

# IMPAIRED GLUCOSE TOLERANCE IN ACUTE ST ELEVATION MYOCARDIAL INFARCTION - A PERSPECTIVE STUDY

Dissertation submitted for  
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Chennai, Tamilnadu.

# CERTIFICATE

This is to certify that this dissertation titled “**IMPAIRED GLUCOSE TOLERANCE IN ACUTE ST ELEVATION MYOCARDIAL INFARCTION - A PERSPECTIVE STUDY**” submitted by **DR.G.Prathap Kumar** to the faculty of Cardiology, The Tamil Nadu Dr. M.G.R. Medical University, Chennai. in partial fulfillment of the requirement for the award of DM degree Branch II [Cardiology] is a bonafide research work carried out by him under my direct supervision and guidance .

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# **DECLARATION**

I, **Dr.G.Prathap Kumar**, solemnly declare that the dissertation titled “**IMPAIRED GLUCOSE TOLERANCE IN ACUTE ST ELEVATION MYOCARDIAL INFARCTION - A PERSPECTIVE STUDY**” has been prepared by me.

This is submitted to the Tamil Nadu, Dr. M.G.R. Medical University Chennai, in partial fulfillment of the regulations for the award of DM degree Branch II [Cardiology].

Place : Madurai

Date :

**DR. G. PRATHAP KUMAR**

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## INTRODUCTION

Acute Myocardial Infarction is a serious and life threatening disorder. There are various risk factors for the development of Acute Myocardial Infarction. Of them, the most important and the one which is causing havoc among South Asian and Indian population is the relationship between abnormal glucose metabolism and Acute Myocardial Infarction. It is a well known fact that Diabetes Mellitus, a full blown form of abnormal glucose metabolism is associated with increased risk of cardiovascular events like AMI, unstable angina, Cerebrovascular events like stroke & also with peripheral vascular disease.

Whether the new concept of Impaired Glucose Tolerance, Impaired Fasting Glucose also have similar effects on the morbidity and mortality of the patients with AMI & their relationship is either causal or casual needs to be urgently evaluated.

Also, the incidence of Impaired Glucose Tolerance, Impaired Fasting Glucose is increasing in the population especially among the Indians and South East Asians. The Impaired Glucose Tolerance, Impaired Fasting Glucose are considered as prediabetic state, in the contemporary literature. Whether the detection of an individual in this prediabetic state & proper treatment with life style modification can bring down the risk of future cardio vascular events like Unstable Angina, AMI are questions to be answered.

With this background of Impaired Glucose Tolerance, Impaired Fasting Glucose metabolic abnormalities, their incidence in the patients with Acute Coronary Syndromes

and their influence on the outcome of the cardio vascular events with respect to mortality and morbidity is studied in this dissertation.

Also the effect of these prediabetic states on the left ventricular function in the post myocardial infarction period and on the functional capacity of the individual is studied in detail in this thesis.



## **AIMS OF THE STUDY**

1. To compare and contrast the in-hospital outcomes of patients with Acute Myocardial infarction with normal Glucose Metabolism and Abnormal Glucose Metabolism – Impaired Glucose Tolerance.
2. To study the effect of Impaired Glucose Tolerance on the left ventricular function in these patients.
3. To assess the functional capacity of the patients with Myocardial infarction with Impaired Glucose Tolerance.

## REVIEW OF LITERATURE

Whether high but non diabetic blood glucose levels are predictive of increased mortality from Coronary Artery Disease is not a new issue. This topic was discussed in International collaborative group in 1979. The conclusion from this analysis was that there was no consistent, strong and graded association between asymptomatic hyperglycemic individuals and coronary artery disease.

**Donahe and Orchard** reviewed the same question and concluded that glucose levels were related to Coronary artery disease risk, but whether the effect was linear or threshold was not clear in their study.

**Jarret et al** concluded that the risk did not appear to be related linearly with the glucose levels, but is apparent only in the upper percentiles of glucose distribution.

In our study we report the association of Fasting Blood Sugar and 2 hour post glucose challenge blood sugar level with the in-hospital events like arrhythmias, heart failure, recurrent angina, pericarditis and death.

We also report the association between fasting blood sugar and 2 hour glucose levels with Left ventricular function by analyzing the ventricular ejection fraction, end diastolic volume, end systolic volume, Wall motion score index and functional capacity as assessed by number of Mets in standard Bruce protocol Symptom limited Treadmill test before discharge from the hospital.

Hyperglycemia in the absence of diagnosis of diabetes (blood sugar < 200mg %) is a common phenomenon in stress related conditions like Acute Myocardial Infarction.

The cause of this hyperglycemia is primarily from excessive release of counter regulatory hormones.

Also, hyperglycemia refers to a pre-diabetic state in patients who are not diagnosed to have diabetes and whose blood sugar levels are in the abnormal range. This state is referred to as Impaired Glucose Tolerance.

Impaired Glucose Tolerance and Impaired Fasting Glucose are a continuum in the spectrum of disorder of glycemia.

**Disorders of Glycemia : Etiologic Types and Stages**

| Stages                | Normoglycemia             | Hyperglycemia  |                               |                                |   |
|-----------------------|---------------------------|--|-------------------------------|--------------------------------|---|
|                       | Normal Glucose regulation | Impaired Glucose Tolerance or Impaired Fasting Glucose | Diabetes Mellitus             |                                |   |
| Non Insulin requiring |                           |  | Insulin requiring for Control | Insulin requiring for survival |   |
| Types                 |                           |  |                               |                                |   |
| Type 1                |                           |  |                               |                                |   |
| Type 2                |                           |  |                               |                                |   |
| Other Specific Types  | ←                         |  |                               |                                | → |
| Gestational Diabetes  | ←                         |  | →                             |                                |   |

*Source : American Diabetic Association*

So, the disorder Impaired Glucose Tolerance and Impaired Fasting Glucose are considered as pre-diabetic state.

Multiple studies have shown that hyperglycemia in the diabetic range was associated with 3-4 fold increase in coronary artery disease risk and the outcome in the patients with diabetes was far more worse than that of the patients with out diabetes.

We in our study are establishing that whether this statement could be extrapolated to the patients who fall in Impaired Glucose Tolerance and Impaired Fasting Glucose range.

Recently, ACC/AHA guidelines have included the presence of Diabetes Mellitus as a major risk factor for coronary artery disease. Also, the Framingham Heart study reiterates that the patients with hyperglycemia in diabetic range have increased mortality and morbidity than the patients with normal glycemia.

A recent concept of Dysglycemia which includes Impaired Glucose Tolerance and Impaired Fasting Glucose is also under vogue. **Sahay et al** in his review has introduced the concept of Dysglycemia. The risk of coronary artery disease is high even at the time of diagnosis of Type 2 Diabetes Mellitus, hence the pre-diabetic states are also associated with increased risk of coronary artery disease.

Cardio vascular disease is leading cause of morbidity and mortality in patients with Diabetes Mellitus and the risk of Myocardial Infarction can be as high as in non diabetic individuals with history of Myocardial Infarction.

This has led to the National Cholesterol Educational Programme Adult Treatment Panel III (NCEP ATP III) to consider diabetes as Coronary Artery Disease equivalent.

It is now being recognised that revised blood glucose even below the threshold necessary to diagnose diabetes or even impaired glucose tolerance is associated with considerable increased risk of coronary artery disease. There appears to be a continuous relation between risk of coronary artery disease and raised post prandial glucose

concentration that extends from barely elevated levels right into the diabetic range.

That the glucose concentration associated with increased risk of Coronary Artery Disease are lower than those required for diagnosis of diabetes should not be surprising. Since these criteria are based on specific glucose concentration above which the individuals are at risk for development of nephropathy and retinopathy and not cardiovascular disease.

Although post prandial glucose levels above which the patients are at an increased risk of coronary artery disease is not yet defined, it may be as low as 98 mg %. Since this value is much lower than that which would qualify for diagnostic of Impaired Glucose Tolerance. The term Dysglycemia is suggested to define the range of glucose concentration associated with risk of Coronary Artery Disease (**Gerstein et al**).

Dysglycemia therefore includes in addition to the pre-diabetic states (Impaired Glucose Tolerance and Impaired Fasting Glucose), blood glucose values much below this threshold.

Impaired Glucose Tolerance & Impaired Fasting Glucose places the individuals at risk of developing diabetes mellitus and its complications. Both Impaired Glucose Tolerance and Impaired Fasting Glucose appear well before type 2 diabetes mellitus is diagnosed and they present an opportunity for intervention to reduce the future burden of diabetes and associated coronary artery disease risk to the individual and to the community.

**Diagnostic values for Oral Glucose Tolerance Test (OGTT) for Diabetes Mellitus & other categories of Hyperglycemia**

|                                   | Whole Blood Glucose     |                         | Plasma Glucose          |                       |
|-----------------------------------|-------------------------|-------------------------|-------------------------|-----------------------|
|                                   | Venous                  | Capillary               | Venous                  | Capillary             |
| <b>Diabetes Mellitus</b>          |                         |                         |                         |                       |
| Fasting                           | ≥ 6.1 (≥ 110)           | ≥ 6.1 (≥ 110)           | ≥ 7.0 (≥ 126)           | ≥ 7.0 (≥ 126)         |
| 2 hr after 75 gm glucose load     | ≥10.0 (≥ 180)           | ≥11.1 (> 200)           | ≥11.1 (≥ 200)           | ≥12.2 (> 220)         |
| <b>Impaired Glucose Tolerance</b> |                         |                         |                         |                       |
| Fasting Value                     | < 6.1 (< 110)           | < 6.1 (<110)            | < 7.0 (<126)            | < 7.0(<126)           |
| 2 hr after 75 gm glucose load     | 6.7 – 10.0<br>(120-180) | 7.8 – 11.1<br>(140-200) | 7.8 – 11.1<br>(140-200) | 8.9-12.2<br>(160-220) |
| <b>Impaired Fasting Glucose</b>   |                         |                         |                         |                       |
| Fasting Value                     | 5.6 – 6.1<br>(100-110)  | 5.6-6.1<br>(100-110)    | 6.1-7.0<br>(110-126)    | 6.1-7.0<br>(110-126)  |
| 2 hr after 75 gm glucose load     | <6.7 (<120)             | <7.8 (<140)             | <7.8 (<140)             | <8.9 (<160)           |

*Source : American Diabetic Association*

Note :

1. Values are given in mmol/lit, values given in the parenthesis are mg/dl.
2. The plasma glucose is 15% higher than the whole blood glucose. In a fasting state the 'Venous and Capillary Whole blood glucose is one and the same. But varies in the postprandial state. But with plasma glucose both the venous and capillary values are and the same.

3. WHO recommends 75 gm glucose load for adults and pregnant women.
4. Fasting value alone is considered less reliable, because true fasting cannot be assured and spurious diagnosis of diabetes may readily occur.
5.  $1 \text{ mmol/l} \times 18 = \text{mg \%}$  (conversion formula).

### **Importance of Impaired Fasting Glucose and Impaired Glucose Tolerance:**

There is no clear consensus with current evidence on whether Impaired Fasting Glucose and Impaired Glucose Tolerance should be classified as diseases. But they clearly represent risk factors and risk markers for diabetes and cardio vascular disease respectively. Both Impaired Fasting Glucose and Impaired Glucose Tolerance are similarly associated with increased risk of diabetes. But Impaired Glucose Tolerance is strongly associated with cardio vascular disease outcomes.

### **Risk of hyperglycemia**

Several explanations may account for the observed increased risk between hyperglycemia and poor prognosis after myocardial ischemia. **Metha et al** in his observation has found that hyperglycemia may be directly toxic to ischemic myocardium. Increase in the lactate content and intra cellular acidosis in the cells of ischemic myocardium are seen in patients with hyperglycemia.

Hyperglycemic patients are relatively deficient in insulin and this leads to both reduced peripheral uptake of glucose and may lead to decrease in the availability of glycolytic substrate for cardiac muscle utilization and increase in free fatty acid

concentration in ischemic musculature. These changes may reduce myocardial contractility even at increased oxygen concentration. It also increases the myocardial oxygen demand for the amount of myocardial work done leading to pump failure, and promote arrhythmias. (**Sarah E.Capes et al**)

According to **Sarah E.Capes et al** in meta analysis of 15 articles hyperglycemia is not simply an epiphenomenon of stress response mediated by cortisol and nor-adrenaline. It is associated increased risk of in-hospital mortality, increased risk of congestive heart failure and cardiogenic shock.

Hyperglycemia is a reflection of relative insulin deficiency and is associated with increased lipolysis, and excess circulating Free Fatty Acids (FFA), this effect may be exaggerated in cases of acute stress like Acute Myocardial Infarction.

Free Fatty Acids (FFA), although normally the substrate of choice for healthy myocardium, are toxic to ischemic myocardial and may lead to damaged cardiac cell membrane, calcium overload, and arrhythmias.

Moreover in animal studies conducted by **Mjos et al**, high concentration of Free Fatty Acids during myocardial ischemia causes increase in myocardial oxygen demands and decreased myocardial contractility.

Beta blockers suppress this release of Free Fatty Acids in patients with myocardial infarction and lessens the harmful effects of hyperglycemia and insulin deficiency. (**Malmberg et al**)

Insulin deficiency may also limit the ability of cardiac muscle to take up glucose



for anaerobic metabolism. In animal studies by **Ebreli et al**, preservation of myocardial function during ischemia correlates with increased uptake and metabolism of glucose.

The potential importance of insulin deficiency is also highlighted by randomised controlled trials in which insulin administered to patients without diabetes who have had acute myocardial infarction have improved clinical outcomes.

A similar observation was reported in DIGAMI study in which 620 patients with diabetes who had acute myocardial infarction were randomly assigned an insulin infusion followed by multidose subcutaneous insulin treatment for at least 3 months or conventional management. In that study, insulin lowered mortality by 28% (p 0.11) after mean follow up of 3-4 yrs and high glucose concentration on admission predicted higher risk of mortality.

Acute hyperglycemia may also precipitate an osmotic diuresis. The resulting volume depletion may interfere with the Frank Starling mechanism, an important compensatory mechanism for the failing Left Ventricle in which increased end diastolic volume leads to increased stroke volume.

Hyperglycemia may be a marker of more extensive cardiac damage in acute myocardial infarction, more extensive cardiac damage may lead to a greater rise in stress hormones promoting glycogenolysis and may also increased the risk of congestive heart failure and mortality.

Hyperglycemia in Impaired Glucose Tolerance is associated with hypertension, dyslipidemia and insulin resistance, a complex situation called Metabolic Syndrome - X.

According to National Cholesterol Educational Programme Adult Treatment Panel III, metabolic syndrome is diagnosed by following criteria.

| <b>Risk factors</b>                                     | <b>Defining Levels</b>                 |
|---|--|
| Abdominal obesity (Waist Circumference)<br>Men<br>Women | > 102cm (> 40 in)<br>> 88 cm (> 35 in) |
| Triglycerides   | > 150 mg%                              |
| HDL – C<br>Men<br>Women                                 | < 40 mg%<br>< 50mg%                    |
| Blood Pressure  | > 130 / 80 mm Hg                       |
| Fasting Glucose   | > 110 mg %                             |

Diagnosis is established when > 3 of these risk factors are present.

Criteria for Hypertriglyceridemic waist in men

| <b>Character</b> | <b>Criteria</b> |
|------------------|-----------------|
| Triglyceride     | > 2.0 mmol/l    |
| Waist            | > 90cm          |

### **Pathophysiology of Hyperglycemia in causing vascular disease**

Abnormalities in endothelial and vascular smooth muscle cell function as well as propensity to thrombosis, contribute to atherosclerosis and its complications. Endothelial cells are biologically active and produce substances that maintain intra vascular hemostasis. It also ensures adequate blood flow and nutrient delivery while preventing thrombosis and leukocyte diapedesis.

Most important of the substances produced by endothelium is Nitric Oxide (NO). Metabolic derangements like hyperglycemia and hyperlipidemia increase free fatty acid liberation and insulin resistances and prevent Nitric Oxide production.

Impaired Glucose Tolerance mediate the abnormalities in endothelial cell function by affecting synthesis or increased degradation of Nitric Oxide.

Experimental evidence supports that hyperglycemia decreases endothelium derived Nitric Oxide. Hyperglycemia induces a series of cellular events that increases the production of Reactive Oxygen Species like super oxide anion that inactive Nitric Oxide to form peroxynitrite.

Hyperglycemia may initiate this process by increasing super oxide anion production via mitochondrial electron transport chain. Super oxide then promotes a cascade of endothelial process that produce oxygen derived free radicals which activate second messengers like Protein Kinase C (PKC).

Mitochondrial production of super oxide anion also increases the production of Advanced Glycation End products (AGE). These glycated proteins adversely affect the cellular function by altering the protein function and by activating the Receptors for Advanced Glycation End products (RAGE). Advanced Glycation End products perse increase the production of oxygen derived free radicals. Receptors for Advanced Glycation End products activation increases intra cellular enzymatic super oxide production.

Hyperglycemia induced oxidative stress increases the levels of Asymmetric Dimethyl Arginine (ADMA), a competitive antagonist of Nitric Oxide Synthase (NOS), by impairing the activity of dimethyl arginine dimethyl aminohydrolase to metabolise Asymmetric Dimethyl Arginine (ADMA).

Hyperglycemia also increases the production of lipid second messenger Diacyl Glycerol (DAC). Diacyl Glycerol causes membrane translocation and activation of Protein Kinase C (PKC), which decreases endothelium dependent vaso relaxation.

### **Free Fatty Acids liberation and Endothelial function**

Circulating Free Fatty Acids are increased in Impaired Glucose Tolerance, because of their excess liberation from adipose tissue and diminished uptake by skeletal muscle. Free Fatty Acids impair endothelial function by increasing the production of oxygen derived free radicals, activation of Protein Kinase C (PKC) and exacerbation of dyslipidemia. Both hypertriglyceridemia and low High Density Lipoprotein (HDL) have been associated with endothelial dysfunction.

### **Impaired Glucose Tolerance, Thrombosis and Coagulation**

Platelet function is also abnormal in Impaired Glucose Tolerance. Expression of both glycoprotein Ib and IIb / IIIa are increased, augmenting the platelet von Willebrand Factor (vWF) and platelet fibrin interaction. Hyperglycemia further changes platelet function by impairing calcium hemostasis. It also alters many aspects of platelet activation and aggregation, including platelet confirmation and release of mediators.

Plasma coagulation factors (eg. Factor VII and Thrombin) and lesion based

coagulants (tissue factors) are increased in impaired glucose tolerance. Endogenous anticoagulation like thrombomodulin and protein C are decreased. Also the production of Plasminogen Activator Inhibitor (PAI-1), a Fibrinolytic inhibitor is increased. Thus, a propensity for platelet activation and aggregation coupled with a tendency for coagulation is relevant for an increased risk of thrombosis complicating plaque rupture and atherosclerotic coronary artery disease.

Hyperglycemia also causes increase in local and systemic inflammation during acute myocardial infarction. Studies suggest that acute “T” cell activation may play a role in plaque instability and acute clinical manifestation of coronary atherosclerosis (**Ross et al**).

According to **Raffaele Marfella et al** hyperglycemia during myocardial infarction is associated with increased levels of inflammatory markers, enhanced expression of cytotoxic ‘T’ cells and reduced expression of ‘T’ cells implicated in limitation of immune process.

Insulin is found to be a central factor in inhibiting cytokine release like IL – 8, decreased C-Reactive Protein (CRP) levels and inhibiting T cell activation. So, patients with insulin resistance like impaired glucose tolerance have increased cytokine release, increase in the levels of C-Reactive Protein (CRP) in blood and increased activation of T cells.

Also hyperglycemia is associated with increased Troponin levels probably as a consequence of more extensive myocardial damage.

**Raffaele Marfella et al** in a study further found out that, hyperglycemia positively correlated with both infarct segment length and Wall Motion Score Index. With increasing blood sugar levels wall motion score index increased and with increasing wall motion score index patient had poor prognosis. Wall motion score index positively correlated with extent of myocardial infarction. A wall motion score index of greater than 1.7 correlated approximately with 20% myocardial infarction by perfusion scan studies.

The increase in Myocardial Performance Index (MPI) which measures both systolic and diastolic parameters of ventricular function indicates worst functional outcome after myocardial infarction in hyperglycemic patients. The diminished diastolic filling time, prolongation of mitral regurgitation and decreased ejection time in hyperglycemic patients suggest, hyperglycemia may influence cardiac synchronisation after acute myocardial infarction.

Dyssynchrony between right ventricular and left ventricular contraction are independent predictors of heart failure and cardiac mortality in patients with congestive cardiac failure.

## **MATERIALS AND METHODS**

The study was carried out with the aim of comparing the in-hospital events like Arrhythmias, congestive heart failure, recurrent angina, Pericarditis & death among patients with acute ST elevation myocardial infarction eligible for thrombolysis with normal glucose metabolism and Impaired Glucose Tolerance.

Patients admitted in our Intensive coronary care unit with acute ST elevation Myocardial infarction were divided into two groups based on their glucose metabolism as patients with normal glucose metabolism and those with Impaired Glucose Tolerance.

Acute ST elevation myocardial infarction was defined as typical chest pain of ischemic origin lasting for > 20 minutes with ECG evidence of ST elevation of at least 1mm in 2 contiguous limb leads and at least 2mm in 2 contiguous chest leads, with typical rise and fall of biochemical markers of myocardial necrosis.

If the patients have any of the 2 above criteria, he/she was included as a patient with acute ST elevation Myocardial Infarction. Eligibility criteria for thrombolysis was scrutinized as per ACC/AHA guidelines for management of ST elevation myocardial infarction. All patients with pre-existing Diabetes Mellitus or blood sugar level in the diabetic range were excluded from the study.

All the study and control patients were treated with thrombolysis with Injection Streptokinase 1.5 million units in 100ml of normal saline to be run over 1 hour. Other medications were given as per the need of the patients and at the discretion of the treating physician.

At admission all patients were drawn blood for random blood glucose estimation. On the next morning after overnight fasting of 10-16 hours an Oral Glucose Tolerance Test was performed as per American Diabetic Association Recommendation.

A fasting blood sample was taken before giving glucose load. The subject was made to drink 75gms of glucose dissolved in 250-300ml of water. The glucose load was consumed over a period of 5 minutes. Blood samples are collected in fluoride – oxalate test tubes to prevent Red Blood corpuscles from metabolizing glucose.

Venous plasma collected from the patients were estimated for glucose by glucose oxidase method in our biochemistry laboratory. The values were expressed in mg %.

### **Lipid Profile**

Blood for lipid estimation was collected after an overnight fast within 24-48 hours of admission. Cholesterol and Triglycerides were measured by enzymatic calorimetric assays. High Density Lipoprotein and Low Density Lipoprotein cholesterol measures were done enzymatically using selective solubilisation method.

### **Glycosylated Hemoglobin**

Glycosylated Hemoglobin was estimated by cation-exchange resin method using Monozyme's Glycohemin kit. It is a rapid 10 minutes assay with conversion chart from Glycosylated Hemoglobin A<sub>1</sub>% to Glycosylated Hemoglobin A<sub>1c</sub>%. The normal value for Glycosylated Hemoglobin A<sub>1</sub> were between 6.0 to 8.7% which corresponded to 4.48 to 6.44 for Glycosylated Hemoglobin A<sub>1c</sub>.

A good clinical history with documentation of duration of chest pain and previous



history of Myocardial Infarction, Angina, Hypertension was taken. Detailed clinical examination at admission was done as per our intensive coronary care protocol. Systolic and diastolic Blood Pressure were measured using mercury sphygmomanometer. Phase I of korotkoff's sound was taken as Systolic Blood Pressure and phase V as Diastolic Blood Pressure. Killip's class was ascertained for every single subject at admission.

After thrombolysis and during hospital stay other parameters like successful thrombolysis, defined as greater than 50% resolution of the ST segment elevation at 90 minutes after Injection Streptokinase, complications like Arrhythmias, Congestive heart failure, Recurrent angina, development of pericarditis and death were monitored.

Complications were treated as per standard treatment protocol like cardioversion for hemodynamically unstable ventricular tachycardia and ventricular fibrillation. Complete heart block was treated with transvenous temporary pacing. Congestive heart failure was treated with diuretics, inotropes, and vasodilators. Recurrent Angina was treated with nitroglycerin infusion, intravenous heparin and beta blockers.

An day 3 of admission all patients underwent two dimensional echo cardiography and Doppler evaluation. Left Ventricular function was assessed using End Diastolic Volume, End Systolic Volume and Ejection Fraction. Ejection Fraction was calculated by Modified Simpson's method.

All the 16 segments as per American society of echocardiography classification were carefully investigated for wall motion abnormalities with naked eye and M – mode examination. Wall motion score was ascertained as 1 if the segment was normally

contracting. A score of 2 was given for hypo kinetic segment, 3 for akinetic segment. Dyskinetic segment was given a score of 4, and aneurysm was scored at 5.

Wall motion score index was calculated using the formula ,

Wall motion score index = Sum of wall motion scores ÷ number of segments visualized.

Presence or absence of Left Ventricular clot was assessed using two dimensional echo cardiography.

All the subjects with out Left Ventricular clot underwent symptom limited Treadmill Test by Bruce protocol and their functional capacity was assessed an day 5-7 before discharge from the hospital. The functional capacity was calculated as number of Mets of exercise the patients was able to perform before achieving either the target heart rate or development of symptoms necessitating stopping of the procedure.

Body mass index was calculated for all patients using the formula

$$\text{Body Mass Index} = \frac{\text{Weight in kg}}{(\text{Height})^2 \text{ in metre}}$$

To ensure correct reading, for height, the patients were made to stand on a flat surface against a wall bare footed and measurement were taken in cms. Weight was measured in kilograms by using the same weighing scale for all the patients.

## **Measurement of waist circumference**

Waist circumference was measured at mid point between the ribcage and 2 cm above the top of the iliac crest. Two measurement of waist circumference were made and the average was taken.

Waist circumference of  $> 103$  cm in male and  $> 88$  cm in female were considered abnormal.

All the parameters and variables were tabulated in a master chart and were analysed by computer analysis. Data were analysed utilizing the software – Epidemiological information package – 2002 (EPI Info 2002) developed by the center for disease control and prevention, Atlanta for World Health Organisation.

Mean, Standard deviation and 'p' values were calculated using this package. Chi-square test was used to find out the significance of relationship between the groups and other variables.

## RESULTS AND OBSERVATIONS

The relationship of parameters in study group i.e., patients with Impaired Glucose Tolerance and control group i.e., normal glucose metabolism revealed the following,

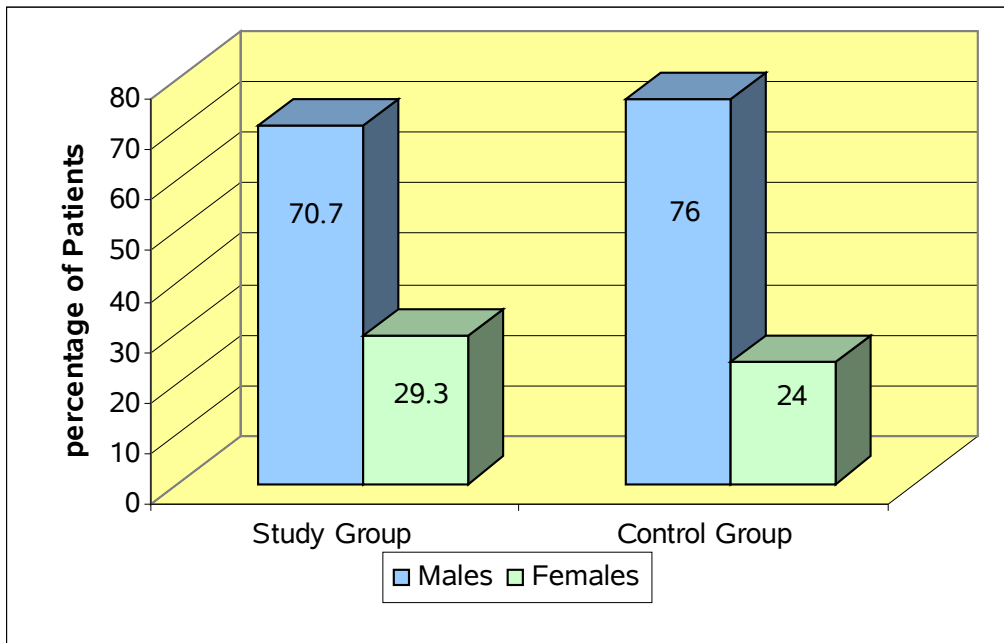
Sex distribution between the study group n=75 and control group n=25 were similar.

53 patients (70.7%) in the study group and 19 patients (76%) in the control group were males. The 'p' value was not significant showing there was no selection bias.

**Table - 1**

### Sex Distribution

| Sex     | Study group<br>(n=75) |      | Control Group<br>(n=25) |    | 'p'   |
|---------|-----------------------|------|-------------------------|----|-------|
|         | No.                   | %    | No.                     | %  |       |
| Males   | 53                    | 70.7 | 19                      | 76 | 0.797 |
| Females | 22                    | 29.3 | 6                       | 24 |       |

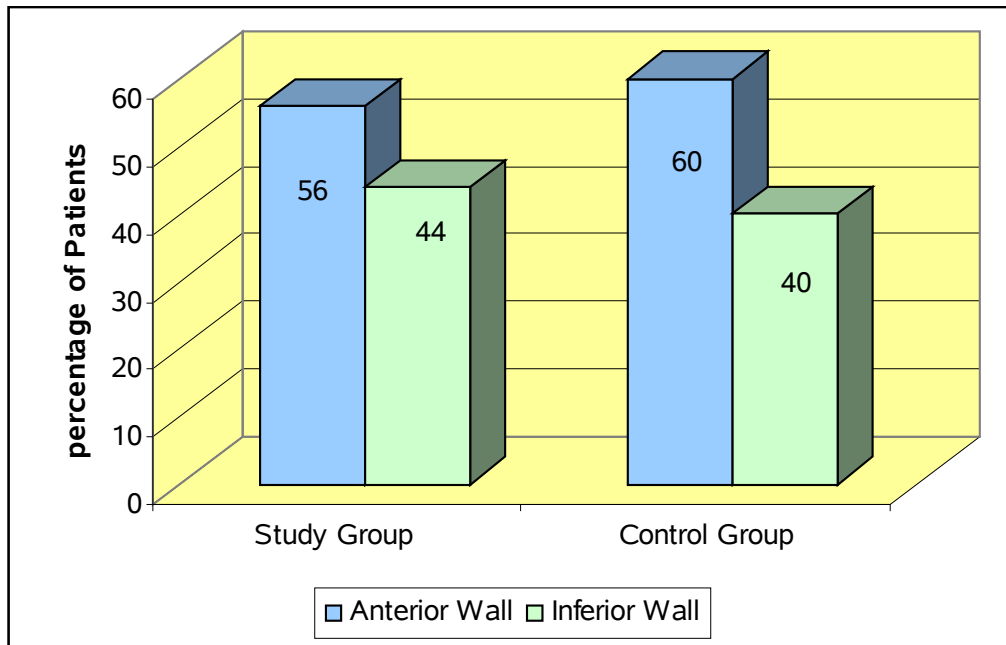


Similarly the distribution of myocardial infarction also did not show any significant 'p' value. 42 patients (56%) in study group and 15 patients (60%) in control group had Anterior Wall Myocardial Infarction.

**Table - 2**

**Distribution of Myocardial Infarction Location**

| Myocardial Infarction Location | Study group (n=75) |    | Control Group (n=25) |    | 'p'    |
|--------------------------------|--------------------|----|----------------------|----|--------|
|                                | No.                | %  | No.                  | %  |        |
| Anterior Wall                  | 42                 | 56 | 15                   | 60 | 0.9071 |
| Inferior Wall                  | 33                 | 44 | 10                   | 40 |        |

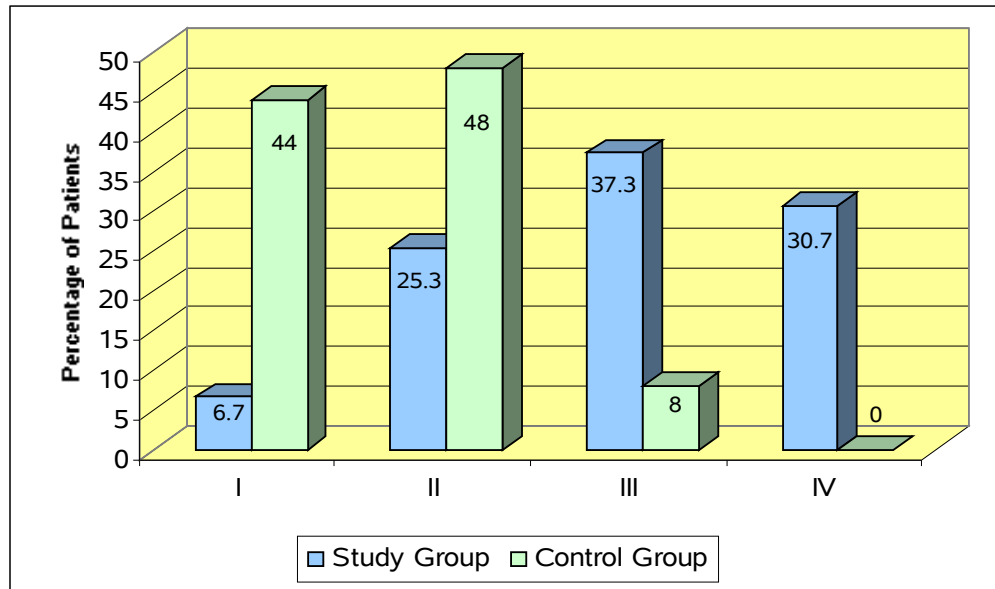


On analyzing the Killip Class, 5 patients (6.7%) were in Killip Class I, 19 patients (25.3%) in Class II, 28 patients (37.3%) in class III and 23 patients (30.7%) in class IV, in the study group and 11 patients (44%) in class I, 12 patients (48%) in class II and 2 patients (8%) in class III in the control group.

**Table - 3**

**Killip's Class Distribution**

| Killip's Class | Study group<br>(n=75) |      | Control Group<br>(n=25) |    | 'p'                 |
|----------------|-----------------------|------|-------------------------|----|---------------------|
|                | No.                   | %    | No.                     | %  |                     |
| I              | 5                     | 6.7  | 11                      | 44 | <b>0.0001(Sig.)</b> |
| II             | 19                    | 25.3 | 12                      | 48 |                     |
| III            | 28                    | 37.3 | 2                       | 8  |                     |
| IV             | 23                    | 30.7 | -                       | -  |                     |



The mean Killip class was  $2.92 \pm 0.91$  in the study group and  $1.64 \pm 0.63$  in control group with significant 'p' value of 0.0001.

Previous history of hypertension also correlated well with in the study group with 41.3% of patients (31) were taking treatment for hypertension.

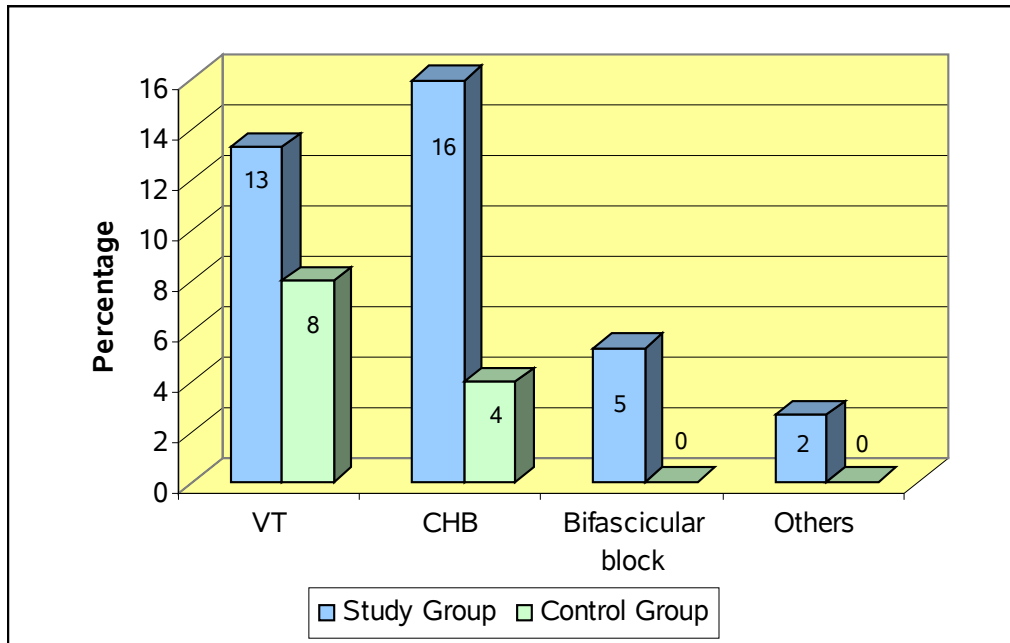
On analyzing the complications, Arrhythmias were more common in the study group. A total of 28 patients (37.3%) had some form of Arrhythmias Ventricular Tachycardia / Ventricular Fibrillation or Complete Heart Block in the study group. Only 3 patients (12%) had Arrhythmias in the control group. The incidence of Arrhythmias was more in the study group with significant 'p' value of 0.0338.

Ventricular Tachycardia was seen in 10 patients (13.33%), Complete Heart Block in 12 patients (16%), Bifascicular Block in 4 patients (5.33%) and Others in 2 patients (2.67%).

**Table - 4**

**Spectrum of Arrhythmias**

| Arrhythmias        | Study group<br>(n=75) |       | Control Group<br>(n=25) |   |
|--------------------|-----------------------|-------|-------------------------|---|
|                    | No.                   | %     | No.                     | % |
| VT / VF            | 10                    | 13.33 | 2                       | 8 |
| CHB                | 12                    | 16    | 1                       | 4 |
| Bifascicular block | 4                     | 5.33  | 0                       | 0 |
| Others             | 2                     | 2.67  | 0                       | 0 |



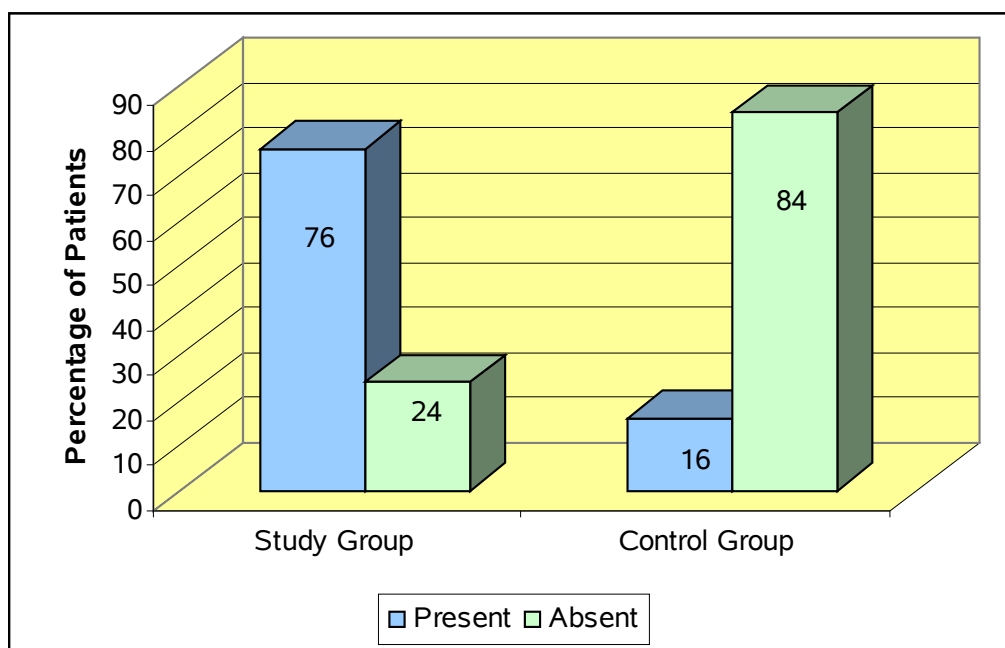


Congestive heart failure was present in 57 patients (76%) in the study group and 4 patients (16%) in the control group. Congestive heart failure was associated with poor outcome in our study. This also had a significant ‘p’ value of 0.0001.

**Table - 5**

**Comparison of Congestive Heart Failure between the two groups**

| Congestive Heart Failure | Study group (n=75) |    | Control Group (n=25) |    | ‘p’           |
|--------------------------|--------------------|----|----------------------|----|---------------|
|                          | No.                | %  | No.                  | %  |               |
| Present                  | 57                 | 76 | 4                    | 16 | <b>0.0001</b> |
| Absent                   | 18                 | 24 | 21                   | 84 |               |



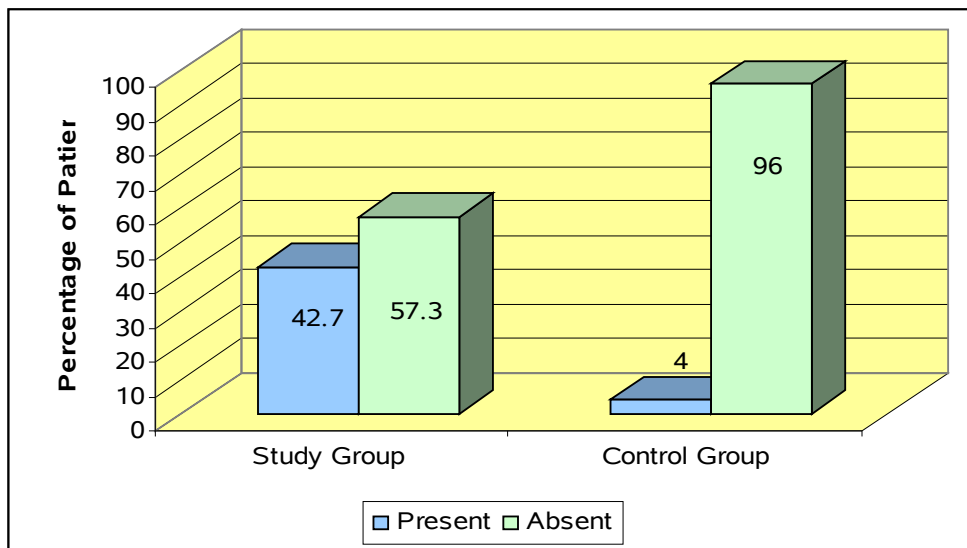
The relative incidence of recurrent angina needing nitroglycerine and heparin therapy were seen in 28 patients (37.3%) in the study group and only 3 patients (12%) in the control group. (p=0.0338).

Similarly there were 32 patients (42.7%) with Left Ventricular clot in the study group and only 1 patient (4%) in the control group. (p=0.0009).

**Table - 6**

**Incidence of Left Ventricular Clot**

| Left Ventricular Clot | Study group (n=75) |      | Control Group (n=25) |    | 'p'           |
|-----------------------|--------------------|------|----------------------|----|---------------|
|                       | No.                | %    | No.                  | %  |               |
| Present               | 32                 | 42.7 | 1                    | 4  | <b>0.0009</b> |
| Absent                | 43                 | 57.3 | 24                   | 96 |               |

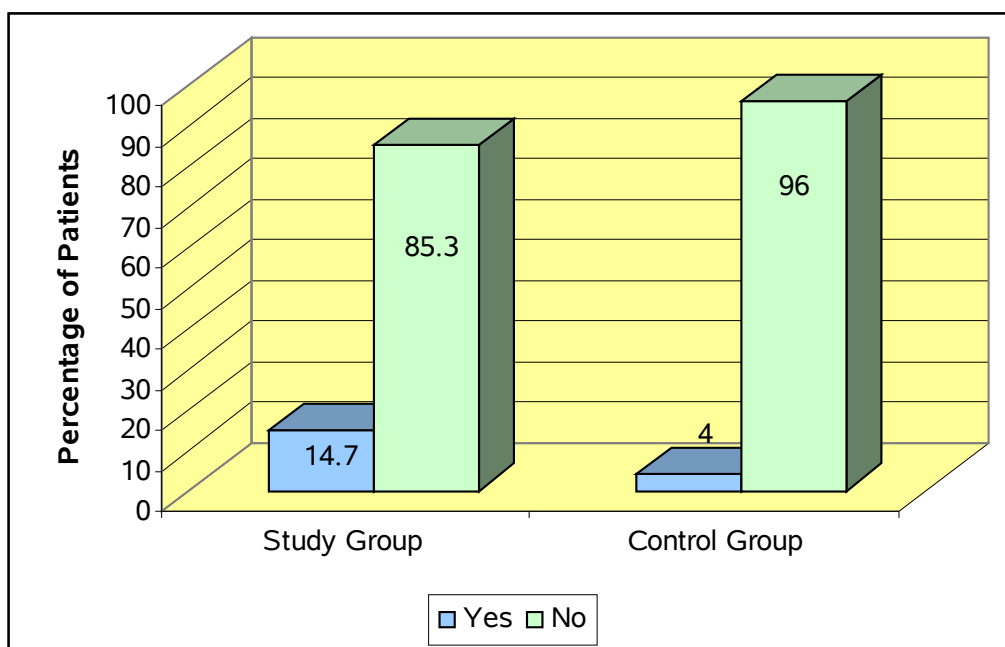


Death was seen a 11 patients in the study group (14.3%) and one patients (4%) in the control group. The percentage of patients was 3.5 times higher in the study group than in the control group. There was a statistically significant difference. (p=0.0414).

**Table - 7**

**Percentage of Death**

| Death | Study group<br>(n=75) |      | Control Group<br>(n=25) |    | 'p'    |
|-------|-----------------------|------|-------------------------|----|--------|
|       | No.                   | %    | No.                     | %  |        |
| Yes   | 11                    | 14.7 | 1                       | 4  | 0.1414 |
| No    | 64                    | 85.3 | 24                      | 96 |        |



**Table - 8**

**Mean Values of the Parameters in the Study and Control Groups**

| Parameters          | Study |      | Control |      | 'p'    |
|---------------------|-------|------|---------|------|--------|
|                     | Mean  | S.D. | Mean    | S.D. |        |
| Age                 | 54.9  | 9.9  | 52.2    | 11.5 | 0.3671 |
| Chest pain duration | 6.4   | 2.3  | 6.5     | 1.9  | 0.9692 |
| SBP                 | 112.4 | 33.1 | 101.7   | 6.4  | 0.4722 |
| DBP                 | 74.5  | 15.9 | 73.2    | 5.7  | 0.9246 |
| PR                  | 83.4  | 23.5 | 83.4    | 13.3 | 0.7829 |
| Hb A <sub>1</sub> C | 5.5   | 0.27 | 5.4     | 0.26 | 0.1954 |
| RBS                 | 176.7 | 16.6 | 123.5   | 11.7 | 0.0001 |
| FBS                 | 110.7 | 9.7  | 84.7    | 12.3 | 0.0001 |
| BS 1 hour           | 182.2 | 12.6 | 119     | 15.3 | 0.0001 |
| BS 2 hour           | 163.9 | 11   | 102     | 11.9 | 0.0001 |
| TC                  | 193   | 46   | 168.7   | 33.2 | 0.0131 |

|       |       |      |       |      |        |
|-------|-------|------|-------|------|--------|
| TGL   | 157.4 | 51.9 | 132.8 | 33.2 | 0.0406 |
| HDL   | 39.9  | 3.8  | 39.9  | 3.5  | 0.8678 |
| LDL   | 112.9 | 39.3 | 99.4  | 31.6 | 0.075  |
| VLDL  | 33    | 12.5 | 27.3  | 6.7  | 0.038  |
| BMI   | 28.2  | 1.6  | 20.7  | 0.9  | 0.0001 |
| Waist | 103.9 | 5.4  | 89.5  | 4.1  | 0.0001 |
| EF    | 40.8  | 4.6  | 49.6  | 3.7  | 0.0001 |
| EDV   | 117.2 | 15.2 | 102   | 9.9  | 0.0001 |
| ESV   | 69.2  | 14.1 | 51.8  | 7.7  | 0.0001 |
| WMSI  | 1.98  | 0.27 | 1.45  | 0.18 | 0.0001 |
| METS  | 5.02  | 0.5  | 6.47  | 0.96 | 0.0001 |

When the biochemical markers of Impaired Glucose Tolerance were analysed all the variables, Random blood sugar, Fasting blood sugar and 2 hour post glucose challenge blood sugar were showing high statistical significance between the study and the control group.

When analyzing the lipid profile of both the groups Total Cholesterol, Triglycerides, Very Low Density Lipoproteins showed positive correlation in the Impaired Glucose Tolerance group.

The Body Mass Index and the Waist Circumference were also found to be abnormal in the study group when compared to the control group.

The age of the study group and the control group were similar without any statistical difference. Also the duration of chest pain i.e., the door to needle time did not show any statistically significant difference between the two groups. These points were reiterated here to show that there was no selection bias among the two groups.

The systolic blood pressures, the diastolic blood pressure and the pulse rate at admission also did not show any significant difference between the two population of patients.

The mean Hemoglobin A<sub>1</sub>C was  $5.5 \pm 0.27$  in the study group and  $5.4 \pm 0.26$  in the control group suggesting that all these patients have not been known diabetics over the previous 120 days.

Random Blood Sugar showed a significant p value when compared with control patients. Study patients had a random blood sugar of  $176.7 \pm 16.6$  mg% and control patients had  $123.5 \pm 11.7$  mg % (p=0.0001).

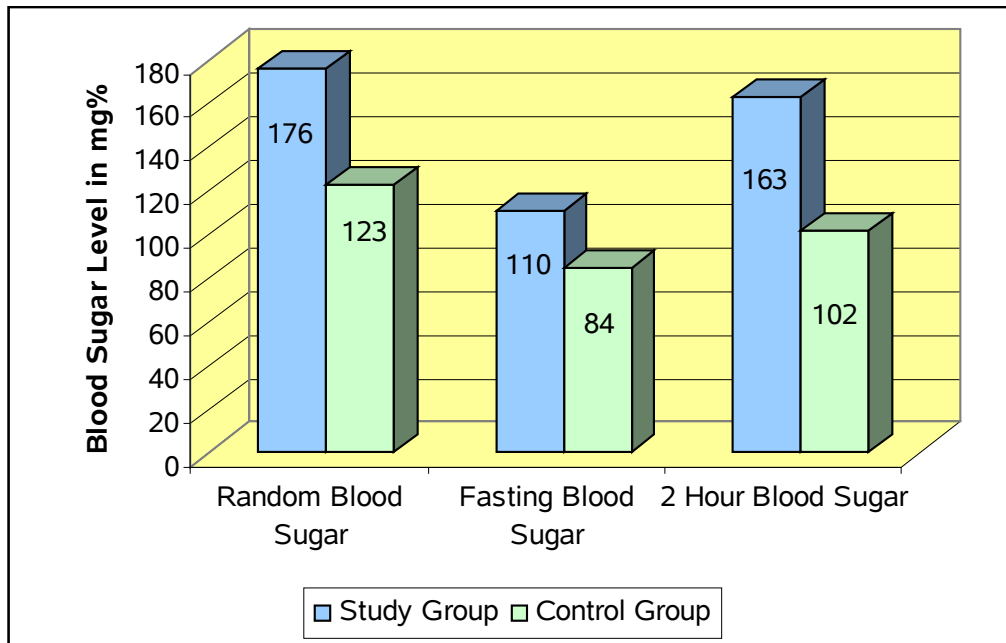
Fasting Blood Sugar also showed a significant 'p' value with study patient having a mean of  $110.7 \pm 9.7$  mg % and controls  $84.7 \pm 12.3$  mg% (p=0.0001).

The mean 2 hour blood sugar was  $163.9 \pm 11.0$  mg% in the study group and  $102 \pm 11.9$  mg% in the control group with a significant 'p' of 0.0001.

**Table - 9**

**Comparison of Blood Sugar Levels**

| <b>Blood Sugar Level</b> | <b>Study</b> |             | <b>Control</b> |             | <b>'p'</b>    |
|--------------------------|--------------|-------------|----------------|-------------|---------------|
|                          | <b>Mean</b>  | <b>S.D.</b> | <b>Mean</b>    | <b>S.D.</b> |               |
| Random                   | 176.7        | 16.6        | 123.5          | 11.7        | <b>0.0001</b> |
| Fasting                  | 110.7        | 9.7         | 84.7           | 12.3        | <b>0.0001</b> |
| 2 hour                   | 163.9        | 11          | 102            | 11.9        | <b>0.0001</b> |



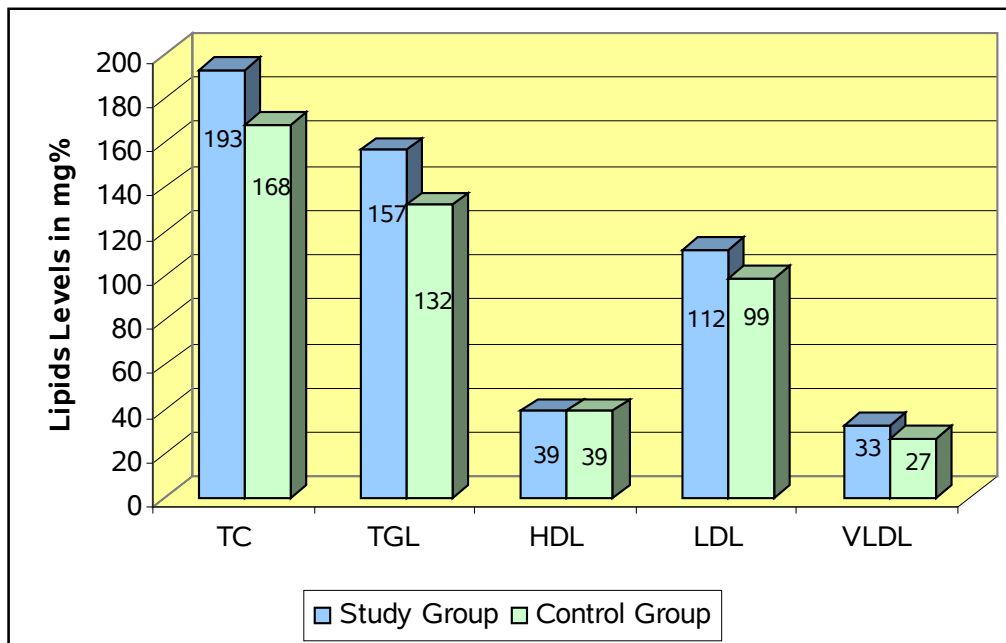
Similarly Total Cholesterol and Triglycerides were significantly different among patients between study group and control group. Total Cholesterol was  $193 \pm 46$  mg% in the study group and Triglycerides was  $157.4 \pm 51.9$  mg% in the study group as against  $168.7 \pm 33.2$  mg% and  $132.8 \pm 33.2$  mg% in the control group ( $p < 0.05$ ).

Even though the mean Low Density Lipoproteins was  $112.9 \pm 39.3$  mg% in the study group as against  $99.4 \pm 31.6$  mg% in the control group, the p value was not significant ( $p = 0.075$ ).

Table - 10

Comparison of Lipid Profile

| Lipids | Study |      | Control |      | 'p'    |
|--------|-------|------|---------|------|--------|
|        | Mean  | S.D. | Mean    | S.D. |        |
| TC     | 193   | 46   | 168.7   | 33.2 | 0.0131 |
| TGL    | 157.4 | 51.9 | 132.8   | 33.2 | 0.0406 |
| HDL    | 39.9  | 3.8  | 39.9    | 3.5  | 0.8678 |
| LDL    | 112.9 | 39.3 | 99.4    | 31.6 | 0.075  |
| VLDL   | 33    | 12.5 | 27.3    | 6.7  | 0.038  |



The Body Mass Index in the study group was  $28.2 \pm 1.6$  as against  $20.7 \pm 0.9$  in the control group which was statistically significant ( $p=0.0001$ ).

Similarly Waist circumference was  $103.9 \pm 5.4$  cm in the study group and  $89.5 \pm 4.1$  in the control group ( $p=0.0001$ ).

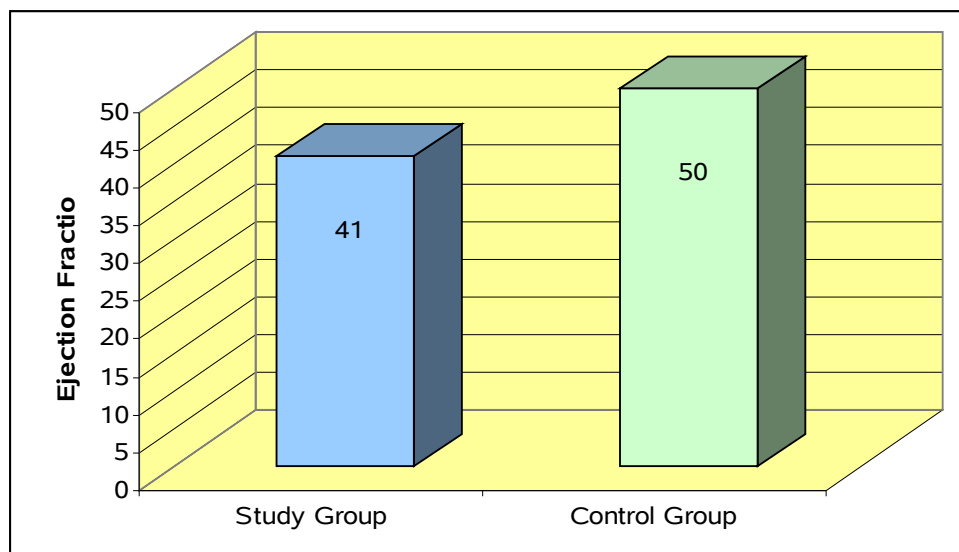
When the indices of left ventricular function were analysed, ejection fraction, end

diastolic volume, end systolic volume were significantly different between the study and the control groups. The mean ejection fraction was  $40.8 \pm 4.6$  in the study group and  $49.6 \pm 3.7$  in the control group which was statistically significant ( $p=0.0001$ ).

**Table - 11**

**Comparison of Ejection Fraction**

| LV function       | Study |      | Control |      | 'p'    |
|-------------------|-------|------|---------|------|--------|
|                   | Mean  | S.D. | Mean    | S.D. |        |
| Ejection Fraction | 40.8  | 4.6  | 49.6    | 3.7  | 0.0001 |



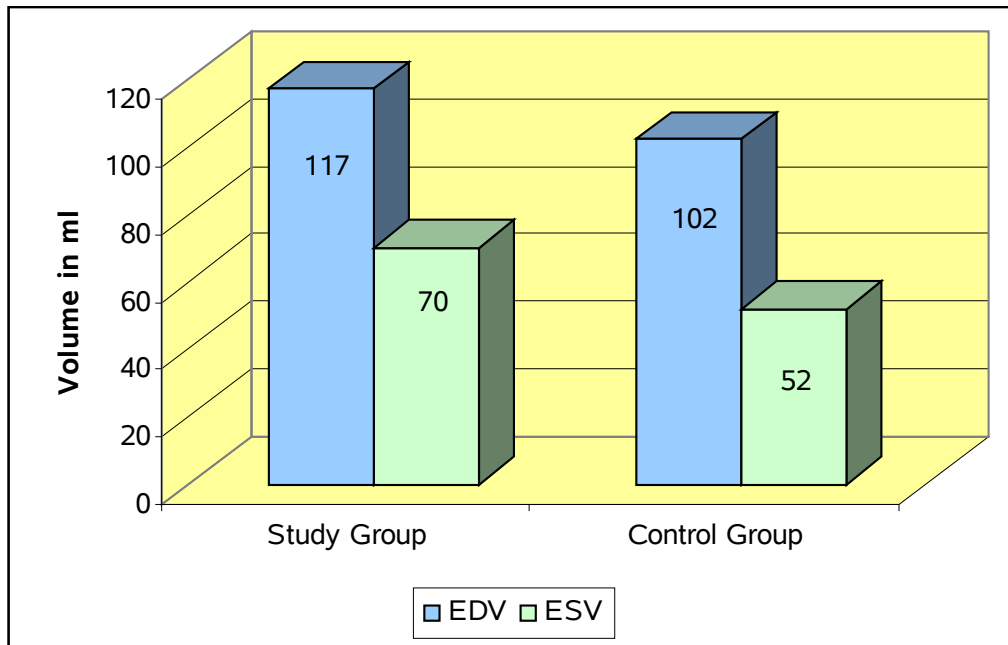
When end diastolic volume and end systolic volume were compared the mean were  $117.2 \pm 15.2$  ml and  $69.2 \pm 14.1$  ml in the study group and  $102 \pm 9.9$  ml and  $51.8 \pm 7.7$  ml in the control group respectively which were statistically very significant ( $p=0.0001$ ).



Table - 12

Comparison of Ventricular Volumes

| Ventricular Volume | Study |      | Control |      | 'p'    |
|--------------------|-------|------|---------|------|--------|
|                    | Mean  | S.D. | Mean    | S.D. |        |
| End Diastolic      | 117.2 | 15.2 | 102     | 9.9  | 0.0001 |
| End Systolic       | 69.2  | 14.1 | 51.8    | 7.7  | 0.0001 |

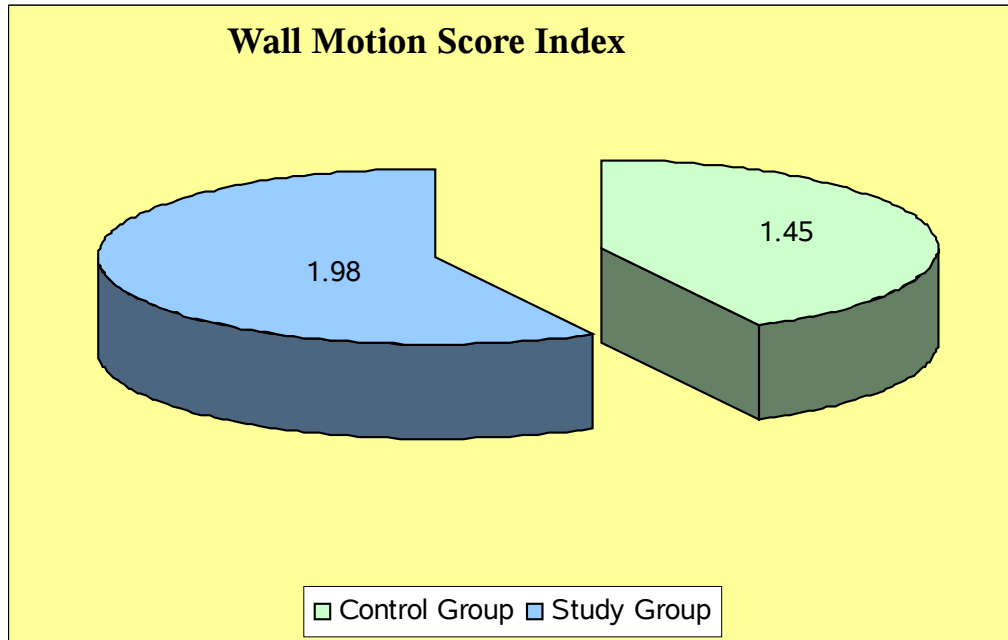


When Wall Motion Score index was compared between the two groups, the mean score was  $1.98 \pm 0.27$  in the study group which showed larger segmental loss of contractility as against  $1.45 \pm 0.18$  in the control group. The p value was again significant ( $p=0.0001$ ).

Table - 13

Comparison of Wall Motion Score Index

| Regional Wall Motion | Study |      | Control |      | 'p'          |
|----------------------|-------|------|---------|------|--------------|
|                      | Mean  | S.D. | Mean    | S.D. |              |
| WMSI                 | 1.98  | 0.27 | 1.45    | 0.18 | 0.0001(Sig.) |

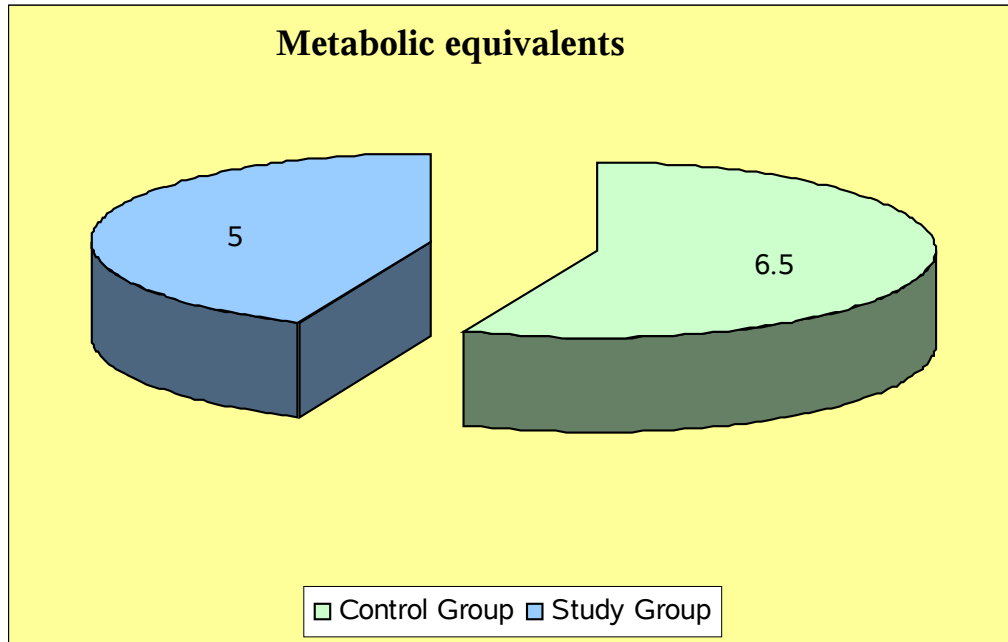


Similarly functional capacity as assessed by pre-discharge Symptom limited Treadmill test using Bruce protocol the mean Mets achieved before stopping the test was  $5.02 \pm 0.5$  in the study group and  $6.47 \pm 0.96$  in the control group ( $p=0.0001$ ).

**Table - 14**

**Comparison of Functional Capacity**

| Functional Capacity | Study |      | Control |      | 'p'    |
|---------------------|-------|------|---------|------|--------|
|                     | Mean  | S.D. | Mean    | S.D. |        |
| Mets                | 5.02  | 0.5  | 6.47    | 0.96 | 0.0001 |



Fasting Blood Sugar  $\leq 110$  mg% was taken as cut of points and analysis was done to find out the distribution in the study and the control group, 41 patients (54.7%) had abnormal Fasting Blood Sugar in the study group and non in the control group ( $p=0.0001$ ).

Similarly 2 hour blood sugar of  $> 140$  mg % showed statistically significant association in the study group.

27 patients (36%) had abnormal total cholesterol values as against 3 patients

(12%) in the control group. 35 patients (46.7%) had abnormal Triglycerides levels against 5 (20%) patients in the control group.

Similarly, 43 patients (52.3%) had Low Density Lipoproteins levels of >100mg% in the study group and only 7 patients (28%) had such values in the control group.

Abnormal Waist circumference (>103 cm in males and > 88cm in females) were present in 49 (65.3%) of the study patients as against none in the control group.

Thus the percentage of patients with abnormal values of Fasting Blood Sugar, 2 hour Blood Sugar, Total Cholesterol, Triglycerides, Low Density Lipoproteins, Waist Circumference and Body Mass Index were significantly different from those of the control cases.

## DISCUSSION

Impaired Glucose Tolerance as per the criteria is associated with increased in-hospital events like arrhythmia, Congestive Heart Failure and mortality according to large number of studies quoted in the bibliography.

The basis of our study was a meta analysis from the *Lancet 2000; 355: 773-78* by **Sarah E. Capes et al.**

**Sarah E. Capes et al** analysed about 15 studies in which patients without diabetes but increased glucose concentration above the normal level with Acute Myocardial Infarction were observed for in-hospital events. The study conclusion was that hyperglycemia with myocardial infarction was associated with increased risk of in-hospital mortality in patients without diabetes. The risk of Congestive Heart Failure and cardiogenic shock were also increased in those patients without diabetes but with hyperglycemia of non diabetic range.

Similar results were obtained in our study, where we analysed about 75 patients with Impaired Glucose Tolerance and compared them with patients with normal glucose tolerance with both these groups had Acute ST elevation Myocardial Infarction.

There was a significant difference in various killip classes between the 2 groups with a mean of  $2.92 \pm 0.9$  in Impaired Glucose Tolerance Group and  $1.64 \pm 0.63$  in the control group. ( $p < 0.0001$ ).

The incidence of in-hospital events like arrhythmias, Ventricular Tachycardia, Ventricular Fibrillation, Non-sustained ventricular Tachycardia, Complete Heart Block,

Bifascicular block were increased in the study group as compared to the control group.

Significant difference is the incidence of Congestive Heart Failure was also seen in the study group with about 76% of patients had clinically significant Congestive Heart Failure requiring treatment, unlike only 16% of patients in the control group with a statistically significant p of 0.0001.

Similarly, the incidence of recurrent angina was also significantly more in the study group than in the control group.

The relative incidence of death was 14.7% in the study group and only 4% in the control groups and most of the deaths were either due to Congestive Heart Failure or Arrhythmias or Recurrent angina or a combination of these complications.

All the patients who died had Congestive Heart Failure with Arrhythmias contributing in about 45% patients along with Congestive Heart Failure and Recurrent Angina contributing 45% among all the death. About 2 patients (18%) had only Congestive Heart Failure as major cause of their death.

When other traditional risk factor like smoking was considered, about 42 patients (56%) were smokers in the study group and 18 patients (72%) were in the control group. Still, death was about 3-4 times higher in the Impaired Glucose Tolerance as compared to that of the normoglycemic group.

Hyperglycemia was the basic culprit causing the increased incidence of all in-hospital event rates in the Impaired Glucose Tolerance group.

Hyperglycemia also contributed for the decreased Left Ventricular function in the

study group when compared to that of the control group. The ejection fraction which is the traditional index of Left Ventricular function was statistically significantly different in both the groups.

In the study group ejection fraction was around  $40.8 \pm 4.6$  and in the control group  $49.6 \pm 3.2$ , which was statistically significant.

Similarly, the end diastolic volume and end systolic volume were also significantly different in both the groups.

The Wall Motion Score Index, a novel index for calculating the amount of myocardial jeopardised by the ischemic insult, also was significantly higher in the study group at  $1.98 \pm 0.27$  versus  $1.45 \pm 0.18$  in the control group, suggesting that patients with Impaired Glucose Tolerance had larger area of Left Ventricular myocardium affected by the ischemia and infarction.

This increase in Wall Motion Score Index positively correlated with the decreased ejection fraction and the increase in the incidence of deaths among the study group when compared to the control group.

All patients who died had a Wall Motion Score Index of at least 1.75 or greater with about 8 out of 11 patients (72%) had a Wall Motion Score Index of  $\geq 2$ . Even in the control group, where only one death was recorded the patient had a Wall Motion Score Index of 1.75.

Thus Wall Motion Score Index had a greater prognostic influence on predicting the deaths not only among the patients with Impaired Glucose Tolerance and Acute ST

elevation Myocardial Infarction, but also in patients with normal blood glucose and Acute ST elevation Myocardial Infarction.

Functional capacity was much lower in patients with Impaired Glucose Tolerance and Acute ST elevation Myocardial Infarction than in patients with normal glycemia. It was  $5.02 \pm 0.5$  Mets in the Impaired Glucose Tolerance group and  $6.47 \pm 0.96$  in the control group with a significant 'p' value of 0.0001.

Thus, patient with Impaired Glucose Tolerance and Acute ST elevation Myocardial Infarction when all other criteria were equally considered had increased in-hospital event rates like Arrhythmias, both tachyarrhythmias and bradyarrhythmias (37.3% versus 12%), increased incidence of Congestive Heart Failure (76% versus 16%).

There was also increased incidence of the Recurrent Angina (37.3% versus 12%), increased incidence of Left Ventricular Clot (42.7% versus 4%) and increased incidence of death (14.7% versus 4%).

This group also had lesser ejection fraction  $40.8 \pm 4.6\%$  versus  $49.6 \pm 3.7\%$ , higher Wall Motion Score Index  $1.98 \pm 0.27$  versus  $1.45 \pm 0.18$ . There was decrease in the functional capacity from  $6.47 \pm 0.96$  in the normoglycemic groups as against  $5.02 \pm 0.5$  in the Impaired Glucose Tolerance group.



## CONCLUSION

- ❖ Impaired Glucose Tolerance in patients with Acute ST elevation Myocardial Infarction is associated with
  - increased incidence of arrhythmias
  - increased incidence of Congestive Heart Failure
  - increased incidence of recurrent angina
  - increased incidence of Left Ventricular Clot
  - increased incidence of in-hospital death
- ❖ Impaired Glucose Tolerance is also associated with decrease in the left ventricular function as suggested by decrease in the ejection fraction in this group of patients and increase in the Wall Motion Score Index.
- ❖ Impaired Glucose Tolerance is associated with decrease in the functional capacity during the Pre discharge Treadmill Test.

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**IMPAIRED GLUCOSE TOLERANCE IN ACUTE ST ELEVATION  
MYOCARDIAL INFARCTION - A PERSPECTIVE**

**PROFORMA**

NAME : AGE : SEX : ADDRESS:

DOA : CDNO. IPNO. BMI WAIST :

CHEST PAIN :

BREATHLESSNESS : DURATION : NYHA CLASS :

PALPITATION : SYNCOPE : OTHERS :

TYPE OF AMI : STEMI NSTEMI

LOCATION OF INFARCT :

KILLIP'S CLASS :

PREVIOUS DISORDERS : MI ANGINA CHF HTN DM  
DYSLIPIDEMIA

SMOKER ALCOHOLIC

DIET : VEG / NON VEG

ECG

ADMISSION

90 MINUTES AFTER SK

THROMBOLYSIS SUCCESSFUL / UNSUCCESSFUL

COURSE IN HOSPITAL ;

DRUGS : SK ASA NITR. ACEI BB STATINS DIG HEP CLOP AB

COMPLICATIONS :



# ANNEXURE 1

Conversion Table of GHb A1 and GHb A1c

| A <sub>1</sub> | A <sub>1c</sub> | A <sub>1</sub> | A <sub>1c</sub> | A <sub>1</sub> | A <sub>1c</sub> |
|----------------|-----------------|----------------|-----------------|----------------|-----------------|
| 5.1            | 3.78            | 7.7            | 5.70            | 10.3           | 7.63            |
| 5.2            | 3.85            | 7.8            | 5.78            | 10.4           | 7.70            |
| 5.3            | 3.92            | 7.9            | 5.85            | 10.5           | 7.78            |
| 5.4            | 4.00            | 8.0            | 5.92            | 10.6           | 7.85            |
| 5.5            | 4.07            | 8.1            | 6.00            | 10.7           | 7.92            |
| 5.6            | 4.15            | 8.2            | 6.07            | 10.8           | 8.00            |
| 5.7            | 4.22            | 8.3            | 6.15            | 10.9           | 8.07            |
| 5.8            | 4.29            | 8.4            | 6.22            | 11.0           | 8.15            |
| 5.9            | 4.40            | 8.5            | 6.29            | 11.1           | 8.22            |
| 6.0            | 4.44            | 8.6            | 6.37            | 11.2           | 8.30            |
| 6.1            | 4.48            | 8.7            | 6.44            | 11.3           | 8.37            |
| 6.2            | 4.59            | 8.8            | 6.52            | 11.4           | 8.44            |
| 6.3            | 4.70            | 8.9            | 6.59            | 11.5           | 8.52            |
| 6.4            | 4.74            | 9.0            | 6.67            | 11.6           | 8.59            |
| 6.5            | 4.81            | 9.1            | 6.74            |                |                 |
| 6.6            | 4.89            | 9.2            | 6.81            |                |                 |
| 6.7            | 4.96            | 9.3            | 6.89            |                |                 |
| 6.8            | 5.04            | 9.4            | 6.96            |                |                 |
| 6.9            | 5.11            | 9.5            | 7.04            |                |                 |
| 7.0            | 5.18            | 9.6            | 7.11            |                |                 |
| 7.1            | 5.26            | 9.7            | 7.18            |                |                 |
| 7.2            | 5.33            | 9.8            | 7.26            |                |                 |
| 7.3            | 5.40            | 9.9            | 7.33            |                |                 |
| 7.4            | 5.48            | 10.0           | 7.41            |                |                 |
| 7.5            | 5.55            | 10.1           | 7.48            |                |                 |
| 7.6            | 5.65            | 10.2           | 7.55            |                |                 |

# ANNEXURE 2

## Abbreviations

|                      |                                  |
|----------------------|----------------------------------|
| <b>Cpdur</b>         | - Chest pain duration            |
| <b>MI</b>            | - Myocardial Infarction          |
| <b>Pre. MI</b>       | - Previous Myocardial Infarction |
| <b>HTN</b>           | - Hypertension                   |
| <b>Succ. Thrombo</b> | - Successful Thrombolysis        |
| <b>CHF</b>           | - Congestive Heart Failure       |
| <b>Rec. Angina</b>   | - Recurrent Angina               |
| <b>SBP</b>           | - Systolic Blood Pressure        |
| <b>DBP</b>           | - Diastolic Blood Pressure       |
| <b>PR</b>            | - Pulse Rate                     |
| <b>RBS</b>           | - Random Blood Sugar             |
| <b>FBS</b>           | - Fasting Blood Sugar            |
| <b>TC</b>            | - Total Cholesterol              |
| <b>TGL</b>           | - Triglycerides                  |
| <b>LDL</b>           | - Low Density Lipoprotein        |
| <b>VLDL</b>          | - Very Low Density Lipoprotein   |
| <b>BMI</b>           | - Body Mass Index                |
| <b>EF</b>            | - Ejection Fraction              |
| <b>EDV</b>           | - End Diastolic Volume           |
| <b>ESV</b>           | - End Systolic Volume            |
| <b>WMSI</b>          | - Wall Motion Score Index        |