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Profile of Coronary Artery Disease in Adults with Severe Left Ventricular Systolic Dysfunction.

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Abstract: *The present study was undertaken to analyze the profile of CAD in adults with severe LV systolic dysfunction with global hypokinesia with EF<35% were enrolled at I.G.M.C. Shimla over a period of one year. A total of 33 patients admitted with severe LV systolic dysfunction at I.G.M.C. Shimla over a period of one year were subjected to coronary angiography to rule out CAD in patients with severe LVD. The analysis of the demographic profile revealed that CAD was more common in males than females across all age groups and prevalence of heart failure increases with the age. The age and sex distribution of patients in our study was similar to the other studies. A large majority of patients were from urban areas 64.2 %. The study observed that all patients had breathlessness of NYHA class II (15.2%) & III(84.8%), indicating that study population of the present study comprised of sick patients in advanced stage of heart failure. The severity of signs and symptoms was more with LVEF of less than 26% and was statistically insignificant. The analysis of risk factors showed that Obesity (78.9) was the most common risk factor followed by hypertension (64.2%), Diabetes (50%), Smoking (36%) and Alcoholism in 14.3% of patients. Frequency & distribution of various risk factors amongst patients with severe LVD with CAD (Group1) & severe LVD without CAD(Group2) study population revealed no significant difference except Diabetes which was present in 50% of patients with Group1 where as in group2 it is present only in 5.2% of the patients. This apparent significant difference is due to the fact that prevalence of Diabetes in general population was associated with increased rates of CAD especially more with TVD. The most common ECG abnormalities was LBBB (30.3%) followed by atrial fibrillation (12.1%) & IVCD in 6.2% of patients. The mean LVEF was 26.3±5.4%. The most common etiological diagnosis was Ischemic cardiomyopathy (42.4%).Diabetes was the most common risk factor in ICM group & it was present in 50% of the patients. Among the patients with severe LVD without CAD (57.4%), 30.3% patients were having Hypertensive Heart Disease, 21.2% were having Idiopathic Dilated Cardiomyopathy followed by Alcoholic cardiomyopathy in 6.2% of the patients. Out of the 33 patients one patient had evidence of hypothyroidism in the Group1. In patients with CAD, 50% had triple vessel disease, 21.4% had double vessel disease & 28.5% patients had single vessel disease. Thus approximately 71.4% patients with CAD had more than double vessel disease & this may be the cause of severe LV systolic dysfunction, as LV dysfunction in them could be due to hibernating myocardium. But before we label them as Ischemic Cardiomyopathy we have to demonstrate viability by various methods & if found viable myocardium these patients should be subjected to revascularisation. The study for viability was done only in two patients, out of 14 patients with CAD & this is the limitation of this study.*

Key Words: *Systolic, Diastolic, Dysfunction, Heart failure, Treatment.*

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I. Introduction

Heart failure is the end stage of all diseases of the heart and is a major cause of morbidity and mortality (1). It poses enormous challenges as heart failure rates are increasing throughout the developed and developing regions of the world (1,3).

Heart failure is a worldwide problem, the estimated heart failure prevalence worldwide is 23 million, the prevalence of heart failure in United States is >5 million and Europe is about 10 million, as most of the

studies had been done in these regions. Very little is known with respect to the prevalence or risk of developing heart failure in emerging nations because of the lack of population-based studies in these countries(3).

Heart failure is a complex clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood. The cardinal manifestations of heart failure are dyspnoea and fatigue, which may limit exercise tolerance and fluid retention, which may lead to pulmonary congestion and peripheral oedema(2).

There is lack of agreement on definition of heart failure to study the epidemiology which is primarily a clinical diagnosis (1).

The prevalence of heart failure is 1-2% in the western world and the incidence approaches 5-10 per 1000 persons per year. The estimate of heart failure in the developing world are largely absent (4). The prevalence and incidence of this disease increases with age & prevalence doubles with each decade of life, seen more frequently in men than women, leads to repeated hospitalization. The number of deaths due to heart failure are more among women than men (1).

There are following types of heart failure, Forward and Backward, Acute and Chronic, Low output and High output, Left and Right, Systolic and Diastolic heart failure (3).From practical stand point, it is useful to divide patients with heart failure into systolic dysfunction and diastolic dysfunction, which usually involves patient's assessment of ejection fraction. Patients with symptoms and examination findings are consistent with heart failure with impaired ventricular ejection fraction of less than 40% are classified having left ventricular systolic dysfunction (2,3).

Systolic heart failure is impairment of the ability of ventricles to eject blood to meet the needs of the body & have low ejection fraction (3). Non ischemic heart failure is due to non-coronary arteries involvement. The systolic heart failure has prevalence of 1.8% over the age of 45 years, higher in men than women, increases with age (1).

In a pooled study of five randomised controlled trial with systolic dysfunction showed that male population was more with systolic dysfunction, 47% were with non ischemic systolic dysfunction, 68.7% were males, women were older among non-ischemic systolic heart failure (5).

In 1970, Burch and colleagues first used the term ischemic cardiomyopathy (ICM) to describe a condition in which CAD results in severe myocardial dysfunction, with clinical manifestations indistinguishable from those of primary DCM(6).

Although the most common cause of heart failure is coronary artery disease, heart failure due to non ischemic causes accounts for about 25-40%. Causes of non ischemic systolic heart failure are dilated cardiomyopathies due to familial or genetic disorders, infiltrative disorders, toxic or drug induced damage including alcohol, viral and Chagas disease. Other causes are hypertension, valvular diseases-obstructive, regurgitant, left to right shunt & extra cardiac shunts, rate and rhythm disorders – chronic brady & tachyarrhythmias and high output states. In Asia and Africa rheumatic heart disease is major cause of heart failure, where as Chagas disease is a cause in South America (7).

Ischemic cardiomyopathy is a term used to describe patients whose heart can no longer pump enough blood to the rest of their body, as a result of a chronic lack of oxygen, due to coronary artery disease. Coronary artery disease is a narrowing of the small blood vessels that supply blood and oxygen to the heart. An effort to provide a standardized definition of ischemic cardiomyopathy was made in a review of 1921 patients with symptomatic heart failure (HF) and a left ventricular ejection fraction (LVEF) <40 percent who underwent coronary angiography. Ischemic cardiomyopathy was considered to be present in patients with HF who have had a myocardial infarction (MI) or have evidence of hibernating myocardium or, on angiography, severe coronary disease. Such patients had a worse outcome than those with non ischemic cardiomyopathy. In contrast, patients with single vessel disease who had no history of myocardial infarction or revascularization had a similar prognosis as those with non ischemic cardiomyopathy. It was suggested that such patients should be classified as non ischemic cardiomyopathy, at least for prognostic purposes (8).

Non Ischemic cardiomyopathy is defined as a myocardial disorder in which the heart muscle is structurally and functionally abnormal, in the absence of other causes of heart dysfunction, like coronary artery disease, hypertension, valvular disease and congenital heart disease(9).

Left Ventricular Dysfunction [LVD] is an important indicator of morbidity & mortality in cardiac patients. Coronary artery disease [CAD] is an important cause of LVD in adults, which may present as myocardial infarction, unstable angina or chronic stable angina. Another important cause of LVD is idiopathic dilated cardiomyopathy [DCM].

Some patients of CAD may not present with chest pain but with LVD only. LVD in them could be due to dysfunctional but viable myocardium, either as a result of chronic flow impairment [hibernating myocardium] or following episodes of ischemia, when LVD persists for a period of time after restoration of blood flow, duration depending upon the period of ischemic insult[stunned myocardium]. Myocardial Stunning is defined as post

ischemic myocardial dysfunction that persists despite restoration of normal blood flow. Hibernation is a state in which some segments of the myocardium exhibit abnormalities of contractile function at rest.

This phenomenon is highly significant clinically because it usually manifests itself in the setting of chronic ischemia that is potentially reversible by revascularization. The reduced coronary blood flow causes the myocytes to enter a low-energy 'sleep mode' to conserve energy.

Left ventricular (LV) function is one of the most important determinants of long-term outcome in patients with coronary artery disease (CAD). Patients with normal or near-normal LV function have an excellent prognosis, whereas patients with impaired LV function are at substantial risk of death during medical therapy. It is now apparent that LV dysfunction is not always an irreversible process and that LV function may improve considerably, and even normalize, after myocardial revascularization in a large subset of patients. The identification of such patients with "hibernating" myocardium that is under perfused and dysfunctional, yet viable, has important implications in the selection of patients with LV dysfunction for revascularization procedures(10).

Both nuclear cardiology techniques and 2-D echocardiography can be used for this purpose. The radionuclide techniques include positron emission tomography to assess blood flow and metabolism (using agents such as [18F] fluorodeoxyglucose) and thallium-201 (and possibly technetium-99m sestamibi) to assess blood flow and cell membrane integrity. Alternatively, echocardiographic imaging during low-dose infusions of dobutamine can be used to assess inotropic reserve. The data available to date suggest that patients with CAD in whom hibernating myocardium is largely the cause of impaired LV function constitute a subgroup of patients who may achieve a substantial improvement in LV function and in outcome if identified and treated with revascularization(10).

But the SOLVD registry and the Swedish study contribute to a better understanding of the natural course of congestive heart failure. Both studies are limited, however, in their ability to draw conclusions about the clinical course of ischemic and non ischemic cardiomyopathy. It is very difficult to diagnose ischemic cardiomyopathy reliably without performing coronary angiography, because many clinical features commonly associated with this condition frequently appear in patients with non ischemic disease. Likoff et al. (11) followed the clinical course of 201 patients with heart failure to determine which clinical variables were the best predictors of mortality. All patients underwent coronary angiography, the results of which were used to determine ischemic or non ischemic etiology. During the 10.8-month follow-up period, the overall mortality rate was 42%; the mortality rate among ischemic patients (n = 121) was significantly higher than that among non ischemic patients (n = 80) (p = 0.005)(12).

Another study showing Angiographically diagnosed ischemic HF is associated with shorter survival than non ischemic HF. A more extensive CAD is independently associated with shorter survival, and patients with single-vessel disease and no history of MI or revascularization should be classified as non ischemic for prognostic purposes. Standardization of the definition of ischemic cardiomyopathy will be useful in the conduct and interpretation of clinical research in HF(8).

JACC proposes a new definition of ischemic cardiomyopathy that reclassifies patients with single-vessel disease as non ischemic unless they have left main or proximal LAD disease or a history of revascularization or MI. The use of such a standardized definition will help limit variability in defining etiologic subgroups for clinical trials and population-based studies. Accurate ascertainment of etiology and its impact on prognosis is important for risk stratification of individual patients and for planning appropriate subgroups for clinical research. Based on these data, coronary angiography should remain a cornerstone of the evaluation of patients with newly diagnosed systolic dysfunction and symptomatic HF(13).

Ischemic etiology is an independent predictor of mortality in patients with left ventricular dysfunction. However, the extent of CAD contributes more prognostic information than the clinical diagnosis of ischemic or non ischemic cardiomyopathy. Coronary angiography should be considered in all patients with left ventricular dysfunction, because the results substantially contribute to diagnosis, prognosis and management decisions (12).

The systolic dysfunction & myocardial damage is global instead of localized abnormality of ventricular contractility in patients with DCM, have progressive pump failure and malignant arrhythmias which are the most frequent causes of death & prognosis is better than ischemic heart failure(14).

But in developing countries like INDIA, not much literature is available regarding angiographic diagnosis of ischemic cardiomyopathy in a subset of patients with dilated cardiomyopathy with severe left ventricular systolic dysfunction.

Only one study from IGMC dept. Of Cardiology by R.Bhardwaj et al (15) showing approximately 38% of the patients with severe LVD were found to have CAD & nearly 22% had more than DVD. Extent of CAD in these patients was also significant as is evident from the fact that 26 vessels were involved in 12 patients with CAD, with 32 lesions.20 of these showed critical stenosis. Thus LVD in at least some of these patients can be due to hibernating myocardium and could improve with revascularisation.

The present study aims to find out the incidence of CAD in patients with severe LV systolic dysfunction without any clinical, ECG or Echocardiographic evidence of angina or old MI & to study the clinical charecteristics & systolic function in patients with severe LV systolic dysfunction found to have CAD, attending to OPD & indoor services of cardiology and internal medicine department.

II. Aims And Objectives

1. To find out the incidence of CAD in patients with severe LV systolic dysfunction without any clinical, ECG or Echocardiographic evidence of angina or old MI.
2. To study the clinical charecteristics & risk factors in patients with severe LV systolic dysfunction found to have CAD.

III. Materials And Methods:

The study was carried out in all consecutive patients with signs & or symptoms of Heart Failure with LVEF <35% without any clinical, ECG or Echocardiographic evidence of CAD attending to cardiology/Medicine OPD or admitted in the Department of Medicine and Cardiology at I.G.M.C. Shimla over a period of one year. Written informed consent was taken from the patients.

Inclusion criteria:

1) Consenting to participate
2) Age>35 years
3) ECG: No evidence of acute or old MI
4) LVEF <35%
5) No RWM abnormality
6) Absence of angina & documented CAD
7) Valvular heart disease

Exclusion Criteria;

1) Not consenting to participate
2) Age <35 years
3) History of angina or myocardial infarction[MI]
4) ECG:Evidence of acute or old MI
5) Echocardiography: Evidence of RWMA
6) EF >35%

MATERIALS and METHODS:

All consecutive patients of heart failure attending OPD or admitted in Cardiology/Medicine department of IGMC from Juiy 2010 to June 2011 fulfilling the inclusion criteria & without any exclusion criteria were taken into the study.

All the eligible patients willing to participate were subjected to focused history and examination.

Demographic profile of the patients was recorded which included age, sex, educational status, background and place of residence (rural/urban) . History regarding diabetes, hypertension, alcohol consumption, tobacco consumption, symptoms suggestive of hypo/hyperthyroidism, receiving anthracyclines, viral prodrome preceding illness, myopathies, autoimmune diseases, family history, pregnancy, Rheumatic Fever/RHD as per pre-designed recording format.

Following history-taking patients were subjected to physical examination to record; BP, HR, Rhythm, elevated JVP, dependent oedema, evidence of Thyroid enlargement, bruit, signs of hypo/hyperthyroidism, systemic AV malformation as per pre-designed recording format.

Systemic examination was focused on any evidence of Cardiomegaly, LVS3, any evidence of valvular heart disease, signs of basal rales, hepatic enlargement and ascites. After physical examination each patients were subjected to undergo following Blood tests. Details of the biochemical investigations, Fasting Blood Sugar (FBS), blood urea, serum creatinine, sodium and potassium levels and HbA1C in selected patients, done by the treating physician or cardiologist within 24 hours of hospital admission were recorded.

Special test; were done if indicated on clinical examination; e.g. thyroid function test, ANA, HIV-Elisa etc. ECG: 12 lead ECG recording of each patient was done to record;

HR, Rhythm, PR interval, QRS duration, LBBB/RBBB, QRS axis.

Echocardiography; All patients were subjected to detailed 2D, M mode to study LV dimensions in diastole(LVEDD) & systolic(LVESD), LV mass, valve regurgitation & regional wall motion abnormalities.

Coronary Angiography; All patients were subjected to Coronary Angiography through radial or femoral approach, to recognize occlusion, stenosis, thrombosis or aneurysmal enlargement of the coronary

artery lumens. Patients having stenosis of >50% in any of the three coronary vessels were labelled as having CAD, critical stenosis was defined as diameter of stenosis >70%.

DEFINITIONS

CARDIOMYOPATHY with CAD: Echo findings suggestive of ;

- LV ejection fraction <35%,
- Global hypokinesia and
- Angiographic evidence of documented coronary artery disease with stenosis of >50%, in atleast one of the coronary vessels.

CARDIOMYOPATHY without CAD: Echo findings suggestive of ;

- LV ejection fraction <40%,
- Global hypokinesia and
- Clinical/angiographic absence of documented coronary artery disease.

Waist circumference; Was taken as circumference of abdomen
midway between lower-most rib and highest point on iliac crest at the end of gentle expiration using a one centimeter width measuring tape.

Hemogram;Estimation of HB level by SM 9, haematology auto analyser,

Biochemistry; Estimation of blood urea, serum creatinine, sodium and potassium levels and fasting blood sugar were recorded. These tests were performed at I.G.M.C. by Konelab 30 fully automatic analyser.

ECG: 12 lead ECG recording of each patient was done to record;

HR, Rhythm, PR interval, QRS duration, LBBB/RBBB, QRS axis, evidence of Q wave, ST-T changes & any AV block.

Echocardiography; Echocardiography was done using ATL HDI-3600 Echo machine from parasternal and apical windows.

RISK FACTORS:

Cigarette Smoking: Smokers were defined as those who had ever smoked more than 100 cigarettes or beedis in their life time or had smoked atleast one cigarette/beedi per day for last three months. Ex- Smokers were defined as those who had not smoked even a single cigarette/beedi for last 12 months but had smoked more than 100 cigarettes/beedis in the past.

Obesity: Visceral obesity was defined according to IDF criteria as waist circumference ≥90 cm in men and ≥80 cm in women.

Hypertension was defined as

1. Diagnosed patients of hypertension.
2. SBP ≥140 mmHg and/or DBP ≥90 mmHg.

Diabetes: Patient were labelled as diabetic if he was known case of diabetes or if fasting blood sugar was more than 126 mg/dl and HbA1C is >7% in non diabetics.

IV. Statistical Analysis:

Data collected was managed on a Microsoft excel spreadsheet. Categorical & continuous variables of the clinical charecteristics of study population was described as percentages & Means±SD respectively & Chi-Square test was used to compare significance of difference in the distribution of discrete variables. Non-parametric test Mann-Whitney test was used to compare the significance of difference in means of continuous variables. 2 tailed significance at <0.05 was used as statistically significant. All analysis was performed with the Epi-Info version 3.5.1.

V. Results:

In the present study, a total of 33 consecutive patients were included with severe left ventricular systolic dysfunction in accordance with Echo findings suggestive of global hypokinesia with LV ejection fraction <35%, were enrolled at I.G.M.C. Shimla over a period of one year from July 2010 to June 2011 & subjected to Coronary Angiography to rule Coronary Artery Disease. Patients were divided into two groups, Group1 is patients with severe LVD with CAD & Group2 is patients with severe LVD without CAD.

PATTERN OF CORONARY ARTERY DISEASE IN GROUP 1 & GROUP 2 PATIENTS;

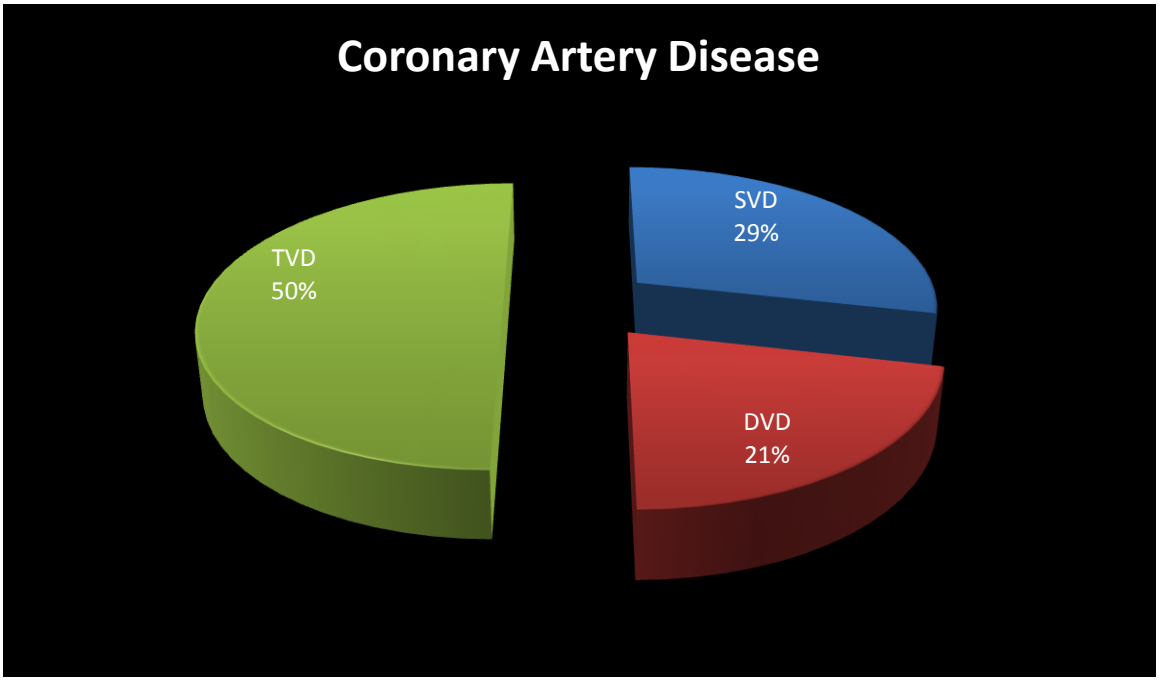
Out of the 33 subjects which were subjected to coronary angiography, 14(42.4%) (group1) patients were found to be having significant CAD & rest of the 19(57.6%)(group2) subjects were having normal coronaries.

Out of 14 patients of group 1, 4(28.57%), 3(21.43%) & 7(50%) had single vessel disease, two vessel disease & three vessel disease respectively. LMCA was not involved in any of the patients.

TABLE1-CAD IN PATIENTS WITH SEVERE LV SYSTOLIC DYSFUNCTION;

Sr No	Parameters	Frequency distribution
1	Total patients	33
2	Normal coronaries	19(57.6%)
3	Coronary artery disease	14(42.4%)
4	Single vessel disease	4(28.57%)
5	Double vessel disease	3(21.43%)
6	Three vessel disease	7(50%)
7	Total vessel involved	31
	LAD	9(29.03%)
	LCX	10(32.25%)
	RCA	12(38.74%)
8	Extent of vessels involved	
	Vessels involved	31
	Total occlusion	8(25.8%)
	Critical+Total stenosis{70-99%}	15(48.38%) +8(25.8%)
	Stenosis {<70%}	8(25.8%)

FIGURE1-PATTERN OF CAD IN PATIENTS WITH SEVERE LV SYSTOLIC DYSFUNCTION;

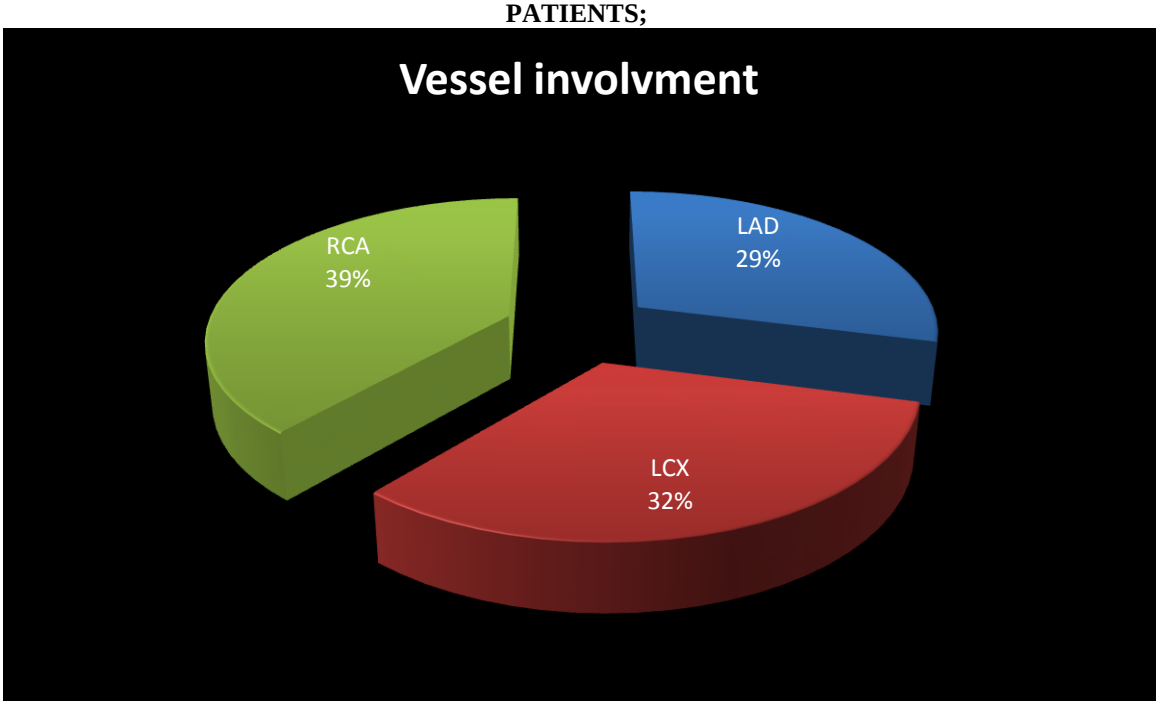


CHARECTERISTICS OF CORONARY ARTERY INVOLVEMENT IN GROUP1 PATIENTS:
Coronaries with stenosis between 50% -70% were seen in 3, 3 & 2 patients respectively in LAD, LCX & RCA territory.
Coronaries with stenosis of >70% were seen in 6, 7 & 10 patients respectively in LAD, LCX & RCA territory.

TABLE2-CHARECTERISTICS OF THE CORONARY ARTERY INVOLVEMENT IN GROUP1 PATIENTS;

VESSELS	SEVERITY	Group1
LAD	Normal	5
	Stenosis (50-69%)	3
	Stenosis (≥70%)	6
LCX	Normal	4
	Stenosis (50-69%)	3
	Stenosis (≥70%)	7
RCA	Normal	2
	Stenosis (50-69%)	2
	Stenosis (≥70%)	10

FIGURE2-CHARECTERISTICS OF THE CORONARY VESSELS INVOLVEMENT IN GROUP1 PATIENTS;



Dobutamine stress echocardiography was done in a single patient to know the improvement in cardiac functional reserve after dobutamine infusion it was showing improvement in the EF from 25% to 37%.
Thallium scan was done in a single patient from PGI Chandigarh to know the myocardial viability & was showing nonviable myocardium.

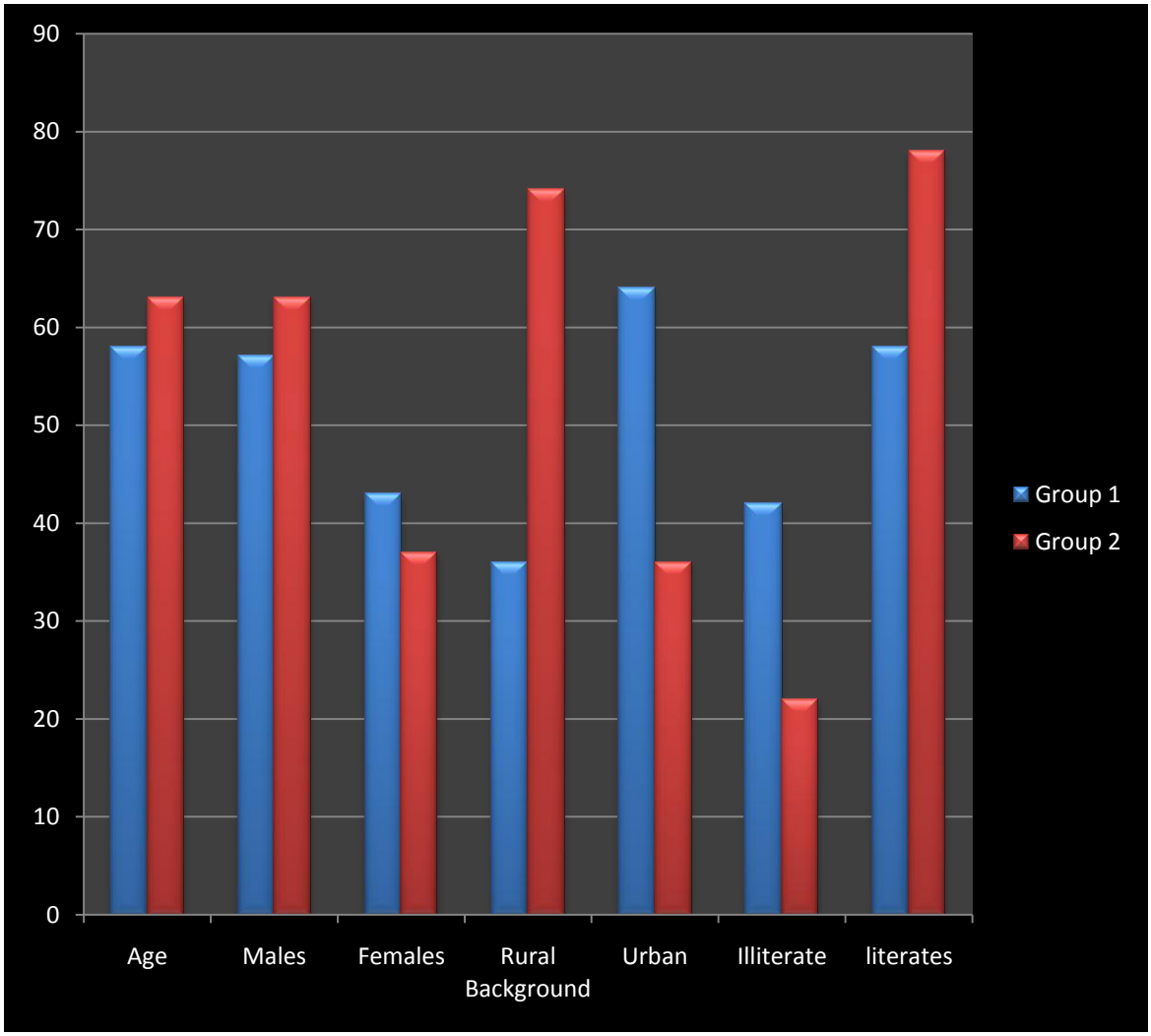
DISTRIBUTION OF VARIABLES IN GROUP1 & GROUP2 PATIENTS;

Demographic characteristics; The mean age of the group 1 & group 2 study subjects were 57.9 vs. 62.8 respectively & the difference in the mean age between group 1 & group 2 study subjects were statistically insignificant (p=0.158). The gender distribution in group 1 & group 2 study groups are,57.1% &42.9% were males & 63.1% & 36.9% were females respectively & the difference was statistically insignificant (p=.727).The 64.2% of subjects in the group1 were from urban background and where as in group2, 73.6% of the patients are from rural area & the difference was statistically significant (p=.029). Both group 1 & group 2 study subjects were illiterate 42% vs. 21.9% & the difference in distribution of illiteracy status was statistically insignificant (p=0.178).

TABLE 3: COMPARISON OF DISTRIBUTION OF DEMOGRAPHIC CHARACTERISTICS IN GROUP1 & GROUP2:

Demographic Features	Group1	Group2	2 sided sig. p value
Age	57.9+9.1	62.8+9.9	0.158
Males	57%	63%	o.727
Females	43%	37%	0.727
Rural Background	35.8%	73.8%	.029
Urban	64.2%	36.2%	.029
Illiterate	42%	22%	.178
Literate	58%	78%	.178

FIGURE 3: COMPARISON OF DISTRIBUTION OF DEMOGRAPHIC CHARACTERISTICS IN GROUP1 & GROUP2:



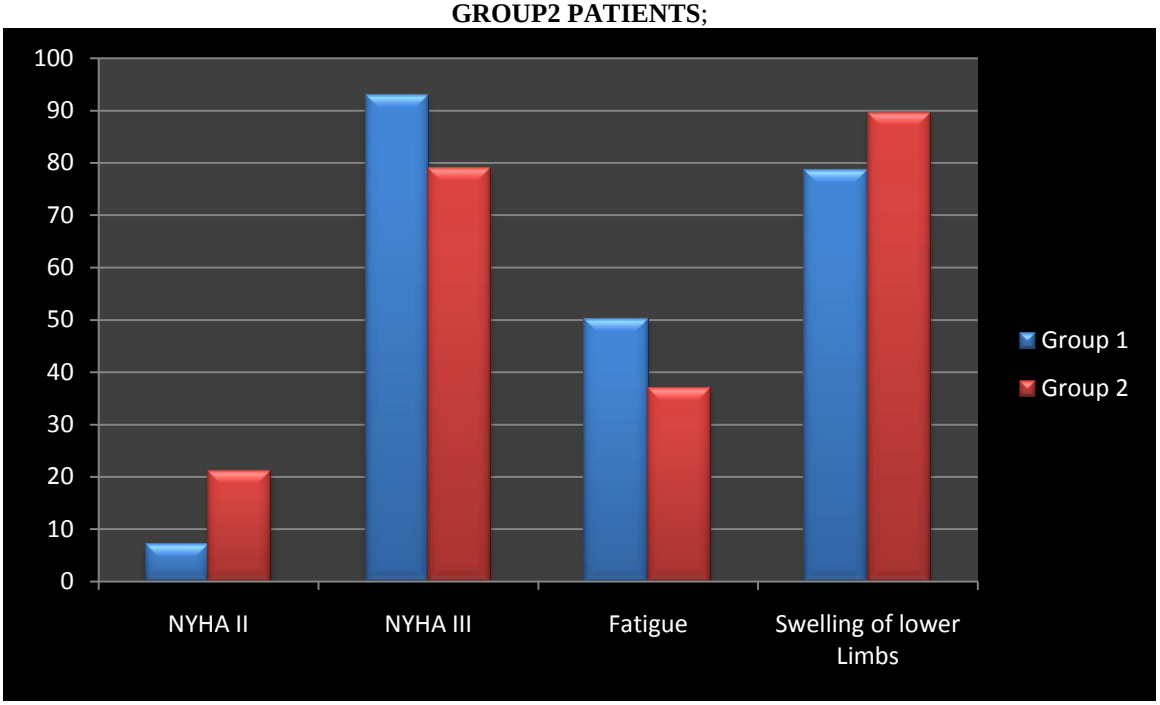
FREQUENCY & DISTRIBUTION OF CLINICAL PRESENTATION IN GROUP1 & GROUP2 PATIENTS;

None of the patients were in NYHA class I and IV. 7.2% of group 1 & 21% of group 2 were in NYHA functional class II while 92.8% of the group1 & 78.9% of the group2 were NYHA class III. The differences in the distribution of NYHA functional class between males & female subjects were statistically insignificant (p=.271). 50% of group1 & 36.8% of group2 had symptoms of fatigue however the difference was statistically insignificant (p=0.317). 78.5% & 89.5% of the group1 & group2 were having swelling of lower limbs respectively but the difference was statistically insignificant (p=.388). None of the group1 & group2 patients had symptoms of syncope & postural syncope.

TABLE 4: COMPARISON OF DISTRIBUTION OF CLINICAL PRESENTATIONS IN GROUP1 & GROUP2 PATIENTS;

Presentation	Group1	Group2	Sig. 2tailed
Breathlessness NYHA Functional Class;			
Class II	7.2%	21.1%	.271
Class III	92.8%	78.9%	.271
Fatigue	50%	36.8%	.317
Swelling of lower limbs	78.5%	89.5%	.388

FIGURE4; FREQUENCY & DISTRIBUTION OF CLINICAL PRESENTATION IN GROUP1 & GROUP2 PATIENTS;



RISK FACTOR DISTRIBUTION IN GROUP1 & GROUP2 SUBJECTS;

Obesity; Overall 78.8% of the patients are having BMI>25, 78.5% in group1 & 78.9% in group 2 patients & the difference in the two groups were statistically insignificant (p=0.971)**.**

Hypertension; 64.2% of the group1 subjects & 63.1% of the group2 subjects were hypertensive but the difference in the frequency distribution of hypertension in the group1 & 2 subjects were statistically insignificant (p=0.947).

Diabetes; 50% of the group1 patients & 5.2% of the group2 were diabetic & the difference in the frequency distribution of diabetes in the group1 & group2 subjects were statistically significant (p=0.003).

Alcohol consumption; 14.2% of the group1 subjects had the history of alcohol consumption while 31.5% in group 2 were consuming alcohol. The difference in the alcohol consumption in the two groups were statistically insignificant (p=0.252).

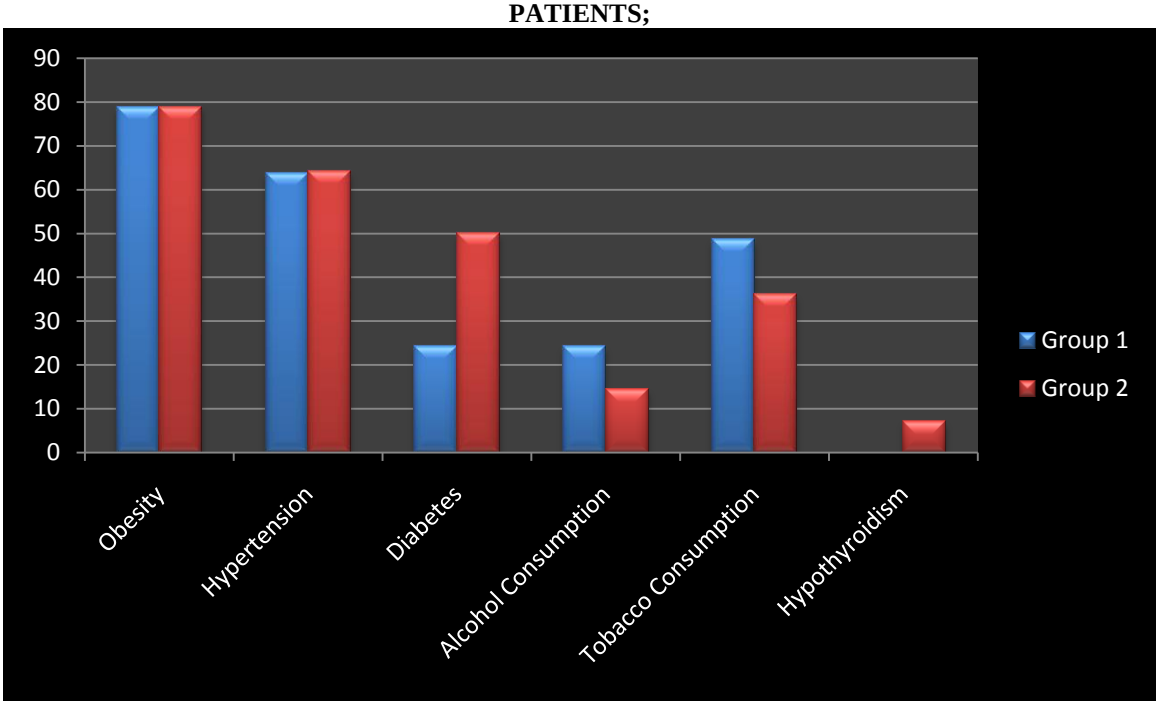
Tobacco consumption; 35.7% of the group1 subjects had the history of tobacco smoking while 57.8% in group2 were smoking tobacco. The difference in the tobacco consumption in the two groups were statistically insignificant (p=.208).

Hypothyroidism; Overall 3.1% of the patients had history of hypothyroidism, none of the group2 patients and 7.1% of the group1 patients had history of hypothyroidism & differences in the two groups were statistically insignificant (p=.237).

TABLE 5: COMPARISON OF RISK FACTOR PROFILE OF SUBJECTS IN GROUP1 & GROUP2 PATIENTS;

Risk factors	Overall	Group1	Group2	Sig.2 tailed
Obesity	78.8%	78.9%	78.5%	.971
Hypertension	63.6%	64.2%	63.1%	.947
Diabetes	24.2%	50%	5.2%	.003
Alcohol Consumption	24.2%	14.3%	31.5%	.252
Tobacco consumption	48.5%	36%	57.9%	.208
Hypothyroidism	3.1%	7.1%	0.00%	.237

FIGURE 5: COMPARISON OF RISK FACTOR PROFILE OF SUBJECTS IN GROUP1 & GROUP2 PATIENTS;



HEMODYNAMIC CHARACTERISTICS IN GROUP1 & GROUP2 SUBJECTS;

BP; The mean SBP & DBP in the group1 & group2 subjects were 131.1 & 79.4 vs. 142 & 87 mmHg respectively & the difference in mean SBP & DBP in two groups were statistically insignificant(p=.237 & .123).

FEATURES OF SYSTEMIC VENOUS HYPERTENSION;

50% of the group1 & 68.4% of group 2 were having evidence of elevated JVP but the difference was statistically insignificant(p=.284).

78.5% & 89.5% of the group1 & group 2 subjects were having dependent oedema respectively but the difference was statistically insignificant (p=.388).

Tender hepatomegaly was observed in the group 1 & group2 subjects ,7.1% & 10.5% respectively & the difference was statistically insignificant (p=.738).

FEATURES OF PULMONARY CONGESTION;

35.7% & 26.3% of the group1 & group 2 subjects respectively had cyanosis & difference was statistically insignificant (p=.561). LVS3 was observed in 78.5% & 36.8% respectively in the group1 & group2 subjects & the differences in the two groups were statistically significant (p=.017). Basal rales were observed in 64.3% vs. 57.9% respectively in the group1 & group2 subjects & difference was statistically insignificant(P=.710).

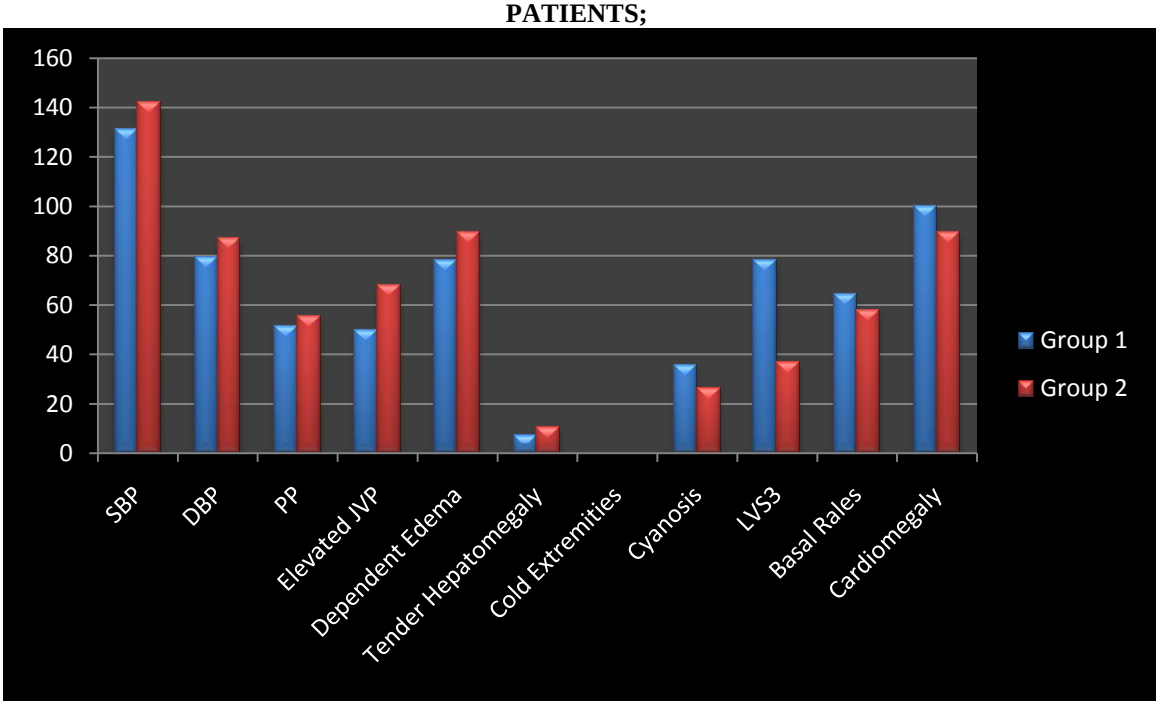
Cardiomegaly; Cardiomegaly was observed in 100% & 89.4% respectively in the group1 & group2 subjects & the differences in the two groups were statistically insignificant (p=.210).

FEATURES OF SYSTEMIC HYPOPERFUSION; None of the patients had evidence of systemic hypoperfusion in the form of cold extremities.

TABLE 6: COMPARISON OF HEMODYNAMIC CHARACTERISTICS IN GROUP1 & GROUP2 PATIENTS;

Hemodynamic Features	Group1	Group2	2 sided sig. p value
SBP	131.1+20.1	142+28.9	.237
DBP	79.4+10.4	87+15.5	.123
PP	51.5+13.1	55.2+16	.487
Elevated JVP	50%	68.4%	.284
Dependent Edema	78.5%	89.5%	.388
Tender Hepatomegaly	7.1%	10.5%	.738
Cold Extremities	0%	0%	1.00
Cyanosis	35.7%	26.3%	.561
LVS3	78.5%	36.8%	.017
Basal Rales	64.3%	57.9%	.710
Cardiomegaly	100%	89.4%	.210

FIGURE 6: COMPARISON OF HEMODYNAMIC CHARACTERISTICS IN GROUP1 & GROUP2 PATIENTS;



BIOCHEMICAL CHARACTERISTICS IN THE GROUP1 GROUP2 PATIENTS;

Hb; The mean HB levels in the group1 & group2 subjects were 11.8 gm/dl & 12.4 gm/dl but the difference was statistically insignificant(p=.247).

FBS; Mean FBS in the group1 & group2 subjects were 145.7gm/dl & 98.4 gm/dl & the differences in the two groups was statistically significant (p=.04)

Renal Function; BU & Serum Creatinine levels in the group1 & group2 subjects were 46.8 & 44, & 1.1 & 1.11 mg/dl respectively & the differences in the mean values were statistically insignificant(p=.557&.970).

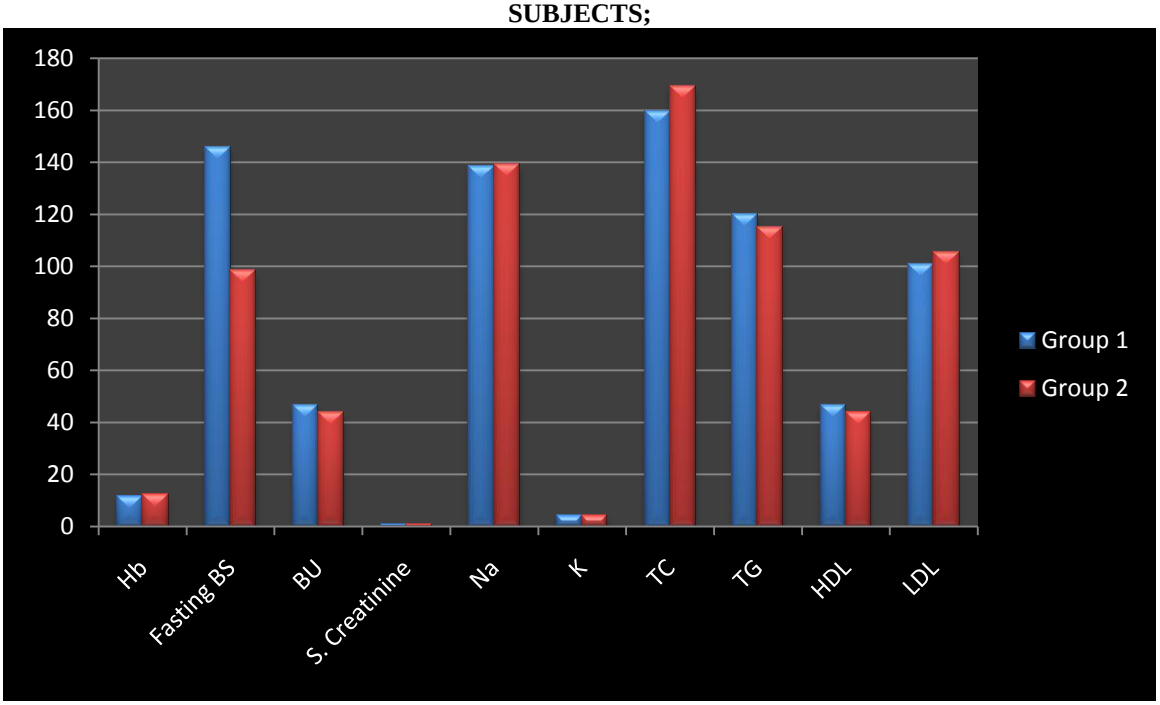
Electrolytes; the mean Na & K levels in the group1 & group2 subjects were 138.1 & 4.2, & 139.3 & 4.9 meq/L respectively & differences were statistically insignificant(p=.577).

LIPID PROFILE; The mean level of TC in the group1 & group2 subjects were 159.7 & 169.4 mg/dl respectively & the differences in the mean values were statistically insignificant(p=.557&.970), while TG was 120 & 115.2mg/dl & the differences in the mean values were statistically insignificant(p=790). The mean HDL was 46.5 & 43.7mg/dl and LDL was 100.8 & 105.4 mg/dl in the group1 & group2 subjects respectively& both of these values were statistically insignificant(p=.228 &.552).

TABLE 7: COMPARISON OF BIOCHEMICAL CHARACTERISTICS IN GROUP1 & GROUP 2 SUBJECTS;

Biochemical Parameter	Group1	Group2	Sig. 2 tailed
Hb	11.8+1.4	12.4+1.4	.247
Fasting BS	145.7+60.2	98.4+23	0.04
BU	46.8+16.8	44+10.8	.557
S. Creatinine	1.1+0.24	1.1+0.25	.970
Na	138.1+3.8	139.3+4.9	.446
K	4.2+0.41	4.21+0.38	.975
TC	159.7+42.9	169.4+52.8	.577
TG	120+56.8	115.2+47.1	.790
HDL	46.5+8.2	43.7+4.9	.228
LDL	100.8+23.7	105.4+20.3	.552

FIGURE7: COMPARISON OF BIOCHEMICAL CHARACTERISTICS IN GROUP1 & GROUP 2 SUBJECTS;



ELECTROCARDIOGRAPHIC CHARACTERISTICS IN GROUP1 & GROUP2 SUBJECTS;

Rhythm; A. Fib. was observed in 21% of group2 & none of the patients in group1 subjects & difference in the prevalence of A. Fib in two groups were statistically insignificant (p=.067).

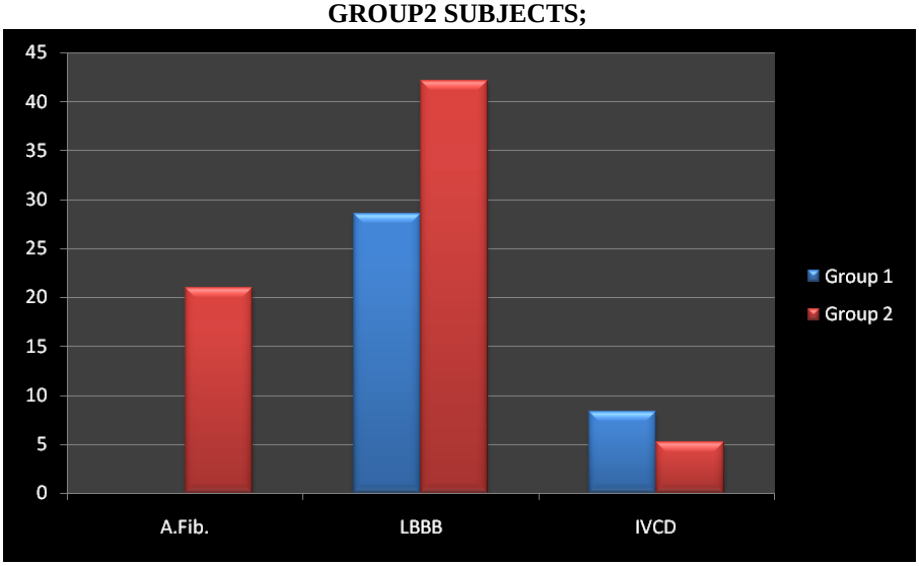
AV Block; None of the group1 & group2 subjects had AV Block, while 8.3% and 5.2% of group1 & group2 subjects respectively were found to have IVCD & difference in the prevalence of IVCD in two groups were statistically insignificant (p=.618).

BBB; 28.5% & 42.1% of the group1 & group2 subjects had LBBB respectively & difference in the prevalence of LBBB in two groups were statistically insignificant (p=.963).while none of the patients had RBBB.

TABLE 8: COMPARISON OF ELECTROCARDIOGRAPHIC CHARACTERISTICS IN GROUP1 & GROUP2 SUBJECTS;

ECG Features	Group1	Group2	Sig. (2-tailed)
A. Fib.	0%	21%	.067
LBBB	28.5%	42.1%	.963
IVCD	8.3%	5.2%	.618

FIGURE 8: COMPARISON OF ELECTROCARDIOGRAPHIC CHARACTERISTICS IN GROUP1 & GROUP2 SUBJECTS;



ECHOCARDIOGRAPHIC MEASURES OF LV STRUCTURE & FUNCTIONS IN GROUP1 & GROUP2 SUBJECTS;

LV mass; The mean LV mass in both group1 & group2 subjects is 193.4gm & the difference was statistically insignificant (p=.999)

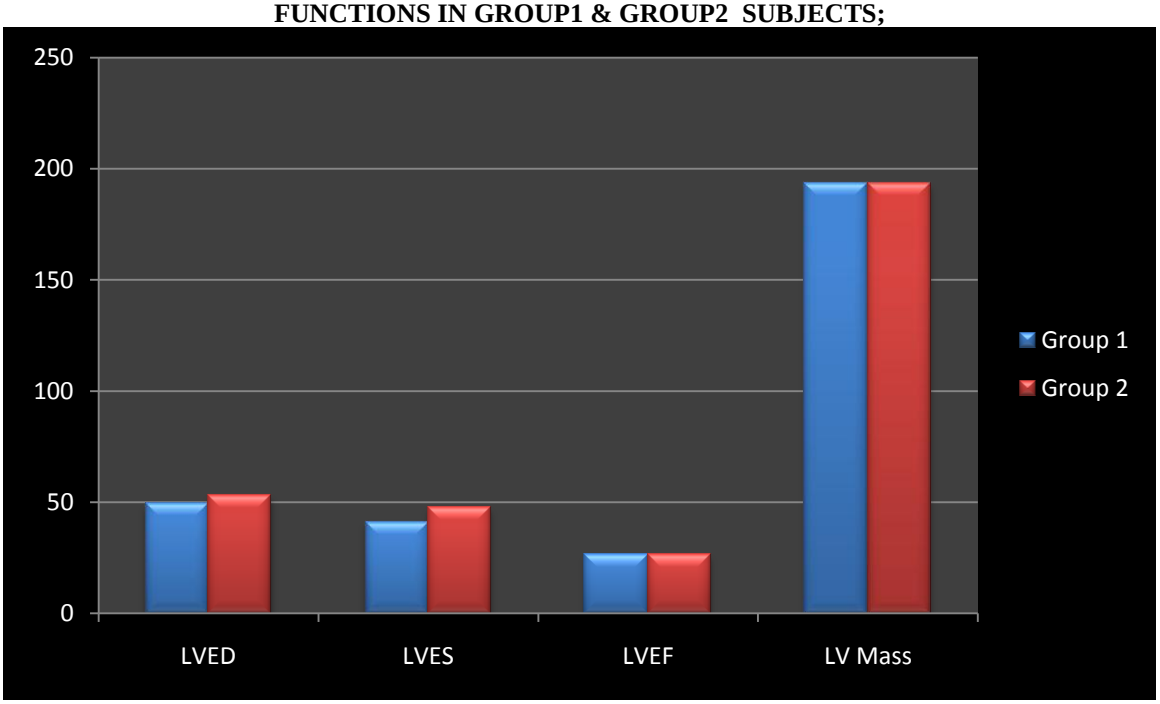
LVEF; The mean LVEF in group1 & group2 subjects were 26.5% vs. 26.2% respectively & the difference was statistically insignificant (p=.882)

LV dimensions; The mean LVED in group1 & group2 subjects were 49.2 & 53.01mm respectively & the difference was statistically significant (p=.03). The mean LVES in group1 & group2 subjects were 41.07 & 47.7mm respectively & the difference was statistically insignificant (p=.22).

TABLE 9: COMPARISON OF ECHOCARDIOGRAPHIC MEASURES OF LV STRUCTURE & FUNCTIONS IN GROUP1 & GROUP2 SUBJECTS;

Echocardiographic features	Group1	Group2	2 sided sig. p value
LVED	49.2+5.35	53.01+4.2	0.03
LVES	41.07+6.6	47.7+5.6	.22
LVEF	26.5+4.0	26.2+6.3	.882
LV Mass	193.4+11.6	193.4+14.3	.999

FIGURE 9: COMPARISON OF ECHOCARDIOGRAPHIC MEASURES OF LV STRUCTURE & FUNCTIONS IN GROUP1 & GROUP2 SUBJECTS;



VI. Discussion:

Present study was an attempt to find out the incidence of CAD in patients with severe LV Dysfunction without any historical, ECG or ECHO evidence of CAD with EF<35%, seen in tertiary care centre over a period of one year. Out of the 33 subjects which were subjected to coronary angiography, 14(42.4%) (group1) patients were found to be having significant CAD & rest of the 19(57.6%) (group2) subjects were having normal coronaries. Out of 14 group1 patients, 4(28.57%), 3(21.43%) & 7(50%) had SVD, DVD & TVD respectively. LMCA was not involved in any of the patients.

Out of 31 vessels involved, LAD in 9 patients, LCX in 10 patients & RCA was involved in 12 patients. Coronaries with stenosis between 50% -70% were seen in 3, 3 & 2 patients respectively in LAD, LCX & RCA territory.Coronaries with stenosis of >70% were seen in 6, 7 & 10 patients respectively in LAD, LCX & RCA territory.

These results are consistent with a study from IGMC dept. Of Cardiology by R.Bhardwaj et al (15) showing approximately 38% of the patients with severe LVD were found to have CAD & nearly 22% had more than DVD. Extent of CAD in these patients was also significant as is evident from the fact that 26 vessels were involved in 12 patients with CAD, with 32 lesions.20 of these showed critical stenosis. Clinical Determinants of Mortality in Patients With Angiographically

Diagnosed Ischemic or Non ischemic Cardiomyopathy study(58) shows, The median age, ejection fraction and proportion of patients with New York Heart Association functional class III or IV symptoms for the nonischemic and ischemic groups were 55 years versus 63 years, 27% versus 32% and 57% versus 25%, respectively.

Ischemic cardiomyopathy is a term used to describe patients whose heart can no longer pump enough blood to the rest of their body, as a result of a chronic lack of oxygen, due to coronary artery disease. Coronary artery disease is a narrowing of the small blood vessels that supply blood and oxygen to the heart(8).

An effort to provide a standardized definition of ischemic cardiomyopathy was made in a review of 1921 patients with symptomatic heart failure (HF) and a left ventricular ejection fraction (LVEF) <40 percent who underwent coronary angiography(60).Ischemic cardiomyopathy was considered to be present in patients with HF who have had a myocardial infarction (MI) or have evidence of hibernating myocardium or, on angiography, severe coronary disease. Such patients had a worse outcome than those with non ischemic cardiomyopathy. In contrast, patients with single vessel disease who had no history of myocardial infarction or revascularization had a similar prognosis as those with non ischemic cardiomyopathy. It was suggested that such patients should be classified as non ischemic cardiomyopathy, at least for prognostic purposes(8). Left Ventricular Dysfunction[LVD] is an important indicator of morbidity & mortality in cardiac patients. Coronary artery disease [CAD] is an important cause of LVD in adults, which may present as myocardial infarction, unstable angina or chronic stable angina. Another important cause of LVD is idiopathic dilated cardiomyopathy [DCM].

Some patients of CAD may not present with chest pain but with LVD only. LVD in them could be due to dysfunctional but viable myocardium, either as a result of chronic flow impairment [hibernating myocardium] or following episodes of ischemia, when LVD persists for a period of time after restoration of blood flow, duration depending upon the period of ischemic insult [stunned myocardium]. Myocardial Stunning is defined as post ischemic myocardial dysfunction that persists despite restoration of normal blood flow. Hibernation is a state in which some segments of the myocardium exhibit abnormalities of contractile function at rest (12). This phenomenon is highly significant clinically because it usually manifests itself in the setting of chronic ischemia , that is potentially reversible by revascularization. The reduced coronary blood flow causes the myocytes to enter a low-energy 'sleep mode' to conserve energy.

Left ventricular (LV) function is one of the most important determinants of long-term outcome in patients with coronary artery disease (CAD). Patients with normal or near-normal LV function have an excellent prognosis, whereas patients with impaired LV function are at substantial risk of death during medical therapy. It is now apparent that LV dysfunction is not always an irreversible process and that LV function may improve considerably, and even normalize, after myocardial revascularization in a large subset of patients. The identification of such patients with "hibernating" myocardium that is under perfused and dysfunctional, yet viable, has important implications in the selection of patients with LV dysfunction for revascularization procedures(10).

After adjustment for baseline clinical risk factors and presenting characteristics, ischemic etiology remained an important independent predictor of 5-year mortality ($p < 0.0001$). The extent of coronary artery disease was a better predictor of survival than ischemic or non ischemic etiology (log likelihood chi-square 700 vs. 675, respectively).

Seventy-five percent of patients in the ischemic cohort had significant stenosis in the left anterior descending coronary artery; 46% had three-vessel CAD.

Non-ischemic systolic dysfunction was a clinical diagnosis based on absence of documented MI, history of angina as a main symptom & absence of regional wall motion abnormality with LV global EF of less than 40% on echocardiographic evaluation with typical signs & symptoms of heart failure. 33 patients enrolled over a period of one year in present study were severely symptomatic & all were in NYHA class II & III.

LV systolic function was severely depressed with mean LVEF of 26.3±5.4% & LV dimensions were significantly elevated with mean LVED of 51.5±5.1 mm & mean LVES of 42.6±6.1 mm.

The mean age of the study subjects was 59.8±9.7 years & 60.4% of the subjects were male. The analysis of frequency & distribution of systolic heart failure with age revealed prevalence increases with increasing age. Prevalence was 15.4% in the age group of <40 years, 38.4% in the age group of 40-60 years & 46.2% in the age group of >60 years thus substantiating the observations made in other hospital based registries & population based cross sectional studies(16, 17). In the Rotterdam Study(18), participants aged ≥55 were included. Prevalence was higher in men and increased with age from 0.9% in subjects aged 55–64 to 17.4% in those aged ≥85. In the study by Walter P Abhayaratna et al in older Australians(19), the mean age was 69.4 years, 50% were men, the frequency of heart failure increased with advancing age, with a 4.4-fold increase from the 60– 64-years age group to the 80–86-years age group. G.Taubert et al(20) did clinical profile in rural and urbanhospital patients, found that mean age was 66±11 years, 74% males & >72% were older than 65 years. Camille G. Frazier et al analyzed pooled data from randomized in 5 clinical trials(5), study population included

76% men, mean age was 58.3±12.5 years, mean LVEF was 23%, and 85% had NYHA functional class III or IV symptoms at enrolment. NA Yadav, K Raghu, LSR Krishna, V Gouthami et al(21) Tertiary Care Centre Study in India, showed that mean age of the patients with dilated cardiomyopathy was 50.3 years. Sex incidence showed a male preponderance of 68%. A study by Docherla .M et al(22) at Kasturba Hospital, Manipal, India in elderly; the average was 68.9±6 years, 60.63% males and 32% were in age group >70 years. Though HF is predominantly a disease of elderly with male preponderance, nonischemic heart failure has been found to be more common in the young with a female preponderance (14).

Interestingly majority of the study subjects (69.7%) were literate & 48.5% were tobacco consumers & 57.6% were from rural population. This association could be by chance factor rather than having any causal association since about 90% of the population in HP resides in rural area & prevalence of tobacco consumption is more common in rural & illiterate community. In the study by Elizabeth O. Ofili et al [23] tobacco use was seen in 42% men vs 18% women (P<0.0001).

Analysis of clinical presentation of study subjects revealed that breathlessness was the commonest presenting symptoms observed in all patients & was of NYHA class II & III. None of the patients had class I symptoms thus indicating that study population of the present study comprised of sick patients in advanced stage of heart failure, as the study was done in tertiary care institution, where more sick patients are coming for treatment. None of the patients had Symptoms of postural presyncope & syncope. In the population study by Cowie et al (16) 7% were in class I, 25% in class III and 68% in class IV. Analysis by G. Taubert et al(20) in patients admitted for heart failure in rural community hospital, 69% patients were in NYHA functional class III, which is in concordance with the our study as most of the patients from rural area. In the study by Nasim Afsarmanesh et al[24] 68% patients in the first group (TC < 133 mg %) were in NYHA class IV while only 28% in the group 2 (TC> 210 mg %) were in NYHA class IV. Camille G. Frazier et al(5) analyzed that 85% had NYHA functional class III or IV symptoms at enrolment. In the study by NA Yadav et al(21) at a Tertiary Care Centre Study in India, the symptoms were dyspnoea in 100% of patients, orthopnea in 92.5%; paroxysmal nocturnal dyspnoea in 55% and syncope/presyncope in 7.5%.

Distribution of clinical features of systemic venous hypertension revealed tender hepatomegaly, elevated JVP & dependent edema occurred in order of frequency 9.1%, 60.6% & 84.8% respectively. The prevalence of features of pulmonary congestion; basal rales were observed in 60.6% & cyanosis in 30.3%. In the Spanish heart failure registry, the clinical features were pulmonary rales (76.5%), neck vein distension (45.5%), hepatomegaly (23.3%), Cardiomegaly (79.4%), LVS3 (13.2%), pleural effusion (19.2%) and pulmonary edema (12.1%). Features of systemic hypoperfusion; was not observed in any of the patients. NA Yadav et al (21) Tertiary Care Centre Study at Hyderabad, India peripheral oedema was present in 77.5%, other physical signs were elevated JVP, ascites, congestive hepatomegaly, S3, and cardiomegaly.A study by Docherla M et al(22) India, hepatomegaly was present in 17.2%, elevated JVP in 10.3% & dependent edema in 34.5% of systolic heart failure, the variation can explained as this was done in elderly patients age >60 years.

Evaluation of ECG features revealed mean HR was 99.6 beats/minute, 12.1% were in Atrial Fibrillation. None of the patients had CHB requiring Pacemaker, 6.25 had evidence IVCD, 30.3% had LBBB & none had RBBB. In the study by Wanwarang Wongchareon MD et al(25) in 137 randomly assigned patients with LV systolic dysfunction the frequency of LBBB was 14.3% and RBBB was 8.2% in non-ischemic patients. In a study by Holly R. Middlekauff [26] to evaluate the prognostic significance of AF in heart failure, Seventy-five patients (19%) had paroxysmal (26 patients) or chronic (49 patients) atrial fibrillation. In the CIBIS – II study(27), LBBB was present in 25% men and 31% women with HF; RBBB in 6% and 4% respectively. 9% men and 6% women had evidence of atrioventricular block. AF was present in 20% and 17% respectively. HF in women was more commonly due to nonischemic etiology (60% vs 48%, p=0.001). Havranek [28] LBBB was present in 16.5% of patients and the frequency of AF increased with age being 23.3% in those between 65 – 69 years and 36.6 in those above > 85 years.

In a study by Frangiskos I. Parthenakis et al [29] to study the relationship between AF and HF, 27.5% had chronic AF. Beatrice Brembilla et al(30) evaluated the prevalence and the clinical significance of LBBB or RBBB in patients with non-ischemic dilated cardiomyopathy. 25% had LBBB, 7% had RBBB, and 68% had no BBB, atrial fibrillation was present in 20% patients, Incidence of spontaneous ventricular tachycardia was 23.2%. In the study by NA Yadav et al 9.6% had A. Fib; 1.6% had CHB; 23.2% had LBBB & 10% had RBBB.

Analysis of frequency distribution of risk factors in the study population revealed 63.6% were hypertensive based on self report & recording of elevated BP at enrolment. The prevalence of hypertension in general population is about 20-30% thus indicating that hypertension apparently has causal association both severe LVD with CAD & without CAD in the present study population. However it is important to appreciate the fact that awareness of hypertension is lower especially in illiterate & rural population as most of the hypertensives are not symptomatic, lack of awareness & access to health care facilities & since the present study population is comprised of advanced stage of heart failure where the measured BP could have been underestimated due to phenomenon of decapitation thus the prevalence of hypertension recorded in this study

may not represent the true prevalence of hypertension. Interestingly 8 study subjects were found to be diabetic; out of that 7 patients were in ICM group this could be due to demographic profile of the study population characterized by presence of obesity in 78.8%, mean BMI was 26.67 only 5 patients had BMI of <23, illiterates from rural background representing poor socioeconomic strata & also number of patients enrolled are small. Thus the findings of present study undermine the importance of diabetes as the risk factor for heart failure especially with severe LVD with CAD(P=0.03). 24.2% of the patients had history of alcohol consumption. However 48.5% were consuming tobacco, the higher prevalence of tobacco consumption observed could be due to demographic characteristics of the study population. The association of tobacco as risk factor for systolic heart failure with or without CAD has not been documented in cohort studies. The EPICAL study a registry based hospital study, had contributing risk factors among non-coronary heart disease, tobacco use in 51,1%, history of diabetes mellitus in 19.8%, history of hypertension in 42.9% and alcohol abuse in 25.7% of patients. Of the dilated cardiomyopathy group, 31.5% of the total cohort had at least one concomitant predisposing or contributing risk factor: alcohol abuse (37.6%), arterial hypertension (61.8%) and/or other rare conditions (20.4%). Michael Felker M.D., et al(31) analysed underlying cause of unexplained cardiomyopathy over 15 years found myocarditis 9%, valvular heart disease 1.5%, thyroid dysfunction 0.5%, and Peripartum cardiomyopathy was 4%. None had history of exposure to Anthracyclins even on such a long follow up. Camille G. Frazier et al(5) did a pooled analysis had prevalence of diabetes mellitus in 32% males, 35% females, tobacco use in 77% males and 52% females, mean BMI was 26.8±4.4 in male, 27.7±6.7 female patients among ischemic & non-ischemic systolic heart failure. Richard K. Cheng et al did study over 14 years in heart failure patients , of which 974 were non-ischemic systolic heart failure 21% had diabetes, 35% had hypertension, 52% had history of tobacco use. Wanwarang Wongchareon MD et al(25) did a study on randomly assigned 137 patients with LV systolic dysfunction had prevelance of diabetes mellitus 16.3%, hypertension 22.4%, and smoking 30.6% in non-ischemic patients. In the study by NA Yadav et al(21) 10% were diabetics, alcoholic consumption was present in 16%, and peripartum cardiomyopathy was implicated in 2%. Viral myocarditis has been postulated to be an antecedent event in substantial proportion of patients with DCM. However none of the patients in the present study were found to have history suggestive of viral prodrome.

Other risk factors that were assessed in the present study were evidence of thyroid dysfunction, and history of exposure to Anthracyclines & Peripartum cardiomyopathy in female patients. 3.1% of the patients were found to have evidence of hypothyroidism while none of the patients had history of exposure to Anthracyclines & Peripartum cardiomyopathy.

Distribution characteristics of demographics amongst patients with severe LVD with or without CAD study population revealed there was no difference in the mean age of male & female study population unlike observations made by Camille G. Frazier et al(5) where the mean age for female patients was 60.3±12.5 years and mean age in male was 58.3±12.5 years among non-ischemic heart failure patients and also in REACH study(17), mean age in male & female patients 69.2±13.6 and 73.7±13.3 years respectively. Similarly in Olmsted County study(32) had mean age in male & female patients 73±12 and 79±11 years respectively. The education status was not statistically different amongst patients with severe LVD with or without CAD. But the rural/urban background was statistically significant as more LVD with CAD patients were from urban background(p=0.029). Comparison of distribution of clinical presentations amongst both groups were not different.

Frequency & distribution of various risk factors amongst both study population revealed no significant difference except Diabetes. This significant difference is due to the fact that prevalence of Diabetes is very high in ICM group especially with TVD. Comparison of cardiac structure & systolic function assessed on echocardiography did not reveal any significant differences in the distribution of LV mass, LV dimensions & systolic function index in the gender groups.

The distribution of characteristics of signs of systemic, pulmonary congestion, systemic hypoperfusion & BP were not significantly different amongst both study population except LVS3 which was most frequently seen with severe LVD with CAD(p=0.017). Comparison of fasting Blood sugar levels revealed it was significantly higher in severe LVD with CAD population than without CAD patients 145.7 vs. 98.4gm/dl (p=0.04). The differences in mean distribution of measures of renal functions BU & Creatinine & electrolytes were statistically insignificant.

VII. Conclusion:

In the present study **"Profile Of Coronary Artery Disease In Adults With Severe Left Ventricular Systolic Dysfunction: A Hospital Based Study".** Severe LVD with CAD & without CAD, clinical and demographic profile of age, sex and causes were in accordance with other studies in India and abroad. This was the second study in India on the profile of coronary artery disease in adults with severe LV systolic dysfunction.

The mean age was 59.84±9.76years, male patients were 60.4% and female patients were 39.4%. The most of the patients enrolled were in NYHA class II & III . Mean LVEF was 26.3±5.4% . 12.1% were having

atrial fibrillation, 30.3% were having LBBB, 6.2% had IVCD & none of the patients had RBBB or CHB. Out of the 33 patients 14 were found to be ischemic & approximately 71.4% were having more than double vessel disease. Diabetes was the most common risk factor in severe LVD with CAD & it was present in 50% of the patients. Among the patients of severe LVD without CAD (57.4%), 30.3% patients were having Hypertensive Heart Disease, 21.2% were having Idiopathic Dilated Cardiomyopathy followed by Alcoholic cardiomyopathy in 6.2% of the patients. Out of the 33 patients one patient had evidence of hypothyroidism in the ischemic group. In patients with severe LV dysfunction with CAD group(42.4%), TVD was the most common etiology constituting about 50%, followed by SVD in 28.5% and DVD in 21.4% of the patients. Nearly 71.4% patients had more than two vessel disease. Extent of CAD in these patients was also significant as is evident from the fact that 31 vessels were involved in 14 patients with CAD, with 31 lesions,23(15 shows stenosis of >70% and 8 vessels showed complete cut off) of these showed critical stenosis (>70%). Most common vessel involved is RCA in 38.7%(12) followed by LCX in 32.2%(10) & LAD in 29.1%(9) in the severe LVD with CAD group. Thus LVD in at least some of these patients could be due to hibernating myocardium and could improve with revascularisation.

References:

[1]. R C Davis, F D R Hobbs, G Y H Lip et al. ABC of heart failure, History and Epidemiology. BMJ 2000; 320:39-42.

[2]. Michael M. Givertz, Wilson S. Colucci, Eugene Braunwald. Clinical Aspects of Heart Failure, in Braunwald's Heart Disease: A Text Book of Cardiovascular Medicine, 7th Edition. Zipes DP, Libby P Bonow RO, Braunwald E (eds), Philadelphia, Saunders, 2005:1.

[3]. Arend Mosterd, Arno W Hoes. Clinical Epidemiology of Heart Failure. Heart 2007; 93:1137-1146.

[4]. Douglas D. Schocken, Chair; Emelia J. Benjamin, Gregg C. Fonarow, et al. Prevention of Heart Failure. Circulation 2008; 117: 2544-2565.

[5]. Camille G. Frazier, Karen P. Alexander, L. Kristin Newby, Susan Anderson, Erik Iverson, Milton Packer, Jay Cohn, Sidney Goldstein, and Pamela S. Douglas. Associations of Gender and Etiology With Outcomes in Heart Failure With Systolic Dysfunction: A Pooled Analysis of 5 Randomized Control Trials. J Am Coll Cardiol 2007; 49:1450–8.

[6]. Burch GE, Giles TD, Colcolough HL; Ischemic cardiomyopathy;Am Heart J; 1970:79:291-292.

[7]. Eugene Braunwald. Heart failure and Cor Pulmonale, in Harrison's Principals of Internal Medicine: A Text Book, 17th Edition. Fauci, Braunwald, Kasper, Hauser, Longo, Loscalzo, 2008:1443-53.

[8]. Felker GM, Shaw LK, O'Connor CM;A standardized definition of ischemic cardiomyopathy for use in clinical research.J Am Coll Cardiol. 2002;39(2):210.

[9]. Mahrholdt H,Wagner A, Judd RM, Sechtem U, Kim RJ. Delayed enhancement cardiovascular magnetic resonance assessment of non-cardiomyopathies. Eur Heart J; 26:1461-1474.

[10]. Bonow RO ; The hibernating myocardium: implications for management of congestive heart failure;Am J Cardiol. 1995;75(3):17A.

[11]. MJ Likoff, SL Chandler and HR Kay, Clinical determinants of mortality in chronic congestive heart failure secondary to idiopathic dilated or to ischemic cardiomyopathy. *Am J Cardiol*, **59**(1987):634–638.

[12]. Bradley A Bart et al**;**Journal of the American College of Cardiology;30, 4, 1997:1002-1008.

[13]. Michael Felker, Linda K. Shaw, and Christopher M. O'Connor; A standardized definition of ischemic cardiomyopathy for use in clinical research ;J Am Coll Cardiol,2002; 39:210-218.

[14]. Follath F. Non-ischemic Heart Failure: Epidemiology, pathophysiology and progression of Disease. Journal of Cardiovascular Pharmacology. Suppliment 0016-9984. 1999; 33:S31-S35. 15. R Bhardwaj et al; Coronary artery disease in adults with severe LV dysfunction; Indian J Cardiol;8:20-22.

[15]. M. R. Cowie, D. A. Wood, A. J. S. Coats, S. G. Thomson, G. C. Sutton et al. Incidence and Aetiology of Heart Failure. European Heart Journal1999; 20:421-428.

[16]. Peter A. McCullough, Edward F. Philbin, John A. Spertus, Scott Kaatz, Keisha R. Sandberg, W. Douglas Weaver. Confirmation of a Heart Failure Epidemic: Findings from the Resource Utilization among Congestive Heart Failure (REACH) Study. JACC.2002; 39, (1):60–9

[17]. Gysele S. Bleuminka, Anneke M. Knetscha, Miriam C.J.M. Sturkenbooma, Sabine M.J.M. Strausa, Albert Hofmana, Jaap W. Deckersa et al. Quantifying the heart failure epidemic: prevalence, incidence rate, lifetime risk and prognosis of heart failure "The Rotterdam Study". European Heart Journal. 2004; 25:1614–1619.

[18]. Walter P Abhayaratna, Wayne T Smith, Niels G Becker, Thomas H Marwick, Ian M Jeffery and Darryl A McGill. Prevalence of heart failure and systolic ventricular dysfunction in older Australians: the Canberra Heart Study. MJA . 2006;184 (4):151-154

[19]. G. Taubert, C. Bergmeiera, H. Andresenb, J. Sengesa, J. Potratzb. Clinical profile and management of heart failure: rural community hospital vs. metropolitan heart center. European Journal of Heart Failure 2001; 611-617.

[20]. N A Yadav, K Raghu, LSR Krishna, V Gauthami, M Jyotsana, D Seshagiri Rao, S. Jaishankar. Clinical Profile of Dilated Cardiomyopathy: A Tertiary Care Centre Study.Indian Heart J 2002; 54:623-628.

[21]. Docherla. M, Hande Manjunath .H,Kavitha S,et al. Comparative Study of Systolic and Diastolic Cardiac Failure in Elderly Hospitalized Patients in a Tertiary Care Hospital in Southwest India. Indian Journal of Clinical and Diagnostic Research. 2009; 3:1529-1536.

[22]. Elizabeth O. Ofili, Robert Mayberry, Ernest Alema-Mensah, Stefanie Saleem, Kamran Hamirani, Christopher Jones, Sanah Salih, Brenda Lankford, Adefisayo Oduwole, and Priscilla Igho-Pemu. Gender Differences and Practice Implications of Risk Factors for Frequent Hospitalization for Heart Failure in an Urban Center Serving Predominantly African-American Patients. Am J Cardiol 1999; 83:1350–55.

[23]. Nasim Afsarmanesh, Tamara B. Horwich, and Gregg C. Fonarow. Total cholesterol levels and mortality risk in non-ischemic systolic heart failure. Am Heart J. 2006; 152:1077-83.

[24]. Wanwarang Wongchareon, Arintaya Phrommintikul, Rungsrit Kanjanavanit, Srun Kuanprasert, Apichard Sukonthasarn. A Predictive Model for Distinguishing Ischemic from Non-Ischemic Cardiomyopathy. J Med Assoc Thai 2005; 88 (11): 1689-96.

[25]. HR Middlekauff, WG Stevenson and LW Stevenson. Prognostic significance of atrial fibrillation in advanced heart failure. Circulation 1991;84;40-48.

[26]. Tabassome Simon, Murielle Mary-Krause, Christian Funck-Brentano and Patric Jaillon. Sex Differences in the Prognosis of Congestive Heart Failure:Results From the Cardiac Insufficiency Bisoprolol Study (CIBIS II).Circulation 2001;103:375-80.
[27]. Edward P. Havranek, MD, Frederick A. Masoudi, MSPH, Kelly A. Westfall, Pam Wolfe, Diana L. Ordin, and Harlan M. Krumholz. Spectrum of heart failure in older patients: Results from the National Heart Failure Project Am Heart J 2002; 143:412-7.
[28]. Frangiskos I. Parthenakis, Alexandros P. Patrianakos, Emmanuel I. Skalidis George F. Diakakis, Evangelos A. Zacharis, Gregory Chlouverakis, Ioannis K. Karalis, Panos E. Vardas Atrial fibrillation is associated with increased neurohumoral activation and reduced exercise tolerance in patients with non-ischemic dilated cardiomyopathy. International Journal of Cardiology 2007; 118: 206–214.
[29]. Beatrice Brembilla-Perrot, Francois Alla, Christine Suty-Selton, Olivier Huttin, Hugues Blangy, Nicolas Sadoul, Frederic Chometon et al. Nonischemic Dilated Cardiomyopathy: Results of Noninvasive and Invasive Evaluation in 310 Patients and Clinical Significance of Bundle Branch Block. PACE 2008; 31:1383–90.
[30]. G Michael Felker, M.D., Richard E. Thompson, Ph.D., Edward K. Kasper, M.D. et al. Underlying causes and long term Survival in Patients with Initially Unexplained Cardiomyopathy. N Eng J Med. 2000; 342,15,1077.
[31]. Veronique L. Roger, Susan A. Weston, Margaret M. Redfield, et al. Trends in Heart Failure Incidence and Survival in a Community-Based Population. JAMA. 2004;292(3):344-350.

ABBREVIATIONS

AHA: American Heart Association, AF: Atrial Fibrillation, AS: Aortic Stenosis, BP: Blood Pressure, BU: Blood Urea, CAD: Coronary Artery Disease, CAG: Coronary Angiography, CCU: Coronary Care Unit, CHB: Complete Heart Block, CHD: Coronary Heart Disease, CI: Confidence Interval, CT: Computerized Tomography, CVA: Cerebrovascular Accident, CVD: Cardiovascular Disease, DBP: Diastolic Blood Pressure, DCM: Dilated Cardiomyopathy, DM: Diabetes Mellitus, DVD: Double Vessel Disease, ECG: Electrocardiography, ECHO: Echocardiography, ECS: European Cardiology Society, ED: Emergency Department, EF: Ejection Fraction, ESC: European Society of Cardiologists, HF: Heart Failure, HFPEF: Heart Failure with Preserved Ejection Fraction, HHD: Hypertensive Heart Disease, HR: Heart Rate, ICM: Ischemic cardiomyopathy, IDCM: Idiopathic Dilated Cardiomyopathy, IHD: Ischemic Heart Disease, IV: Intra-venous, JVP: Jugular Venous Pressure, LA: Left Atrium, LAD: Left Anterior Descending Artery, LBBB: Left Bundle Branch Block, LCX: Left Circumflex Artery, LMCA: Left Main Coronary Artery, LVEF: Left Ventricular Ejection Fraction, LVF: Left Ventricular Failure, MI: Myocardial Infarction, MR: Mitral Regurgitation, NICM: Non-ischemic Cardiomyopathy, NIHF: Non-ischemic Heart Failure, NYHA: New York Heart Association, PP: Pulse Pressure, PSVT: Paroxysmal Supra-ventricular Tachycardia, PTCA: Percutaneous Transluminal Coronary Angioplasty, RBBB: Right Bundle Branch Block, RCA: Right Coronary Artery, RF: Rheumatic Fever, RHD: Rheumatic Heart Disease, RV: Right Ventricle, RWMA: Regional Wall Motion Abnormalities, SBP: Systolic Blood Pressure, SOLVD: Studies of Left Ventricular Dysfunction, SVD: Single Vessel Disease, TDI: Tissue Doppler Imaging, TR: Tricuspid Regurgitation, TVD: Tripple Vessel Disease, VT: Ventricular Tachycardia, VHD: Valvular Heart Disease

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