"A STUDY OF ECHOCARDIOGRAPHY CHANGES IN PATIENTS WITH TYPE 2 DIABETES IN A TERTIARY CARE LEVEL HOSPITAL''

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BRANCH – I

Reg No: 201711258

DEPARTMENT OF GENERAL MEDICINE

CHENGALPATTU MEDICAL COLLEGE

CHENGALPATTU - 603 001

MAY 2020

CERTIFICATE

This is to certify that the dissertation titled **"A STUDY OF ECHOCARDIOGRAPHY CHANGES IN PATIENTS WITH TYPE 2 DIABETES IN A TERTIARY CARE LEVEL HOSPITAL"** is the bonafide original work of **Dr. SURESH SHANMUGAM** for partial fulfillment of the requirements for **M.D. Branch - I (General Medicine)** Examination of the Tamil Nadu Dr. M. G. R. Medical University to be held in May 2020. The period of study was from October 2018 to September 2019.

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This is to certify that dissertation **"A STUDY OF ECHOCARDIOGRAPHY CHANGES IN PATIENTS WITH TYPE 2 DIABETES IN A TERTIARY CARE LEVEL HOSPITAL"** is the bonafide original work of **Dr. SURESH SHANMUGAM**, postgraduate student of General medicine, Chengalpattu Medical College, Chengalpattu, under my guidance and supervision for fulfillment of regulations of The Tamil Nadu Dr. M.G.R Medical University for the award of M.D. Degree during the Academic period 2017 - 2020.

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DECLARATION

I, Dr. SURESH SHANMUGAM, solemnly declare that dissertation titled **"A STUDY OF ECHOCARDIOGRAPHY CHANGES IN PATIENTS WITH TYPE 2 DIABETES IN A TERTIARY CARE LEVEL HOSPITAL"** is a bonafide record of work done by me in the Department of General Medicine, Government Chengalpattu Medical College and Hospital during October 2018 to September 2019 under the guidance of **Prof Dr. B. PRABAKAR. M.D.,** Professor of General Medicine, Government Chengalpattu Medical College and Hospital, Chengalpattu. This dissertation is submitted to Tamil Nadu Dr. M.G.R. Medical University, for partial fulfillment of the University regulations for the award of M.D. Degree (Branch 1) General Medicine - May 2020.

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INTRODUCTION

INTRODUCTION

Diabetes is one of the most important public health problems among non-communicable disease. Globally, 422 million adults were having diabetes in 2012 when compared to 180 million in 1980. Diabetes caused 1.5 million deaths in 2012 and additional 2.2 million deaths were attributed who was having higher than optimal blood glucose by increasing the risk factor for CVD[1]. The most common modality of death among these patients were found to be cardiovascular disease [2].

Cardiovascular disease accounts for up to 80% of the excess mortality in patients with type 2 diabetes. The increased cardiovascular mortality risk in type 2 diabetic patients include generalised microvascular disease, coronary atherosclerosis, and autonomic neuropathy [3]. Apart from these, myocardial abnormalities (‘diabetic cardiomyopathy’) and heart failure also plays an important role in the mortality risk [4]. In general, undiagnosed case of heart failure is common among type 2 diabetes patients [4].

The cardiac changes in the diabetic patients are increased myocardial thickening with predominantly manifests in the form of diastolic dysfunction defined as the diabetic cardiomyopathy [5]. Diastolic dysfunction has been reported in many studies among these patients [6,7].

Type 2 diabetes mellitus patients are more prone for the increased Cardiovascular risk. So, in our study, type 2 diabetes mellitus patients will be screened with echocardiography for early diagnosis of myocardial changes and

cardiac dysfunction. Left ventricular diastolic dysfunction prevalence in type 2 diabetes patients asymptomatic, normotensive patients with or without significant coronary artery disease is much higher than previously suspected[7]. By early detection of these myocardial changes in echocardiography will help in treating these patients accordingly. Hence, we can reduce the cardiovascular mortality risk in these patients.

AIM OF THE STUDY

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To determine the echo-cardiographic abnormalities in type 2 diabetes mellitus patients attending the out- patient department.

REVIEW OF LITERATURE

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DIABETES MELLITUS:

A chronic metabolic disease characterized by high blood glucose progressive over decades [8,9]

TESTS	NORMAL GLUCOSE TOLERANCE (MG/DL)	IMPAIRED GLUCOSE TOLERANCE (MG/DL)	DIABETES MELLITUS
Fasting plasma Glucose	100	100 - 125	≥ 126
2hrs plasma Glucose	140	140 - 199	≥ 200
HbA1c	< 5.6%	5.7 - 6.4%	≥ 6.5%

TYPES OF DIABETES: [9]

- **Type 1 diabetes**
  - Presents at younger age
  - Genetic Susceptibility (HLA complex on Chr.6)
  - Absolute insulin deficiency
    - A) Immune mediated
    - B) Idiopathic
- **Type 2 diabetes**
  - Insulin insensitivity of tissues with relative insulin deficiency
  - Insulin secretory defect with insulin resistance

- **Gestational diabetes**
- **Other types** [9]

A) Mutations in genes of Beta cell function/ development

- Hepatocyte nuclear transcription factor (HNF)4α (MODY 1)
- Glucokinase (MODY 2)
- HNF-1 α (MODY 3)
- Insulin promoter factor-1 ((MODY 4)
- HNF-1 β (MODY 5)
- NeuroD1 (MODY 6)
- Pro-insulin or insulin
- Mitochondrial DNA
- Subunits of ATP-sensitive Potassium channel
- Other pancreatic islet proteins/regulators such as PAX4, BLK, KLF 11, SLC2A2, GATA 4, GATA 6, RFX6, GLIS3

B) Genetic defects of Insulin action

- Leprechaunism
- Type A insulin resistance
- Lipodystrophy syndromes
- Rabson - Mendenhall syndrome

C) Exocrine Pancreas Disease

- Pancreatitis
- Cystic fibrosis
- Neoplasia

- Cystic fibrosis
- Hemochromatosis

D) Endocrinopathies

- Cushing's syndrome
- Acromegaly
- Glucagonoma
- Hyperthyroidism
- Pheochromocytoma
- Aldosteronism

E) Chemical/Drug induced

- Glucocorticoids
- Pentamidine, thiazides, anti-psychotics
- β agonists, calcineurin and mTOR inhibitors

F) Infections

- Congenital rubella
- CMV, Coxsackie virus

G) Uncommon forms of Immune-mediated disease

- Stiff person syndrome
- Anti- insulin receptor antibodies

H) Other genetic syndromes associated with DM

- Down's syndrome,
- Klinefelter syndrome
- Wolfram syndrome

EPIDEMIOLOGY:

Diabetes was found to be the seventh leading cause of death by WHO in 2016. In 1980 people with diabetes was 108 million but in 2014 it has been raised to 422 million. Likewise the worldwide prevalence of diabetes among people over 18 years of age has been raised from 4.7% (1980) to 8.5% (2014)[10]. The prevalence has been raised rapidly in low-income and middle economic countries. About 1.6 million deaths were due to diabetes directly in 2016. In 2012, another 2.2 million deaths were due to higher blood sugar level. The half of all death attributable due to higher blood glucose level usually occur before 70 years of age[10]. In India about 69.1 million people are living with DM and found to be the second highest number of patients of DM in the world. In India, DM is found to be high in Southern part people and in urban areas. The prevalence of DM was found to be 5- 7%. Increasing urbanization, dietary changes, ageing populations, reduced physical activity and unhealthy behaviour were found to be the rapid social and cultural changes, causes DM to increase in occurrence. In diabetic patients, cardiovascular diseases found to be the leading cause of death. Diabetic patients have a 2 - 5 times higher risk of developing heart failure when compared to non-diabetic patients, irrespective of other co-morbidities [11].

RISK FACTORS:

TYPE 1 DM:

- Family H/O
- Environmental factors
- Exposure to some viral infections

TYPE 2 DM:

- Family H/O of diabetes
- Unhealthy diet
- Physical inactivity
- Overweight (BMI ≥ 25 kg/m^2)
- High blood pressure
- Increasing age
- Ethnicity/ Race
- H/o of gestational diabetes or delivery of big baby
- Poor nutrition during pregnancy
- PCOD
- Acanthosis nigricans
- Metabolic syndrome
- H/o CVD
- Triglyceride level > 250 mg/dl, HDL cholesterol < 35 mg/ dl
- Impaired glucose tolerance (IGT) [12]

PATHOGENESIS

There are eight distinct patho-physiologic abnormalities which is associated with type 2 diabetes mellitus (T2DM). The insulin resistance is characterized by decreased peripheral glucose uptake and also increased glucose production and there by hyperglycemia and high circulating insulin levels. The

insulin resistance further causes increased lipolysis, leading to increased free fatty acid levels and intermediary lipid metabolites in blood contributes to a further increase in glucose production endogenously, a reduction in peripheral glucose utilization and impaired beta-cell function in long run.

As the beta-cell function of pancreas is well decreased at this stage, it won't able to maintain normal plasma glucose levels by compensatory insulin secretion and again progressively worsens over time. Concomitantly, pancreatic alpha-cells produces glucagon inappropriately, particularly in the post-prandial period. It has been said that both impaired insulin and increased glucagon secretion in T2DM are due to the gastrointestinal incretin hormones deficiency primarily as inadequate release or response to food intake.

Hypothalamic insulin resistance (central nervous system) also decrease the ability of insulin to suppress glucose production, and renal tubular glucose reabsorption capacity may be increased despite hyperglycemia in T2DM. These pathophysiologic abnormalities should be taken into account for the treatment of patients with T2DM [13].

Ultimately the main pathophysiology in T2DM are mainly due to insulin resistance, hyperinsulinemia and glucose toxicity [14]. Impaired glucose homeostasis, altered metabolic status, impaired energy production and accumulations of advanced glycation end products and oxidative stress and endothelial dysfunction form the some of the postulating mechanisms in the pathogenesis of diabetic cardiomyopathy [15].

SCREENING FOR DM

American diabetes association provided guidelines to screen a asymptomatic adults for testing prediabetes and diabetes. Testing for pre-diabetes and diabetes should be done in all individuals who are over-weighted (BMI ≥ 25 kg/ m^2) and having additional risk factors.

In the absence of those risk factors, screening should be done in all individuals of age > 45 years. Even if they are negative for pre-diabetes and diabetes screening should be done at-least every three years of interval.

GLYCOSYLATED HEMOGLOBIN

The term glycation means adding of sugar residue to an - NH_2 group of a protein molecule. Glycation will also occur in hemoglobin, membrane protein and lens protein. In HbA1c, glycation occurs in each beta chain on N- terminal valine residue.

HbA1c provides a retrospective index of blood glucose level over a period of 6 – 8 weeks. It is a reliable indicator of diabetic control over past 90 days, treatment effectiveness and the risk of development of short and long –term complications.

HbA1c values & interpretations:

- Risk of Hypoglycaemia: < 4.5%
- Non-diabetic range: 4.5 - 5.8%

- Pre-diabetic range: 5.8 - 6.5%
- Diabetic range: > 6.5%

CONDITIONS WHICH GIVES TO FALSE ELEVATION / REDUCTION OF HbA1c:

- Hereditary persistence of fatal Hb
- Hemoglobinopathies (Thalassemia)
- Uraemia

GUIDELINES FOR ONGING, COMPREHENSIVE MEDICAL CARE FOR DIABETIC PATIENTS:

- Glycaemic control targets should be optimized and individualized by frequent blood glucose testing – every 2 weeks in uncontrolled patients until target glycaemic value is attained and every 3 months in controlled patients (HbA1c < 7%)
- Self-monitoring of blood glucose with individualized frequency is advised.
- HbA1c should be done every 3 months in uncontrolled diabetic patients and every 6 months to 1 year (ie., 2- 4 times/ year)
- Fundus examination should be done in the first visit itself and every 1-2 years during follow-up.
- Screening for diabetic kidney disease using urine micro-albumin and serum creatinine annually.

- Foot examination for neuropathy should be done daily by the patient themselves, at-least 1- 2 times/ year by physician.
- Blood pressure measurement should be done every quarterly.
- ECG should be done annually.
- Treadmill test have to done five years after diagnosing diabetes mellitus, later at-least once in two years.
- Lipid profile should be done annually, if abnormal then it is done every 6 months.
- Prophylactic anti-platelet therapy should be considered based on the individuals.
- Patient education regarding diabetes management should be given annually.
- Medical nutrition education and therapy must be done annually.

TREATMENT GOALS FOR ADULT DM PATIENTS:

Goals should be individualized for every patient. Goals may be differing for certain patient population depending on geographic distribution, genetic inheritance and dietary habits.

The recommendations by **American Diabetic Association**[16] are:

INDEX		GOAL
Glycemic control		
1.	HbA1c	< 7.0%
2.	Fasting plasma glucose	80-130 mg/dl
3.	Peak post-prandial plasma glucose	< 180 mg/dl
Blood pressure		< 130 / 80 mmHg
Lipid levels:		
1.	High Density Lipo- protein	> 40 mg/dl for men > 50 mg/dl for women
2.	Low Density Lipo- protein	< 100 mg/dl for both gender < 70 mg/dl for cardiovascular disease patients
Triglycerides		< 150 mg/dl

COMPLICATIONS

There are many complications in patients with T2DM depending on various factors such as dietary habits, associated lifestyle, smoking, alcoholism, other metabolic diseases. Apart from these, complications depend on duration of T2DM, hyperglycemia and hyperinsulinemia.

The complications may be classified into acute and chronic complications.

Acute complications are mainly

- Hypoglycaemia
- Hyperglycaemia- hyperosmolar non ketonic coma, diabetic ketoacidosis.

CHRONIC COMPLICATIONS

Chronic complications mainly due to vascular (micro-vascular & macro-vascular) and metabolic changes because of long duration of hyper-glycemia and due to secondary factors, such as hypertension, hyper - lipidemic and sedentary life style habits. These causes the functional alterations progress to early structural changes in many organs. When the early changes once set in, progression to irreversible damage and end stage depends on various factors apart from hyperglycemia.

MICROVASCULAR:

EYE:

- Macular oedema
- Retinopathy (Proliferative / non – proliferative)
- Glaucoma
- Cataract

NEPHROPATHY (Declining Renal function)

- Diabetic kidney disease

NEUROPATHY

- Diabetic peripheral neuropathy
- Autonomic neuropathy

MACROVASCULAR

Cardiac complications

1. Coronary artery disease
2. Small vessel disease
3. Diabetic cardiomyopathy
4. Heart failure
5. Cardiac autonomic neuropathy

OTHERS:

Gastrointestinal

- Gastroparesis,
- Nocturnal diarrhea

Genitourinary

- Cystopathy,
- Female sexual dysfunction

Infections – Possess high risk for Gram negative organism, *M. tuberculosis, S. aureus, Candida spp*

Dermatologic complications

- Xerosis, Pruritis,
- Pigmented periorbital papules,
- Bullous disease,
- Necrobiosis lipoidica diabeticorum,
- Ancanthosis nigricans, Lipoatrophy

Cheiroarthropathy

Hearing loss, Periodontal disease etc.,

CARDIOMYOPATHY

The literal meaning of cardiomyopathy is myocardial disease. Cardiomyopathy is defined as an abnormality in the myocardium leading to changes in cardiac chamber size, wall thickness, electrical and/ or mechanical dysfunction.

Clinical manifestations may be confined to the heart or it may be a part of a generalized systemic disorder; but cardiac dysfunction is the key problem.

- Primary cardiomyopathies - Predominantly confined to the heart muscle
- Secondary cardiomyopathies - Myocardial involvement is one of a component of any systemic or multiorgan disorder.

TYPES:

There are four main types of cardiomyopathy.

1) Dilated
2) Hypertrophic
3) Restrictive
4) Arrhythmogenic right ventricular cardiomyopathy [17]

ETIOLOGY:

The causes of cardiomyopathies are varied in adults, dilated cardiomyopathy the most common cause is CAD (Ischemic cardiomyopathy) and hypertension, although valvular disease, viral myocarditis and genetic predisposition may also be responsible. neuromuscular diseases and idiopathic myocarditis are the most common cause of dilated cardiomyopathy, and present during the first year of life in children. Neuromuscular diseases that may lead to

dilated cardiomyopathy in children include Becker muscular dystrophy, Duchenne muscular dystrophy & Barth syndrome (an X-linked genetic disorder consisting of dilated cardiomyopathy, neutropenia and skeletal myopathy.

The common causes are

INFLAMMATORY MYOCARDITIS

- Infectious
  - Viral myocarditis-more commonly coxsackie, adenovirus, hepatitis - C
  - Bacterial- diphtheria, lyme's disease
  - Systemic fungal infections
- Non- infectious
  - Granulomatous inflammatory diseases - sarcoidosis, giant cell myocarditis
  - Eosinophilic myocarditis
  - Collagen vascular disease
  - Peripartum cardiomyopathy

TOXIC

- Alcohol, catecholamines
- Chemotherapeutic agents- anthracyclines
- Interferon
- Heavy metal poisoning like pb, Hg
- occupational exposure

METABOLIC CAUSES

- Endocrinopathies- diabetes, pheochromocytoma, hyperthyroidism and hypothyroidism
- Nutritional deficiencies- thiamine, carnitine, selenium
- Electrolyte deficiencies-calcium, Magnesium, PO4
- Hemochromatosis
- Obesity

INHERITED METABOLIC PATHWAY DEFECTS

FAMILIAL

- Becker muscular dystrophy
- Duchenne muscular dystrophy
- Myocardial myopathies
- Skeletal and cardiac myopathy
- Arrhythmogenic right ventricular dysplasia

OVERLAP WITH NONDILATED CARDIOMYOPATHY

- Hemochromatosis
- Amyloidosis

IDIOPATHIC

MISCELLANEOUS

- Arrhythmogenic right ventricular dysplasia
- LBBB
- Tachycardia related cardiomyopathy

DIABETIC CARDIOMYOPATHY

Diabetic cardiomyopathy is defined as presence of abnormal cardiac muscular structure and function in diabetic patients in the absence of other cardiovascular risk factors such as CAD, hypertension, and valvular heart disease [15]. It is characterized by left ventricle (LV) concentric hypertrophy and perivascular and interstitial fibrosis which leads to diastolic dysfunction.

There are 4 stages of diabetic cardiomyopathy and there is overlap with the HF classifications of both New York Heart Association Class and the American Heart Association Stage /American College of Cardiology.

DIABETIC CARDIOMYOPATHY STAGES [18]

STAGE 1: Diastolic Heart Failure with normal Ejection Fraction > 55%

NYHA CLASS 1: The individual is asymptomatic and there is no limitation of physical activity.

ACC/AHA HF stage A: At risk of Heart Failure, but there is no structural heart disease or symptoms.

STAGE 2: Symptomatic Heart Failure with systolic and diastolic dysfunction

NYHA CLASS 2: Slight limitation during ordinary physical activity with palpitation, fatigue, dyspnea or angina.

ACC/AHA HF STAGE B: Asymptomatic structural heart disease.

STAGE 3: Symptomatic Heart Failure due to hypertension, microvascular disease or viral disease.

NYHA CLASS 3: There is marked limitation of ordinary physical activity. Symptoms occur during minimal physical activity.

ACC/AHA HF STAGE C: Symptomatic Heart Failure with structural heart disease.

STAGE 4: Symptomatic Heart Failure, with contribution from multiple confounder including coronary artery disease.

NYHA CLASS 4: Symptoms of heart failure at rest. Unable to carry out any physical activity without discomfort.

ACC/AHA HF STAGE D: Refractory Heart Failure requiring specialist interventions

PATHOGENESIS OF DIABETIC CARDIOMYOPATHY

CHANGES IN STRUCTURE & SIGNALING PATHWAYS

1. LEFT VENTRICULAR HYPERTROPHY:

The Framingham study found that significant increase in LV wall thickness was seen only in women with diabetes. But, in a Study conducted in Native Americans, suggested that both woman and men with diabetes had higher LV wall thickness and mass. Among the diabetic patients Increased LV mass was seen only in patients with diabetes but not those with impaired glucose tolerance

or impaired fasting glucose. In diabetics, the changes in myocardial geometry are due to consequence of long-term diabetes changes such as obesity / hyperglycemia not due to early defect. LV hypertrophy development predicts the increased risk of stroke and heart failure with reduced ejection fraction and mortality. There is a significant interaction between central obesity and diabetes on the risk for LVH. Apart from this, concentric LVH is produced by obesity, independent of hypertension. Cytokines produced by expanded adipose tissue of obesity plays a role in the development of LVH. In obese humans, leptin induces cardiac hypertrophy.

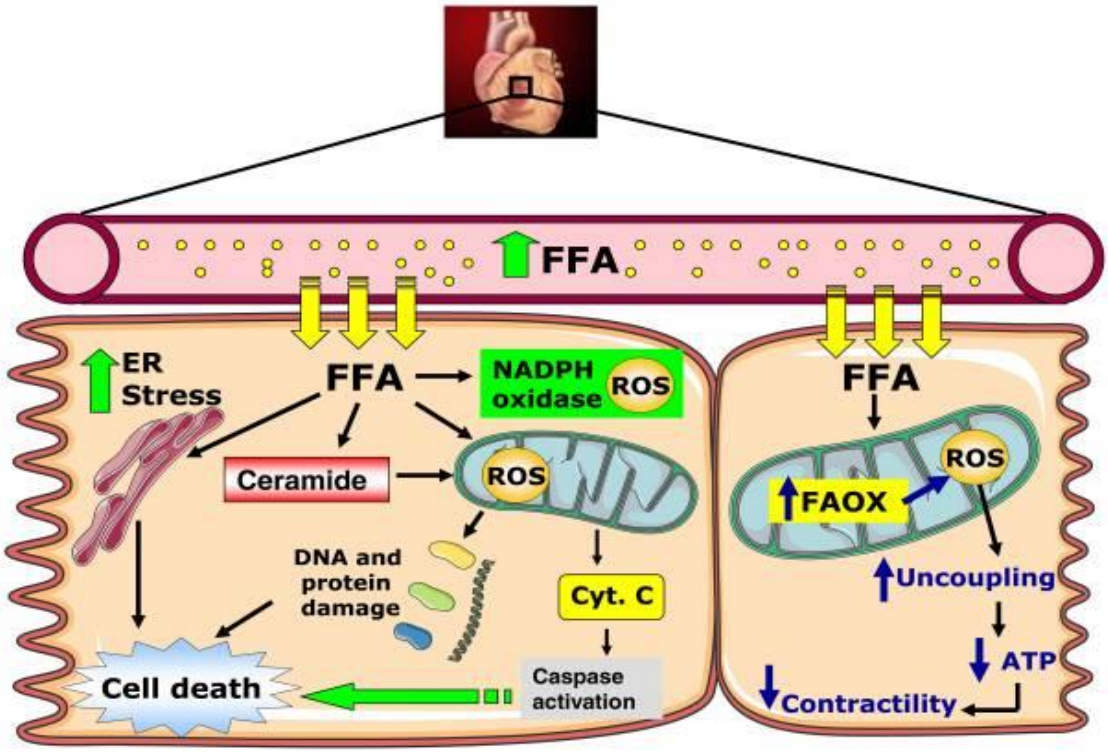
Likewise, an adipokine (resistin), released from macrophages, induce cardiomyocyte hypertrophy *in vitro* via MAPK and IRS-1 signaling pathways. A significant correlation was seen between circulating levels of interleukin 6 and the risk of obesity-associated heart failure. Hyperinsulinemia and Insulin resistance also been correlated with increased LV mass and partially account for the association of cardiac hypertrophy and obesity and there is an association with increased risk of heart failure [19].

2. MYOCARDIAL LIPOTOXICITY:

Diabetes, insulin resistance, obesity and impaired glucose tolerance are found to be associated with increased intra-myocardial lipid, independent of circulating triglycerides [20]. The increase in cardiac triglyceride accumulation is associated only with diastolic dysfunction not systolic dysfunction [20, 21]. In well-controlled diabetics, myocardial triglycerides appear to be normal [22].

3. INCREASED OXIDATIVE STRESS:

An increase in oxidative stress play an important role in producing diabetic cardiomyopathy, but the actual mechanisms involved in reactive oxygen species (ROS) production among diabetic patients' heart is not well understood.



Mechanism for Free Fatty Acid (FFA) - induced cardiac dysfunction in diabetes. Increased FFA uptake in cardiac myocyte precipitates cardiomyocyte dysfunction by multiple mechanisms in vivo including increased mitochondrial and cytosolic reactive oxygen species (ROS) generation and ER stress. FFA-mediated ROS generation leads to uncoupling of mitochondria, which reduces mitochondrial ATP production. ER- Endoplasmic Reticulum; Cyt C- Cytochrome C; FAOX- Fatty Acid oxidation

4. CELL DEATH

In diabetics, there is an increased apoptosis of cardiac myocytes, but mechanism is not well understood.

Renin-angiotensin system (RAS) activation correlated with increased oxidative stress, necrosis and apoptosis in cardiomyocytes and endothelial cells in the hearts of diabetic patients and end stage heart failure. It is found that inhibition of RAS has reduced first hospitalization rate from heart failure and echocardiographic findings of LV diastolic function improved in Type 2 diabetes patients.

5. INTERSTITIAL FIBROSIS

DM cardiomyopathy is characterized by perivascular and interstitial fibrosis. It has been found that in diabetic patient's heart biopsies, there is an increase in collagen deposition between myofibers and around intramural vessels.

6. DIASTOLIC DYSFUNCTION (DD)

Diastolic dysfunction develops first before systolic dysfunction. Altered calcium homeostasis and increased cardiac lipid accumulation was found to be associated with DD in T2DM patients**.**

7. SYSTOLIC DYSFUNCTION (SD)

Systolic dysfunction is measured in the form of reduced ejection fraction which occurs due to the alterations in the cardiac contractility. It occurs mainly due to microvascular injury to the myocardium which impairs the cardiac perfusion.

8. IMPAIRED CONTRACTILE RESERVE

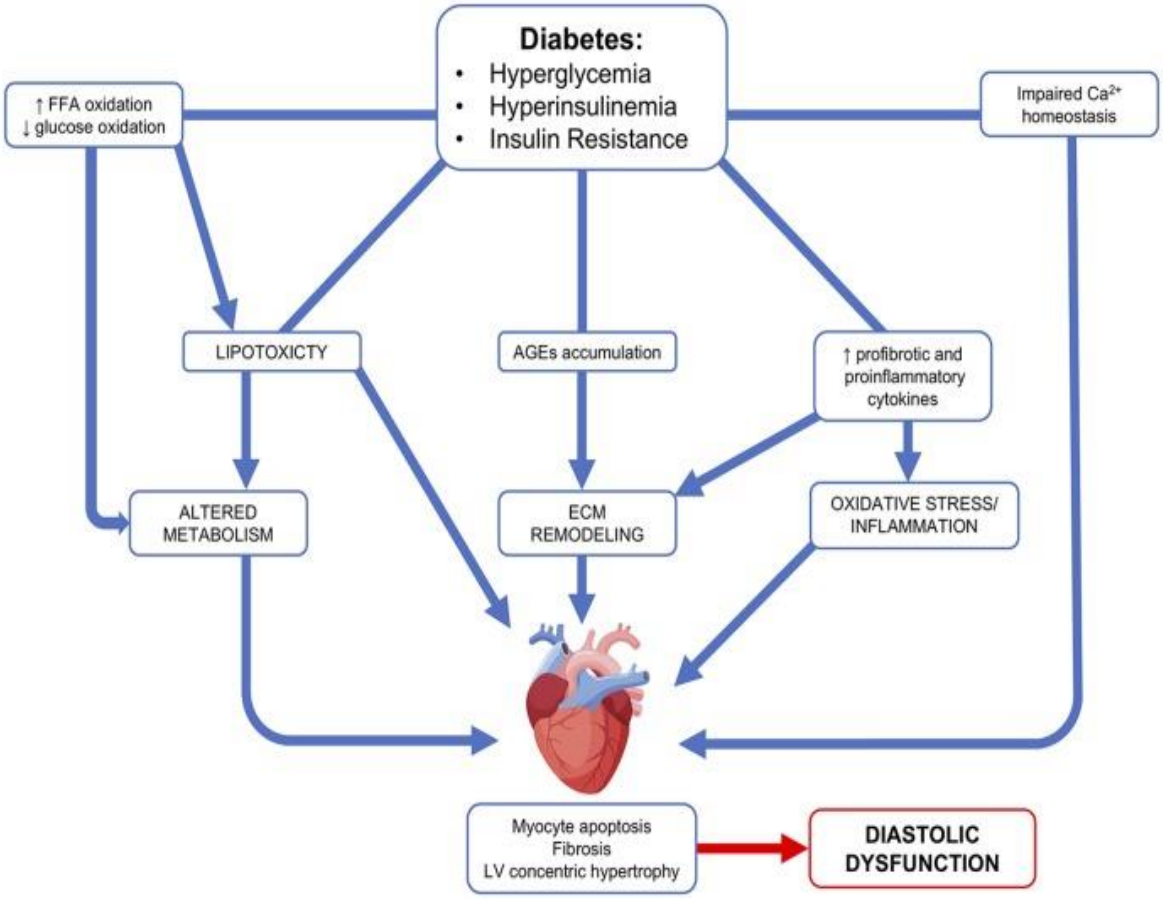
The diabetic autonomic dysfunction causes the impaired contractile reserve of the cardiac myocytes and it is also due to microvascular injury in coronary vasculature.

9. CHANGES IN MYOCARDIAL METABOLISM

10. ALTERED SUBSTRATE UTILIZATION

11. MITOCHONDRIAL DYSFUNCTION

PATHOPHYSIOLOGICAL MECHANISMS OF DIABETIC CARDIOMYOPATHY.



Hyperglycaemia, insulin resistance and hyperinsulinemia lead to an elevation in free fatty acid (FFA) oxidation, pro-inflammatory and pro-fibrotic cytokines, as well as there will be an accumulation of advanced glycation end products (AGEs). Altogether, these abnormalities will lead to an altered metabolism, oxidative stress, extracellular remodelling and inflammation. Finally, this will leads to cardiac effects, such as cardiac myocyte fibrosis, apoptosis and Left Ventricular concentric hypertrophy.[15]

CLINICAL FEATURES

Early symptoms involve

- Exertional intolerance with dyspnoea varying severity
- Fatigueness
- Pedal oedema due to right ventricle dysfunction
- Ascites

Later severe symptoms of heart failure ensue such as (due to reduced ejection fraction)

- Shortness of breath
- Orthopnoea
- Paroxysmal nocturnal dyspnoea
- Palpitation
- Sudden cardiac death[20]

PHYSICAL EXAMINATION

- Cyanosis may be present in acute presentation
- Pedal oedema.

- Raised jugular venous pressure(JVP).
- Apical impulse shifted to down and outward (may not be in restrictive type).
- S3, S4 may be heard.
- Loud P2, with wide split.
- Murmurs may be heard due to valvular involvement (Mitral valve involvement is more common due to annular dilatation).

DILATED CARIOMYOPATHY

In dilated cardiomyopathy the common first symptoms are

- Exertional intolerance.
- Left sided congestive features occur earlier than the right sided.
- Valvular regurgitation occurs due to annular dilation. Mitral regurgitation will appear earlier during decompensation; tricuspid regurgitation occurs with right ventricular dysfunction.
- Atrial size may be increased; atrium may also be primarily involved.
- Left ventricular wall thickness may be normal or decreased.

- Left ventricular diastolic dimension which is normally < 55mm, here it is increased usually > 60mm.

- Left ventricular ejection fraction (LVEF) which is normally > 55%, here it is decreased. When the symptoms are very severe LVEF will be usually < 30%.

- Arrhythmias may also occur in these patients that may be ventricular tachyarrhythmia or conduction block, sometimes atrial fibrillation.

RESTRICTIVE CARIOMYOPATHY

In restrictive cardiomyopathy the following things will occur which slightly differs from dilated one. The common first symptoms are

- Exertional intolerance, fluid retention does early.

- Right sided congestive features occur earlier than the left sided symptoms, such as pedal oedema, ascites.

- Valvular regurgitation occurs due to endocardial involvement. Both mitral regurgitation and tricuspid regurgitation occurs with ventricular dysfunction but rarely severe.

- Atrial size usually be increased; atrium may also be massively enlarged.

- Left ventricular wall thickness may be normal or increased.

- Left ventricular diastolic dimension which is normally < 55mm, here it may be normal or decreased.

- Left ventricular ejection fraction (LVEF) which is normally >55%, here it is decreased up to 25-50%.

- Arrhythmias may also occur in these patients, but ventricular tachyarrhythmia is uncommon except in sarcoidosis. Conduction block will happen in sarcoidosis and amyloidosis. Atrial fibrillation may also occur.

HYPERTROPHIC CARIOMYOPATHY

These patients will have the following features with common symptoms of

- Exertional intolerance, may have chest pain.

- Left sided congestive features may develop at late such as orthopnoea, paroxysmal nocturnal dyspnoea.

- Valvular regurgitation occurs due to interaction of valve and the inter ventricular septum. Usually mitral regurgitation will appear.

- Atrial size usually be increased; relating to the elevated filling pressures

- Left ventricular wall thickness will be markedly increased.
- Left ventricular diastolic dimension which is normally < 55mm, here it is often decreased.
- Left ventricular ejection fraction (LVEF) which is normally > 55%, here it is more than 60%
- Arrhythmias may also occur in these patients, but ventricular tachyarrhythmia is uncommon except in sarcoidosis. Conduction block will happen in sarcoidosis and amyloidosis. Atrial fibrillation may also occur.

INVESTIGATIONS:

Usually in asymptomatic individuals' cardiomyopathy is diagnosed incidentally during routine medical screening or family evaluation of already diagnosed patients [21]. Hence diagnosis of cardiomyopathy starts from the detailed history of symptoms and diseases, family history, personal history of habits, drug or treatment history.

Assessment of performance abilities is done in routine and desired activities.

- Assessment of volume status of the patient is done along with orthostatic blood pressure and BMI
- Chest radiograph
- Electrocardiograph

- 2D and Doppler echocardiogram
- MRI of myocardium for evidence of inflammation and fibrosis

- Haematology tests for
  - Haematocrit/ haemoglobin
  - WBC with differential count and eosinophilic count
  - Erythrocyte sedimentation rate

- Biochemical tests:
  - Serum electrolytes with calcium and magnesium
  - Blood urea nitrogen and serum creatinine
  - Total liver function tests including albumin and total protein
  - Thyroid function tests- thyroid stimulating hormone
  - Lipid profile
  - Cardiac biomarkers- troponin levels
  - Creatine kinase isoforms
  - Serum iron and transferrin saturation

- Screening for Infections
    - Viral – Influenza, ECHO virus, Coxsackie
    - HIV
    - Lyme's disease
    - Chaga's disease- *T. cruzi*
    - Toxoplasmosis
- Serology for active rheumatological disease
- Screening for breathing disorders
- In patients with angina, catheterization with coronary angiography

ELECTROCARDIGRAPHY IN CARDIOMYOPATHY

The findings in ECG of these patients vary widely. Most of the findings are

- Sinus bradycardia
- Atrial enlargement
- ST depression
- Pathologic Q waves

- Prolonged corrected QTc interval
- Left ventricular hypertrophy
- Ventricular tachyarrhythmia
- Atrial fibrillation[22,23].

ECHOCARDIOGRAPHY

Echocardiography is one of the non-invasive examinations of the heart using reflected ultrasound waves. It delineates and evaluates the structures of the heart and as well as the direction of blood flow within it by placing a transducer over the body.

Echocardiography is one of the widely recognized appropriate imaging modality in evaluating heart disease. The assessment includes chamber quantification, valvular heart disease, pulmonary hypertension, left ventricular systolic and diastolic function, pericardial disease, congenital heart disease and intracardiac masses.

There are different types of Echocardiography. They are

- 2D-Transthoracic Echocardiography
- Stress Echocardiography
- Doppler Echocardiography

- Transoesophageal Echocardiography
- Real time 3D Echocardiography

2D-TRANSTHORACIC ECHOCARDIOGRAPHY (TTE):

Single dimensional echocardiography records the motion of the heart at high frame rate 1000-2000 frames per second in M (motion based) mode. But two-dimensional echocardiography records at the rate of 30-2000 frames per second for the cross-sectional display of the cardiac structures.

CHAMBER QUANTITATION:

VENTRICULAR MEASUREMENT:

Left ventricular linear dimensions are one of the parameters which help in the management of patients with valvular heart disease especially in volume overload status and symptoms. LV internal dimensions at end diastole (LVIDd) and end systole (LVIDs) are measured at perpendicular to the long axis of the left ventricle at the level of cords or mitral leaflet at the tissue blood interface.

Normal LV internal dimensions at end diastole (LVIDd) is less than or equal to 5.3cm for male and 5.9cm for female.

American Society of Echocardiography recommended the following formula for the estimation of Left Ventricular Mass (LVM).

LV mass =0.8*[1.04*{(LVIDd+PWTd+IVSd)^3 - (LVIDd)^3}]+ 0.6gm

Where, LV = Left ventricle

LVIDd = LV internal diameter at end diastole

PWTd = Posterior Wall Thickness of LV at end diastole

IVSd = Inter Ventricular Septal wall thickness at end diastole

The normal reference range varies between men and women.

For men, 88-224 gm. For women, 67-162 gm.

Normal left ventricular wall thickness and Inter Ventricular Septal wall thickness at end diastole is $\leq$ 0.9 cm and more than 1.0 cm is considered to be hypertrophied left ventricle.

Right ventricular wall thickness > 5mm indicates right ventricular hypertrophy. Right ventricular mid cavity diameter at end diastole should be less than that of the left ventricle or else RV dilatation is said to be present.

RV functional volume measurement is challenging and it is measured by fractional area change of RV at end systole and end diastole. If < 35% it is said to be abnormal. Factors affecting right ventricular size are mainly the afterload and pressure changes and also diseases like myocardial infarction and right ventricular dysplasia.

LEFT ATRIUM:

The enlargement of left atrium is one of the predictors of cardiovascular outcomes. Left atrium is measured when it is at its largest dimension i.e. at ventricular end systole. Left atrium volumes are usually calculated by using area length. The normal LA volume is 22+/- 6ml/m^2 when it is indexed for BSA.

EJECTION FRACTION:

Left ventricular ejection fraction (LVEF) guides us for making therapeutic decision. It helps to identify the patients for drug therapy initiation (E.g. ACE inhibitors and Beta blockers in patients with LVEF < 40%) and for internal cardiac defibrillators implantation.

LVEF is calculated from end systolic volume and end diastolic volume.

$$EF = (EDV - ESV) / EDV$$

EDV- End Diastolic Volume

ESV – End Systolic Volume

Normal reference Range:

EF: $\geq$ 55%

EDV: $\leq$ 104 ml for female

$\leq$ 155 ml for male

DIASTOLIC FUNCTION:

Doppler Echocardiography is used to measure or assess the diastolic function. About half of the newly diagnosed heart failure patients have normal or near normal left ventricular systolic function but these patients are frequently having abnormal diastolic function. Mitral inflow velocities, pulmonary vein velocities and myocardial tissue velocities of left ventricle are also used to assess the left ventricular diastolic dysfunction. There are four major mitral inflow velocity parameters. They are

- Peak early filling (E) inflow velocity
- Late diastolic filling (A) inflow velocity
- E/A ratio
- Deceleration time (DT) of early filling inflow velocity (E')
- Deceleration time (DT) of late filling inflow velocity (A')

The mitral Peak early filling (E) inflow velocity mainly reflects the left atrial- left ventricle pressure gradient in early diastole. It is affected by preload and alterations in LV relaxation. Mitral late diastolic filling (A) inflow velocity mainly reflects the left atrial- left ventricle pressure gradient in late diastole and it is affected by Left ventricular compliance and LA contraction.

Diastolic Dysfunction grading:

Grade1: Normal where E velocity is dominant i.e. E/A is > 1, E/e' >1

Grade 2: impaired LV relaxation where A velocity is dominant i.e. E/A is < 1, E/e' <1

Grade 3: Pseudo normal where normal E velocity is dominance i.e. E/A is >1, E/e'<1

Grade 4: Restrictive filling where increased E velocity i.e. E/A is >2, E/e'<1.

TREATMENT OF CARDIAC FAILURE:

General principles for the management of cardiomyopathy focus on the following.

- Controlling the symptoms of congestion and maintaining euvolemic state.
- Stabilization of heart rate
- Controlling /maintaining the blood pressure
- Improve the exercise tolerance of the patient
- Prevention of sudden cardiac death and stroke

ACUTE DECOMPENSATION:

DIURETICS

Volume management should be done by using intravenous diuretic agents in the form of bolus or continuous infusion. Diuretics may be continued until the patient is euvolemic. Renal function and serum electrolytes must be monitored. Commonly used diuretic agents are furosemide, torsemide and bumetanide.

IONOTROPIC THERAPY

Ionotropic agents should be used when the patient presents with low output states hypotension and end organ dysfunction or shock states. Commonly used inotropes in practice are dobutamine, milrinone.

VASODILATORS

Vasodilators can be used in the presence of pulmonary congestion with preserved blood pressure for the rapid relief of symptoms like dyspnoea. Commonly used agents are nitro-glycerine infusion, nesiritide and nitroprusside.

ULTRAFILTRATION

It is a method of invasive fluid removal which may supplement the diuretic need in the management with the neutral effects on serum electrolytes.

NEUROHORMONAL ANATAGONISM

ACE inhibitors (angiotensin converting enzyme inhibitors) reduce 23% mortality in heart failure patients. Addition of beta blocker provides further 35% reduction in mortality. Hence, two drug combination should be used as initial therapy in HFrEF

First ACE inhibitors + beta blockers are used, if beta blockers is intolerant then ACE inhibitors + Angiotensin Receptor Blockers, if ACE inhibitors is intolerant then ARBs + beta blockers should be used. In all symptomatic patients of HFrEF NYHA class II to IV mineralocorticoid receptor antagonists are strongly recommended.

Commonly used beta blockers are carvedilol, bisoprolol and metoprolol, ACE inhibitors – lisinopril, enalapril and captopril, ARBs- losartan, valsartan, candesartan, Aldosterone antagonist- spironolactone, eplerenone.

ATRIOVENOUS VASODILATORS

Hydralazine and iso-sorbide dinitrate are used. Hydralazine acts by reducing systemic vascular resistance and induces arterial vasodilatation. Nitrates stimulate cyclic GMP production and causes arterio-venous vasodilatation.

HEART RATE MODIFICATION

Ivabradine, a funny I_f current inhibitor in sinoatrial node reduces the heart rate without a negative ionotropic effect. It should be considered before digoxin

after the standard first line therapy in patients with residual heart rate > 70 beats/min.

Digoxin, a cardiac glycoside decreases carotid sinus baroreceptor activity with mild ionotropic effect. It is sympatho- inhibitory by decreasing serum nor-epinephrine and plasma rennin levels. Digoxin is now used in refractorily symptomatic patients who are using other standard medications. Digoxin should be avoided in hypertrophic cardiomyopathy as increases the contractility and worsen the outcomes.

CALCIUM CHANNEL ANTAGONIST

Second generation calcium channel blockers like amlodipine and felodipine can be safely used to reduce blood pressure effectively in HFrEF. First generation calcium channel blockers like verapamil and diltiazem should be avoided as these agents having negative ionotropic effects. But verapamil should be considered in case of hypertrophic cardiomyopathy in front of second-generation drugs.

STATINS

Many studies like CORONA trial[24] showed rosuvastatin does not improve the outcomes in the heart failure patients. Still statins can be used in the case of ischaemic dilated cardiomyopathy and in progressive coronary artery disease patients.

ANTICOAGULANT AND ANTIPLATELET THERAPY

Hypercoagulable state accompanies the HFrEF posing high risk for thromboembolic events like pulmonary embolism, cerebrovascular accident and peripheral arterial embolism. Though there is increased risk the anticoagulant and antiplatelet therapy are not routinely advised in HFrEF as there is no difference in final outcomes 6 years of study. Warfarin is prescribed in setting of atrial fibrillation with appropriate internationalized ratio. Aspirin can be given in ischaemic dilated cardiomyopathy patients.

NUTRITIONAL THERAPY

Long chain omega 3 poly saturated fatty acids appear to be beneficial in improving the clinical outcomes in HFrEF by increasing the docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA).

Thiamine and selenium reverse the cardiomyopathy in their deficiency. But there is no evidence for suggesting routine supplementation/testing of these in HFrEF patients.

ENHANCED EXTERNAL COUNTERPULSATION (EECP)

Graded external pneumatic compression at high pressure at peripheral lower extremity with each session lasting for 1 hour and 5 per week for 7 weeks, totally 35 sessions improve the angina symptoms and exercise tolerance in patients with HFrEF in coronary artery disease.

CARDIAC RESYNCHRONIZATION THERAPY

Impaired systolic function and decreased mechanical efficiency of ventricular contraction happens due to the non-synchronous contraction of intraventricular or interventricular walls and these affects the ventricular filling significantly. This desynchrony worsens the functional mitral regurgitation and increase the LV wall stress.

Patients having wide QRS complexes (>149ms) and LBBB are good candidates for CRT. But RBBB patients, atrial fibrillation patients and patients having scar in the lateral wall are not good candidates for CRT.

PREVENTION OF SUDDEN CARDIAC DEATH (SCD)

Ventricular tachyarrhythmias are the mode of death in SCD in 50% of heart failure patients. Hence implantable cardioverter defibrillator (ICD) is advisable in high risk patients and who survived SCD episode. Now patients with LVEF < 35% and NYHA II or III symptoms of heart failure are good candidates for prophylactic ICD implantation irrespective of aetiology.

SURGICAL TREATMENT IN HEART FAILURE

Coronary artery bypass grafting (CABG) is beneficial in ischaemic cardiomyopathy with triple vessel disease. As mitral regurgitation in heart failure is functional one valvuloplasty or replacement is not much helpful. Restructuring

the shape of the heart in dominant anterior LV dysfunction and ischaemic cardiomyopathy is proposed but fails to show much clinical benefits in outcomes.

GLYCEMIC CONTROL IN CARDIAC PATIENTS

Compared with normal populations, diabetic populations have increased risk of developing coronary heart disease and death from the same by 2-4 times.

Hence the glycaemic control and maintaining the near normal metabolic status of the body is more important along with the other general and supportive measures in diabetic patients. The targeted HbA1c in cardiac patients should be < 7%, even close to 6% may targeted with meticulous monitoring for hypoglycaemia using insulin and other oral hypoglycaemic drugs. As the poor glycaemic status favours the progression of set cardiac dysfunction at faster rates which precipitates the mortality.

To achieve adequate glycaemic control sometimes insulin is required in patients with DM and HF. Insulin usage is associated with risk of hypoglycaemia and weight gain. Hence, it should be used with caution and close monitoring. Other oral hypoglycaemic agents, such as SGLT-2 inhibitors and metformin can be preferred if adequate glycaemic control will be achieved without insulin[25].

Metformin can be used in patients with diabetes mellitus who are at risk of or with established heart failure as long as estimated glomerular filtration rate (eGFR) more than 30 ml/min/1.73 m^{-2}. Metformin should be avoided in patients with acute conditions like lactic acidosis, cardiogenic or distributive shock.

It is preferable to use SGLT-2 (sodium glucose cotransporter type 2) inhibitors as the first class of glucose-lowering agents than sulfonylurea drugs in patients who are at high risk of HF and those with established HF. But the potential risks like genital candidiasis and other rare potential complications, such as diabetic ketoacidosis, lower-limb amputation, and fractures (the last two complications are seen only with canagliflozin).

Thiazolidinediones are not usually recommended in patients with established HF and as they may increase the risk of heart failure events in individuals with DM without HF. There is no strong evidence that dipeptidyl peptidase-4 (DPP-4) inhibitors shown any cardiovascular benefit. In patients with DM at high risk of cardiovascular disease, some DPP-4 inhibitors would increase the risk of HF hospitalization.

Management of DM and HF can be particularly difficult in patients with severely decreased renal function. In patients with eGFR <30 ml/min/1.73 m^{-2}, insulin can be used safely but might require lower doses and frequent monitoring of blood glucose levels. Other selected agents including glipizide, glimepiride and selected GLP-1 receptor agonists can be considered, but should be used carefully and may require dose adjustment.

MATERIAL AND METHODS

MATERIALS AND METHODS

STUDY DESIGN:

Cross sectional study

STUDY PERIOD:

1 year (April 2018 to May 2019)

STUDY POPULATION:

Type 2 Diabetes mellitus patients more than 30 years of age attending the medical Out Patient Department.

STUDY SETTING:

The study is to be conducted in the Department of General Medicine and Cardiology, Chengalpattu Medical College, Chengalpattu.

SAMPLE SIZE:

200 [minimum sample size arrived 171- ($n= Z^2 pq/d^2$)**]**

SAMPLING METHOD:

Convenient sampling.

OPERATIONAL DEFINITION:

Diabetes mellitus is defined as person who is having **FBS ≥126;**

PPBS ≥ 200

INCLUSION CRITERIA:

- Age above 30 years.
- Both in known type 2 diabetic patients and newly diagnosed T2DM patients.

EXCLUSION CRITERIA:

- Known coronary artery disease patients
- Known Valvular heart disease patients
- Hypertension
- Chronic Alcoholics
- Smokers
- Chronic Obstructive Pulmonary Disease patients

Well informed and written consent was obtained from the study population. Patients were interviewed by structured questionnaire for the history, symptoms if any, duration and details of the disease and treatment that they are undergoing.

All patients satisfying the inclusion criteria were included in this study, documented and subjected to 2D echocardiography at cardiology department, Chengalpattu Medical College, Chengalpattu.

All the eligible candidates are screened with complete physical examination, blood pressure measurement, renal function test, liver function tests to rule out hypertension, renal disease and liver disease which could affect the outcomes of this study.

2D-Echocardiography was done in all patients. The following parameters are measured in the Parasternal long axis (PLAX view).

LA – Left Atrial chamber volume

E - Peak mitral early filling velocity

A - Peak mitral late filling velocity by atrial contraction

IVSd - Interventricular septal thickness at end diastole

LVPWd - Left ventricular posterior wall thickness at end diastole

EDV- End diastolic volume

ESV - End systolic volume

EF - Ejection fraction

LVM - Left ventricular mass

LVIDd - Left ventricular internal diameter at end diastole.

RESULTS

RESULTS

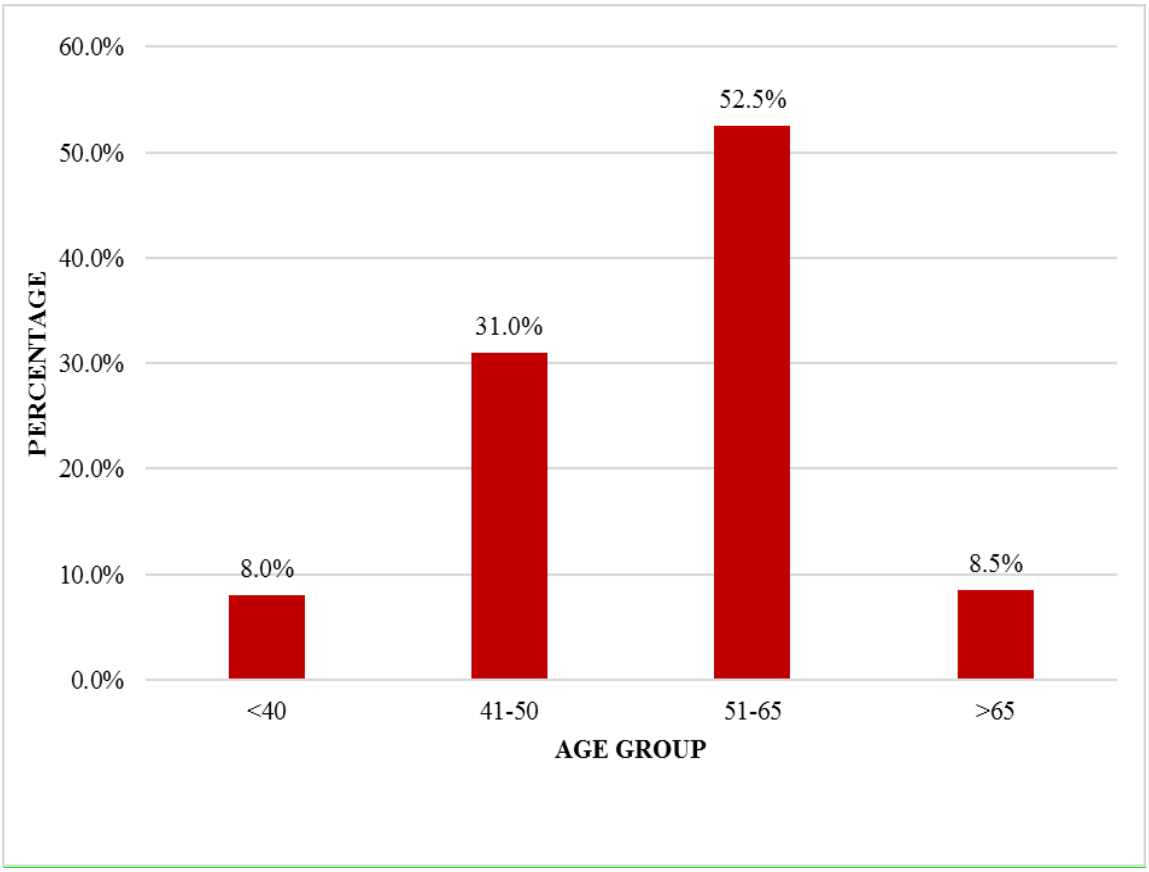
A total of 200 subjects were included in the final analysis.

TABLE 1: DISTRIBUTION OF AGE (YEARS) IN STUDY POPULATION (N=200)

AGE GROUP	NO. OF PATIENTS	PERCENTAGE
< 40	16	8.0%
41 - 50	62	31.0%
51 - 65	105	52.5%
> 65	17	8.5%

In the study population majority of the patients were in the age group 51-65 years (52.5%) followed by 41-50 years (31%) , more than 65 years (8.5%) and less than 40 years (8%).

FIGURE 1: BAR CHART OF AGE GROUP IN THE STUDY POPULATION (N=200)

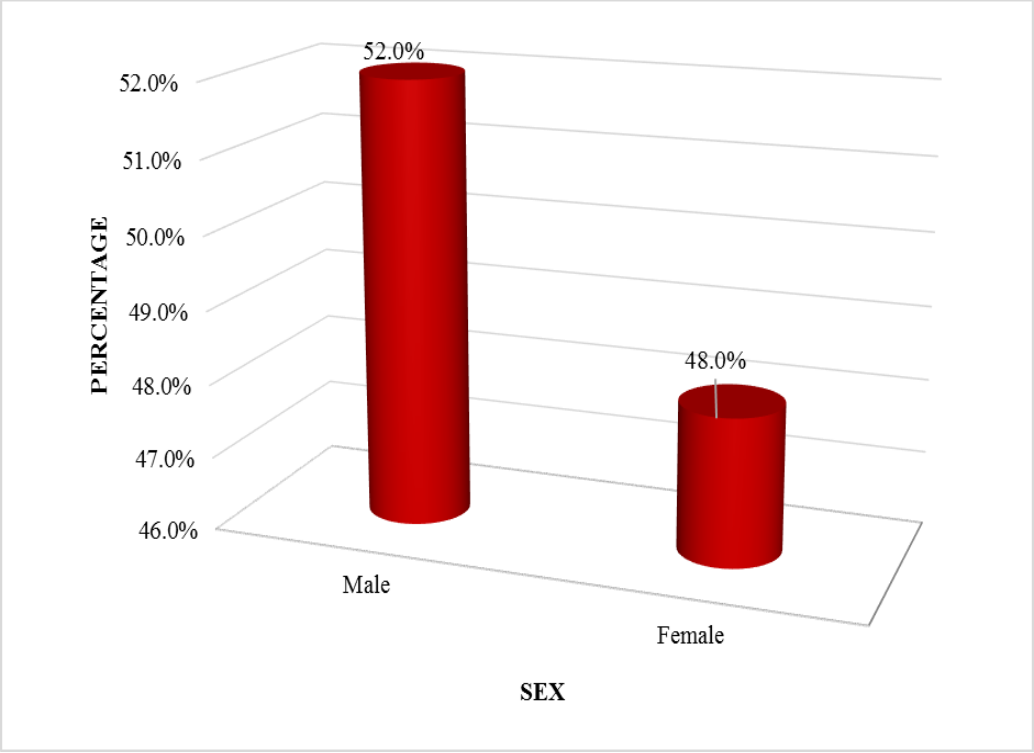


The bar diagram shows, majority of patients were in the age group 51-65 years (52.5%) followed by 41-50 years (31%), more than 65 years (8.5%) and less than 40 years (8%).

TABLE 2: DISTRIBUTION OF SEX IN THE STUDY POPULATION (N=200)

GENDER	NO. OF PATIENTS	PERCENTAGE
Male	104	52.0%
Female	96	48.0%

FIGURE 2: BAR CHART OF SEX IN THE STUDY POPULATION (N=200)



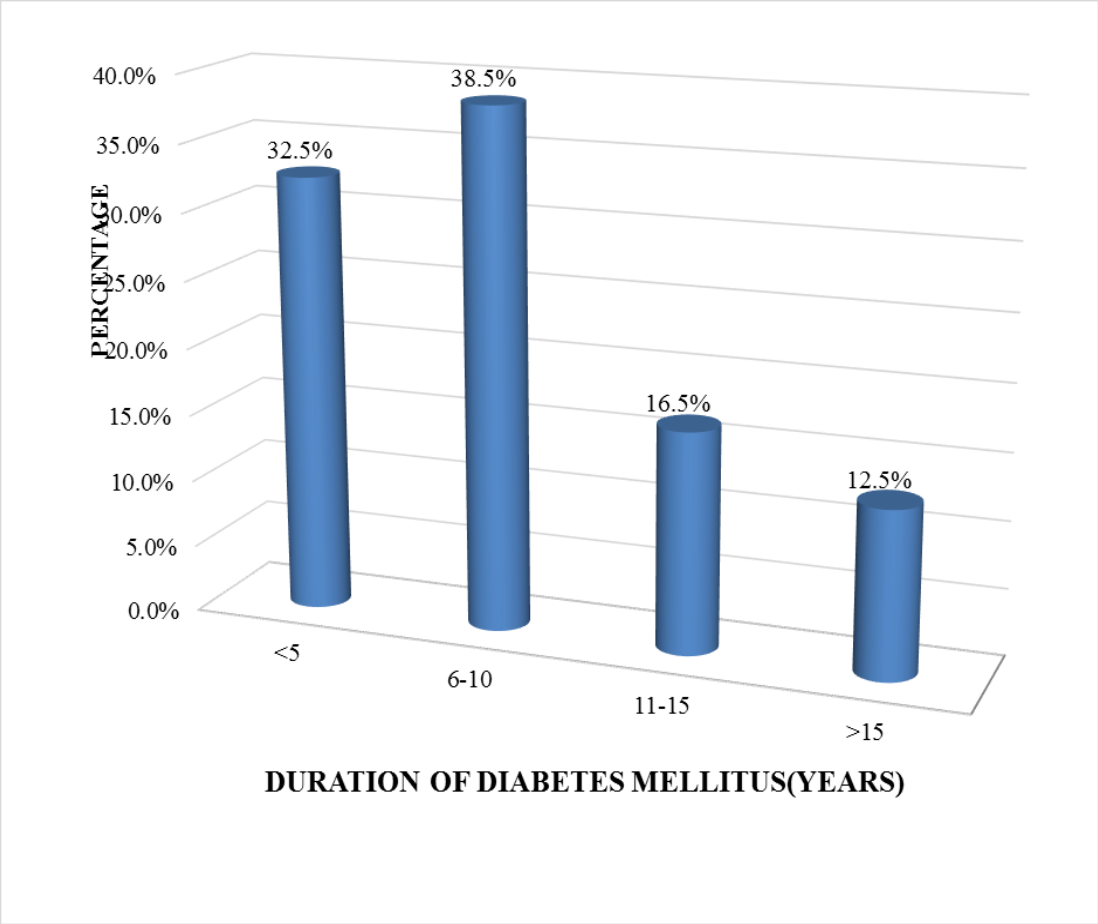
In this study, majority of the patients were male with 52% and female were 48%.

TABLE 3: DESCRIPTIVE ANALYSIS OF DURATION OF DIABETES MELLITUS IN THE STUDY POPULATION (N=200)

DURATION OF DIABETES MELLITUS (YEARS)	NO. OF PATIENTS	PERCENTAGE
< 5	65	32.5%
6 - 10	77	38.5%
11 - 15	33	16.5%
> 15	25	12.5%

In our study, about 77 patients had diabetes mellitus for duration of 6- 10 years (38.5%).

FIGURE 3: BAR CHART OF DURATION OF DIABETES MELLITUS IN THE STUDY POPULATION (N=200)

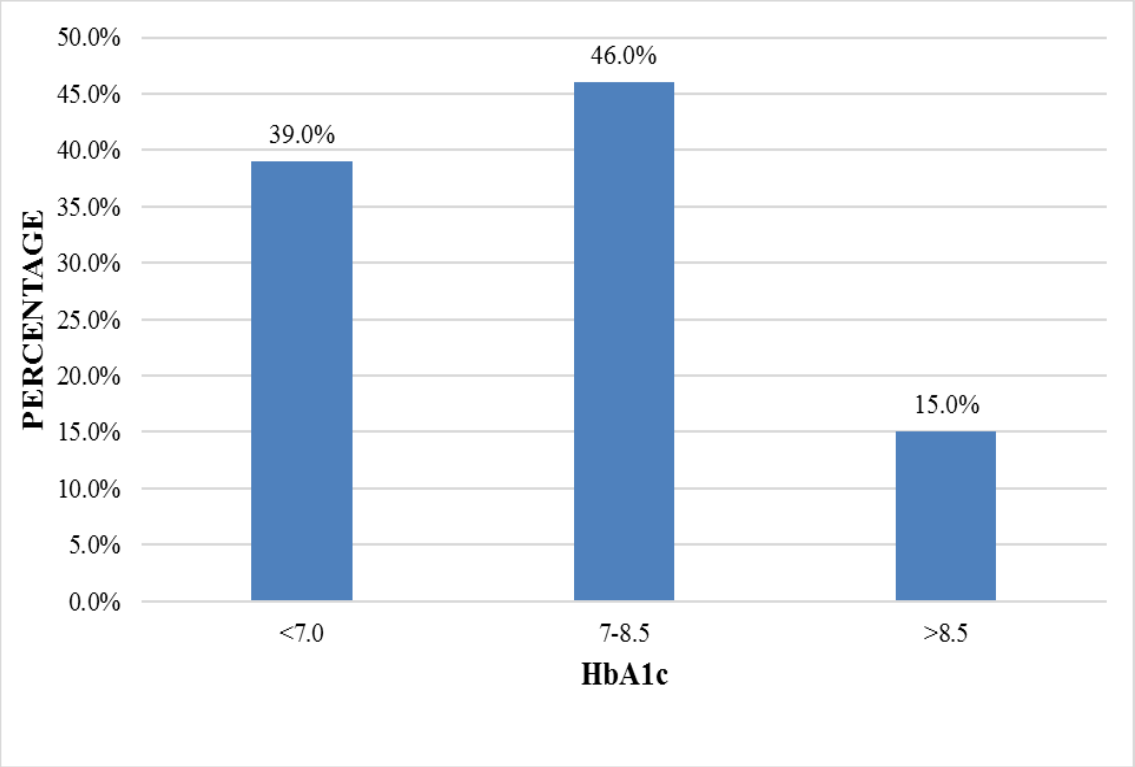


This bar chart shows, about 77 patients had Diabetes mellitus for duration of 6- 10 years (38.5%).

TABLE 4: DESCRIPTIVE ANALYSIS OF HbA1c IN THE STUDY POPULATION (N=200)

HbA1c	NO. OF PATIENTS	PERCENTAGE
< 7.0	78	39.0%
7 - 8.5	92	46.0%
> 8.5	30	15.0%

FIGURE 4: BAR CHART OF HbA1c IN THE STUDY POPULATION (N=200)

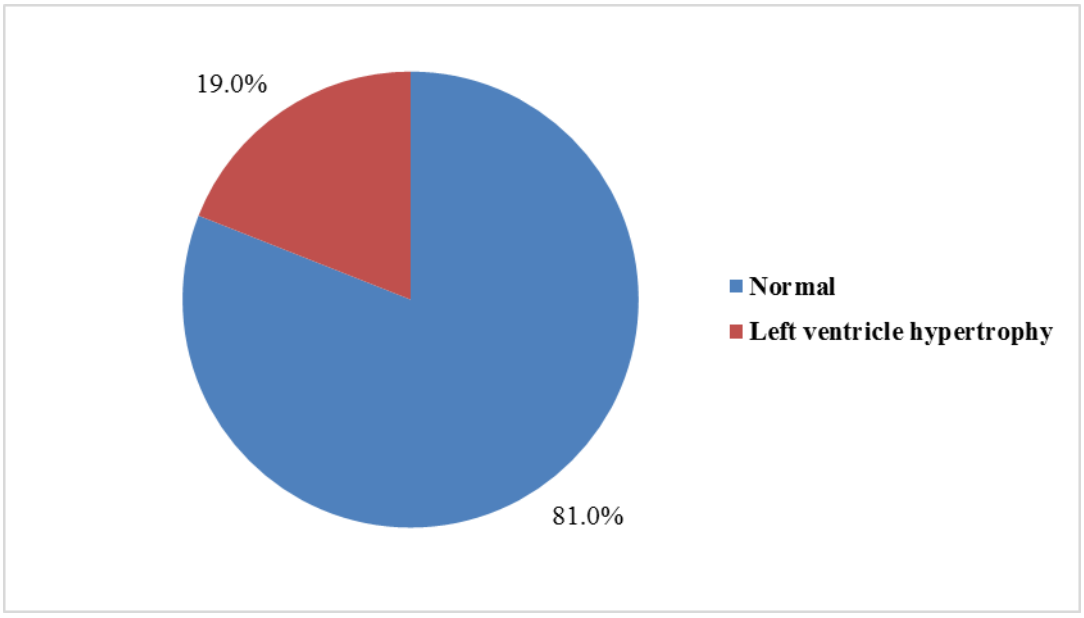


In our study population, most of the patients having the HbA1c fall in the group with range of 7 - 8.5.

TABLE 5: DISTRIBUTION OF LEFT VENTRICUALR HYPERTROPHY BASED ON IV SEPTAL WALL THICKNESS

SEPTAL WALL THICKNESS	NO. OF PATIENTS	PERCENTAGE
Normal (< 1cm)	162	81.0%
Left ventricle hypertrophy (>1cm)	38	19.0%

FIGURE 5: PIE CHART OF LEFT VENTRICULAR HYPERTROPHY IN THE STUDY POPULATION (N=200)

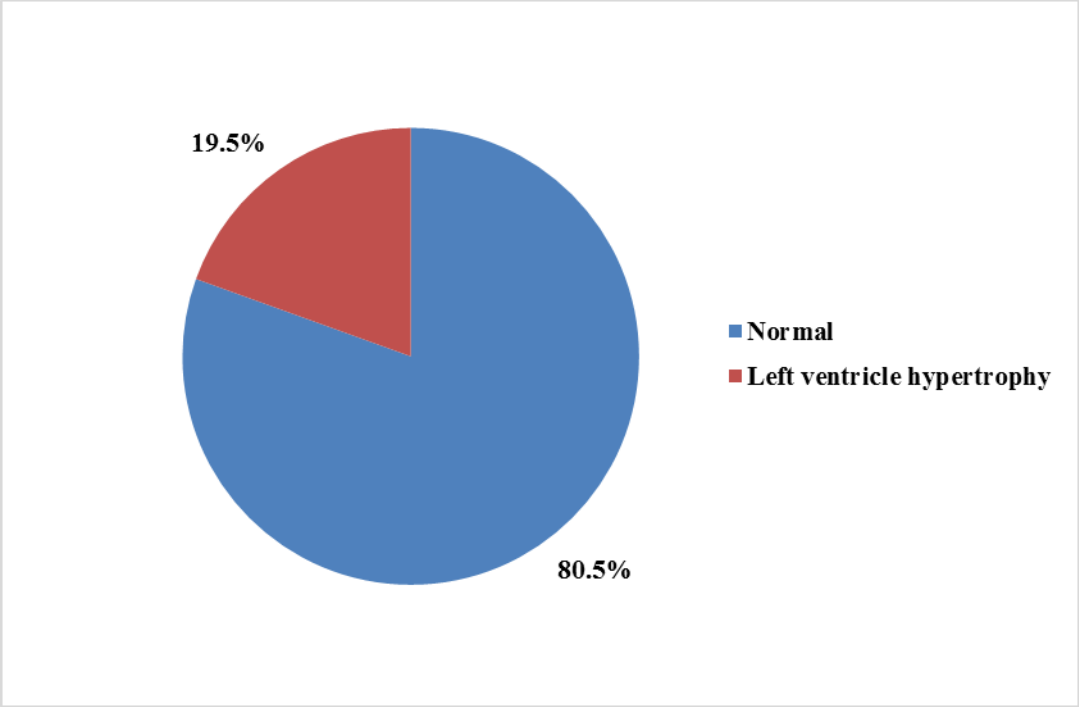


In our study, 38 patients had left ventricular hypertrophy (19%) with increased septal wall thickness.

TABLE 6: DISTRIBUTION OF LEFT VENTRICUALR HYPERTROPHY BASED ON LV POSTERIOR WALL THICKNESS (LVPWd):

LVPWd	FREQUENCY	PERCENTAGE
Normal (<1cm)	161	80.5%
Left ventricle hypertrophy (>1cm)	39	19.5%

FIGURE 6: PIE CHART OF LEFT VENTRICULAR HYPERTROPHY IN THE STUDY POPULATION (N=200) BASED ON LVPWD.

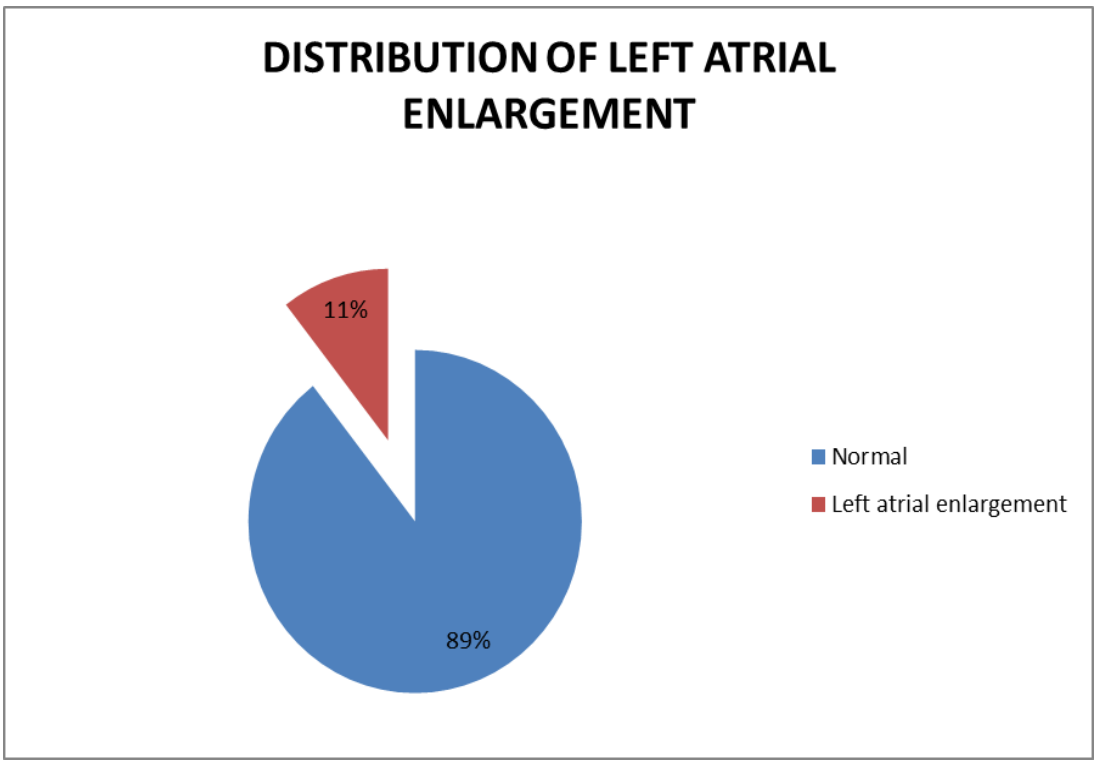


In our study, 39 patients had left ventricular hypertrophy (19.5%) with increased left ventricular posterior wall thickness.

TABLE 7: DISTRIBUTION OF LEFT ATRIAL ENLARGEMENT IN THE STUDY POPULATION (N=200)

LEFT ATRIUM	NO. OF PATIENTS	PERCENTAGE
Normal	179	89.5%
Left atrial enlargement	21	10.5%

FIGURE 7: PIE CHART OF LEFT ATRIAL ENLARGEMENT IN THE STUDY POPULATION (N=200)



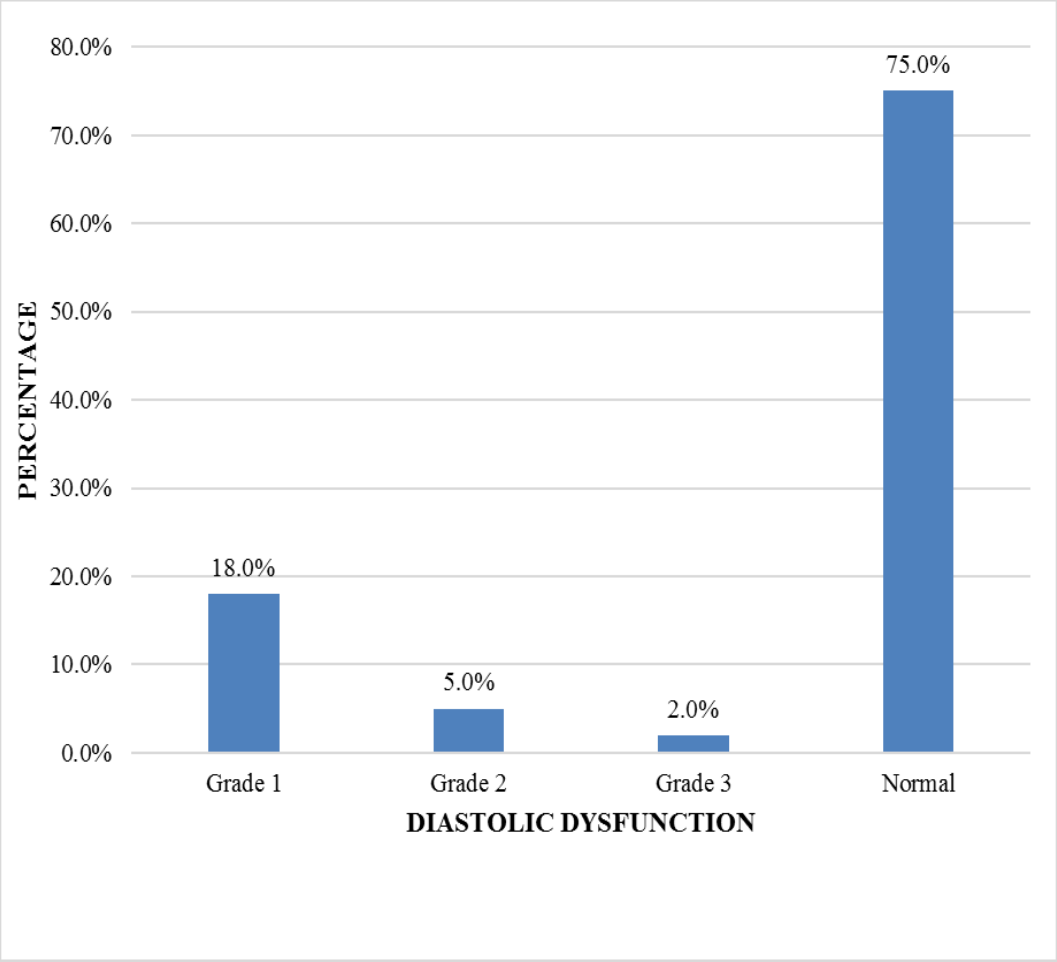
In our study group, 10.5% of patient had enlarged left atrium.

TABLE 8: DESCRIPTIVE ANALYSIS OF DIASTOLIC DYSFUNCTION IN THE STUDY POPULATION (N=200)

DIASTOLIC DYSFUNCTION	NO. OF PATIENTS	PERCENTAGE
Grade 1	36	18.0%
Grade 2	10	5.0%
Grade 3	4	2.0%
Normal	150	75.0%

In our study, 25% of population found to have diastolic dysfunction of which18% of patients with grade 1, 5% of patients had grade 2 and 2% of patients had grade diastolic dysfunction.

FIGURE 8: BAR CHART OF DIASTOLIC DYSFUNCTION IN THE STUDY POPULATION (N=200)

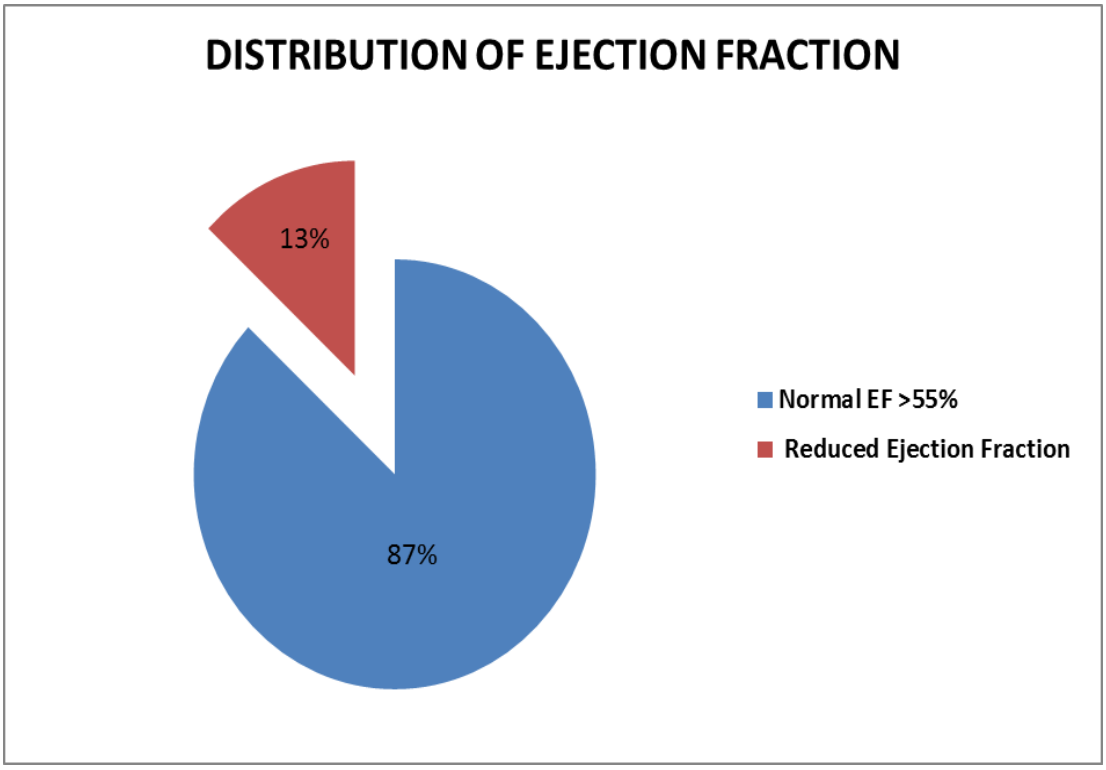


This bar diagram shows, 18% of patients with grade 1 diastolic dysfunction, 5% of patients had grade 2 and 2% of patients had grade 3 diastolic dysfunction.

TABLE 9: DESCRIPTIVE ANALYSIS OF EJECTION FRACTION IN THE STUDY POPULATION (N=200)

EJECTION FRACTION	NO. OF. PATIENTS	PERCENTAGE
Normal (> 55%)	174	87.0%
Reduced EF (< 55%)	26	13.0%

FIGURE 9: PIE CHART OF EJECTION FRACTION IN THE STUDY POPULATION (N=200)

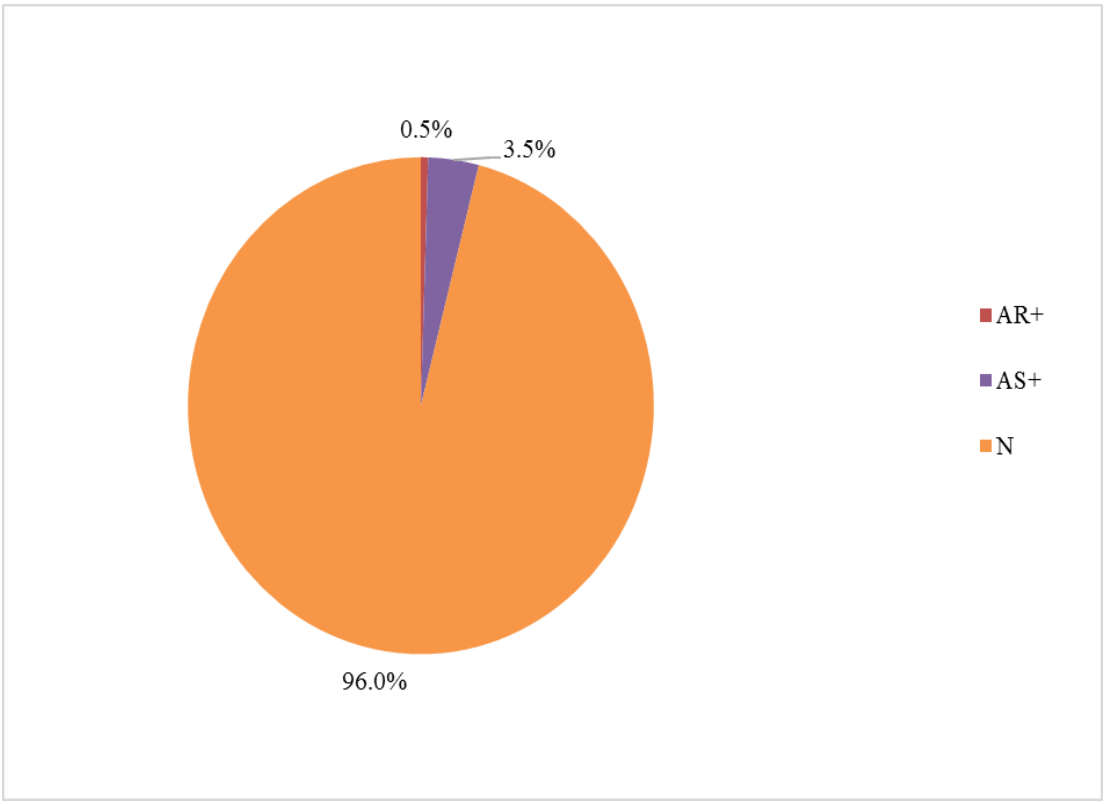


In this study, 13% of population had reduced ejection fraction in the echocardiography.

TABLE 10: DESCRIPTIVE ANALYSIS OF AORTIC VALVE IN THE STUDY POPULATION (N=200)

AORTIC VALVE	FREQUENCY	PERCENTAGE
Aortic regurgitation (AR)	1	0.5%
Aortic stenosis (AS)	7	3.5%
Normal (N)	192	96.0%

FIGURE 10: PIE CHART OF AORTIC VALVE IN THE STUDY POPULATION (N=200)

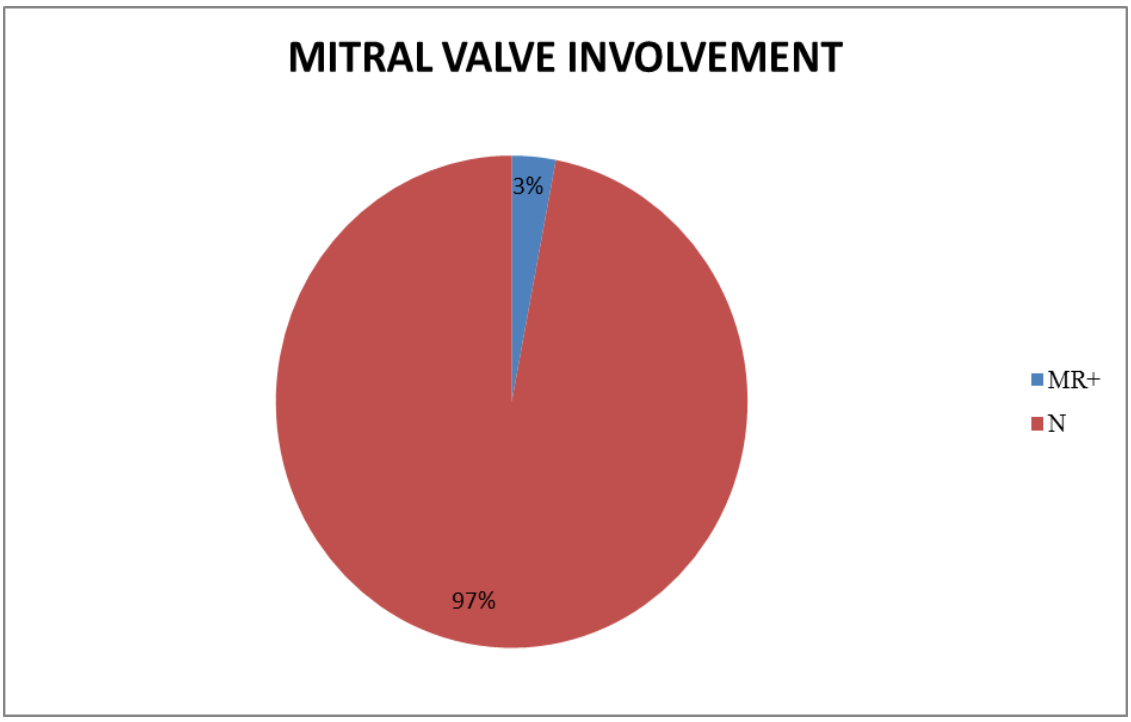


In our study, 4% of population had aortic valvular involvement. The most common lesion is Aortic stenosis (3.5%)

TABLE 11: DESCRIPTIVE ANALYSIS OF MITRAL VALVE IN THE STUDY POPULATION (N=200)

Mitral Valve	Frequency	Percentages
Mitral regurgitation (MR)	6	3%
Normal (N)	194	97%

FIGURE 11: PIE CHART OF MITRAL VALVE IN THE STUDY POPULATION (N=200)

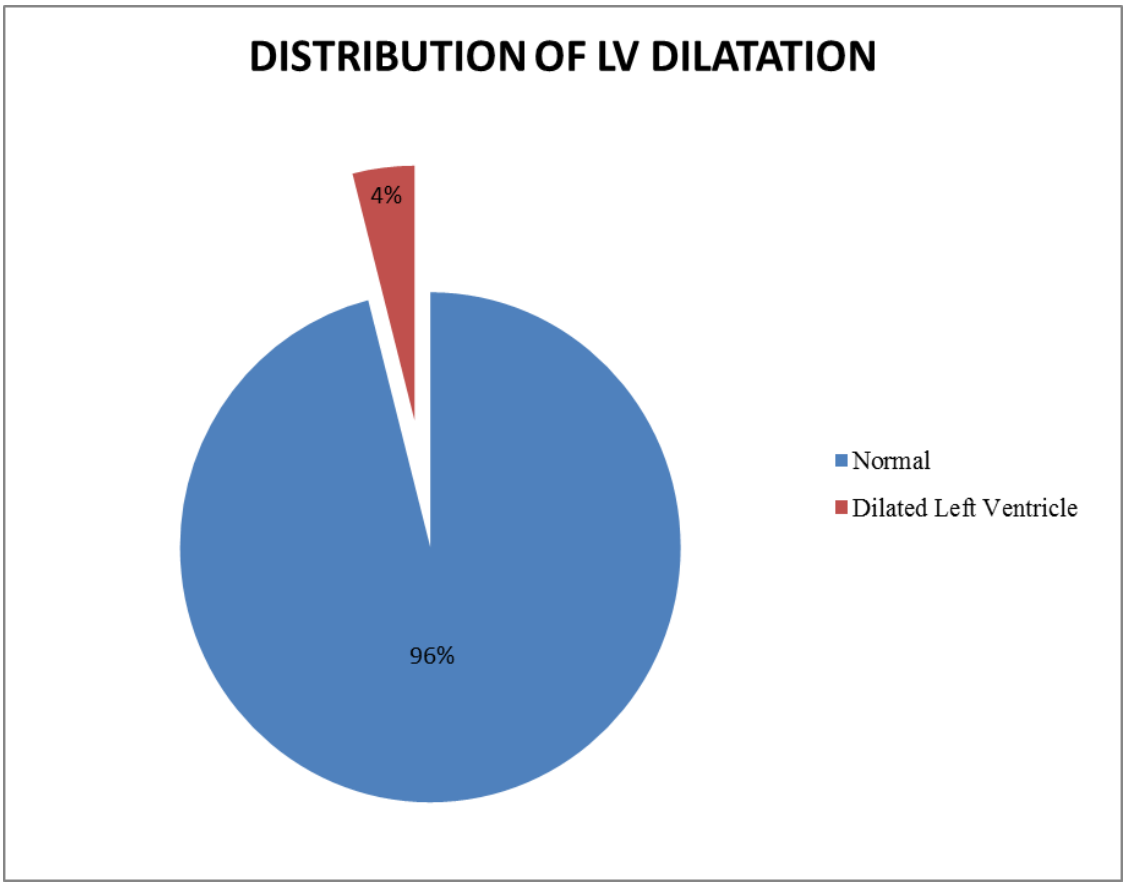


In our study, 3 % of population had mitral valve regurgitation.

TABLE 12: DESCRIPTIVE ANALYSIS OF LEFT VENTRICLE DILATATION IN THE STUDY POPULATION (N=200)

LVID	FREQUENCY	PERCENTAGES
Normal	192	96%
Dilated left ventricle	8	4%

FIGURE 12: DISTRIBUTION OF LEFT VENTRICLE DILATATION



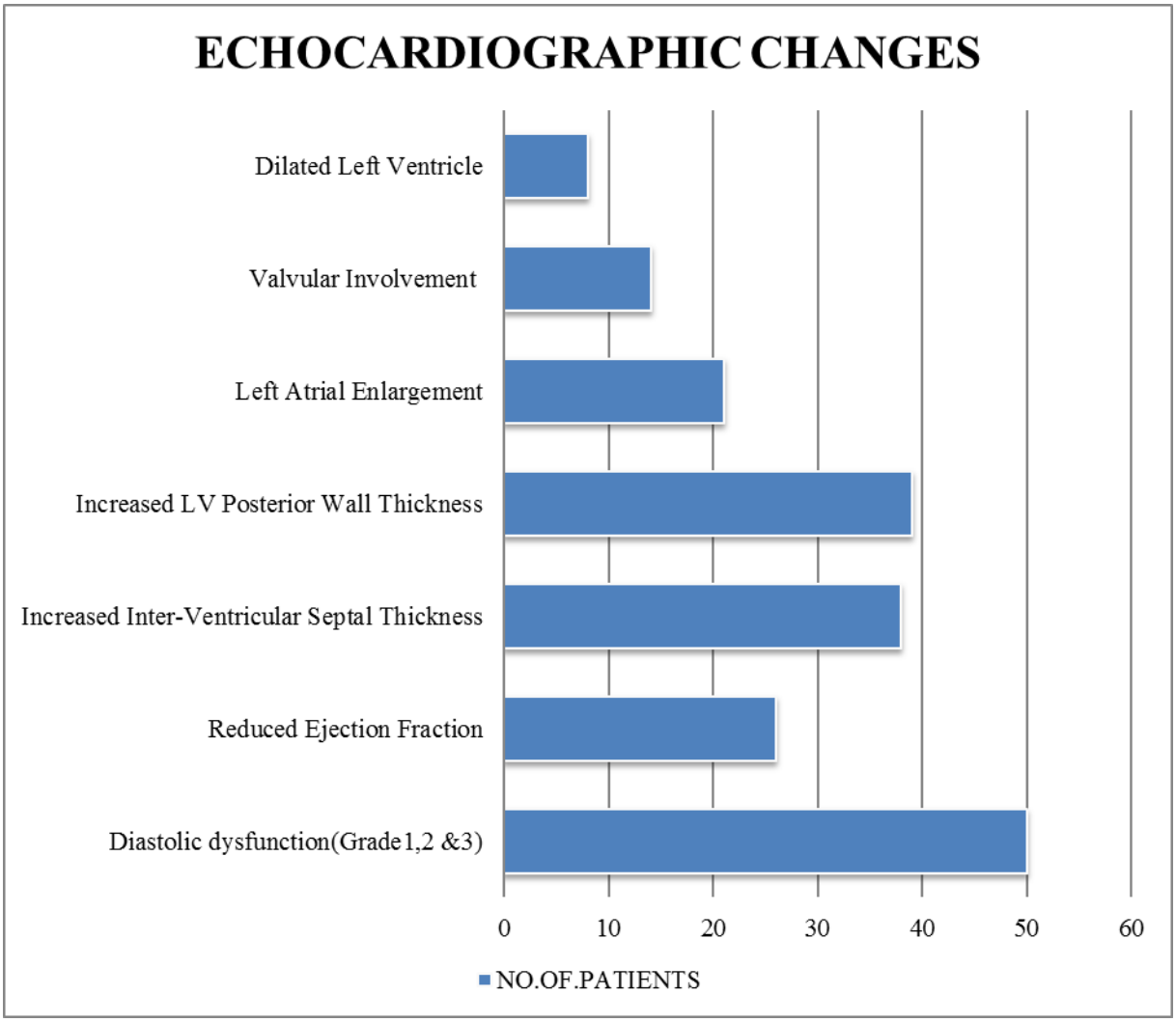
In this study group, 4% had enlarged left ventricle.

TABLE 13: ECHOCARDIOGRAPHIC CHANGES IN THE STUDY GROUP

ECHO CHANGES	NO. OF PATIENTS	PERCENTAGE
Diastolic dysfunction (Grade1,2 &3)	50	25%
Reduced Ejection Fraction	26	13%
Increased Inter-Ventricular Septal Thickness	38	19%
Increased LV Posterior Wall Thickness	39	19.5%
Left Atrial Enlargement	21	10.5%
Valvular Involvement	14	7%
Dilated Left Ventricle	8	4%

In our study, about 50 patients were found to have diastolic dysfunction and about 39 patients showed left ventricular hypertrophy in the form of increased inter-ventricular septal thickness and increased LV posterior wall thickness.

FIGURE 13: BAR DIAGRAM SHOWING ECHOCARDIOGRAPHIC CHANGES IN THE STUDY GROUP



The bar diagram shows that most common finding was diastolic dysfunction followed by the left ventricular hypertrophy and other findings.

TABLE 14: COMPARISON OF ECHOCARDIOGRAPHIC CHANGES IN TYPE 2 DIABETES PATIENTS ACROSS AGE GROUP (N=200)

Parameters	Age Groups				Chi square	P value
	< 40 (N=16)	41-50 (N=62)	51- 65 (N=105)	> 65 (N=17)		
Left Atrial Enlargement						
LA	1 (6.25%)	1 (1.61%)	9 (8.57%)	10 (58.82%)	48.177	< 0.001
Diastolic Dysfunction						
Grade 1	5 (31.25%)	11 (17.74%)	18 (17.14%)	2 (11.76%)	*	**
Grade 2	0 (0%)	3 (4.84%)	6 (5.71%)	1 (5.88%)		
Grade 3	0 (0%)	2 (3.23%)	1 (0.95%)	1 (5.88%)		
Reduced Ejection Fraction						
EF (%)	1 (6.25%)	6 (9.68%)	16 (15.23%)	3 (17.64%)	4.128	0.248
Left Ventricle Hypertrophy						
IVSd	2 (12.5%)	5 (8.06%)	21 (20%)	10 (58.82%)	22.843	< 0.001
LVPWd	2 (12.5%)	5 (8.06%)	21 (20%)	11 (64.71%)	27.813	< 0.001
LV mass						
Left Ventricle hypertrophy	4 (25%)	9 (14.52%)	17 (16.19%)	6 (35.29%)	12.526	0.051
LVID						
Dilated Left Ventricle	1 (6.25%)	3 (4.84%)	3 (2.85%)	1 (5.88%)	3.442	0.328
Valvular Involvement						
Aortic Stenosis	0 (0%)	0 (0%)	5 (4.76%)	2 (11.76%)	*	**
Aortic Regurgitation	0 (0%)	0 (0%)	0 (0%)	1 (5.88%)		
Mitral Regurgitation	0 (0%)	0 (0%)	1 (0.95%)	5 (29.41%)		

*No statistical test was applied- due to 0 subjects in the cells

TABLE 15: COMPARISON OF ECHOCARDIOGRAPHIC CHANGES IN TYPE 2 DIABETES PATIENTS ACROSS DURATION OF DM (N=200)

Parameters	Duration of DM				Chi square	P value
	<5 (N=65)	6-10 (N=77)	11-15 (N=33)	>15 (N=25)		
Left Atrial Enlargement						
LA	3 (4.62%)	5 (6.49%)	5 (15.15%)	8 (32%)	16.767	<0.001
Diastolic Dysfunction						
Grade 1	10 (15.38%)	15 (19.48%)	8 (24.24%)	3 (12%)	*	**
Grade 2	1 (1.54%)	2 (2.6%)	4 (12.12%)	3 (12%)		
Grade 3	0 (0%)	1 (1.3%)	1 (3.03%)	2 (8%)		
Reduced Ejection Fraction						
EF (%)	4 (6.15%)	10 (12.99%)	6 (18.18%)	6 (24%)	6.152	0.104
Left Ventricle Hypertrophy						
IVSd	6 (9.23%)	9 (11.69%)	11 (33.33%)	12 (48%)	24.772	<0.001
LVPWd	6 (9.23%)	10 (12.99%)	11 (33.33%)	12 (48%)	23.406	<0.001
LV mass						
Left Ventricle Hypertrophy	8 (12.31%)	14 (18.18%)	8 (24.24%)	6 (24%)	13.002	0.043
LVID						
Dilated Left Ventricle	1 (1.54%)	3 (3.9%)	2 (6.06%)	2 (8%)	5.757	0.124
Valvular Involvement						
Aortic Stenosis	0 (0%)	1 (1.3%)	3 (9.09%)	3 (12%)	*	**
Aortic Regurgitation	0 (0%)	1 (1.3%)	0 (0%)	0 (0%)		
Mitral Regurgitation	1 (1.54%)	1 (1.3%)	1 (3.03%)	3 (12%)		

*No statistical test was applied- due to 0 subjects in the cells

TABLE 16: COMPARISON OF ECHOCARDIOGRAPHIC CHANGES IN TYPE 2 DIABETES PATIENTS ACROSS HbA1c VALUES (N=200)

Parameters	HbA1c			Chi square	P value
	<7.0 (N=78)	7-8.5 (N=92)	>8.5 (N=30)		
Left Atrial Enlargement					
LA	5 (6.41%)	7 (7.61%)	9 (30%)	14.346	<0.001
Diastolic Dysfunction					
Grade 1	15 (19.23%)	15 (16.3%)	6 (20%)	*	**
Grade 2	3 (3.85%)	6 (6.52%)	1 (3.33%)		
Grade 3	1 (1.28%)	3 (3.26%)	0 (0%)		
Reduced Ejection Fraction					
EF (%)	3 (3.85%)	14 (15.21%)	9 (30%)	0.810	0.667
Left Ventricle Hypertrophy					
IVSd	11 (14.1%)	12 (13.04%)	15 (50%)	22.070	<0.001
LVPWd	11 (14.1%)	13 (14.13%)	15 (50%)	20.916	<0.001
LV mass					
Left Ventricle hypertrophy	12 (15.38%)	18 (19.57%)	6 (20%)	5.910	0.206
LVID					
Dilated Left Ventricle	1 (1.28%)	5(5.43%)	2(6.66%)	1.419	0.492
Valvular Involvement					
Aortic Stenosis	2 (2.56%)	4 (4.35%)	1 (3.33%)	*	**
Aortic Regurgitation	0 (0%)	1 (1.09%)	0 (0%)		
Mitral Regurgitation	0 (0%)	3 (3.26%)	3 (10%)		

*No statistical test was applied- due to 0 subjects in the cells.

TABLE 17: COMPARISON OF ECHOCARDIOGRAPHIC CHANGES IN TYPE 2 DIABETES PATIENTS BETWEEN GENDER (N=200)

Parameters	Gender		Chi square	P value
	Male (N=104)	Female (N=96)		
Left Atrial Enlargement				
LA	7 (6.73%)	14 (14.58%)	3.276	0.070
Diastolic Dysfunction				
Grade 1	19 (18.27%)	17 (17.71%)	2.395	0.495
Grade 2	7 (6.73%)	3 (3.13%)		
Grade 3	3 (2.88%)	1 (1.04%)		
Reduced Ejection Fraction				
EF (%)	10 (9.61%)	16 (16.66%)	5.319	0.021
Left Ventricle Hypertrophy				
IVSd	20 (19.23%)	18 (18.75%)	0.007	0.931
LVPWd	21 (20.19%)	18 (18.75%)	0.066	0.797
LV mass				
Left Ventricle hypertrophy	18 (17.31%)	18 (18.75%)	0.680	0.712
LVID				
Dilated Left Ventricle	3 (2.88%)	5 (5.20%)	0.296	0.673
Valvular Involvement				
Aortic Stenosis	1 (0.96%)	6 (6.25%)	*	**
Aortic Regurgitation	1 (0.96%)	0 (0%)		
Mitral Regurgitation	3 (2.88%)	3 (3.13%)		

*No statistical test was applied- due to 0 subjects in the cells.

DISCUSSION

DISCUSSION

This study was done to find out the prevalence of echocardiographic abnormalities in the type 2 diabetes patients who are normal, without any symptoms.

The mean age of the study population in this study: 53.64 years with minimum age of 34 years and maximum age of 82. Sebhat Erqou et al., done a meta-analysis study where the mean age was found to be 60 years [26].

Zabalgoitia et al., done a study with mean age 46 +/- 6 years, to find the prevalence of diastolic dysfunction in type 2 diabetes mellitus [27].

In our study population, 52 percentage were males and 48 percentage were females, among these men tend to have more left ventricular hypertrophy and more diastolic dysfunction, women more often had more reduced ejection fraction, and left atrial enlargement.

This correlates with a study conducted by Jorgensen et al[5].

The Framingham Heart Study reveals that in diabetes mellitus the risk of developing HF raises up to 5-times in women and 2 times in men compared with age-matched controls, [11] [28] but highlighting a sex discrepancy between male and female is incompletely understood.

In this study, mean duration of diabetes mellitus of the participants is approximately 9 years.

In our study, 46 % of study population having HbA1c in the range 7 - 8.5 %, 39 % of patients had HbA1c < 7% and 15% of patients had > 8.5%. We found that the incidence of diastolic dysfunction is lower in the group of patients with HbA1c 7 - 8.5 and increased occurrence in groups with HbA1c > 8.5.

Aguilar et al found that association between mortality and HbA1c in diabetes mellitus patients with heart failure appears to be U - shaped, with the lowest risk of mortality in patients having HbA1c levels of approximately 7.1% [11,29].

Poor glycaemic control is associated with higher risk of developing heart failure. For each 1% increase in glycosylated haemoglobin (HbA1c), the risk of incident heart failure increases by 8% to 36%.

Various studies show that Intensive glycaemic control does not appear to decrease the risk of all-cause mortality, stroke or cardiovascular mortality. But it might reduce the risk of nonfatal myocardial infarction. Therefore, HbA1c goal should be individualized in patients with DM and HF based on the patient's functional and clinical status (presence of complications of DM, life expectancy, comorbidities), self-management capacity, history of hypoglycaemic episodes and overall the treatment burden.

Many other studies proposes that diabetes mellitus is independently associated with higher risk of death and readmission in hospitals compared with non-diabetics with heart failure[30] .

It was estimated that the prevalence of asymptomatic diastolic dysfunction as 27% in the general population. Left ventricular diastolic dysfunction (LVDD) is used to describe as an early preclinical indication of a specific cardiomyopathy in patients with diabetes mellitus without known coronary artery disease and hypertension [31].

A study by Exiara et al., shown that there is a strong correlation between duration of diabetes mellitus and diastolic dysfunction [31]. In our study group, among the patients with diastolic dysfunction 36% of them had duration of diabetes more than 10 years and 23% of them had duration of DM 5 -10 years. Our study also shows a positive correlation between the duration of diabetes mellitus and diastolic dysfunction.

A study by Aaron M From et al., showed that there is a direct relationship between the LV diastolic dysfunction and the duration of DM. They further said that significant LV diastolic dysfunction manifest 4 years after the onset of diabetes mellitus independent of hypertension and coronary disease [32].

Kannel et al., suggests that echocardiographic findings with left ventricular hypertrophy signifies a high risk of developing heart failure, proportional to the amount of increase in left ventricular mass not relating to a critical value for compensatory pathological hypertrophy [33].

In our study about 39 (19.5%) patients showed left ventricular hypertrophy in the form of increased inter-ventricular septal thickness and increased LV

posterior wall thickness and about 36 patients were found to have left ventricular hypertrophy in the form of increased left ventricular mass.

Our study correlates with Fang et al., findings in screening for heart disease among diabetic patients without known heart disease shows echocardiographic LVH in about 22% [34].

Eguchi et al., performed a study and showed diabetes mellitus was associated with 1.7 times increase in risk of having Left Ventricular hypertrophy [35] and likelihood of having Left ventricular mass above the 75th percentile of the distribution was 1.46-times more in diabetics. Similar results were also shown by Palmieri et al., [36].

DM is also an important predictor of the development of symptomatic HF in patients with asymptomatic left ventricular (LV) systolic dysfunction [37]. Many previous studies have shown that LV systolic dysfunction in the patients with DM were ranged from 5% to 21.8% [38].

In this study, 13% of population had reduced ejection fraction in the echocardiography. A study by Niklas etal., revealed that diabetic patients had a significantly reduced left ventricular EF when compared with non-diabetics irrespective of the extent of Coronary Artery Disease [39].

Diabetes mellitus remains strongly associated with non-rheumatic aortic valve disease. In our study population 4% had aortic valve involvement with commonly stenotic lesion (3.5% AS) and 3% population had mitral regurgitation.

A study by Movahed shows that non-rheumatic aortic valve disease was found in 2.5% of DM patients[40] .

In 2017, a study by Rossi et al., 814 diabetic patients were included and observed that 73% patients had normal mitral valve, 18% of patients had isolated calcification of the mitral valve, and 9% of patients had other mitral valve diseases [41].

Impaired LA function may be found in newly diagnosed asymptomatic Diabetes mellitus patients. Changes in left atrial size and function are also associated with adverse clinical events [42].

Recently, duration of Diabetes mellitus (DM2) has been found to be associated positively with increased left atrial volume and impaired left atrial function. In our study group, 10.5% of patient had enlarged left atrium.

CONCLUSION

CONCLUSION

Type 2 diabetes mellitus is one of the wide spread medical disorder leading to many complications. It is the one of the major risk factor cardiovascular mortality and also increase in the incidence of cardiac failure.

Though heart failure is asymptomatic in the early period of type 2 diabetes mellitus, the symptoms are same and severe as that of any type of cardiomyopathy in the poorly controlled diabetic patients. The severity of heart failure progresses much faster in diabetic patients than the non-diabetics.

The most common echocardiographic changes in our study is diastolic dysfunction of about 25% of patients, followed by left ventricular hypertrophy which is 19.5%, systolic dysfunction in 13%, left atrial enlargement in 10.5%.

From this study it was observed that the incidence of diastolic dysfunction is increased in patients with uncontrolled glycaemic levels (HbA1c > 7%).

Early subclinical findings of asymptomatic systolic and diastolic dysfunction appear in echocardiography before the overt heart failure and cardiomyopathy occurs in type 2 Diabetes mellitus patients.

Thus, Echocardiography is one of the important screening tool and predictor of heart failure in type 2 diabetic patients who is at increased risk of having asymptomatic left ventricular systolic and diastolic dysfunction.

Early screening with echocardiography in the newly diagnosed type 2 Diabetes and at regular intervals(at least once yearly) in T2 DM we can reduce the hospitalization of these patients with heart failure and thereby the cardiovascular mortality along with appropriate drug therapy in right time.

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BIBILOGRAPHY

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ABBREVIATIONS

A - Peak mitral late filling velocity by atrial contraction

CVD - Cardiovascular disease

DM - Diabetes mellitus

DD - Diastolic dysfunction

E - Peak mitral early filling velocity

ECG - Electrocardiogram

EDV- End Diastolic Volume

ESV - End Systolic Volume

EF - Ejection Fraction

HF - Heart Failure

HFrEF- Heart Failure reduced Ejection Fraction

HFpEF- Heart Failure preserved Ejection Fraction

HbA1c - Glycosylated Haemoglobin

IGT- Impaired Glucose Tolerance

IVSd - Interventricular septal thickness at end diastole

LA - Left atrial chamber volume

LVPWd - Left ventricular posterior wall thickness at end diastole

LVM - Left ventricular mass

LVIDd - Left ventricular internal diameter at end diastole

MODY- Maturity Onset Diabetes Mellitus in Young

SD- Systolic Dysfunction

PROFORMA

Name of the patient:	
Age	
Sex	
Occupation:	
Address	
Phone number:	
Personal history: Alcohol intake Cigarette smoking	
Details of Diabetes mellitus: Duration Treatment History	
Co-morbidities - • CCF, MI, Valvular heart disease, CAD, COPD	
Associated Symptoms Dyspnoea Chest pain Swelling of legs	

HbA1c	
ECHOCARDIOGRAPHY: **A)LEFT VENTRICLE** • Hypertrophy • Systolic anterior motion of mitral leaflet • Left ventricular EF • Diastolic dysfunction	
B)LEFT ATRIUM • Left atrial enlargement	
C)VALVULOPATHY • Aortic stenosis • Aortic regurgitation • Mitral regurgitation • Mitral stenosis	
D)AORTA • Dilated aorta ascendens	

MASTER CHART

S.NO	AGE(YRS)	SEX	DURATION OF T2DM(YRS)	HbA1c	LA volume	MV PG(E)	MV PG (A)	E/A	EDV(ml)	ESV(ml)	EF%	SV(ml)	IVSd(cm)	LVIDd(cm)	LVPWd(cm)	LVM(cube)	Aortic valve	Mitral valve
1	42	1	7	7.4	13.1	2.63	0.79	3.33	108	28.44	73.7	79.65	0.76	4.81	0.86	129.20	N	N
2	51	1	6	7.9	11.62	0.86	1.34	0.64	75.74	22.93	69.7	52.81	0.57	4.14	0.8	80.74	N	N
3	53	1	15	6.8	32.62	0.84	2.74	0.31	185.6	121.9	34.3	63.62	1.15	4.9	1.69	288.50	N	N
4	48	2	9	7.6	15.12	0.94	1.29	0.73	63.83	19.22	69.9	44.61	0.86	3.86	0.86	97.30	N	N
5	51	2	4	6.5	14.89	3.56	2.37	1.50	59.38	22.93	61.4	36.45	1.03	3.75	1.43	155.97	N	N
6	45	2	3	6.3	10.32	3.28	2.38	1.38	65.99	18.2	72.4	47.79	0.67	3.9	0.74	75.81	N	N
7	44	1	3	6.9	12.76	4.22	2.84	1.49	62.8	18.97	69.8	43.83	0.69	5.31	0.94	154.13	N	N
8	39	2	5	7.5	11.54	2.43	2.21	1.10	85.69	37.14	56.7	48.55	0.74	4.36	0.86	107.78	N	N
9	61	1	13	8.3	14.57	1.34	1.76	0.76	68.45	21.11	69.2	47.34	1.45	4.68	1.38	267.72	N	N
10	34	1	2	6.5	15.35	1.84	1.44	1.28	67.45	11.13	83.5	56.32	0.86	4.78	0.92	144.61	N	N
11	35	2	1	6.8	12.44	1.34	1.27	1.06	64.9	28.56	56.0	36.34	0.84	3.95	0.94	105.85	N	N
12	41	1	1	7	16.32	0.98	3.45	0.28	53.67	17.17	68.0	36.5	1.03	3.75	1.34	147.44	N	N
13	56	2	9	8.2	16.74	3.12	2.3	1.36	84.78	44.28	47.8	40.5	0.73	4.32	0.84	103.53	N	N
14	43	1	5	6.5	15.73	1.45	1.26	1.15	76.75	34.08	55.6	42.67	0.8	3.86	0.94	98.86	N	N
15	44	1	4	6.7	22.6	0.97	0.68	1.43	73.35	26.51	63.9	46.84	0.93	3.45	0.88	87.52	N	N
16	59	1	2	6.8	28.48	0.96	0.73	1.32	64.25	24.69	61.6	39.56	0.68	3.7	0.72	68.82	N	N
17	64	2	5	7.2	29.42	0.84	0.68	1.24	56.47	31.91	43.5	24.56	1.25	4.66	1.18	212.93	N	N
18	50	1	10	9.6	26.41	1.8	2.34	0.77	88.45	54.8	38.0	33.65	0.87	4.88	0.66	123.04	N	N
19	57	1	7	6.1	14.82	0.82	2.56	0.32	68.46	20.22	70.5	48.24	0.75	3.96	0.81	88.87	N	N
20	49	2	4	6.2	15.48	2.35	1.56	1.51	78.65	37.3	52.6	41.35	0.79	3.79	0.85	88.51	N	N
21	63	1	15	9.9	16.48	2.73	1.12	2.44	105.4	65.06	38.3	40.34	1.4	4.12	1.34	211.01	N	N
22	65	2	4	7	16.15	1.68	1.43	1.17	128.6	50.15	61.0	78.45	0.94	4.9	0.95	163.17	N	N
23	60	1	5	6.5	14.32	2.38	1.23	1.93	78.23	27.93	64.3	50.3	1.45	4.8	1.51	297.37	N	N
24	58	1	3	6.5	18.46	2.53	1.76	1.44	90.35	34.57	61.7	55.78	0.68	4.7	0.76	106.81	N	N
25	51	2	8	8.6	19.45	2.88	1.86	1.55	98.45	41.79	57.6	56.66	0.73	3.6	0.84	76.75	N	N
26	39	2	2	6.9	28.48	1.95	1.44	1.35	64.27	19.49	69.7	44.78	0.79	4.1	0.88	103.08	N	N
27	52	2	6	7.1	30.24	2.67	1.93	1.38	68.45	29.89	56.3	38.56	0.66	3.82	0.71	70.53	N	N
28	48	2	9	8.3	18.64	1.93	1.46	1.32	84.25	47.9	43.1	36.35	0.89	4.53	0.66	110.25	N	N
29	51	2	5	6.8	17.98	1.85	1.44	1.28	67.44	27.56	59.1	39.88	0.93	3.9	0.82	101.31	N	N
30	50	1	10	9.4	20.56	1.51	1.94	0.78	89.72	49.36	45.0	40.36	1.32	4.13	1.44	214.12	N	N
31	39	2	4	7.1	18.98	2.03	1.87	1.09	74.32	37.79	49.2	36.53	0.83	3.89	0.52	71.33	N	N
32	46	2	7	8.4	21.25	1.98	1.77	1.12	76.48	28.81	62.3	47.67	0.73	4.13	0.65	81.17	N	N
33	42	1	2	6.7	19.87	2.13	1.49	1.43	63.46	12.12	80.9	51.34	0.71	3.71	0.77	74.43	N	N
34	61	1	10	7.9	28.56	2.83	2.54	1.11	156.45	77.51	50.5	78.94	0.73	4.11	0.67	82.02	N	N
35	57	1	8	6.5	18.98	2.47	2.03	1.22	88.42	31.97	63.8	56.45	0.69	4.71	0.71	103.45	N	N
36	72	2	24	8.8	25.48	2.24	1.84	1.22	98.68	52.88	46.4	45.8	1.54	3.72	1.43	206.89	N	N
37	76	2	22	11.3	28.56	1.25	1.12	1.12	128.34	83.78	34.7	44.56	1.44	5.6	1.62	394.84	AS+	N
38	53	1	17	9.6	20.44	1.78	2.54	0.70	111.56	47.12	57.8	64.44	1.15	5	1.34	246.20	N	N
39	57	1	3	6.9	18.45	1.93	1.87	1.03	97.58	40.74	58.2	56.84	0.69	3.99	0.77	82.43	N	N
40	52	1	16	8.4	16.48	1.98	1.65	1.20	134.54	76.09	43.4	58.45	0.71	3.91	0.77	81.15	N	N
41	43	1	8	9.7	32.64	1.83	2.32	0.79	126.42	57.54	54.5	68.88	0.89	3.6	0.91	92.79	N	N
42	44	1	3	7.1	14.76	2.13	1.87	1.14	88.5	39.15	55.8	49.35	0.77	4.3	0.82	104.46	N	N
43	49	1	5	8	12.58	1.98	1.74	1.14	65.78	19	71.1	46.78	0.75	4.13	0.87	100.16	N	N
44	57	2	12	8.9	25.36	2.16	1.98	1.09	100.6	25.36	69.0	69.46	1.36	5.2	1.45	311.20	N	N
45	59	1	9	6.7	13.77	1.45	1.11	1.31	92.46	45.9	50.4	46.56	0.71	3.7	0.74	72.10	N	N
46	53	2	2	7	13.32	1.89	1.56	1.21	104.2	43.78	58.0	60.42	0.91	3.3	0.93	83.68	N	N
47	44	1	3	7	17.55	1.94	1.46	1.33	101.4	41.42	59.2	59.98	0.85	4.1	0.88	108.12	N	N
48	48	2	7	8.5	13.56	2.14	1.87	1.14	156.4	70.06	55.2	86.34	1.28	3.92	1.49	199.60	N	N
49	55	2	6	6.9	12.84	2.56	1.73	1.48	138.68	61.9	55.4	76.78	0.72	3.44	0.82	69.49	N	N
50	56	2	9	9.2	25.68	2.33	2.97	0.78	125.6	55.71	55.6	69.89	0.89	3.81	0.93	103.06	N	N
51	49	1	12	7.8	13.6	2.45	1.93	1.27	110.5	47.9	56.7	62.6	0.81	3.99	0.89	101.02	N	N
52	70	2	22	9.4	15.82	1.53	1.47	1.04	86.8	38.44	55.7	48.36	1.36	4.23	1.12	188.99	N	N
53	63	1	15	10.3	22.36	2.96	1.32	2.24	82.44	37.76	54.2	44.68	0.69	4.21	0.77	90.18	N	N
54	37	2	8	8.6	16.44	2.14	1.88	1.14	84	28.4	66.2	55.6	1.43	4.67	1.58	292.75	N	N
55	35	2	3	7.3	17.03	2.56	1.93	1.33	90.2	25	72.3	65.2	0.73	3.9	0.84	87.42	N	N
56	48	2	9	6.4	27.55	2.53	3.04	0.83	110.3	45.84	58.4	64.46	0.71	3.2	0.85	63.07	N	N
57	54	1	6	6.9	15.48	2.67	2.1	1.27	86	29.57	65.6	56.43	0.85	3.45	0.97	88.21	N	N

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58	51	2	9	6.3	17.5	1.89	1.57	1.20	80.4	33.06	58.9	47.34	0.87	3.21	0.91	76.46	N	N
59	63	1	11	8.5	26.54	1.96	2.55	0.77	110.7	41.71	62.3	68.99	1.69	4.58	1.76	351.46	N	N
60	56	1	7	7.6	16.94	2.76	2.14	1.29	136.2	64.7	52.5	71.5	0.82	3.91	0.91	100.13	N	N
61	60	2	8	7.4	18.45	2.16	1.84	1.17	92.4	45.6	50.6	46.8	0.91	3.99	0.88	108.41	N	N
62	65	2	5	6.1	14.62	2.56	2.16	1.19	68.2	3.6	94.7	64.6	0.54	4.57	0.79	92.07	N	N
63	71	1	9	7.9	29.6	1.94	1.78	1.09	80.4	35.8	55.5	44.6	0.71	5	0.67	112.67	N	MR+
64	67	1	16	10.3	18.42	1.83	2.42	0.76	86.6	17.82	79.4	68.78	1.14	4.1	1.24	169.64	N	N
65	60	2	11	7	31.45	1.75	1.62	1.08	80.8	36.5	54.8	44.3	0.75	3.9	0.78	84.45	N	N
66	54	1	10	7.5	16.66	1.92	1.78	1.08	64.6	29	55.1	35.6	1.56	4.3	1.64	285.45	N	N
67	60	2	6	6.9	17.36	2.14	1.77	1.21	90.6	40.15	55.7	50.45	0.82	3.9	0.89	98.14	N	N
68	68	2	13	7.4	30.24	1.36	1.88	0.72	110.2	45.84	58.4	64.36	1.48	3.87	1.56	226.89	N	N
69	55	1	9	8.3	17.44	2.17	1.89	1.15	58.8	24	59.2	34.8	0.67	4.21	0.77	88.58	N	N
70	43	2	5	7.1	14.83	2.13	1.68	1.27	110.56	42.06	62.0	68.5	0.74	3.87	0.78	82.66	N	N
71	57	2	16	8.3	15.85	1.83	1.45	1.26	90.45	25.67	71.6	64.78	0.91	3.67	0.99	103.25	N	N
72	46	2	8	6.9	17.3	1.94	1.33	1.46	85.45	31.75	62.8	53.7	0.76	4.5	0.79	109.03	N	N
73	58	2	14	6.8	16.73	1.36	0.93	1.46	90.34	37.54	58.4	52.8	1.32	4.77	1.47	269.79	N	N
74	56	1	2	6.8	17.63	1.86	1.56	1.19	89.46	31.82	64.4	57.64	1.11	3.99	1.47	183.70	N	N
75	78	2	27	9.2	32.75	1.73	2.46	0.70	95.37	49.59	48.0	45.78	0.95	5.8	0.84	202.06	N	MR+
76	49	1	12	8.3	15.98	2.96	2.45	1.21	110.45	48.1	56.5	62.35	0.65	4.53	0.69	91.54	N	N
77	48	1	10	8.2	14.55	1.83	1.56	1.17	85.67	38.1	55.5	47.57	0.91	3.9	0.92	107.77	N	N
78	60	2	5	7.1	20.46	2.45	1.98	1.24	105.32	43.95	58.3	61.37	0.89	3.77	0.95	102.92	N	N
79	64	2	23	8.4	32.62	3.12	2.84	1.10	108.47	61.75	43.1	46.72	1.22	5.6	1.39	314.91	AS+	N
80	47	1	4	7.3	17.45	1.84	1.51	1.22	98.34	38.3	61.1	60.04	0.69	3.99	0.73	79.49	N	N
81	49	1	8	7.4	18.36	2.13	1.83	1.16	84.28	33.83	59.9	50.45	0.76	4.12	0.79	94.07	N	N
82	53	2	7	6.7	14.86	1.54	1.98	0.78	96.38	38.3	60.3	58.08	0.77	4.57	0.79	112.84	N	N
83	41	1	5	6.9	15.33	2.67	1.92	1.39	81.64	33.08	59.5	48.56	0.83	4.66	0.77	120.51	N	N
84	44	2	9	7	18.37	1.97	1.39	1.42	65.42	23.78	63.7	41.64	1.3	4.17	1.48	219.57	N	N
85	82	1	35	6.8	30.42	2.17	1.74	1.25	78.43	34.95	55.4	43.48	1.48	4.11	1.68	262.53	N	N
86	48	1	9	6.9	17.45	2.75	2.14	1.29	84.88	28.09	66.9	56.79	0.81	4.55	0.91	127.31	N	N
87	59	2	12	9.5	19.45	2.11	2.88	0.73	98.45	33.77	65.7	64.68	0.89	3.99	0.94	111.77	N	N
88	53	2	13	8.12	18.66	1.88	1.48	1.27	119.35	44.01	63.1	75.34	0.91	3.99	0.97	116.03	N	N
89	75	2	13	10.5	30.2	4.12	1.55	2.66	103.82	43.28	58.3	60.54	1.27	5.3	1.46	307.53	N	MR+
90	69	1	9	8.1	22.01	1.46	1.39	1.05	94.37	30.92	67.2	63.45	0.9	5.7	1.1	226.35	AR+	N
91	49	1	15	7.6	20.47	1.22	1.86	0.66	91.48	24.59	73.1	66.89	0.58	4.33	0.61	73.00	N	N
92	51	2	7	6.9	14.56	2.18	1.89	1.15	75.38	28.01	62.8	47.37	0.89	3.79	0.95	103.78	N	N
93	60	1	8	7.2	17.53	1.93	2.57	0.75	82.36	29.12	64.6	53.24	0.79	3.67	0.88	86.16	N	N
94	38	1	5	6.6	18.42	1.97	1.52	1.30	96.45	31.67	67.2	64.78	0.77	4.18	0.81	98.83	N	N
95	44	2	7	6.7	17.55	1.85	1.63	1.13	91.8	28.56	68.9	63.24	0.81	3.99	0.89	101.02	N	N
96	70	2	20	10.8	30.66	2.15	1.75	1.23	119.47	52.69	55.9	66.78	0.72	6.1	0.75	172.67	N	MR+
97	62	2	24	12.4	18.54	3.86	1.78	2.17	107.38	57.85	46.1	49.53	1.62	3.79	1.55	235.82	N	N
98	57	1	5	6.8	12.46	2.81	1.93	1.46	95.45	31.63	66.9	63.82	0.85	4.12	0.95	115.03	N	N
99	60	1	9	7.4	11.58	1.89	1.27	1.49	69.43	29.03	58.2	40.4	0.89	3.56	0.91	91.18	N	N
100	58	2	7	7.1	10.58	2.17	1.49	1.46	86.35	36.15	58.1	50.2	1.4	3.59	1.39	178.17	N	N
101	43	1	5	6.8	14.68	1.92	1.38	1.39	88.25	32.79	62.8	55.46	0.73	4.1	0.87	97.34	N	N
102	40	1	4	6.9	20.53	2.47	1.82	1.36	97.46	38.79	60.2	58.67	0.65	4.19	0.71	81.63	N	N
103	38	1	5	6.6	18.44	2.13	2.45	0.87	114.68	37.94	66.9	76.74	1.25	4.79	1.6	280.19	N	N
104	46	2	6	6.8	17.43	2.67	1.92	1.39	110.58	45.16	59.2	65.42	0.66	4.99	0.71	111.26	N	N
105	38	1	4	7	14.52	2.27	1.77	1.28	102.35	31.75	69.0	70.6	0.91	3.67	0.95	100.18	N	N
106	49	2	5	6.8	16.88	1.89	2.84	0.67	95.48	40.08	58.0	55.4	0.89	3.77	0.91	99.80	N	N
107	58	1	7	6.9	14.36	2.16	1.78	1.21	91.38	36.88	59.6	54.5	0.77	4.25	0.81	101.60	N	N
108	64	1	14	7	16.35	1.82	1.45	1.26	85.49	36.97	56.8	48.52	0.88	4.29	0.91	121.91	N	N
109	48	2	21	7.9	14.35	1.98	1.65	1.20	75.48	31.88	57.8	43.6	0.87	6	0.92	214.20	N	N
110	55	2	10	6.8	15.68	1.96	1.49	1.32	99.7	37.16	62.7	62.54	0.79	3.49	0.81	74.95	N	N
111	70	1	15	7	14.56	1.73	1.82	0.95	92.46	37.68	59.2	54.78	1.24	4.9	1.32	248.13	AS+	N
112	59	2	16	8.9	14.84	1.99	1.91	1.04	117.35	51.97	55.7	65.38	0.65	4.77	0.67	98.22	N	N
113	62	1	19	7.1	17.52	2.14	1.79	1.20	124.58	48.98	60.7	75.6	0.75	4.12	0.81	94.88	N	N
114	53	2	13	9.3	18.42	2.1	1.63	1.29	103.28	43.58	57.8	59.7	0.89	6.1	0.91	221.96	N	N
115	51	1	7	8.2	14.38	1.93	1.74	1.11	88.64	33.86	61.8	54.78	0.69	4.59	0.78	105.30	N	N
116	68	1	24	11.3	38.42	5.46	1.72	3.17	92.44	37.78	59.1	54.66	1.68	4.78	1.72	365.12	N	N
117	54	2	13	7.4	15.82	2.15	1.95	1.10	76.35	37.9	50.4	38.45	0.95	3.79	0.88	102.99	N	N
118	59	1	6	8.1	16.89	2.48	1.91	1.30	84.12	36.62	56.5	47.5	0.67	4.85	0.77	112.73	N	N
119	64	1	9	7.3	17.45	2.19	1.69	1.30	95.32	39.95	58.1	55.37	0.74	3.71	0.78	77.14	N	N
120	60	2	4	7.2	16.38	2.14	2.95	0.73	104.56	35.97	65.6	68.59	0.71	4.1	0.77	87.81	N	N

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121	58	2	9	8.5	14.76	2.28	1.82	1.25	72.43	30	58.6	42.43	0.69	3.99	0.82	86.17	N	N
122	45	1	5	7.2	15.68	2.16	1.83	1.18	110.68	36.36	67.1	74.32	0.81	3.57	0.89	84.52	N	N
123	49	1	6	7	14.78	2.36	1.73	1.36	101.24	34.81	65.6	66.43	0.77	3.9	0.91	95.80	N	N
124	50	2	7	6.9	13.64	1.93	1.78	1.08	84.65	34.39	59.4	50.26	0.87	4.12	0.9	112.42	N	N
125	53	1	8	7.7	18.24	2.14	1.79	1.20	79.35	34.79	56.2	44.56	0.91	3.97	0.97	115.11	N	N
126	58	1	16	8.3	29.84	1.34	1.95	0.69	117.46	33.64	71.4	83.82	1.25	5.9	1.37	344.29	N	N
127	62	1	14	7.2	14.36	2.25	1.84	1.22	95.29	30.51	68.0	64.78	1.36	4.77	1.42	268.37	N	N
128	45	1	8	7.6	14.58	2.19	1.92	1.14	75.62	26.98	64.3	48.64	0.95	3.11	0.98	82.09	N	N
129	50	2	8	7.1	15.26	2.15	1.72	1.25	118.35	39.6	66.5	78.75	0.78	3.89	0.81	88.54	N	N
130	59	2	13	6.8	16.84	2.63	1.92	1.37	120.68	51.9	57.0	68.78	0.74	4.65	0.88	122.03	AS+	N
131	65	1	17	6.7	14.58	1.94	1.45	1.34	104.85	44.87	57.2	59.98	1.22	4.79	1.34	239.52	N	N
132	61	2	15	7.8	12.48	1.97	1.63	1.21	100.46	40.93	59.3	59.53	0.59	4.88	0.77	106.06	N	N
133	58	1	13	6.9	14.62	2.14	1.57	1.36	87.43	37.79	56.8	49.64	0.89	3.86	0.92	104.41	N	N
134	46	1	6	7.4	15.88	1.95	1.52	1.28	84.57	33.23	60.7	51.34	0.76	4.17	0.85	100.93	N	N
135	41	1	4	7.5	13.98	2.57	1.94	1.32	75.48	29.53	60.9	45.95	0.94	3.91	0.97	114.88	N	N
136	59	2	8	7.9	14.36	2.13	1.59	1.34	79.46	38.9	51.0	40.56	0.95	6	0.98	235.79	N	N
137	65	1	13	8.6	14.21	1.74	2.89	0.60	83.75	32.77	60.9	50.98	0.83	4.15	0.87	107.70	N	N
138	60	2	12	9	29.48	2.81	2.34	1.20	88.55	35.94	59.4	52.61	1.44	5.6	1.78	425.35	N	N
139	49	2	7	7.2	14.45	2.18	1.86	1.17	78.65	31.9	59.4	46.75	0.75	3.88	0.81	85.94	N	N
140	65	1	17	9.4	18.48	2.15	1.77	1.21	96.82	42.36	56.2	54.46	1.42	4.56	1.51	271.31	N	N
141	42	1	6	7.1	13.46	1.99	1.79	1.11	100.34	37.85	62.3	62.49	0.71	4.78	0.81	117.77	N	N
142	54	1	9	7.8	12.45	1.99	1.85	1.08	104.57	43.99	57.9	60.58	0.69	4.79	0.74	109.38	N	N
143	58	2	8	6.8	14.68	2.13	2.97	0.72	94.85	39.18	58.7	55.67	0.77	3.98	0.91	99.01	N	N
144	63	2	25	8.5	13.92	2.48	1.85	1.34	83.89	43.33	48.3	40.56	0.88	3.65	0.97	98.57	AS+	N
145	47	1	4	6.7	12.95	2.55	1.94	1.31	82.54	32.56	60.6	49.98	0.81	3.71	0.93	92.80	N	N
146	51	2	6	5.6	13.45	2.51	1.96	1.28	87.45	38.88	55.5	48.57	0.81	4.15	0.91	109.42	N	N
147	54	2	7	6.3	13.56	1.83	2.85	0.64	97.35	37.75	61.2	59.6	0.89	4.1	0.95	117.63	N	N
148	48	1	3	7	16.78	2.64	1.92	1.38	93.75	36.92	60.6	56.83	0.91	3.88	0.89	104.47	N	N
149	56	1	4	7.9	16.74	2.94	1.87	1.57	110.4	44.7	59.5	65.7	0.76	4.01	0.85	94.64	N	MR+
150	45	2	2	7.4	15.34	2.3	1.42	1.62	96.8	42.6	56.0	54.2	0.73	4.66	0.88	121.49	N	N
151	64	1	9	7.1	14.78	1.97	3.46	0.57	88.98	36.53	58.9	52.45	1.34	4.65	1.44	258.21	N	N
152	51	1	3	6.4	13.57	2.57	1.72	1.49	96.76	42.56	56.0	54.2	0.72	3.89	0.77	81.19	N	N
153	57	1	6	7.8	14.85	2.14	1.83	1.17	98.18	32.98	66.4	65.2	0.77	3.99	0.85	94.65	N	N
154	68	2	14	9.9	32.93	1.58	1.76	0.90	94.78	39	58.9	55.78	1.28	4.34	1.32	210.69	N	N
155	61	2	10	8.4	26.48	2.97	2.68	1.11	110.23	49.45	55.1	60.78	0.67	4.56	0.86	109.63	N	N
156	38	1	3	8.4	12.56	1.84	1.36	1.35	97.56	36.32	62.8	61.24	0.84	4.12	0.74	96.50	N	N
157	39	1	7	7	13.25	2.84	2.37	1.20	98.68	36.12	63.4	62.56	0.91	4.1	0.96	120.29	N	N
158	53	1	12	7.5	14.75	2.99	2.91	1.03	106.24	46.26	56.5	59.98	0.77	4.78	0.79	121.76	N	N
159	47	2	4	7.3	18	2.16	1.82	1.19	110.78	45.45	59.0	65.33	0.75	3.98	0.83	91.15	N	N
160	41	2	2	7.1	13.48	2.56	1.95	1.31	108.98	47.74	56.2	61.24	0.74	4.79	0.85	125.23	N	N
161	48	1	1	6.8	14.65	1.98	1.53	1.29	110.68	41.79	62.2	68.89	0.59	4.89	0.76	105.47	N	N
162	46	1	5	6.9	14.54	1.94	2.43	0.80	98.65	39.67	59.8	58.98	0.67	3.87	0.76	76.24	N	N
163	53	2	4	6.9	12.86	2.15	1.87	1.15	96.87	40	58.7	56.87	0.76	4.19	0.79	96.75	N	N
164	64	2	9	7.8	14.65	1.88	1.46	1.29	116.42	55.66	52.2	60.76	0.85	4.2	0.87	111.58	N	N
165	62	2	7	7.7	15.68	2.48	2.03	1.22	99.98	41	59.0	58.98	0.67	4.58	0.76	101.28	N	N
166	58	2	4	8.1	15.24	1.65	2.33	0.71	106.87	47.98	55.1	58.89	0.93	3.77	0.97	107.68	N	N
167	55	1	6	7	14.82	2.58	2.15	1.20	108.78	46.54	57.2	62.24	0.87	4.28	0.95	124.22	N	N
168	48	1	2	6.8	13.58	2.5	2.71	0.92	104.98	40	61.9	64.98	0.69	4.59	0.75	102.56	N	N
169	44	1	1	6.9	15.28	2.48	1.97	1.26	92.79	27.01	70.9	65.78	0.81	3.75	0.89	91.41	N	N
170	39	2	4	7.2	15.14	2.44	1.92	1.27	111.87	45.11	59.7	66.76	0.82	3.92	0.87	97.38	N	N
171	40	2	2	6.7	13.68	2.4	1.88	1.28	102.76	40.42	60.7	62.34	0.79	4.1	0.81	97.34	N	N
172	43	1	4	6.4	11.82	1.95	1.47	1.33	98.89	38.02	61.6	60.87	0.75	4.56	0.86	117.14	N	N
173	46	2	1	7.5	17.52	2.18	1.86	1.17	97.87	37.89	61.3	59.98	0.76	3.96	0.87	94.26	N	N
174	58	1	4	7.4	18.58	1.94	1.62	1.20	109.98	46.77	57.5	63.21	0.96	4.09	0.91	119.82	N	N
175	52	2	3	6.8	17.35	4.13	1.54	2.68	89.98	35.53	60.5	54.45	0.91	3.85	0.96	108.83	N	N
176	47	1	12	7.1	17.25	2.56	1.94	1.32	112.67	48.11	57.3	64.56	0.84	4.12	0.95	114.16	N	N
177	45	2	4	7.6	16.82	2.42	2.92	0.83	108.78	44.32	59.3	64.46	0.89	4.44	0.92	130.90	N	N
178	63	1	14	6.9	15.38	2.56	2.18	1.17	98.87	38.75	60.8	60.12	0.65	4.67	0.75	101.94	N	N
179	54	1	10	7.8	15.46	2.94	2.47	1.19	96.89	38.02	60.8	58.87	0.69	4.78	0.73	108.02	N	N
180	57	2	16	10.1	16.28	2.16	1.73	1.25	109.98	43.53	60.4	66.45	0.78	4.65	0.79	117.16	N	N
181	68	2	23	10.2	28.45	3.57	1.46	2.45	110.12	47.67	56.7	62.45	0.59	3.98	0.77	74.84	N	N
182	63	1	17	9.5	18.42	2.65	2.16	1.23	102.46	42.34	58.7	60.12	0.69	4.91	0.73	113.14	N	N
183	48	1	9	7.4	19.58	1.84	1.67	1.10	88.35	25.55	71.1	62.8	0.78	4.65	0.79	117.16	N	N

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184	45	2	4	7.6	17.46	2.63	1.98	1.33	88.5	30.15	65.9	58.35	0.76	4.85	0.79	123.79	N	N
185	65	2	12	8.4	16.38	2.18	2.58	0.84	98.62	35.05	64.5	63.57	0.77	4.19	0.78	96.75	AS+	N
186	52	1	8	8.9	16.84	1.94	1.77	1.10	102.4	33.97	66.8	68.43	0.81	4.09	0.89	105.17	N	N
187	50	2	6	7.3	15.36	1.98	1.76	1.13	110.4	40.08	63.7	70.32	1.4	4.18	1.35	216.74	N	N
188	58	2	12	7.6	15.42	2.36	1.83	1.29	102.47	37.12	63.8	65.35	0.85	6	0.97	218.76	N	N
189	61	1	15	8.1	18.28	2.17	1.68	1.29	106	42.32	60.1	63.68	0.79	3.89	0.85	92.33	N	N
190	69	1	19	8.5	22.37	3.45	1.33	2.59	87.56	38	56.6	49.56	0.76	4.8	0.79	121.62	N	MR+
191	46	1	4	7	17.64	2.83	1.46	1.94	92.48	40.02	56.7	52.46	0.81	4.09	0.87	103.50	N	N
192	52	2	8	7.2	17.2	2.56	1.68	1.52	95.42	37.01	61.2	58.41	0.84	4.11	0.88	107.70	N	N
193	38	2	6	6.9	14.86	1.95	1.48	1.32	96.33	40.44	58.0	55.89	0.86	3.99	0.96	110.92	N	N
194	60	1	15	6.9	13.64	1.95	1.69	1.15	99.57	42.59	57.2	56.98	0.91	4.11	0.93	118.09	N	N
195	55	1	13	7.1	15.62	1.94	1.56	1.24	97.61	41.08	57.9	56.53	0.76	3.99	0.84	93.08	N	N
196	58	2	12	8.4	17.6	2.14	2.44	0.88	98.42	39.99	59.4	58.43	0.79	4.11	0.82	98.55	N	N
197	73	2	21	7.8	16.38	2.15	1.84	1.17	99.58	44.16	55.7	55.42	0.79	4.56	0.86	120.96	N	N
198	56	2	7	7.5	17.82	2.45	1.92	1.28	106.47	46.91	55.9	59.56	0.84	4.21	0.89	112.89	AS+	N
199	43	2	5	7.3	19.46	2.18	1.71	1.27	87.48	36.03	58.8	51.45	0.79	4.09	0.81	96.95	N	N
200	51	1	10	6.8	15.65	1.86	1.72	1.08	93.88	35.36	62.3	58.52	0.71	3.99	0.87	91.53	N	N

INFORMATION SHEET

We are conducting a study on"**A study of echocardiography changes in patients with type 2 diabetes in a Tertiary Care Level Hospital''**

- Aim of the study is to determine the echocardiographic abnormalities in type 2 diabetes mellitus patients attending the OPD.
- The privacy of the patient in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.
- Taking part in the study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time. Your decision will not result in any loss of benefits to which you are otherwise entitled.
- The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of investigator Signature of patient /guardian

Date:

INFORMATION TO THE PARTICIPANTS

Principal investigator

DR.S.SURESH,
MD post graduate,
Department of General Medicine,
Chengalpattu medical college, Chengalpattu.

Name of the participant:

TITLE: "A study of echocardiography changes in patients with type 2 diabetes in a Tertiary Care Level Hospital''

You are invited to take part in this study. The information in the document is meant to help you decide whether or not to take part. Please feel free to ask if you have any queries or concerns.

You are being asked to participate in this study being conducted in the Department of General Medicine and Cardiology, Chengalpattu medical college.

Purpose of the research:

Cardiovascular disease accounts for up to 80% of the excess mortality in patients with type 2 diabetes. In our study, type 2 diabetes mellitus patients will be screened with ECHO for early diagnosis of myocardial changes and cardiac dysfunction. By early detection of these myocardial changes in ECHO will help in treating the patients accordingly. Hence, we can reduce the cardiovascular mortality risk in these patients.

Study procedure: Type 2 diabetes mellitus patients of more than 30 years of age will be screened with ECHO for early diagnosis of myocardial changes and cardiac dysfunction .

PATIENT CONSENT FORM

Study Title: "**A study of echocardiography changes in patients with type 2 diabetes in a Tertiary Care Level Hospital''**

STUDY CENTER: Department Of General Medicine
Chengalpattu Govt. Medical College And Hospital
Chengalpattu.

Participant name: Age: Sex:
O.P.NO:

- I have been explained about the nature of the study.
- I confirm that I have understood the purpose and nature of the above study.
- I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.
- I understand that my participation in the study is voluntary and that I am free to withdraw at anytime without giving any reason.
- I understand that investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to current study and any further research that may be conducted in relation to it, even if I withdraw from the study.
- I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from the study.

Time:

Date: Signature / thumb impression of patient

Place: Patient name:

Signature of the investigator:

Name of the investigator:

சுயஒப்புதல் படிவம்

***ஆய்வு செய்யப்படும் தலைப்பு*: "A STUDY ON ECHOCARDIOGRAPHY CHANGES IN PATIENTS WITH TYPE 2 DIABETES MELLITUS IN TERTIARY CARE LEVEL HOSPITAL".**

ஆய்வு செய்யப்படும் இடம்: பொது மருத்துவத் துறை,
செங்கல்பட்டு அரசினர் மருத்துவக் கல்லூரி
மருத்துவமனை, செங்கல்பட்டு.

புற நோயாளர் எண்:

நோயாளர் பெயர்: ***வயது / பாலினம்:***

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கபட்டுள்ளது. நான் இவ்வாய்வில் தன்னிச்சையாக பங்கேற்கின்றேன். எந்த காரணத்தினாலோ, எந்த சட்டசிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகிக்கொள்ளலாம் என்றும் அறிந்துகொண்டேன்.

இந்த ஆய்வு சம்பந்தமாகவோ, இதைசார்ந்து மேலும் ஆய்வு மேற்கொள்ளும்போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர், என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவை இல்லை என அறிந்துக் கொண்டேன். இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக் கொள்ள மறுக்கமாட்டேன்.

இந்த ஆய்வில் பங்குகொள்ள ஒப்புக்கொள்கிறேன். இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிக்கிறேன்.

நோயாளர் கையொப்பம்: ***சாட்சியாளரின் கையொப்பம்:***

நோயாளர் பெயர் மற்றும் முகவரி:

ஆய்வாளரின் கையொப்பம் மற்றும் பெயர்:

இடம்:

தேதி: