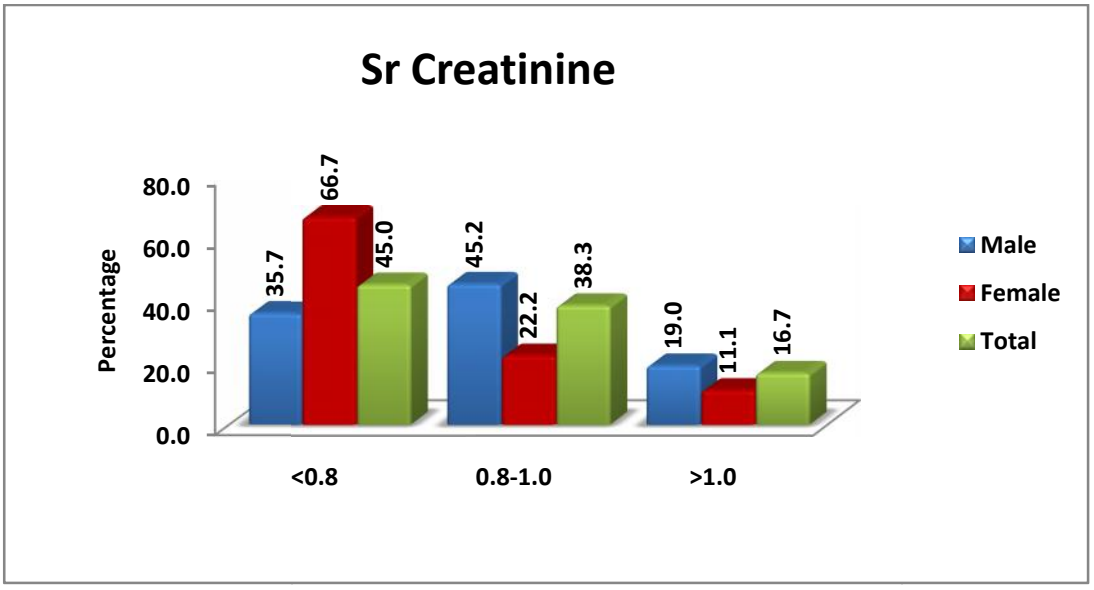


Trop-t with sex in **total 60** patients **39** are **positive** that it around **65%** in that **males 29** are positve **out of 42** it around **69%** and in **females 10** are positive out of **18** it around **65%** and it shows that there is **significant relations** between **trop-t** and **microalbuminuria.**

Table 14 : Association of Sr Creatinine with Sex

Sr Creatinine	Male		Female		Total		p value
	N	%	N	%	N	%	
<0.8	15	35.7	12	66.7	27	45.0	0.086
0.8-1.0	19	45.2	4	22.2	23	38.3	
>1.0	8	19.0	2	11.1	10	16.7	
Total	42	100.0	18	100.0	60	100.0	

Figure 14 : Association of Sr Creatinine with Sex



There is no significant difference between the sr creatinine and the microalbuminuria and the **mean** is **0.9** and **SD is 0.4**

Table 15 : Association of Total Cholesterol with Sex

Total Cholesterol	Male		Female		Total		p value
	N	%	N	%	N	%	
≤200	28	66.7	15	83.3	43	71.7	0.189
>200	14	33.3	3	16.7	17	28.3	
Total	42	100.0	18	100.0	60	100.0	

Figure 15 : Association of Total Cholesterol with Sex

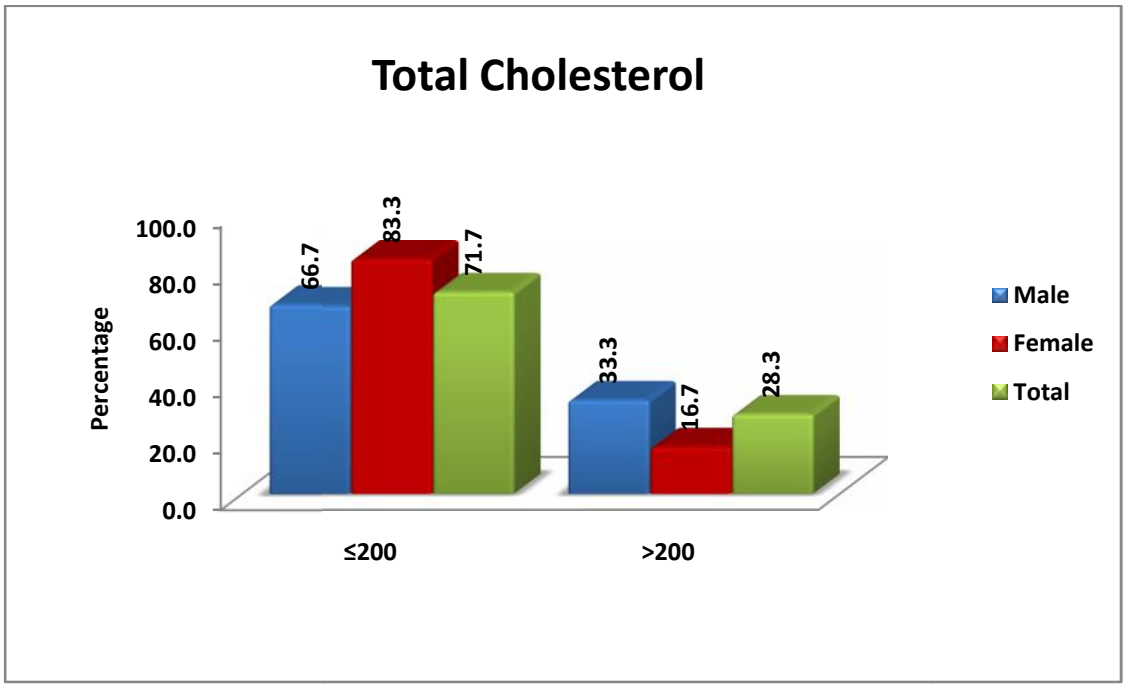


Table 16 : Association of Triglycerides with Sex

Triglycerides	Male		Female		Total		p value
	N	%	N	%	N	%	
≤150	25	59.5	12	66.7	37	61.7	0.602
>150	17	40.5	6	33.3	23	38.3	
Total	42	100.0	18	100.0	60	100.0	

Figure 16 : Association of Triglycerides with Sex

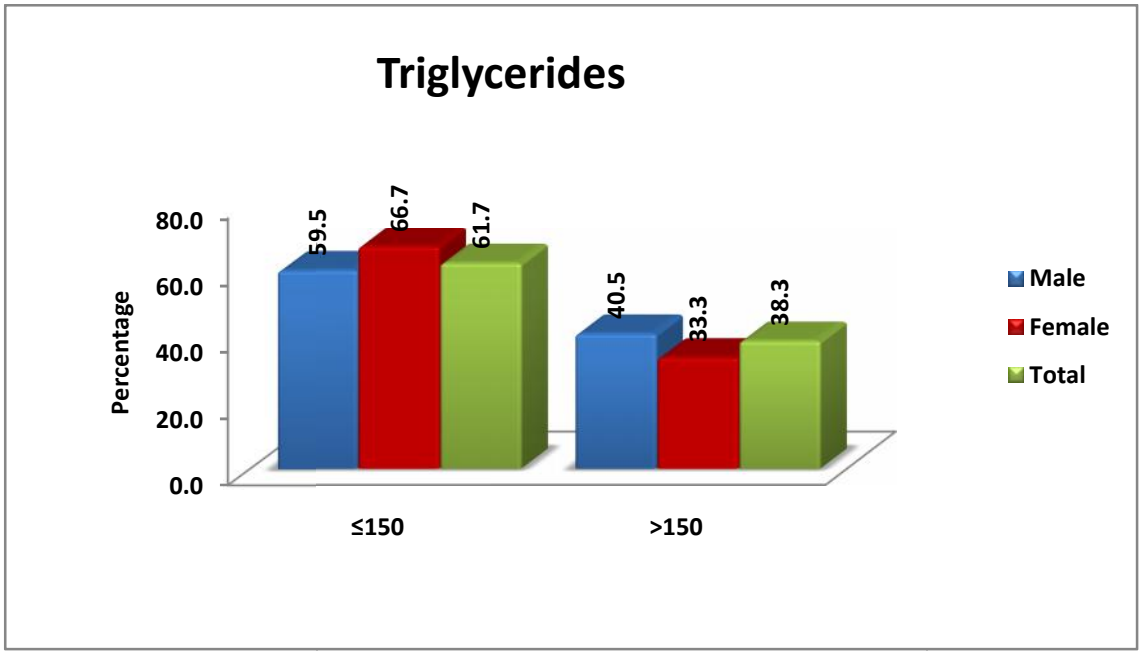


Table 17 : Association of HDL-cholesterol with Sex

HDL-cholesterol	Male		Female		Total		p value
	N	%	N	%	N	%	
≤40	29	69.0	12	66.7	41	68.3	0.856
>40	13	31.0	6	33.3	19	31.7	
Total	42	100.0	18	100.0	60	100.0	

Figure 17 : Association of HDL-cholesterol with Sex

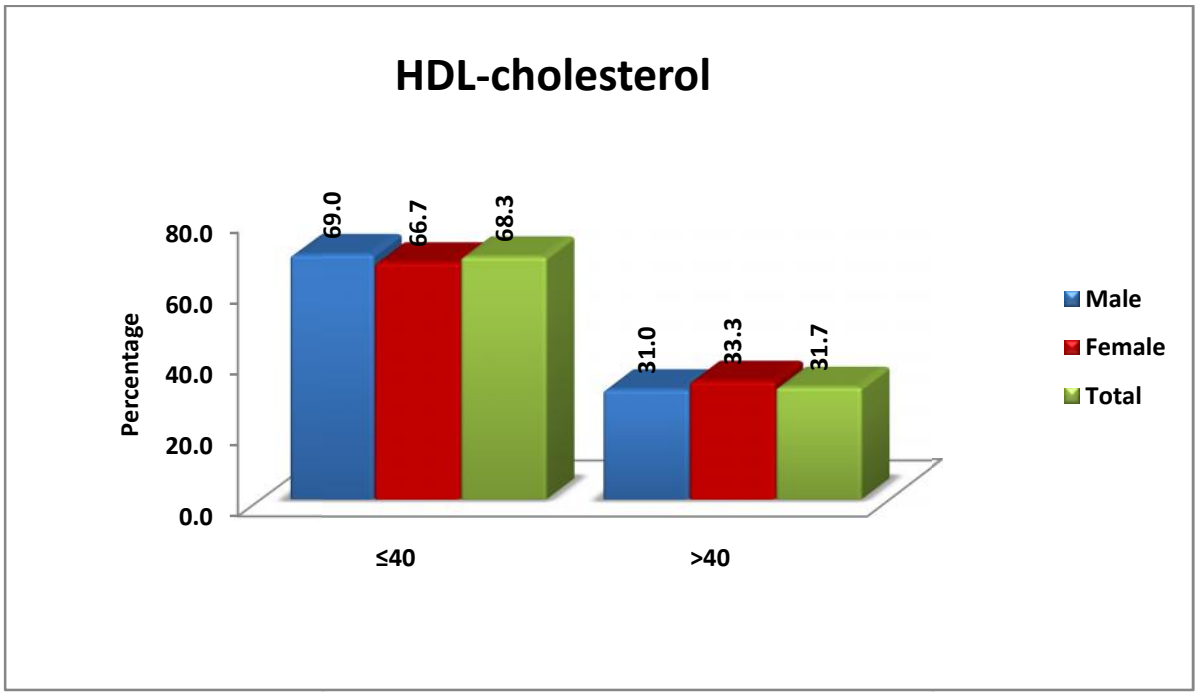
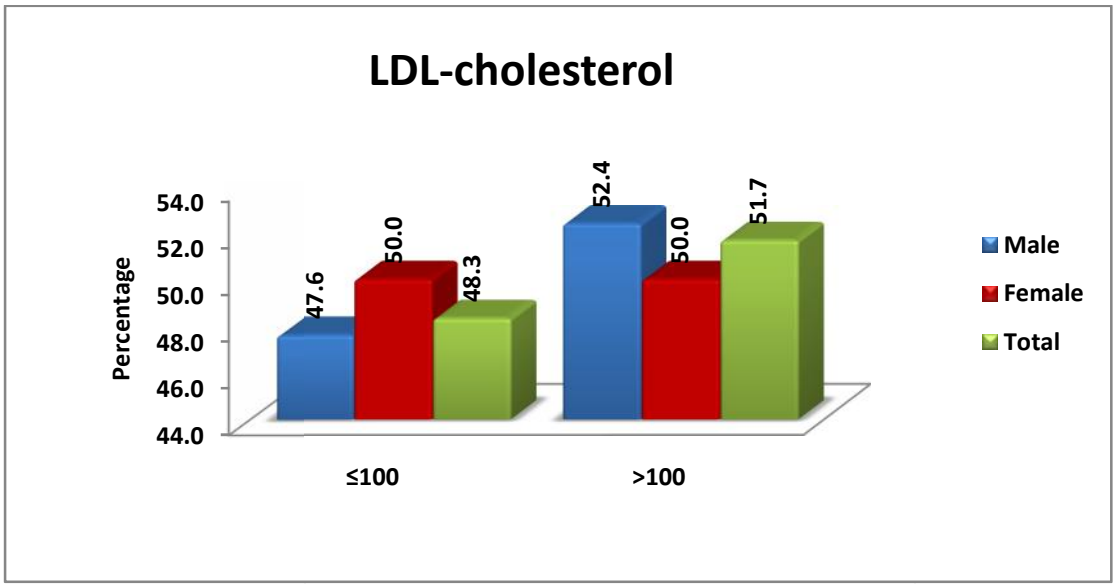


Table 18 : Association of LDL-cholesterol with Sex

LDL-cholesterol	Male		Female		Total		p value
	N	%	N	%	N	%	
≤100	20	47.6	9	50.0	29	48.3	0.866
>100	22	52.4	9	50.0	31	51.7	
Total	42	100.0	18	100.0	60	100.0	

Figure 18 : Association of LDL-cholesterol with Sex

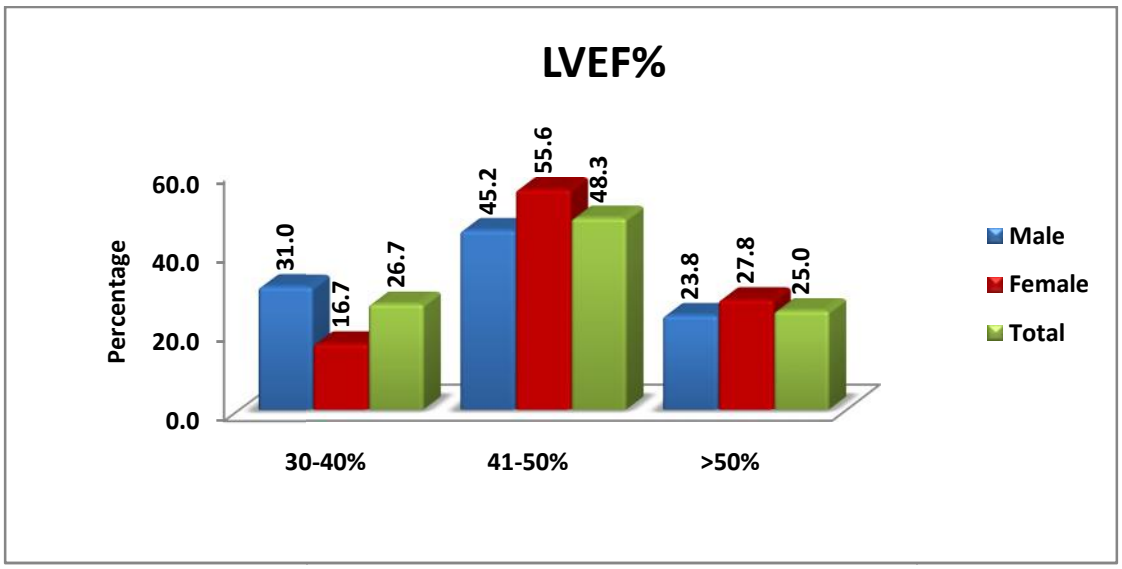


Mean LDL was **172.07 ± 46.57** which was higher than the normal considering the cut off value with existing risk factors. The minimum LDL value was **70 mg/dl** whereas the maximum was **291.8mg/dl**

Table 19 : Association of LVEF% with Sex

LVEF%	Male		Female		Total		p value
	N	%	N	%	N	%	
30-40%	13	31.0	3	16.7	16	26.7	0.517
41-50%	19	45.2	10	55.6	29	48.3	
>50%	10	23.8	5	27.8	15	25.0	
Total	42	100.0	18	100.0	60	100.0	

Figure 19 : Association of LVEF% with Sex

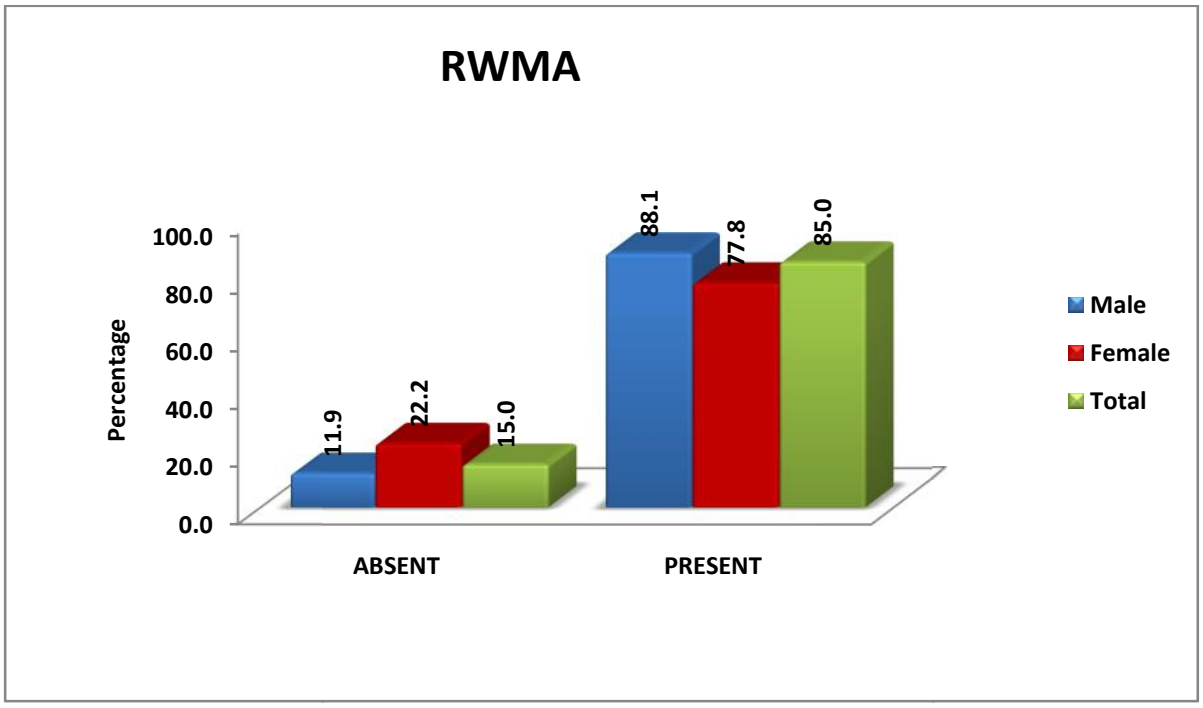


There is no significant difference between the LVEF % in males and females but there is significant relation between microalbuminuria and LVEF%

Table 20 : Association of RWMA with Sex

RWMA	Male		Female		Total		p value
	N	%	N	%	N	%	
ABSENT	5	11.9	4	22.2	9	15.0	0.305
PRESENT	37	88.1	14	77.8	51	85.0	
Total	42	100.0	18	100.0	60	100.0	

Figure 20 : Association of RWMA with Sex

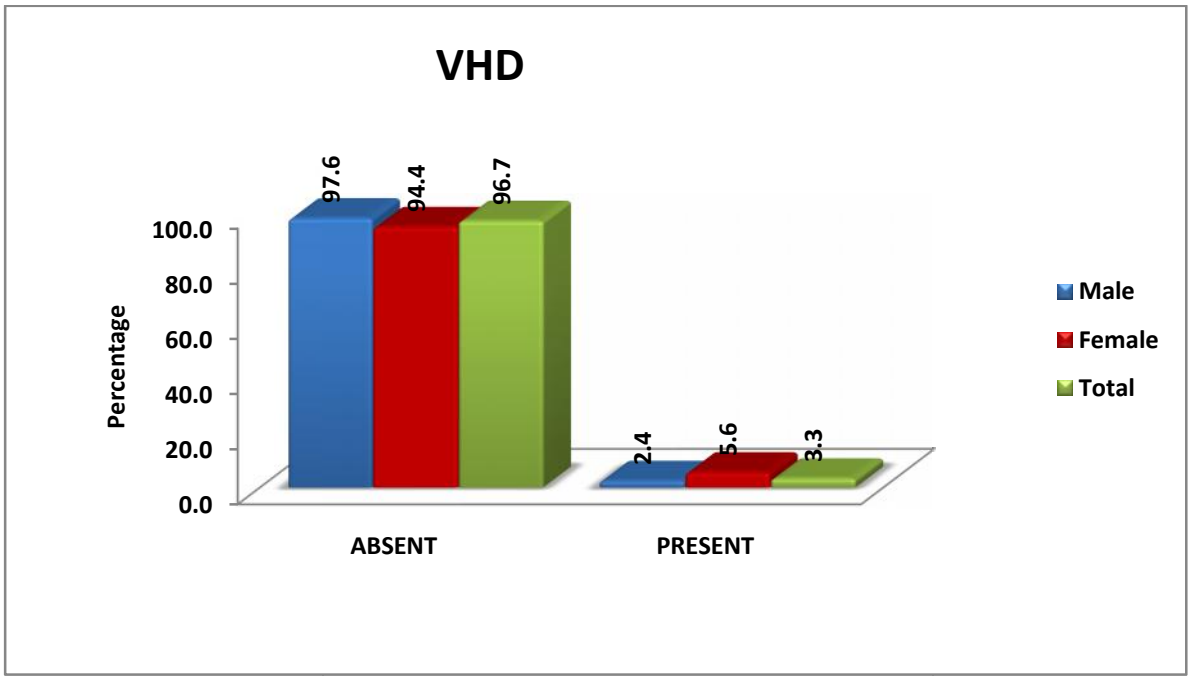


There is no significant difference between the RWMA in males and females but there is significant relation between microalbuminuria and RWMA

Table 21 : Association of VHD with Sex

VHD	Male		Female		Total		p value
	N	%	N	%	N	%	
ABSENT	41	97.6	17	94.4	58	96.7	0.53
PRESENT	1	2.4	1	5.6	2	3.3	
Total	42	100.0	18	100.0	60	100.0	

Figure 21 : Association of VHD with Sex

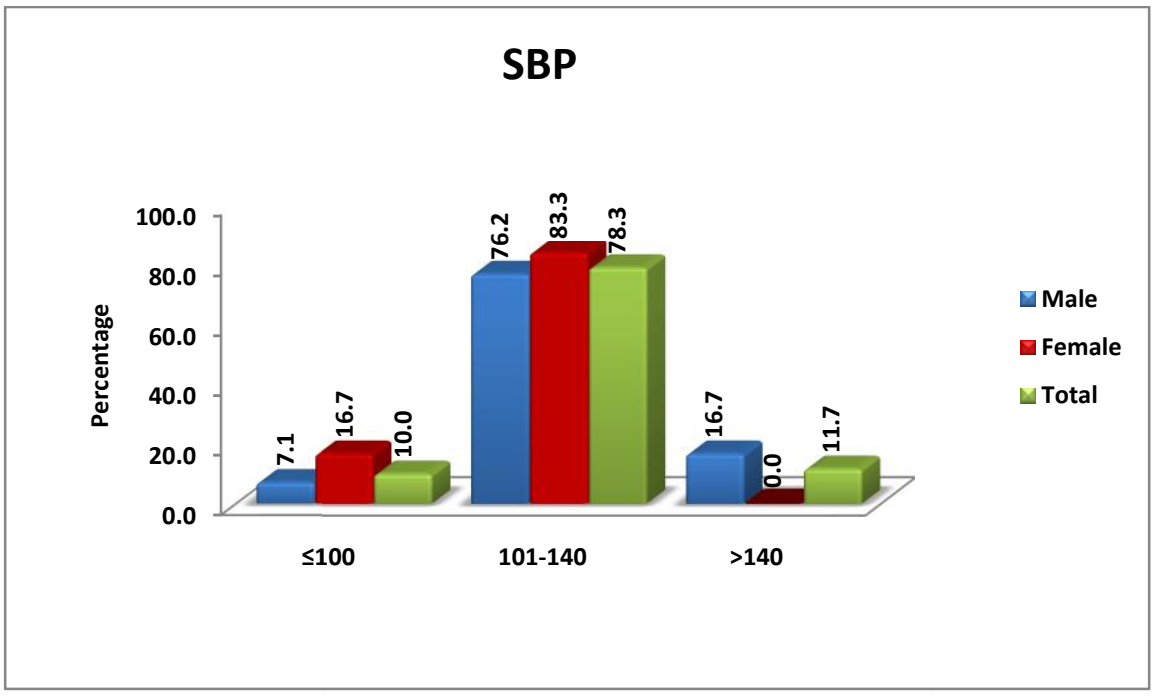


There is no significant difference between the VHD in males and females but there is no significant relation between microalbuminuria and VHD

Table 22 : Association of SBP with Sex

SBP	Male		Female		Total		p value
	N	%	N	%	N	%	
≤100	3	7.1	3	16.7	6	10.0	0.121
101-140	32	76.2	15	83.3	47	78.3	
>140	7	16.7	0	0.0	7	11.7	
Total	42	100.0	18	100.0	60	100.0	

Figure 22 : Association of SBP with Sex

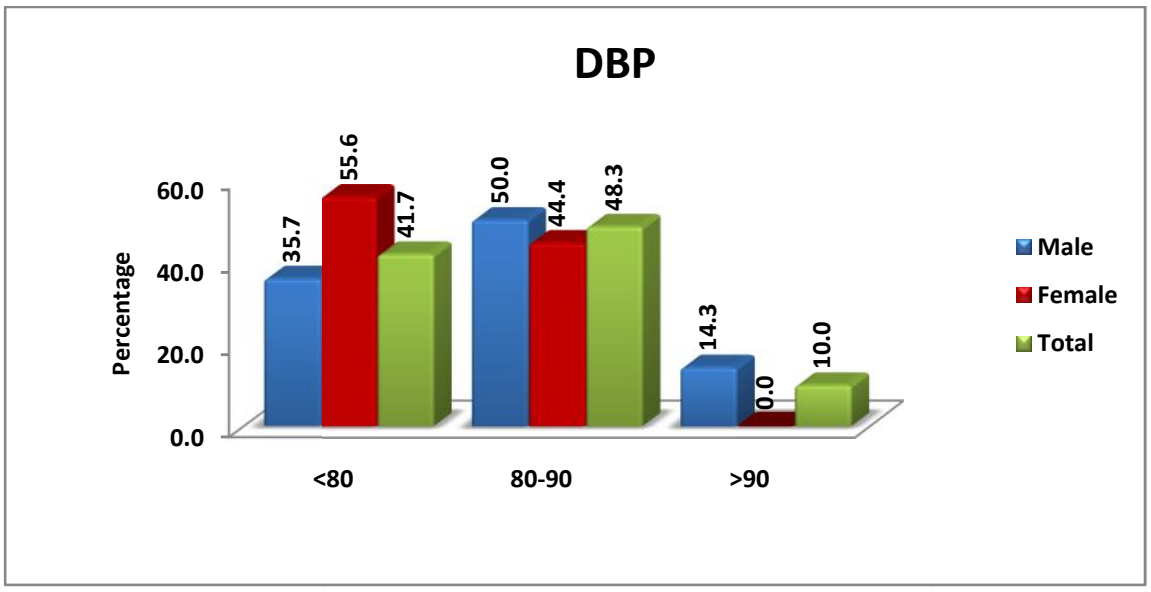


There is no significant statistical variation between males and females in SBP in the study population

Table 23 : Association of DBP with Sex

DBP	Male		Female		Total		p value
	N	%	N	%	N	%	
<80	15	35.7	10	55.6	25	41.7	0.146
80-90	21	50.0	8	44.4	29	48.3	
>90	6	14.3	0	0.0	6	10.0	
Total	42	100.0	18	100.0	60	100.0	

Figure 23 : Association of DBP with Sex



There is no significant statistical variation between males and females in DBP in the study population

Table 24 : Mean comparison of background variables between Males and Females

Variables	Male		Female		p value
	Mean	SD	Mean	SD	
AGE	55.7	12.6	58.6	10.4	0.396
Systolic BP	127.3	22.4	118.9	13.7	0.146
Diastolic BP	80.5	15.6	75.0	9.2	0.172
Urinary Albumin	43.5	21.3	47.4	24.3	0.535
CPK MB	74.3	66.0	58.1	31.8	0.327
Sr Creatinine	1.0	0.5	0.8	0.2	0.103

Figure 24 : Mean comparison of background variables between Males and Females

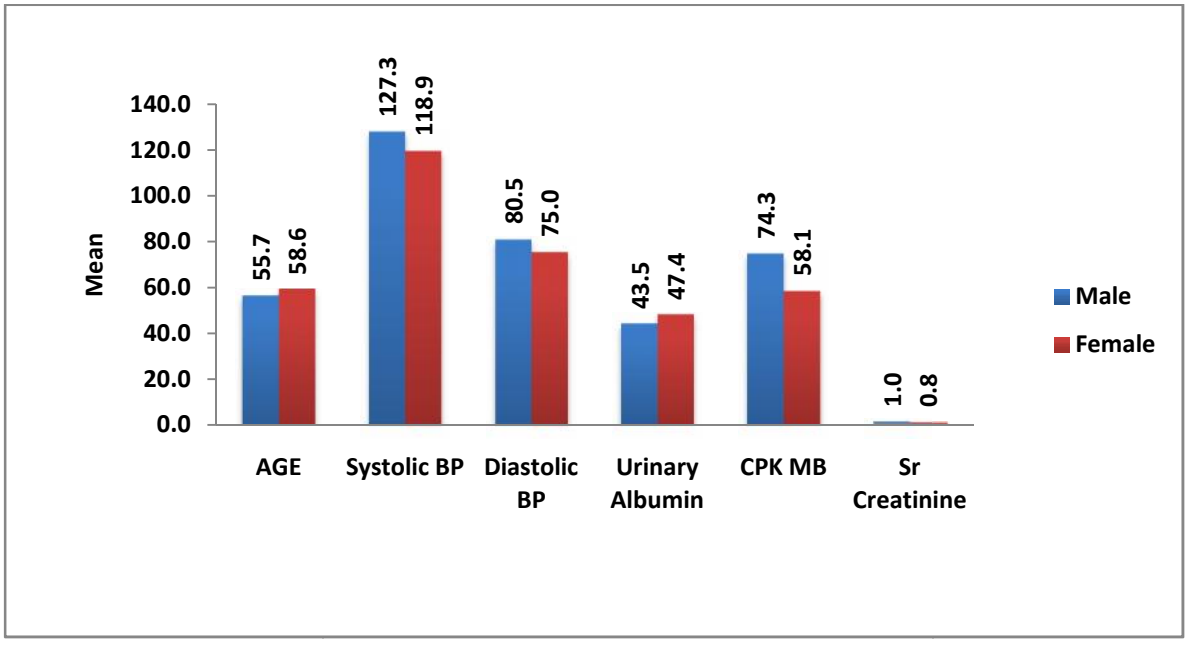


Table 25 : Mean comparison of CBC variables between Males and Females

Variables	Male		Female		p value
	Mean	SD	Mean	SD	
Hemoglobine	13.9	2.1	10.4	1.7	<0.001*
Total Count	12410.0	6277.9	14371.7	5039.7	0.246
Neutrophils	77.8	10.3	80.9	9.3	0.277
Lymphocytes	17.1	9.3	16.1	8.2	0.686
Eosinophils	1.9	2.2	0.8	0.9	0.039*
Basophils	0.0	0.3	0.4	1.2	0.098
Monocytes	2.5	1.5	2.2	1.6	0.6
Platelet count	2.7	0.7	2.7	0.7	0.929
ESR	27.9	32.2	35.6	29.2	0.387

Note: *means significant at 5% level of significance (p<0.05)

Figure 25 : Mean comparison of CBC variables between Males and Females

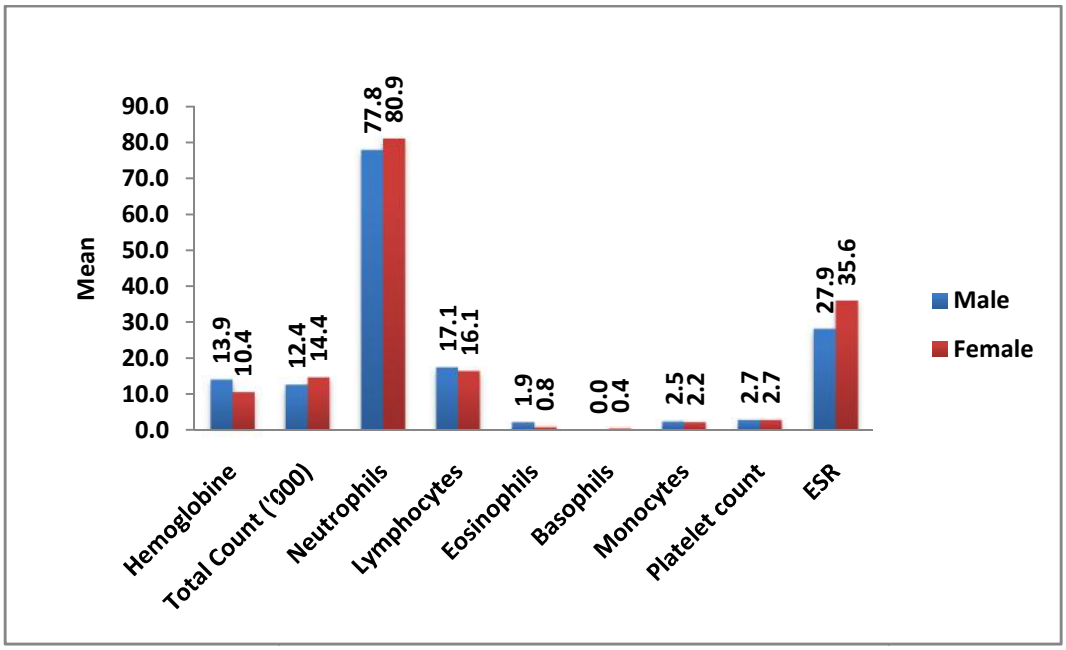
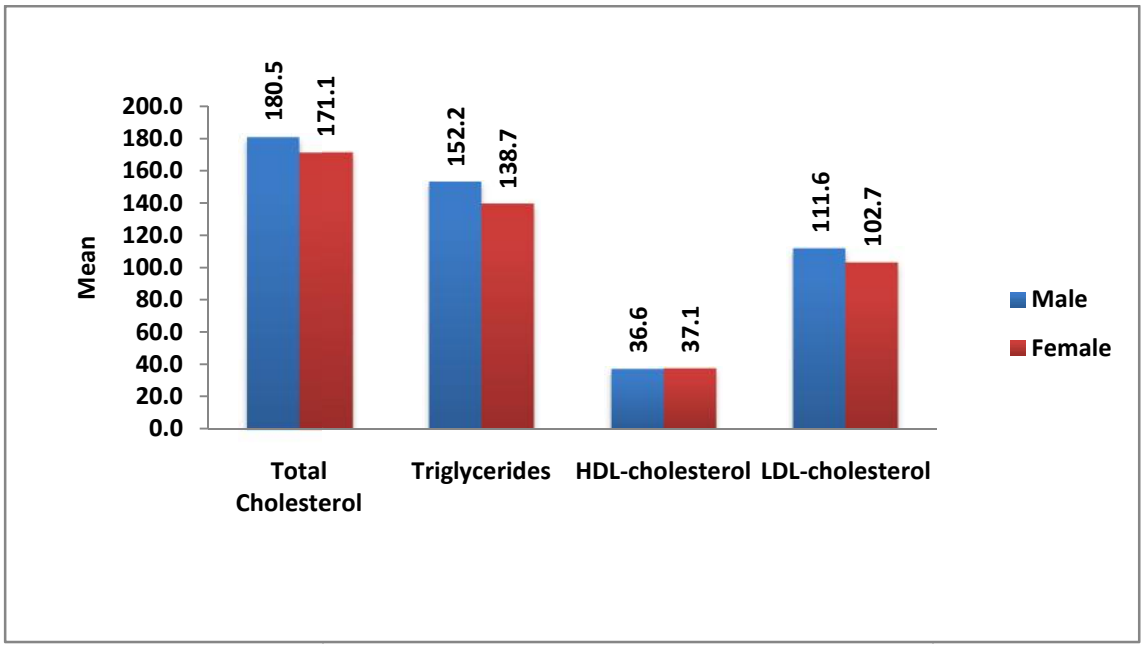


Table 26 : Mean comparison of Lipid Profile variables between Males and Females

Variables	Male		Female		p value
	Mean	SD	Mean	SD	
Total Cholesterol	180.5	48.9	171.1	37.9	0.47
Triglycerides	152.2	92.1	138.7	83.9	0.594
HDL-cholesterol	36.6	10.5	37.1	10.0	0.867
LDL-cholesterol	111.6	44.5	102.7	34.8	0.456

Figure 26 : Mean comparison of Lipid Profile variables between Males and Females



The **lipid profile** between males and females in the total cholesterol **mean** is **177.7 ± 4.2** and in that **males the mean is 180.5** and in **females 171.1** and **SD is 45.8 in both** and in **males 48.9** and in **females 37.9 .**

In **triglycerides** the **mean in both is 148.2 ± 5.0** and in that **males the mean is 152.2** and in **females the mean is 138.7** and **SD is 89.2 in both** in **males is 92.1** in **females is 83.9** .

In **HDL** the **mean in both is 36.8 ± 2.1** and in that **males the mean is 36.6** and in **females the mean is 37.1** and **SD is 10.3 in both** in **males is 10.5** in **females is 10.0 .**

In **LDL** the **mean in both is 108.9 ± 3.2** and in that **males the mean is 111.6** and in **females the mean is 102.7** and **SD is 41.7 in both** in **males is 44.5** in **females is 34.8**

TABLE 27:

Parameters	Male				Female				Total			
	Min	Max	Mean	SD	Min	Max	Mean	SD	Min	Max	Mean	SD
AGE	30	85	55.7	12.6	38	80	58.6	10.4	30	85	56.5	12.0
Systolic BP	90	210	127.3	22.4	100	140	118.9	13.7	90	210	124.8	20.4
Diastolic BP	50	140	80.5	15.6	60	90	75.0	9.2	50	140	78.8	14.2
Urinary Albumin	13	98	43.5	21.3	12	90	47.4	24.3	12	98	44.6	22.1
CPK MB	10	300	74.3	66.0	23	133	58.1	31.8	10	300	69.4	58.1
Sr Creatinine	0.6	3.2	1.0	0.5	0.5	1.2	0.8	0.2	0.5	3.2	0.9	0.4
Hemoglobine	9	18	13.9	2.1	6.2	13.5	10.4	1.7	6.2	18	12.9	2.5
Total Cholesterol	90	319	180.5	48.9	115	252	171.1	37.9	90	319	177.7	45.8
Triglycerides	61	634	152.2	92.1	64	438	138.7	83.9	61	634	148.2	89.2
HDL-cholesterol	22	70	36.6	10.5	25	52	37.1	10.0	22	70	36.8	10.3
LDL-cholesterol	25	246	111.6	44.5	58	185	102.7	34.8	25	246	108.9	41.7
VLDL-cholesterol	12	126	30.8	18.5	13	87	27.9	16.7	12	126	29.9	17.9

DISCUSSION

DISCUSSION

This study was conducted between November 2015 to July 2017 and included 60 patients admitted to BLDE hospital with Acute coronary syndrome (ACS) and who tested positive for microalbuminiuria, of 60 cases 42 were male (70%) and 18 females (30%).

The age ranged from 30 to 85 years of age. The mean age of the group was 55.5 $\pm$ 13.19 SD. The patients in the age group of 51 to 60 years had the highest incidence of ACS. This finding was similar to other studies and accepted fact that the incidence of ACS increases with age.

Previous study by Borsch-Johnsen et al also showed a male preponderance .

The duration of disease was defined as the number of days the patient was symptomatic before he or she finally arrived at the hospital.

The mean duration of disease in microalbuminuria positive ACS patients were 1day .

The symptom of chest pain was either divided in to STEMI, NSTEMI and Unstable angina who were diagnosed to have ACS but free of chest pain .The majority of males came with chest pain .

Patients of ACS who were microalbuminuria positive were analyzed for their past history, 6 patients had history of MI out this 3 were males and 3 were females .

There was a significant difference ($p<0.05$) between microalbuminuria positive male and female ACS population .

The known risk factors of ACS like smoking status, tobacco chewing, family history of ACS and hypertension were studied and correlated. 28.3 % of all ACS patients were smokers, 21.8% were hypertensive, 21% chewed tobacco, and 8 % had family history of ACS .

Hypertension had statistically significant correlation with ACS. All risk factors were more associated with STEMI compared to unstable angina or NSTEMI. On comparing with similar studies all risk factors had positive correlation with ACS.

In our study the mean systolic BP was 127.8mmof Hg ± 7.6 amongst males and 118.9mm of Hg ± 5.8 amongst females, these vlues did not differ significantly($p>0.005$)

In the earlier study by borch – johnsen et al they found that mild systolic hypertension (141-160 mmof Hg) was associated with a 3.3 times increased risk of ACS and this increased to 5.3 times when SBP was >160mmofHg.

The mean DBP was 80.5mm of Hg ± 5.4 in males and 75.0 mm of Hg ± 4.7 in females. These values showed that the difference between them were not significant.

Majority of males presented with myocardial infraction i.e. 82.4 % whereas the majority of females i.e. 68.6 % presented with ischaemia on ECG. There was significant statistical difference between males and females in these.

The majority of males and females had normal serum creatinine values (0.9 – 1.0) and there was no significant statistical difference between the two study groups of the population under consideration.

In our study the majority of patients had RBS of <150 and there was no significant statistical difference between the two groups ($p>0.05$). the patients were grouped into <150 and >150 because the value for our hospital for normal RBS was between 150-180.

Microalbuminuria was calculated by the immunoturbidimetry method. A early morning urine sample was taken for this study. The samples were tested in our hospital. The normal albuminuria range is up to 30mg/L for reagent used by this particular test. In our study of Non-diabetic ACS patients who tested positive for microalbuminuria, mean value for males were 43.5 and for females were 47.4 and there was no significant difference between the two groups of our population.

The CPK-MB levels were taken as significant when there were more than 2 times the normal levels our laboratory >40 U/L. the majority of males 77.6% and a majority of females 93.6% had a cpk mb levels.

Total Leucocyte count (TLC) in ACS is usually high as infarction of the myocardium is an inflammatory process. In our study the mean TLC was 12410.7 $\pm$ 6277.27 SD.

Mean LDL was 111.6 $\pm$ 46.57 which was higher than the normal considering the cut off value with existing risk factors.

Both TLC and LDL were statistically significant when compared to microalbuminuria levels and hence our study was similar to other studies in this regard.

The ejection fraction in the majority of our patients <50% but there was no significant statistical difference between the two.

Regional wall motion abnormality was present in majority of our patients 88.1 in males and 77.8 in females with statistical difference between two.

Majority of the patients did not have any valvular heart disease.97.6% males and 94.4% females and these values did not show any significant statistical difference.

Microalbuminuria has been long postulated to have marker in the ischemic coronary arteries diseases and thus its correlation with ACS is of interest. This study tried to find association of urinary microalbuminuria levels in patients with acute coronary syndrome.

The mean microalbuminuria in patients with ACS was 44.6 $\pm$ 22.1 SD mg/l compared to microalbuminuria levels of 30mg/l in normal population.

There is statistically significance microalbuminuria of ACS than normal standard mean value (0.5) ($p<0.0001$). Hence our research hypothesis accepted.

Increased levels of microalbuminuria were shown to be associated with higher prevalence of coronary artery disease emerging as new potential risk factor marker.[25]

SUMMARY

AND

CONCLUSIONS

SUMMARY AND CONCLUSION:

There are many studies that showed a positive correlation between microalbuminiuria and acute coronary syndrome (ACS) hence many researchers postulated that microalbuminiuria was a marker of ACS. This view was however disputed by many others.

In our study of 60 patients admitted to BLDE hospital with symptoms of ACS and were subsequently proved to the ACS patients were tested for microalbuminiuria and those who tested for positive were included in the study.

Microalbuminiuria is known to be associated with diabetes, so we to include patients who are non-diabetic, hypertensive and non hypertensive and aimed at forming a clinical profile of patients who are microalbuminiuria positive ACS patients. From the results that we have derived from this study we can say that microalbuminiuria positive patients had a mean age of approximately 55 years old amongst both sexs. The usual duration of illness before presentation in casuality was less than one day. The majority of them came with the chest pain but not classified as angina. Dyspones was not a significant symptom but past history of ACS and any hospitalization was significant in this study.

Majority of the patients had a significant family history of hypertension and ACS, smoking and alcoholism was a significant part of their personal history. Majority of them had a microalbuminiuria level of >30mg.

Patients of ACS were naturally tested for cardiac enzymes and in our group of microalbuminiuria positive ACS patients, CPKMB and Trop t positive. Maximum

number of ACS patients are included in our study was admitted for chest pain, followed by admissions due to ACUTE CORONARY SYNDROME.

Majority of patients are in normal cholesterol and triglycerides range. Majority of them are not anaemic. The mean BP of this group was 120/84 mm of Hg.

The renal function tests were normal almost for all of them.

Finally on echocardiograohy most of our patients had an ejection fraction of 41-50%. Most of them have regional wall abnormality and a vast majority of them did not have valvular heart disease.

Our aim was to study the clinical profile of the patients with slightly elevated urinary albumin excretion and whether consequently it may be a clinically relevant risk factor.

Our study demonstrated the presence of microalbuminiuria independent of other classic risk factor for ACS. We also found a definitive correlation between LDL Cholesterol and microalbuminiuria; a positive correlation between microalbuminiuria and TGL; and a positive correlation between microalbuminiuria and creatinine, which may become significant in a study based on a larger sample.

Thus individuals with elevated microalbuminiuria levels should get their LDL, Triglycerides and serum creatinine levels measured because this piece of information contributes to the classification of the individual as a high risk or high susceptibility individual.

It is unknown wheather individuals with microalbuminiuria will benefit more from intervention, but we would recommend that future controlled clinical trials should focus on answering this question as it could to a more targeted and focused strategy for the prevention of ACS.

APPARATUS USED FOR MICROALBUMINURIA ASSAY

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ANNEXURE I

ETHICAL CLEARANCE CERTIFICATE:

ANNEXURE II

CONSENT FORM

TITLE OF RESEARCH: "MICROALBUMINURUA IN NON DIABETIC ACUTE CORONARY SYNDROME PATIENTS"

GUIDE :

P.G. STUDENT :

PURPOSE OF RESEARCH:

I have been informed that the purpose of this study is to find out whether there is any association between acute coronary syndrome and urinay microalbuminuria.

PROCEDURE:

I understand that I will undergo detailed history and clinical examination and investigations.

RISKS AND DISCOMFORTS:

I understand that there is no risk involved and I may experience mild pain during the above mentioned procedures.

BENEFITS:

I understand that my participation in this study will help to find out whether there is any association between acute coronary syndrome and urinay microalbuminuria.

CONFIDENTIALITY:

I understand that the medical information produced by the study will become a part of hospital record and will be subjected to confidentiality and privacy regulations of hospital. If the data is used for publications the identity of the patient will not be revealed.

REQUEST FOR MORE INFORMATION:

I understand that I may ask for more information about the study at any time.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and I may refuse to participate or withdraw from study at any time

INJURY STATEMENT:

I understand in the unlikely event of injury to me during the study I will get medical treatment but no further compensations.

(Signature of Guardian)

(Signature of patient)

(If the patient is conscious, well oriented and fully aware)

ANNEXURE III

ACUTE CORONARY SYNDROME PATIENTS"

PROFORMA :

Name: **IP. No:**

Age: **Address**

Sex: **Date of Admission:**

Occupation: **Unit:**

Religion:

Chief complaints: Chest pain

Site/Location

Type/Character

Radiation

Vomiting

Sweating

Present history:

Past history:

History of (H/o) hypertension

H/o myocardial infarction / Angina

H/o diabetes mellitus

H/o hepatitis

DRUGS	YES/NO
ACE INHIBITORS	

H/O drug intake :

H/o a)Recent Trauma, Surgery, Burns

Personal history:

Diet:

Appetite:

Sleep:

Bladder and bowel habits:

Habits:

Family history:

GENERAL PHYSICAL EXAMINATION

Pallor:

Icterus:

Cyanosis:

Clubbing:

Lymphadenopathy:

Edema:

JVP:

VITAL SIGNS:

Pulse rate:

Blood pressure:

Temperature:

Respiration rate:

SYSTEMIC EXAMINATION :

CARDIOVASCULAR SYSTEM:

RESPIRATORY SYSTEM:

PER ABDOMEN EXAMINATION:

CENTRAL NERVOUS SYSTEM:

INVESTIGATIONS:

HAEMATOLOGY

PATHOLOGY:

1)Complete blood count:	
Hb	gm/dl
Total count	Cells/cumm
Differential count Neutrophils	%
Lymphocytes	%
Eosinophils	%
Monocytes	%
Basophils	%
Platelet Count	Cells/cumm
ESR	At end of 1st hour.
2) Urine albumin sample (spot collection for albumin)	

BIOCHEMISTRY:

1)Random Blood sugar (or) Fasting Blood Sugar Post prandial Blood Sugar	
2) FASTING LIPID PROFIL	
Triglycerides	
Total Cholesterol	
HDL-Cholesterol	
LDL-Cholesterol	
VLDL-Cholesterol	
3) CPK-MB	
4) TROP T	
5) SERUM CREATININE	

ECG-

2D-ECHO

APPENDIX :

With incidence of acute coronary syndrome 5% (prevalence is around 3-4% in rural and 8-10% in urban areas)[10] At 95% confidence level, and at 5% Margin of error the sample size is 69 (approx 70).

Formula:

$$n = \frac{Z^2 P (1-P)}{d^2}$$

where n = sample size

z = z statistic for a level of confidence**.**

p =incidence rate**.**

d=margin error**.**

The prevalence of coronary artery disease in India it ranges from (3-10% in rural & urban areas)[10].

STATISTICAL ANALYSIS:

All characteristics wiil be summarized descriptively and graphically. For continuous variables, the summary statistics of N, arithmetic mean (referred to as mean), standard deviation (SD) will be used. For categorical data the number and percentage will be used in the data summaries.

A chi-square (x^2) test will be employed p- value <0.05 would be considered to be statistically significant.

MASTER CHART

S.NO	NAME	IP NO	AGE	SEX	DD	CP	Sweating	Vomiting	PAST HISTORY					PERSONAL HISTORY				GEN. Phy. Examination							CVS			RS		P/A		ROUTINE INVESTIGATION					COMPLETE BLOOD COUNT								Lipid Profile					2D ECHO		
									HTN	MI	ANGINA	HEPT	ACE inhibitor	Food habitis	Smoker	Alcoholic	Bowel&bladdE	Pallor	Cyanos	Clubbing	edema	jvp	Systolic BP	Diastolic BP	PH	S3 sound	Mrm	Breath Sounds	Add sounds	Liver	spleen	Urinary Album	Rbs/fbs/ppbs	CPK MB	Trop t	Sr Creatinine	Hb	Total Count	Neutrophils	Lymphocyte	Eosinophils	Basophils	Monocytes	Platelet coun	ESR	Total Cholest	Triglycerides	HDL	LDL	VLDL	LVEF%	RWMA	VHD
1	chinnapa sidappa	24257	45	M	1D	YES	YES	YES	AB	AB	AB	AB	AB	NV	NO	YES	R	AB	AB	AB	AB	AB	140	100	AB	AB	AB	N	AB	N	N	26	R 89	184	P_+	0.8	16.2	14490	87	9	0	0	4	2.92	5	110	92	47	44	18	40%	P	AB
2	Layappa kesappa giri sagar	9361	30	M	6H	YES	YES	AB	AB	AB	AB	AB	AB	NV	NO	YES	R	AB	AB	AB	AB	AB	100	80	YES	AB	AB	N	AB	N	N	30	R 127	13	N_-	1.2	15.8	16900	73	25	2	0	0	2.38	70	245	210	49	154	42	60%	P	AB
3	mahaveer ramappa shahapur	20668	45	M	4H	YES	YES	YES	AB	AB	AB	AB	AB	V	YES	NO	R	AB	AB	AB	AB	AB	130	80	AB	AB	AB	N	AB	N	N	29	F 110	78	P_+	0.7	13	14700	88	10	0	0	2	3.46	20	214	136	26	160	27	40%	P	AB
4	kamala kashiram shinda	20558	55	F	2d	YES	YES	YES	AB	AB	AB	AB	AB	V	NO	NO	R	AB	AB	AB	AB	AB	130	70	AB	AB	AB	N	AB	N	N	32	R 180	23	P+	0.6	11	20320	50	43	2	5	6	3.04	10	145	115	37	85	23	60%	AB	AB
5	Siddengouda sharan gouda	8761	65	M	1D	YES	AB	AB	AB	YES	AB	AB	AB	V	YES	YES	R	AB	AB	AB	AB	AB	120	70	AB	AB	AB	N	AB	N	N	49	R 109	96	P_+	0.8	18	11320	67	21	7	0	5	2.19	15	180	204	34	96	46	40%	P	AB
6	nanagouda basangouda patil	19443	55	M	4H	YES	YES	YES	YES	AB	AB	AB	AB	V	YES	YES	R	AB	AB	AB	AB	AB	130	80	AB	AB	AB	N	AB	N	N	41	F 106	85	P_+	0.7	15	16190	95	0	0	0	2	1.33	5	165	187	42	85	37	40%	P	AB
7	Revansidda tippanna	21755	55	M	1D	YES	YES	YES	AB	AB	AB	AB	AB	NV	YES	YES	R	AB	AB	AB	AB	AB	120	70	AB	AB	AB	N	AB	N	N	80	R 143	41	N-	0.7	14	12130	70	26	1	0	3	2.45	20	165	106	31	112	21	50%	P	AB
8	shivappa yanamappa	9887	42	M	5H	YES	YES	AB	AB	AB	AB	AB	AB	NV	YES	NO	R	AB	AB	AB	AB	AB	110	70	AB	AB	AB	N	AB	N	N	38	R 94	12	P_+	0.7	14.8	10950	69	26	1	0	4	1.76	20	120	110	22	76	22	50%	P	AB
9	hanumantha timanna	9888	60	M	4H	YES	YES	AB	AB	AB	AB	AB	AB	NV	YES	YES	R	AB	AB	AB	AB	AB	110	70	AB	AB	AB	N	AB	N	N	34	R 110	203	P_+	0.6	16	11720	81	17	0	0	2	3.39	5	152	87	54	80	17	45%	P	AB
10	Nanasaheeb bala saheeb desai	11968	67	M	3H	YES	AB	AB	AB	YES	AB	AB	AB	V	NO	NO	R	AB	AB	AB	AB	AB	120	70	AB	AB	AB	N	AB	N	N	13	R 84	49	P_+	0.8	16	8760	63	28	5	0	4	2.24	25	151	216	27	80	43	50%	P	AB
11	sampapad madangopal das	23306	50	M	5H	YES	YES	YES	AB	N	AB	AB	AB	NV	NO	NO	R	AB	AB	AB	AB	AB	110	70	AB	AB	AB	N	AB	N	N	83	R 111	46	P_+	0.7	13	6180	87	12	0	0	1	1.78	20	211	118	43	107	23	45%	P	AB
12	Sayad buranuddin dadamiya	11718	55	M	4H	YES	YES	YES	YES	AB	AB	AB	AB	NV	YES	NO	R	AB	AB	AB	AB	AB	130	80	YES	AB	AB	N	AB	N	N	36	R130	45	P_+	1.6	14	16980	86	12	0	0	2	2.43	20	201	168	27	141	33	60%	P	AB
13	Abdul hameed faridsab	9780	48	M	2D	YES	YES	AB	AB	AB	AB	AB	AB	NV	YES	NO	R	AB	AB	AB	AB	AB	110	80	AB	AB	AB	N	AB	N	N	23	R 132	50	P_+	0.8	16	12540	77	17	1	0	5	3.24	10	215	160	43	140	32	60%	AB	AB
14	Malkappa Revgond metri	15559	48	M	1D	YES	YES	AB	AB	AB	AB	AB	AB	NV	YES	YES	R	AB	AB	AB	AB	AB	110	70	AB	AB	AB	N	AB	N	N	58	R 133	35	N_-	0.8	16	11900	73	23	4	0	0	2.77	5	135	212	26	66	42	50%	P	AB
15	Siddanna sharanappa Biradar	23700	66	M	8H	YES	AB	AB	AB	AB	AB	AB	AB	NV	NO	NO	R	AB	AB	AB	AB	AB	130	80	AB	AB	AB	N	AB	N	N	37	R 138	53	P_+	0.9	17	7450	64	22	10	0	4	1.85	5	210	164	40	130	46	45%	P	AB
16	Nagu Kandiba shinde	23755	40	M	1D	YES	AB	AB	YES	AB	AB	AB	AB	NV	NO	YES	R	AB	AB	AB	AB	AB	120	80	AB	AB	AB	N	AB	N	N	56	F 139	38	N_-	1.1	13	9110	87	12	0	0	1	3.13	60	188	91	50	129	18	60%	AB	AB
17	Shivabasamma Basappa	25021	80	F	1D	YES	YES	AB	YES	AB	AB	AB	AB	V	NO	NO	R	AB	AB	AB	AB	AB	110	80	AB	AB	AB	N	AB	N	N	41	R 126	26	N_-	0.6	6.2	19370	85	13	0	0	2	2.58	40	140	196	26	74	39	60%	AB	AB
18	Ranganath basappa kurti	24379	38	M	6H	YES	YES	AB	YES	AB	AB	AB	AB	NV	NO	NO	R	AB	AB	AB	AB	AB	144	90	AB	AB	AB	N	AB	N	N	36	R 112	37	P_+	1	15	10600	58	38	2	0	2	3.16	5	130	62	37	80	12	45%	P	AB
19	Lakshmibai mallapoa sarashetti	24389	68	M	1D	YES	AB	AB	YES	YES	AB	AB	YES	NV	NO	NO	R	AB	AB	AB	AB	AB	160	100	AB	AB	AB	N	AB	N	N	40	R 89	23	N_-	0.6	13	10760	75	20	2	0	3	2.8	20	158	174	40	83	34	40%	P	AB
20	Amasidda ramanna	21939	60	M	2D	YES	YES	AB	AB	AB	AB	AB	AB	NV	NO	NO	R	AB	AB	AB	AB	AB	110	70	AB	AB	AB	N	AB	N	N	32	R 116	189	N_-	0.6	13	12000	90	7	1	0	2	2.3	5	195	97	26	149	19	45%	P	AB
21	Irrappa basappa twlawad	24435	80	M	3D	YES	YES	AB	AB	AB	AB	AB	AB	NV	NO	NO	R	AB	AB	AB	AB	AB	120	70	AB	AB	AB	N	AB	N	N	57	R 144	24	P_+	0.9	12	10160	88	10	0	2	0	1.47	45	144	78	29	89	17	45%	P	AB
22	Basanna tammanna ajanalkar	26049	65	M	1D	YES	YES	AB	AB	AB	AB	AB	AB	NV	YES	YES	R	AB	AB	AB	AB	AB	130	80	AB	AB	AB	N	AB	N	N	24	F 90	30	N_-	0.6	11	8350	92	5	1	0	2	1.5	35	150	78	36	98	15	40%	P	AB
23	hema ishwar ambalajeri	13851	40	F	4H	YES	YES	AB	YES	AB	AB	AB	AB	V	NO	NO	R	AB	AB	AB	AB	AB	110	70	AB	AB	AB	N	AB	N	N	76	F 108	42	N_-	0.8	10	9030	78	20	0	0	2	1.51	15	191	159	40	80	38	45%	P	AB
24	Shrishail muragappa aloor	26244	56	M	1D	YES	AB	AB	AB	AB	AB	AB	AB	V	NO	NO	R	AB	AB	AB	AB	AB	112	80	AB	AB	AB	N	AB	N	N	17	R 87	26	P_+	0.9	16	10500	60	35	1	0	4	3.47	10	170	150	24	116	30	60%	AB	AB
25	Hanumanthiyya basappa	26581	53	M	8H	YES	AB	AB	AB	AB	AB	AB	AB	V	NO	NO	R	AB	AB	AB	AB	AB	90	50	AB	AB	AB	N	AB	N	N	16	R 102	34	N_-	0.8	13	12600	82	13	3	0	2	2.6	65	155	176	30	82	35	45%	P	AB
26	Malakubai vittal indi	27131	62	F	1D	YES	YES	YES	AB	AB	AB	AB	AB	NV	NO	NO	R	AB	AB	AB	AB	AB	100	70	AB	AB	AB	N	AB	N	N	56	R 106	73	N_-	0.6	11	11290	86	12	0	0	2	2.43	20	160	165	30	97	33	50%	P	AB
27	krishna subbha rao kale	2387	55	M	8H	YES	YES	YES	AB	AB	AB	AB	AB	NV	YES	NO	R	AB	AB	AB	AB	AB	150	90	AB	AB	AB	N	AB	N	N	34	R 112	246	P_+	1	15	19890	88	10	0	0	2	3.66	5	319	140	45	246	28	40%	P	AB
28	Ragavendra gururaj kulkarni	2157	55	M	6H	YES	YES	AB	AB	AB	AB	AB	AB	NV	NO	NO	R	AB	AB	AB	AB	AB	120	70	AB	AB	AB	N	AB	N	N	42	R 107	300	P_+	0.8	14	10890	66	24	6	0	4	3.01	5	207	134	34	146	26	50%	P	AB
29	Rachayya shivayaa hiramanth	2267	56	M	6H	YES	AB	AB	AB	AB	AB	AB	AB	V	NO	NO	R	YES	AB	AB	AB	AB	180	100	AB	AB	AB	N	AB	N	N	30	R 125	52	P_+	0.6	9	36950	95	4	0	0	1	4.61	150	258	263	43	162	52	55%	P	AB
30	guralingayya shivaling hiremath	20140	38	F	1D	YES	AB	YES	AB	AB	AB	AB	AB	V	NO	NO	R	AB	AB	AB	AB	AB	110	70	AB	AB	AB	N	AB	N	N	67.5	R 180	32	N-	0.6	13.5	17630	78	20	0	2	0	2.39	30	178	438	25	65	87	60%	N	AB
31	Sanjeev kumar naik	2326	36	M	2D	YES	AB	AB	AB	AB	AB	AB	NV	YES	NO	R	AB	AB	AB	AB	AB	120	80	AB	AB	AB	N	AB	N	N	18	R 94	105	P_+	0.9	15	11950	80	6	1	0	3	3.17	5	281	221	36	200	44	50%	P	AB
32	Neelama nandayya	2388	50	F	6H	YES	YES	AB	YES	AB	AB	AB	AB	V	NO	NO	R	AB	AB	AB	AB	AB	130	90	AB	AB	AB	N	AB	N	N	78	R 92	50	P_+	1	11	18460	92	8	0	0	0	2.56	85	242	149	27	185	29	45%	P	AB
33	jitendra narayansing hajeri	21722	33	M	1D	YES	AB	AB	AB	AB	AB	AB	AB	NV	NO	NO	R	AB	AB	AB	AB	AB	140	70	AB	AB	AB	N	AB	N	N	58	R 105	37	N-	2.7	14	11900	75	19	6	0	0	3.33	10	218	167	52	130	33	45%	P	AB
34	Lakshmibai ramachandra pujari	4218	65	F	1D	YES	YES	YES	AB	AB	AB	AB	AB	NV	NO	NO	R	AB	AB	AB	AB	AB	120	80	AB	AB	AB	N	AB	N	N	89	R 165	44	N_-	0.7	11	21700	83	16	1	0	2	3.34	30	200	158	39	129	31	45%	P	AB
35	Parvathi chandrakanth bansode	3085	65	F	1D	YES	YES	YES	AB	AB	AB	AB	AB	V	NO	NO	R	AB	AB	AB	AB	AB	110	70	AB	AB	AB	N	AB	N	N	50	R 129	133	P_+	0.6	13	12500	88	8	2	0	2	4.28	30	252	120	52	176	24	45%	P	AB
36	Guruputra chandrappa	2950	60	M	1D	YES	YES	AB	YES	AB	AB	AB	AB	V	NO	NO	R	AB	AB	AB	AB	AB	140	80	AB	AB	AB	N	AB	N	N	80	R 143	32	N_-	3.2	11	7980	80	14	3	0	0	3.6	90	126	113	34	69	22	35%	P	AB
37	Mithili ravi das	2583	53	M	6H	YES	YES	AB	AB	AB	AB	AB	AB	NV	NO	YES	R	AB	AB	AB	AB	AB	210	140	AB	AB	AB	N	AB	N	N	80	R 93	75	P_+	1	14	3140	90	7	1	0	2	1.8	15	200	82	70	113	16	35%	P	AB
38	Annapurna irappa guddewadi	3992	60	F	2D	YES	YES	AB	AB	AB	AB	AB	AB	V	NO	NO	R	YES	AB	AB	AB	AB	110	70	AB	AB	AB	N	AB	N	N	12	R 112	68	P_+	0.7	9.5	9500	81	17	2	0	0	1.45	30	176	86	47	111	17	30%	P	AB
39	Siddhu hanumanth talewad	4754	40	M	8H	YES	AB	AB	AB	AB	AB	AB	AB	NV	NO	YES	R	AB	AB	AB	AB	AB	150	100	AB	AB	AB	N	AB	N	N	23	R 102	51	P_+	0.9	16	11860	86	10	1	0	3	2.28	5	164	75	50	99	15	50%	P	AB
40	Saida abdul syed md peerjade	4809	66	M	1D	YES	YES	AB	AB	AB	AB	AB	AB	NV	NO	NO	R	AB	AB	AB	AB	AB	120	80	AB	AB	AB	N	AB	N	N	65	R 118	154	P_+	1.1	12	13150	78	18	0	0	4	3.47	30	170	146	37	103	29	45%	P	AB
41	Boramma malappa hadapad	4834	45	F	2D	YES	YES	YES	AB	AB	AB	AB	AB	V	NO	NO	R	AB	AB	AB	AB	AB	100	60	AB	AB	AB	N	AB	N	N	20	R 98	41	N_-	0.7	11	11300	83	15	1	0	1	2.3	25	181	95	49	113	19	45%	P	AB
42	Basappa ningappa rugi	5118	55	M	8H	YES	YES	AB	AB	AB	AB	AB	AB	NV	NO	NO	R	AB	AB	AB	AB	AB	110	90	AB	AB	AB	N	AB	N	N	75	R 106	45	P_+	0.7	15	10450	75	20	3	0	2	2.45	10	173	231	22	104	46	45%	P	AB
43	Sudash chandra shranappa `	4496	50	M	1D	YES	AB	AB	AB	AB	AB	AB	AB	V	YES	NO	R	AB	AB	AB	AB	AB	120	90	AB	AB	AB	N	AB	N	N	30	R 81	100	P_+	0.7	14	8290	79	16	2	0	3	2.55	5	128	98	31	77	19	60%	AB	AB
44	Md yusuf modinsab peerjade	5900	68	F	1D	YES	YES	AB	YES	AB	AB	AB	AB	NV	NO	NO	R	YES	AB	AB	AB	AB	140	80	AB	AB	AB	N	AB	N	N	27	R 125	31	N_-	1.2	8.5	14520	80	14	2	0	4	3.5	5	121	67	49	58	13	40%	P	AB
45	Babu lakshman jadhav	6261	52	M	1D	YES	AB	YES	AB	AB	AB	AB	AB	V	NO	YES	R	AB	AB	AB	AB	AB	90	50	AB	AB	AB	N	AB	N	N	42	F 100	77	P_+	0.7	16	5320	64	32	3	0	0	1.7	5	177	634	25	25	126	50%	P	AB
46	Vittal lakshman gunnapur	6375	58	M	1D	YES	YES	YES	AB	AB	AB	AB	AB	V	NO	NO	R	AB	AB	AB	AB	AB	140	100	AB	AB	AB	N	AB	N	N	75	R 110	52	P_+	1.4	15	36130	78	6	2	0	5	3.34	5	189	132	55	107	26	40%	P	AB
47	Shabira kashimsab walikar	6730	60	F	2D	YES	YES	AB	AB	AB	AB	AB	AB	NV	NO	NO	R	AB	AB	AB	AB	AB	130	70	AB	AB	AB	N	AB	N	N	48	R 125	72	N_-	0.9	10	11700	88	8	0	0	4	2.65	30	130	95	28	83	19	45%	P	AB
48	Appa limbaji lokande	7025	45	M	2D	YES	YES	AB	AB	AB	AB	AB	AB	NV	NO	NO	R	AB	AB	AB	AB	AB	140	60	AB	AB	AB	N	AB	N	N	23	R 89	54	N_-	1	15	9490	60	35	2	0	3	3.4	35	253	205	33	179	41	40%	P	AB
49	Shanthabai arjun jigajini	8409	52	F	8H	YES	AB	AB	AB	AB	AB	AB	AB	V	NO	NO	R	AB	AB	AB	AB	AB	110	70	AB	AB	AB	N	AB	N	N	34	R 149	132	P_+	0.9	9.9	11980	90	9	0	0	1	2.54	115	197	137	50	119	27	50%	P	AB
50	Pulabai maruti chavan	6979	65	F	1D	YES	YES	AB	AB	AB	AB	AB	AB	V	NO	NO	R	YES	AB	AB	AB	AB	130	80	AB	AB	AB	N	AB	N	N	190	R 80	64	P_+	1.2	9	26430	86	10	0	0	4	2.74	30	140	64	32	75	14	45%	P	P
51	Mallappa bhimappa elaganti	9508	65	M	1D	YES	AB	AB	AB	AB	AB	AB	AB	V	NO	NO	R	AB	AB	AB	AB	AB	120	90	AB	AB	AB	N	AB	N	N	49	R 142	151	P_+	1	13	9680	67	27	2	0	4	1.8	20	182	134	31	124	26	35%	P	AB
52	Shankar tuljaram dhudekar	9608	59	M	6H	YES	YES	AB	AB	AB	AB	AB	AB	NV	YES	NO	R	AB	AB	AB	AB	AB	110	80	AB	AB	AB	N	AB	N	N	98	R 180	36	P_+	0.6	12	10330	68	25	2	0	5	3.19	90	265	167	34	197	33	35%	P	AB
53	RANIBAI MOTHILAL RATHOD	9665	59	F	1D	YES	AB	AB	AB	AB	AB	AB	AB	NV	NO	NO	R	YES	AB	AB	AB	AB	130	80	AB	AB	AB	N	AB	N	N	21	F 110	60	P_+	0.7	9	11800	81	17	0	0	2	2.65	85	182	168	35	113	33	60%	AB	AB
54	RAYAWWA SIDDAPPA ILAGER	25005	65	M	4H	YES	YES	YES	AB	AB	AB	AB	AB	V	YES	NO	R	AB	AB	AB	AB	AB	140	90	AB	AB	AB	N	AB	N	N	53	R 128	72	P_+	0.6	11	11810	82	15	1	0	2	3.44	20	150	100	28	102	20	55%	P	AB
55	BURAPPA BIMAPPA HOMAWID	8958	85	M	16H	YES	AB	AB	AB	AB	AB	AB	AB	V	YES	YES	R	AB	AB	AB	AB	AB	140	90	AB	AB	AB	N	AB	N	N	21	R 119	10	N_-	0.6	13	11910	90	7	1	0	2	2.45	10	90	72	25	50	14	45%	P	P
56	Shivappa gurappa shetty	9893	76	M	2D	YES	YES	YES	AB	AB	AB	AB	AB	V	NO	NO	R	AB	AB	AB	AB	AB	110	60	YES	AB	AB	N	AB	N	N	49	R 78	78	P_+	1	11	12670	72	24	2	0	2	3.39	25	148	127	35	87	25	60%	AB	AB
57	sharawwa yamanappa	20731	60	F	1D	YES	YES	YES	YES	AB	AB	AB	AB	V	NO	NO	R	AB	AB	AB	AB	AB	130	90	AB	AB	AB	N	AB	N	N	30	R 180	55	P_+	0.7	12	9460	73	22	1	0	4	1.82	15	180	100	49	111	20	45%	P	AB
58	GOURABAI DUNDAPPA YARANAI	10362	65	F	2D	YES	YES	AB	AB	AB	AB	AB	AB	NV	NO	NO	R	AB	AB	AB	AB	AB	100	60	YES	AB	AB	N	AB	N	N	28	R 100	28	P_+	0.5	11	9890	73	23	2	0	2	2.67	25	115	84	25	73	17	40%	P	AB
59	SHANTAPPA NINGAPPA ELAGI	10712	70	M	1D	YES	AB	AB	YES	AB	AB	AB	AB	V	YES	NO	R	YES	AB	AB	AB	AB	130	80	AB	AB	AB	N	AB	N	N	38	R 70	44	N_-	0.8	9.9	12950	80	15	2	0	3	2.24	70	120	61	35	72	12	60%	P	AB
60	neelakantayya siddayya hiremath	27516	78	M	8H	YES	YES	AB	YES	AB	AB	AB	AB	V	NO	NO	R	AB	AB	AB	AB	AB	150	90	AB	AB	AB	N	AB	N	N	40	R150	30	P+	1.4	10	10000	86	13	0	1	0	2.45	90	150	115	28	99	23	60%	P	AB