

**ASSESSMENT OF LEFT VENTRICULAR DOPPLER
DERIVED MYOCARDIAL PERFORMANCE INDEX IN
DIABETIC PATIENTS WITH ALBUMINURIA**

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

**D.M. CARDIOLOGY
BRANCH II – CARDIOLOGY**



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CERTIFICATE

This is to certify that the dissertation titled “**ASSESEMENT OF DOPPLER DERIVED MYOCARDIAL PERFORMANCE INDEX IN DIABETIC PATIENTS WITH ALBUMINURIA**” is the bonafide original work of Dr. **B.VIJAYASEKARAN**, in partial fulfillment of the requirements for D.M. Branch-II (CARDIOLOGY) examination of THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY to be held in August 2014. The period of post-graduate study and training was from August 2011 to July 2014.

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DECLARATION

I, **Dr.B.VIJAYASEKARAN**, solemnly declare that this dissertation entitled, “**ASSESEMENT OF LEFT VENTRICULAR DOPPLER DERIVED MYOCARDIAL PERFORMANCE INDEX IN DIABETIC PATIENTS WITH ALBUMINURIA**” is a bonafide work done by me at the department of Cardiology, Madras Medical College and Government General Hospital during the period 2011 – 2014 under the guidance and supervision of the Professor and Head of the department of Cardiology of Madras Medical College and Government General Hospital, Professor M.S.RAVI M.D.D.M. This dissertation is submitted to The Tamil Nadu Dr.M.G.R Medical University, towards partial fulfillment of requirement for the award of **D.M. Degree (Branch-II) in Cardiology**

Place:

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Date:

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INTRODUCTION

India has highest percentage of diabetic population in the world. The complications of diabetes increases with the duration of diabetes and the level of glycemetic control. Albuminuria is one of the early indicator of microangiopathy. Diabetic patients predicts the cardiovascular dysfunction when it is associated with micro Albuminuria. Albuminuria judge mainly diabetic kidney disease in people with type II Diabetes mellitus, and can also one of a critical judge of all-cause death, mainly from cardiac causes. The reason of the correlation of albumin uria with heart disease is in doubt, it is mainly due the coronary vascular and structural alteration (raise endothelial permeability, cholesterol accumulation , blood clot production) that causes kidney problems can be associated with in the heart structures and, thus, contribute to heart failure.

Various critical Studies have shown there is link between micro Albuminuria and atherothrombotic disease but also other confounding factors needs to be ruled out. The presence of a unique feature called diabetics cardio myopathy may lead to increase in cardiac disease and death in diabetic population.

Major mortality in diabetic patients is due cardiovascular disease and its complications. Atherosclerosis and coronary artery disease are the most common causes of diabetic cardiac death. Cardiac mortality is major contribution from diabetic and hypertensive pt, which will have coronary artery disease.

Studies also give evidence that distinct cardiomyopathy in patients with diabetes mellitus, which is a myocardial disease without the presence of coronary artery disease. Hence it is proven that the patients with diabetes without coronary atheroma will have increased risk of cardiac death than the non diabetic patient therefore the diabetes mellitus is considered to be coronary artery equivalent.

Early identification of cardiovascular dysfunctions in diabetes pt is critical since cardiac disease mainly influences prognosis of diabetic population. Hence, it warrants a clear, easy, and specific imaging index. Early echo indices which define the L.Ventricle function; like E F can define the L.Ventricle function in systole and mitral valve velocity during diastole for judging L.Ventricle function during diastole. The errors in using the conventional parameters are, it can't be used in abnormal ventricle shape (sphere shaped) in dilated cardiac disease and abnormal regional wall motion. Mitral valve inflow pattern can't be used

in tachycardia since there is fusion of both E and A waves and pseudonormalization.

The other method which is proposed by Tei and associates has proposed the index called TEI INDEX which can assess the global LV function both systolic and diastolic functions.

TEI INDEX when it is abnormally increased can give prognostic value in various cardiac disorders. The advantage of this index is, it is not dependent on Heart rate and can simply be reproducible.

The aim of our study is to assess the diabetic Albuminuria patients with cardiac dysfunction who are not having any clear evidence of cardiac problems using the TEI INDEX and to compare this index with other traditional echo parameters which are generally used to assess the cardiac function.

AIMS AND OBJECTIVES

- To assess the echo cardiographic index like IVS & posterior wall thickness, L.Ventricle functions, E wave flow velocity Deceleration time ;mitral inflow velocity ratio between E and A wave;fractional shortening and LV ejection index ;time of LV emptying (ejection time) ;time during isovolumic contraction (IVCT); time during isovolumic relaxation (IVRT);thickness of IVS; left atrium dimension; left ventricle dimension during diastolic;LVMI(left ventricle mass and index); left ventricle dimension during systolic); thickness of posterior wall.
- To determine the cardiac function in diabetic patient with Albuminuria and those patient without Albuminuria without obvious previous heart disease using the TEI INDEX and to compare this index with other usual method which are used to assess the cardiac function?

REVIEW OF LITERATURE

DIABETES:

Type II DM is the most common form and India contributes major diabetic population in the world. Insulin resistance and inefficient production of insulin by beta cells is the main mechanism of DM. Insulin secretion decreases with increasing age, and also genetically influenced. Hyperglycemia is preceded by insulin resistance and together with other factors like lipid disorders, hypertension.

Metabolic syndrome is term for the patient with impairment of this insulin production. Metabolic syndrome patients will have impaired glucose tolerance than overt diabetes and precedes the DM several years .The special point here is the diabetic complication starts to set-in before the overt diabetic state, can be during the impaired glucose state.

MICRO ALBUMINURIA:***DEFINITION AND ESTIMATION OF URINE ALBUMIN EXCRETION***

Micro Albuminuria which is urinary excretion of albumin ranging from 30 to 300 mg in a 24-h urine sample. Urinary albumin excretion of ≥ 300 mg/24 h can be defined by macro Albuminuria. Urinary Albuminuria usually comprises 20–70% of daily urinary total protein excretion. Urine albumin measurement by dipstick without measuring the creatinine excretion will lead to error. Dipstick methods are used to estimate the urinary albumin excretion but the precise methods to estimate the urinary albumin excretion are :

- 1) Measurement of the albumin-to-creatinine ratio (ACR) in a first spot morning sample,
- 2) 24 hrs estimation urinary albumin excretion with the measurement of creatinine can eliminate the diurnal variation of albumin excretion but collection faults can occur.
- 3) 4-hrs of overnight (timed) urine sample.

A C R measurement in a *first-morning spot urine sample* is adequate and a 4 hrs urine collection is not necessary as proposed by The Kidney Disease Outcomes Quality Initiative guidelines.

Table 1—Measurement of albuminuria

	Normal	Microalbuminuria	Macroalbuminuria	Advantages	Disadvantages
Dipstick for Protein	-	-	+	Convenience	Dependent on level of hydration
24-h protein (mg)	<150	<500	≥500	Overcomes problem of diurnal variation in excretion	Subject to collection errors
24-hour albumin (mg)	<30	30–300	>300	Overcomes problem of diurnal variation in excretion	Subject to collection errors
Timed collection (μg/min)	<20	20–200	>200	Overcomes problem of diurnal variation in excretion	Subject to collection errors
Spot collection (μg albumin/mg creatinine)	<30	30–300	>300	Convenience Not dependent on hydration level Most reproducible	Ratios vary based on sex

Micro Albuminuria - Cardiovascular Risk Indicator:

Micro Albuminuria is strong risk features for cardiovascular disease. Dinneen and Gee stein, showed microalbuminuria among patients with type!! Diabetes mellitus is associated with a 3.4-fold risk for cardiovascular mortality as compared with normo Albuminuria. Identical things exist in hypertensive people (with out diabetes) and in the general population.

Association between urinary albumin excretion and cardiac disease.

- Diabetic pt with micro Albuminuria has increase risk for cardiac morbidity when the albumin excretion increases and initial albumin is not essential in predicting the outcome. In the Losartan Intervention For End point reduction in hypertension (LIFE) study, the reduction of BP is not related in reduction of primary endpoint but the reduction of urine albumin excretion can leads to reduction of primary end point. Samuelsson *et al.* Proposed the in hypertensive people have 3 fold increase I cardiac mortality when associated with Albuminuria.

- RENAAL study - In the Reduction of End points in NIDDM with the Angiotensin II Antagonist Losartan study, renal outcomes is related in decrease in micro Albuminuria levels. . The degree of Albuminuria increase with aging, presence of co morbid disease like diabetes, hypertension.
- The association of urinary albumin excretion and atherosclerosis need to be confirmed because both can coexists with other confounding disease.

Cardiac mortality and micro Albuminuria association – assessment.

Can Micro Albuminuria Cause Cardiovascular Disease?

The renal response is evoked by increase renal albumin trafficking that can increases the atherosclerosis process.

Can Athero thrombosis Cause Micro Albuminuria:

Micro Albuminuria can reflect the generalized atherosclerosis in the body and can leads to increase mortality in atherosclerotic patient because of its generalized process rather than micro Albuminuria itself.

Association with micro Albuminuria and cardiovascular disease by common denominator.

Many studies indicate that micro Albuminuria is associated with several cardiovascular factors, like age, male gender, hypertension, DM, smoking, obesity, dyslipidemia and hyperhomocysteinemia. Metabolic syndrome can also constitute micro Albuminuria but there is dispute or controversial in this statement.

The complete causes for cardiovascular disease remain unclear, the micro Albuminuria can constitute the undiscovered factor which can possibly reflect the atherosclerosis but its correlation remains to be explained.

Micro Albuminuria and Cardiovascular Disorders; common pathogenesis:

Micro Albuminuria and cardiovascular Disorders be by a common pathologic process, proposed hypothesis are:

- Micro Albuminuria cause endothelial malfunction, the endothelial malfunction can depend on the function of endothelium like homeostasis, fibrin degradation, smooth cell multiplication, WBC adhesion, permeability.

- NO (nitric oxide) is produced by endothelium because it has antiproliferative, antiplatelet, anti-inflammatory functions. In microalbuminuria and atherosclerosis, this function will be impaired and can contribute to the risk of cardiac death in diabetic patients.
- The evidence of low grade, chronic inflammation markers like C-reactive protein, TNF- α , interleukins are elevated as a product of endothelial dysfunction and further increase the risk of atherothrombotic in diabetic patients.

CARDIOVASCULAR DISEASE:

Coronary atherosclerosis is a major cause of major pathogenesis in the diabetic population. In diabetic patients, the main cause of LV failure (90%) is due to CAD and hypertension. But some patients have cardiac dysfunction in the absence of CAD, which is proven in autopsy and is termed as diabetic cardiomyopathy. Hence in diabetic patients, independent of CAD, there is a further increase in the risk of heart failure.

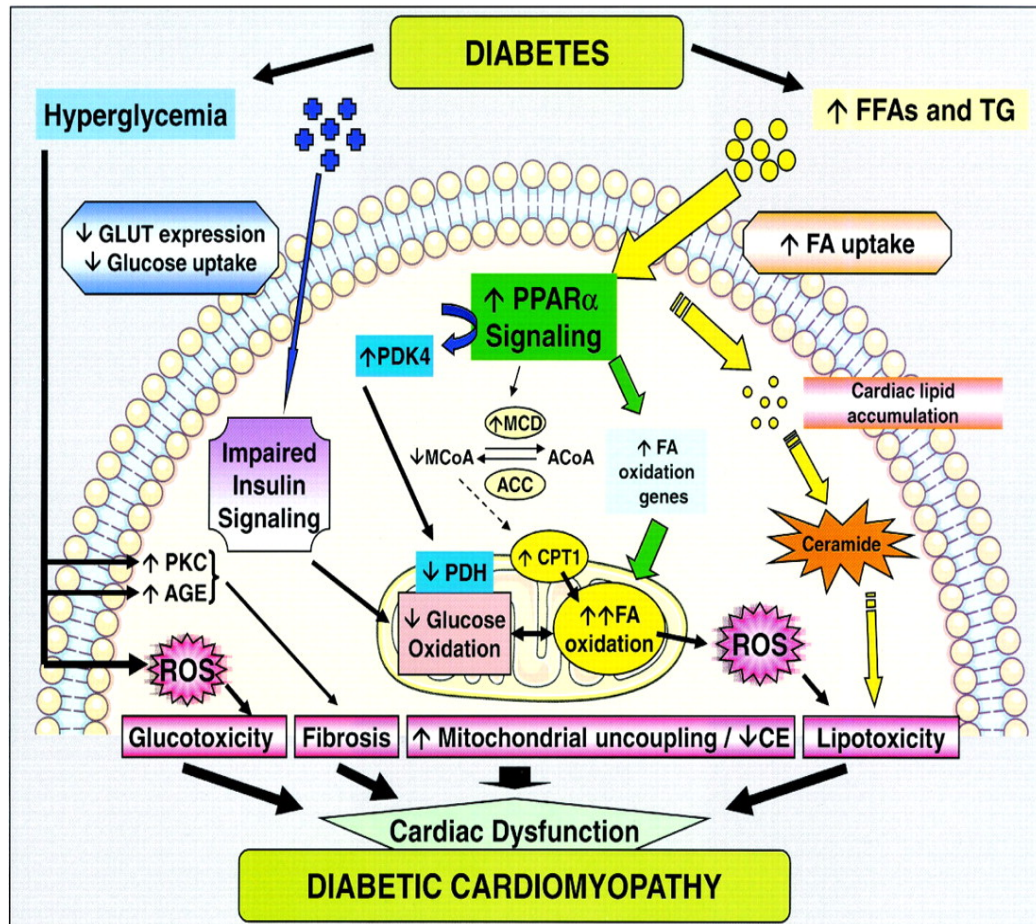
The pathology of diabetic cardiomyopathy:

Damage in ventricular myocardium:

In diabetic patients, the CAD are not essentially different from the general population, but the CAD have extensive atheroma, diffuse lesions and small distal vessel disease and treatment strategy will vary for

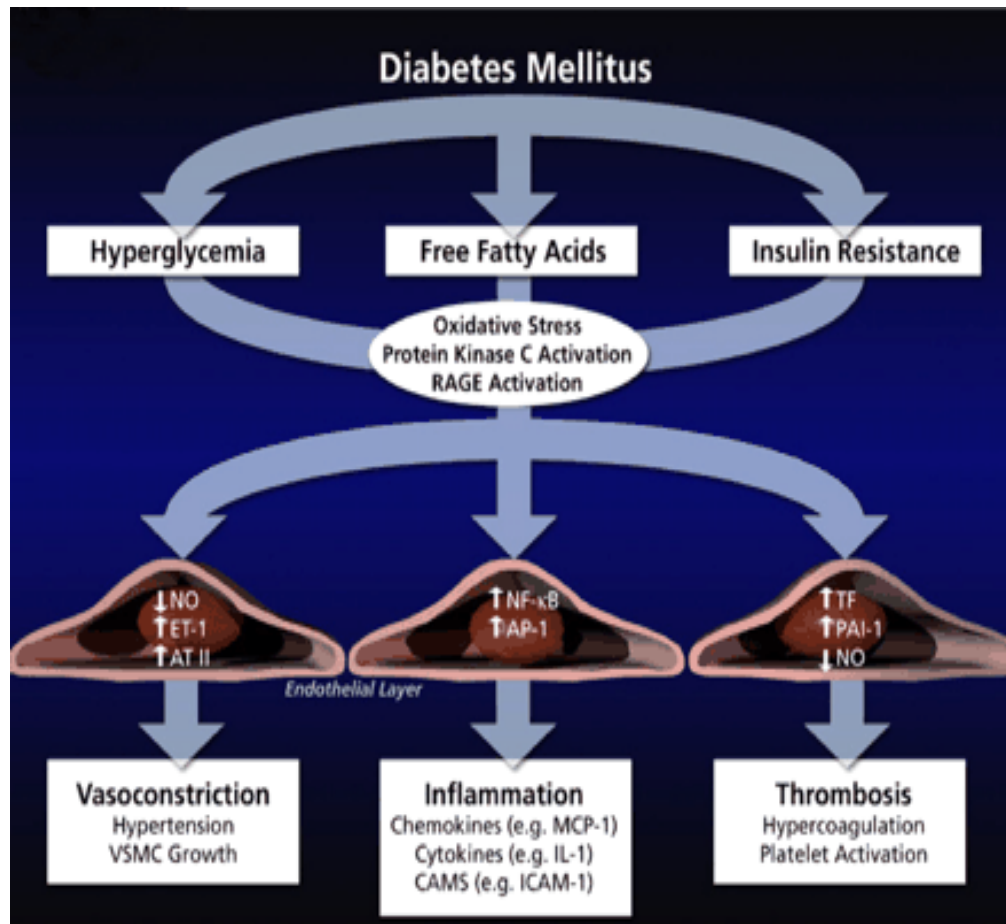
diabetic CAD patients. Myocardial variation in diabetic patient are due AGE (advance d end stage glycation)which can hinder the ventricular compliance ,contractility, and LV hypertrophy can occur due to interstitial and perivascular fibrosis as the diabetic progression occurs.

LV diastolic functions is impaired due to impaired relaxation and increased atrial filling. Previous studies which have used conventional echo method found to 30% Lv diastolic function. Using newer Doppler parameters, LV DD in diabetic patients will be around 60% when compared to the general population.



(Contributors to the evolution of diabetic myocardial dysfunction.)

Diabetic patient having high incidence of LV diastolic dysfunction is due to cardiac myocardial fibrosis and amount of fibrosis is directly related to HBA1C level in serum. The myocardial fibrosis is due to AGE in myocardium which can be decreased by lyses of collagen cross links .furthermore the endothelial dysfunction is aggravated by hyperglycemia by increase in myocardial free radical contents, decrease EDRF. It is also associated with lipotoxixcity which is direct toxic to myocardium can lead to evolution of diabetic cardio myopathy.



(Metabolic Abnormalities in Hyperglycemia)

(Hyperglycemia And Endothelium-Derived Vasoactive
Substance).

Free Fatty Acid Liberation and Endothelial Function

Endothelial function through several mechanisms may be impaired by Free fatty acids, free radicals, dyslipdemias, small dense LDL which is more atherogenic. Other dyslipdemias like hypertriglyceredemia, high HDL also associated with malfunction of endothelium.

Insulin Resistance and NO

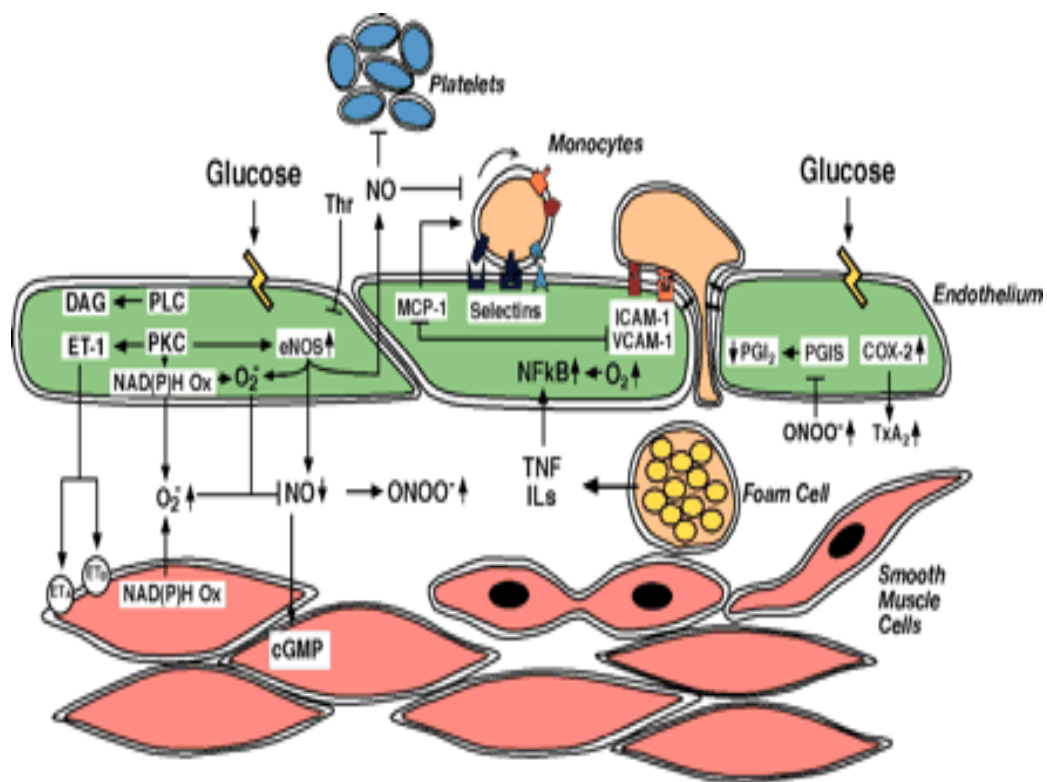
Nitric oxide productions are stimulated by insulin which leads to endothelial derived vasodilation. But in insulin resistance develops; the NO related vasodilatations were reduced. Hence insulin sensitizing drugs like metformin, glitazone can lead to NO related vasodilatations.

Diabetes mellitus and Smooth Muscle Function

The diabetic patient have increased tendency for migration and proliferation of smooth cell in the atherosclerotic plaque which leads to progression of coronary disease and lesion become hard which gives difficulty during the coronary percutaneous interventions.

Diabetes, Thrombosis, & Coagulation

Platelet function is impaired in diabetic pt because of this expression GpII b,IIIa is elevated which in turn augments the vWF and platelet fibrin interaction and more rapid formation of thrombus and occlusion causing acute coronary syndrome.



TEI INDEX

Left ventricular measurement can be calculated by Penn conventional method taking leading edge with trailing edge.. It includes Left V diameter in end diastole and end of systole. Left V volumes were measured according to the popular Teichholz M-mode formula $\text{volume} = \frac{7}{4} D^3 / (2.4 + D)$. Ejection fraction was measured $(\text{LV ED volume} - \text{LV ES volume}) / \text{LV ED volume}$.

Left V mass can be measured by M-mode formula of Troy et al by American Society of Echocardiography (ASE) guidelines. LV mass / body surface area gives the LV mass index.

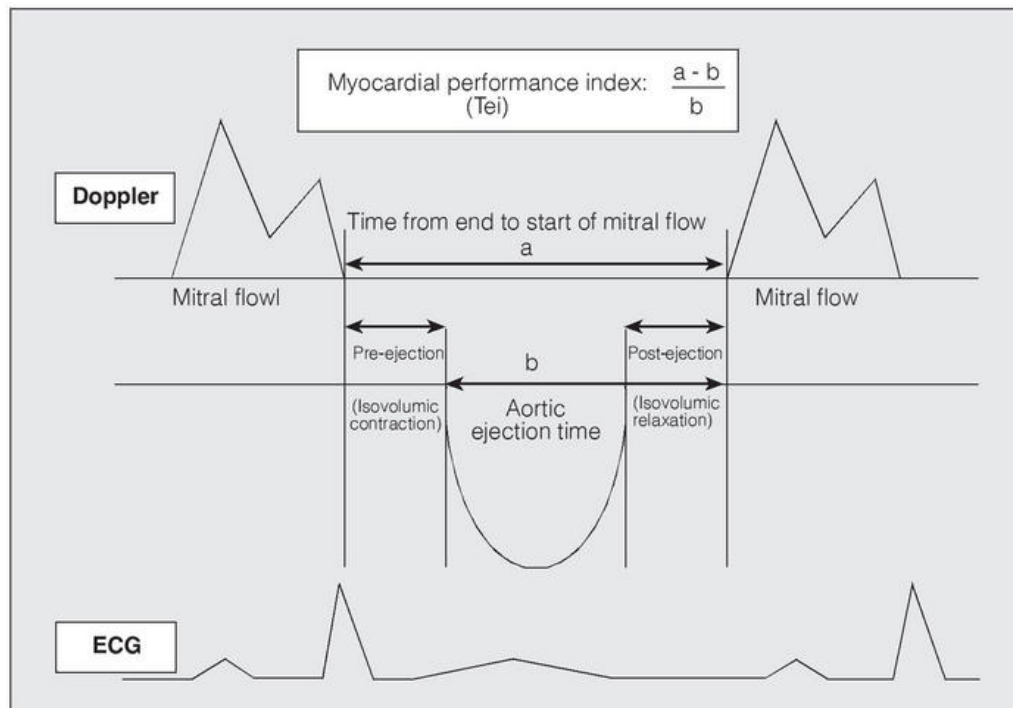
Left V wall motion score measured as the average score in a 17-segment model of the left ventricle using 2D echo images.

Calculation of the Tei index

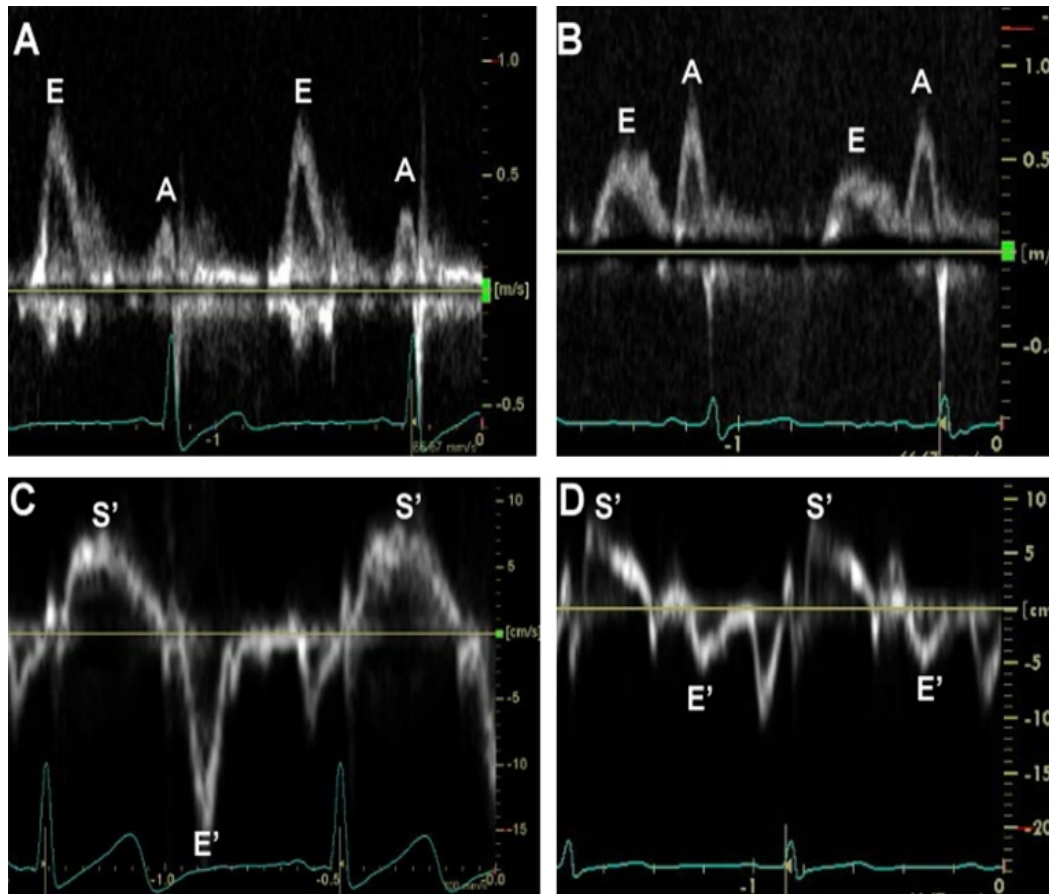
The Tei index is a number and is calculated from the ratio of time intervals $(a-b/b)$ derived with the help of pulsed Doppler echocardiography. Locating the sample volume at the tips of the mitral valve leaflets, in the apical 4-chamber view, enables the measurement of a , which is the time interval between the end and the start of Trans mitral flow. The sample volume is then located in the LV outflow tract, just below the aortic valve (apical 5-chamber view A5C)

For the measurement of b , the LV ejection time. The interval a includes the isovolumic contraction time (IVCT), the ejection time (ET) and the isovolumic relaxation time (IVRT), and the Tei index may also be expressed by the formula $IVCT+IVRT/ET$.

For the evaluation of the right ventricular (RV) Tei index the a interval, from the end to the start of trans-tricuspid flow (the interval from the end of the A wave to the start of the E wave), is obtained from the apical 4-chamber view with the Doppler sample volume located between the tips of the tricuspid valve leaflets. The b interval (RVET) is measured from the parasternal long-axis view, with the sample volume located just below the pulmonary valve.



Pictorial Demonstration of How To Measure The Tei Index



Pulse Wave Doppler and Tissue Doppler Demonstrating the

Measurement of Tie Index

TEI INDEX & and alteration factor, **preload**

In order to determine the Tei index determinant like preload of heart, which can likely to change the value, study population of previous myocardial infarction were taken. In the patients

Preload of heart is reduced by: Vals alva man oeuvre, and administration of sublingual nitroglycerine.

Preload of heart is increased by: passive leg raising). In the control population group the index shows significantly raise during the Vals alva man oeuvre (due to a reduction in Ejection Time), elevating the limb passively (due to mainly raise in Iv CT) and after giving nitro-glycerin (as due to decline in Ejection time and elongation in of IV C T).on the contrary, the previous myocardial infarction patient doesn't show any change in Tei index. .in the population having the preload which is reduced the IV CT/E T ratio clearly found that it is reduced while the ratio between IV RT/E T found to be raised , leaving the index remain unchanged. This study has clearly demonstrated that the change in preload ha only mild alteration in the Tei index and it is considered to preload independent parameters.

TEI INDEX & hemodynamic measurements.

Idiopathic cardiac dysfunction and ischemic Lv dysfunction were subjected cardiac cath study and Doppler examination.

The measurements like $+dP/dT$ (rate of raise of pressure during systole), $-dP/dT$ (rate of pressure drop), t a u time constant were measured. The Tei index is comparable with the study variables hence it can be concluded that the Tei index is a more sensitive indices for diastolic function than any conventional parameters.

Tei index in cardiac dysfunction

In cardiomyopathy, the tie index reflects the severity of myocardial malfunctions and it is clearly evidenced that it gives the independent outcome of the cardiomyopathy patient. Higher the value, worse the prognosis. It is similar the ejection fraction. The tie index has positive correlation with NYHA class, l.v entricular dimensions, one year, and five year survival outcome.

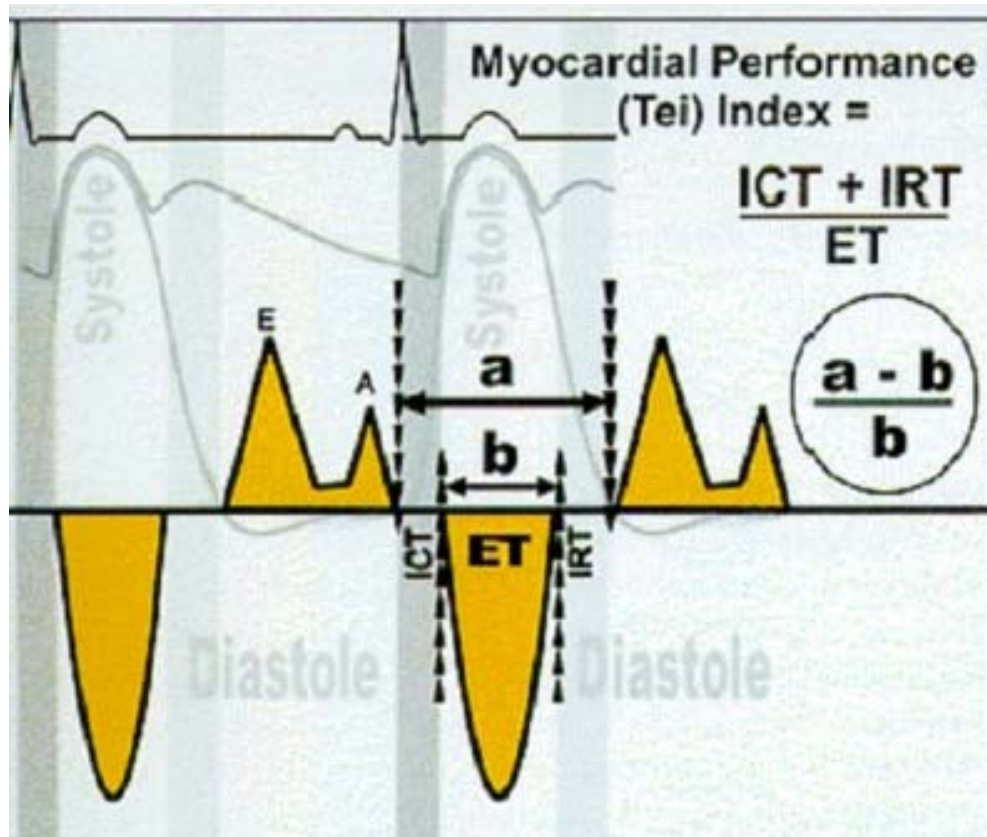
Tei index measurement

The index is derived from pulse Doppler can help to assess the global l. ventricular function is the Tei Index which is coined by Tei and his colleagues.

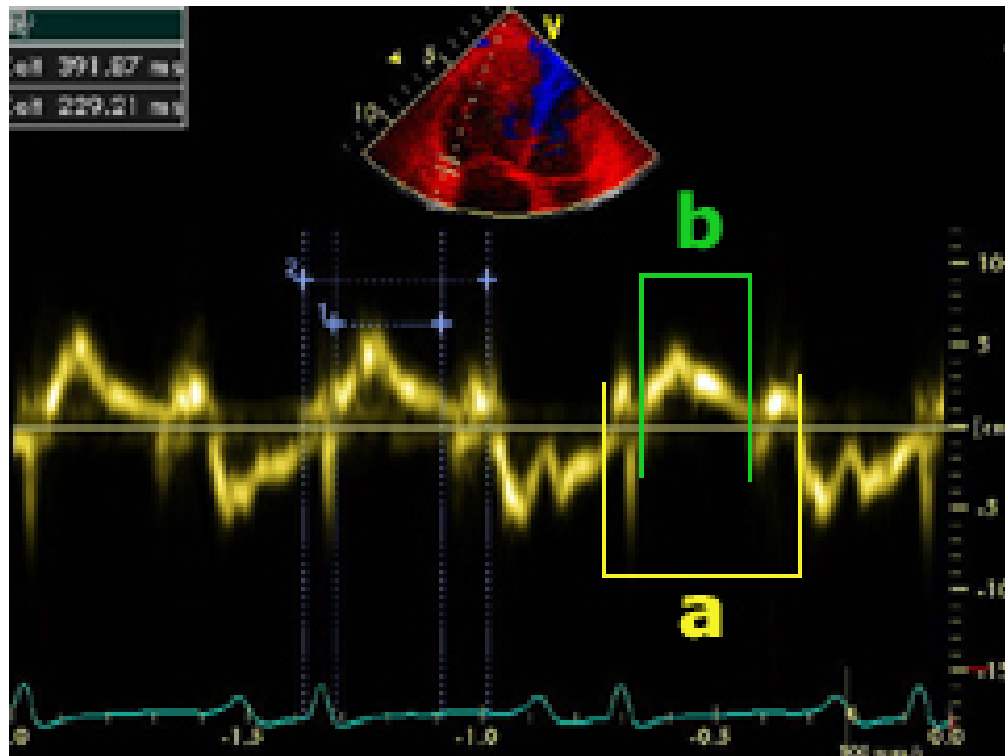
The average normal value of the Tei in-dex is $0.39 \pm$ while for the right ventricle (RV) it is 0.28 ± 0.04 .

In adults' population, values of the L.Ventricle index less than 0.40 and for the R.Ventricle less than 0.30 are considered normal.

The Tei index value will be increased depending upon the pathologic condition of ventricular myocardium.

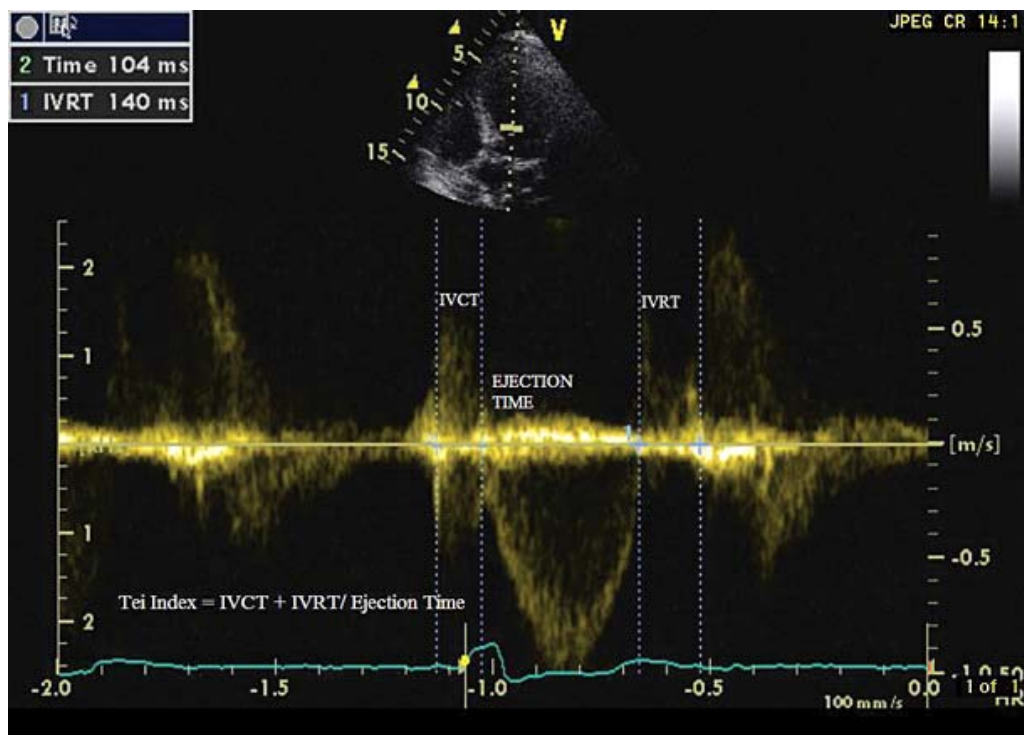


Tei index measurement



Tei Measurement by TDI

Tei index measurement



METHODOLOGY

Patients:

102 random diabetic populations who are attending cardiology Department, madras medical college for cardiac assessment are taken for study and included as study population. Those diabetic population with clinically normal heart like normal LV systolic function, without any chest pain, previous heart attack, cardiac failure. And also they should not have imaging signs of myocardial infarction in ECG and ECHO. There are no age limit and for the patient in our study.

Relevant history, clinical examination, biochemistry parameters, E.C.G, echocardiography was taken for all the patients.

Echocardiography examinations were performed Philips HD7XE Echocardiography machine) with a 2.5-Megta Hz probe. the echoardiographic index like IVS & posterior wall thickness, L.Ventricle functions, E wave flow velocity Decel eration time ;mitral inflow velocity ratio between E and A wave; fractional shortening and LV ejection index ;time of LV emptying (ejection time) ;time during isovolumic contraction (IV CT); time during isovolumic relaxation (IV RT);thickness of IVS; left atrium dimension; left ventricle dimension

during diastolic; LV MI (left ventricle mass and index); left ventricle dimension during systolic); thickness of posterior wall were measured for the study population with angina symptoms both clinically and imaging wise.

INCLUSION CRITERIA:

All patients with Diabetics, with L.Ventricle EF > 55%, no previous H/O MI, angina, heart failure attending cardiology OPD in Rajiv Gandhi Government General Hospital, Chennai.

EXCLUSION CRITERIA

We excluded patients with

- rheumatic heart disease
- arrhythmias which can confound in mitral inflow like fibrillation, av block
- heart disease from birth;
- hyper thyroidism; hypo-thyroidism; and
- lung diseases.

The study population of 102 patients was divided into three groups;

- * NO albuminuria - 40 patients (22 male and 18 female, average age 51)
- * MICRO albuminuria - 40 patients (25 male & 10 female with average, age 49yrs)
- * MACRO albuminuria - 22 patients (12 male & 10 female with average age 51yrs)

Echocardiography

The study population were examined using echo machine (Phillips with a probe frequency of 2.5 Mega Hz .) the echocardiography index like IVS & posterior wall thickness, L.Ventricle functions, E wave flow velocity Deceleration time ;mitral inflow velocity ratio between E and A wave; fractional shortening and LV ejection index ;time of LV emptying (ejection time) ;time during isovolumic contraction (IV CT); time during isovolumic relaxation (IV RT);thickness of IVS; left atrium dimension; left ventricle dimension during diastolic;LV MI(left ventricle mass and index); left ventricle dimension during systolic); thickness of posterior wall.

Biochemical Parameters: Measurement of albumin-to -creatinine ratio in a random or first MOR ning spot collection.

Micro Albuminuria can be proposed as daily excretions of 30 to 300 mg/day of albumin in urine and macro

Albuminuria be proposed as daily excretions of rate 3 00 mg/day.

Analysis:

The final value are mentioned as mean standard deviation. The values were studied with Kol mogorov-S mirnov method. Correlation of various groups (as noalbuminuria, micro Albuminuria, macro bu- minuria) done by Krus kal-Wal lis method. The test was used to determine the differences of the cat egoric variables between the groups. Pear son and Spear man correlation co-efficient were used for calculation. The cor relation of urine albumin and various echo indices were studied using regression stepwise multiple analysis. The p value which are less than 0.4 are considered to significant.

DATA ANALYSIS FROM THE OBSERVED VALUE

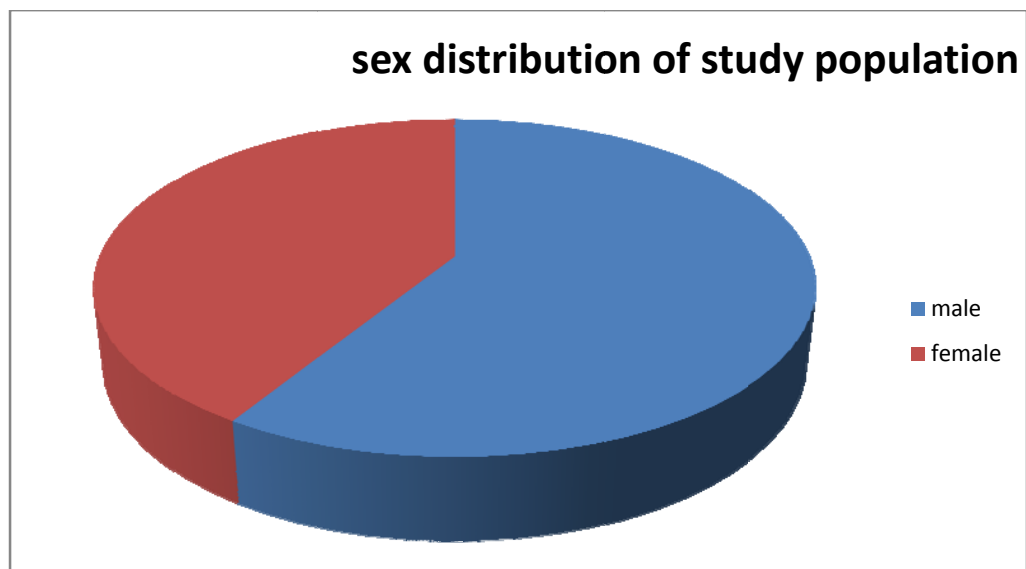
102 patients of established diabetes patient were selected for the study. Among them 60 were males and 42 were females. Female have significant higher percentage of no Albuminuria and micro Albuminuria than with macro albuminuria. 60% of all patients have systemic hypertension. The degree of urine albumin is increased as the duration diabetic years increases from NO albuminuria to micro & macroalbuminuria. Serum creatinine was increased with the patients having macroalbuminuria than patients with micoalbuminuria and no albuminuria. The values are

- Noalbumiuric patients: 12 ± 6 8.0 mg/d
- Micro albuminric patients: 85 ± 50 mg/d
- Macro Albuminuria patients: 889 ± 564 mg/d

Age of the patients ranged from 29 years to 70 years with the mean value of age is 48.5 years. Among female patients age range from 34 years to 68 years with the mean age of 46.5 years. For the male patients it ranged from 29 years to 70 years with mean age of 49.5 years.

Table 1: Age and Sex distribution of Study Population

Age Group	Male		Female		Total	
	No of Patient	%	No of Patients	%	No of Patients	%
< 31	1	4.3	0	5.9	1	5
31- 40	10	26.1	8	23.5	18	25
41 -50	14	26.1	10	23.5	24	25
51 – 60	19	26.1	14	35.3	33	30
61 – 70	16	17.4	10	11.8	26	15
Total	60	100	42	100	102	100
Mean ± S D	49.0 ± 11.38		49.52 ± 10.9		49.22 ± 11.04	



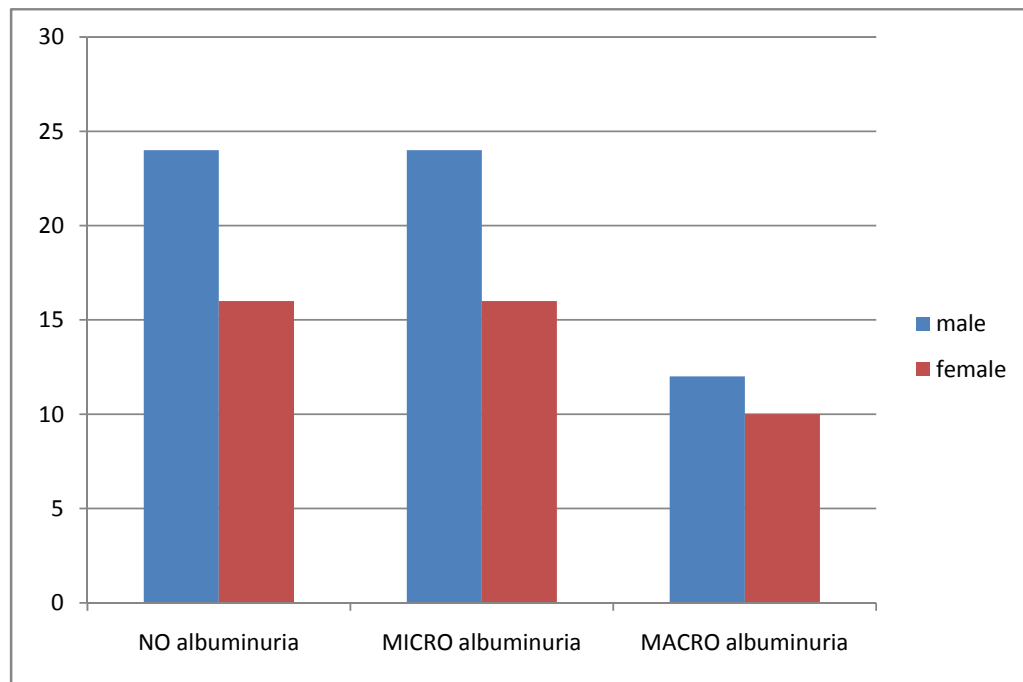
Among the male patients Albuminuria is more common in the age group between 40-60 years of age. Albuminuria is less common before 40years of age in both males and females. In female gender high prevalence was noted between 50-60 years of age. In the age group of 60-70 years Albuminuria is slightly higher in the females when compared to males.

Among these 102 patients 40 patients had no Albuminuria, 40 patients had micro Albuminuria and 22 patients macro Albuminuria. These comprises of 39% of patients with no Albuminuria, 39% of patients with micro Albuminuria and 21% of patients with macro Albuminuria

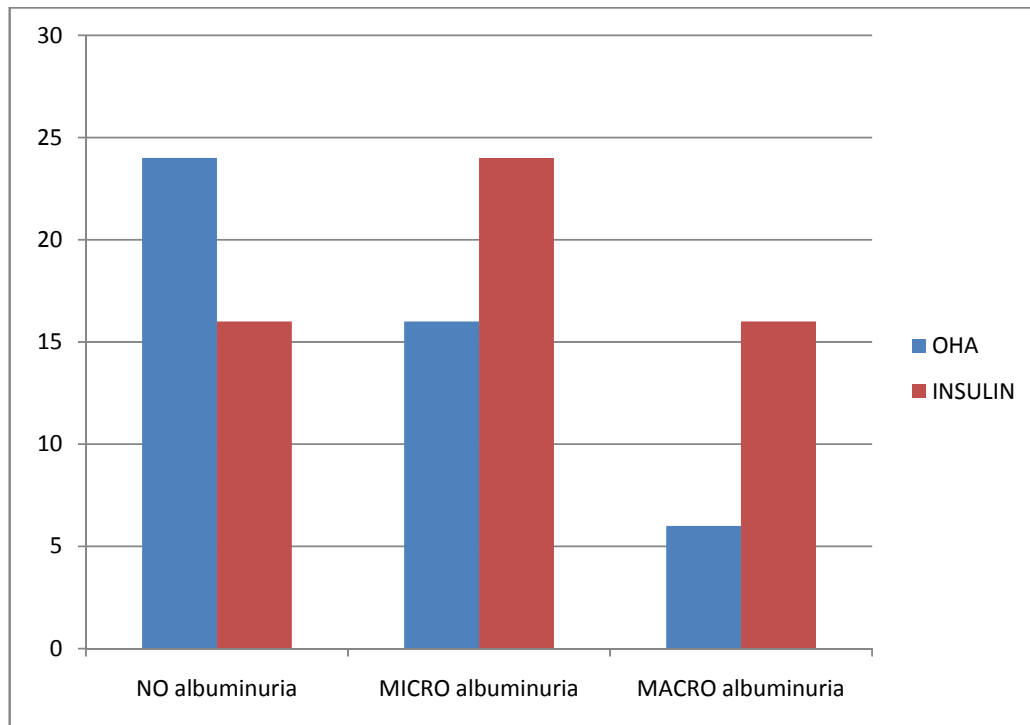
Table 2: Severity of Albuminuria

ALBUMINURIA	Male		Female		Total	
	No of Patient	%	No of Patients	%	No of Patients	%
NO	24	40	16	38	40	39.5
MICRO	24	40	16	38	40	39.5
MACRO	12	20	10	24	22	21
Total	60	100	42	100	102	100

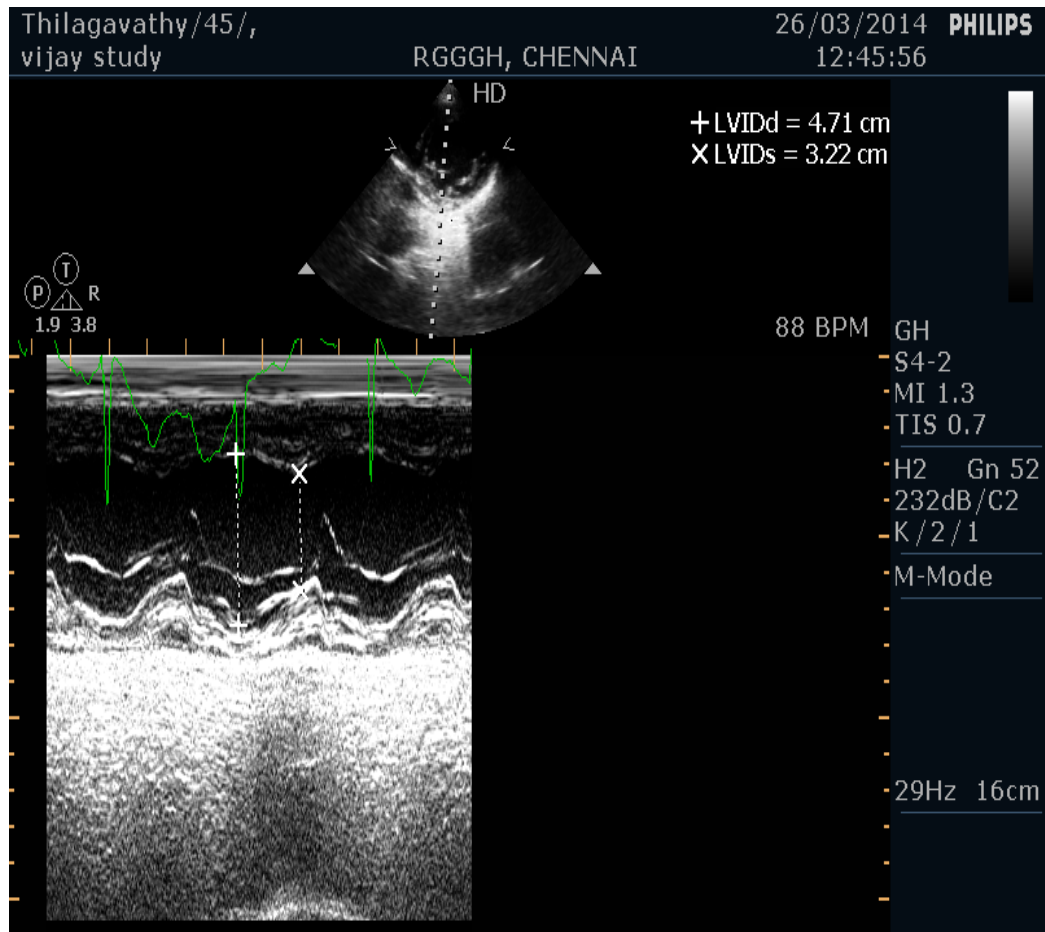
ALBUMINURIA BETWEEN MALES AND FEMALES



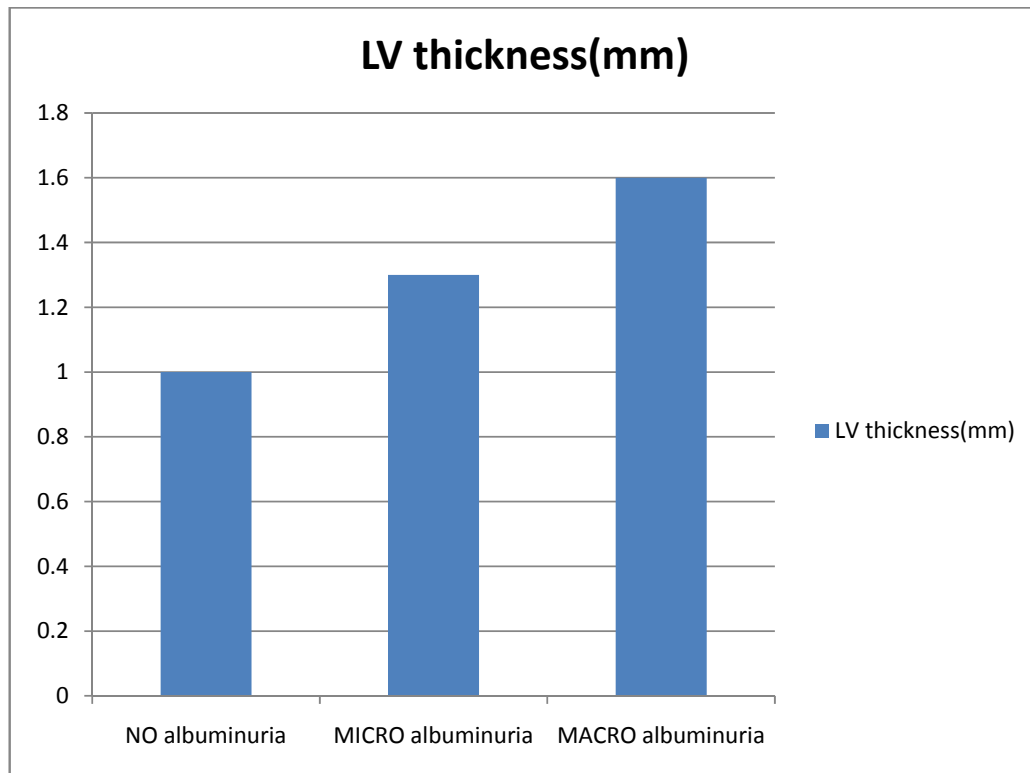
The prevalence of Albuminuria is more common in males. The severe Albuminuria prevalence is more prevalent in the males.



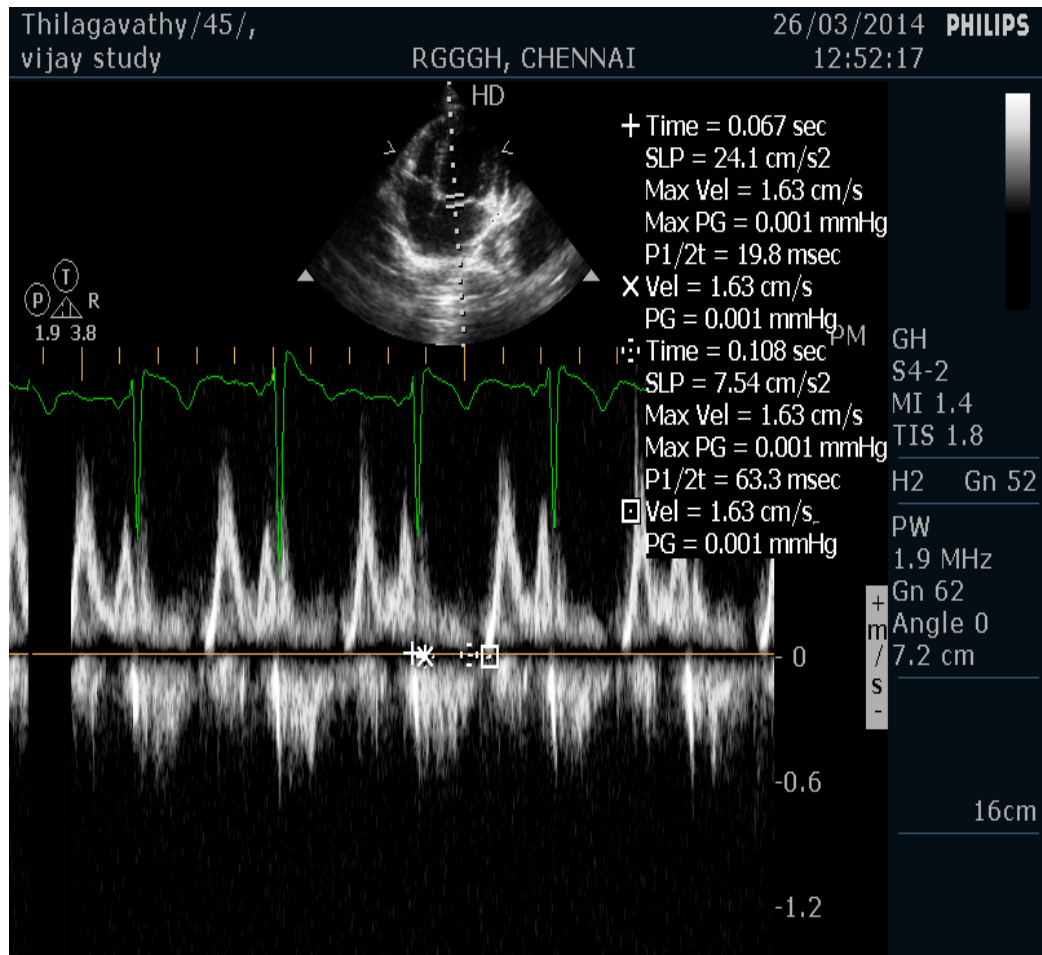
The levels of Albuminuria increase with the patients who is taking insulin which is positively correlated ($p < 0.005$). This can be due to long standing diabetes and diabetic complications for which the patients were started with insulin.



Picture represents normal LV contraction with normal ejection fraction in our study population.

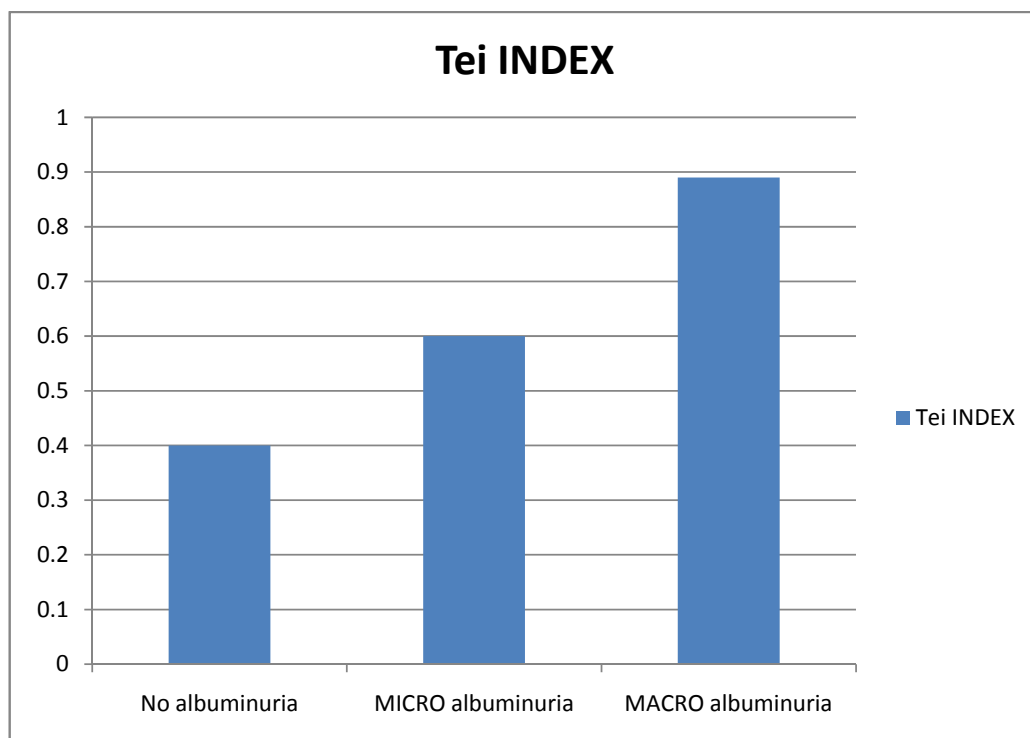


In our study population thicknesses of left ventricular PW in macro Albuminuria population ($P < 0.01$) were clearly increased than micro and noalbuminuric patients. Left ventricular mass index is proportionately increased from no to macro Albuminuria population.



Echo picture represents, calculation of various parameters for calculating TEI INDEX

The average TEI INDEX values were significantly larger than the general population. The TEI value were proportionately elevated from no Albuminuria pt to micro and macro Albuminuria patients ($p < 0.001$)

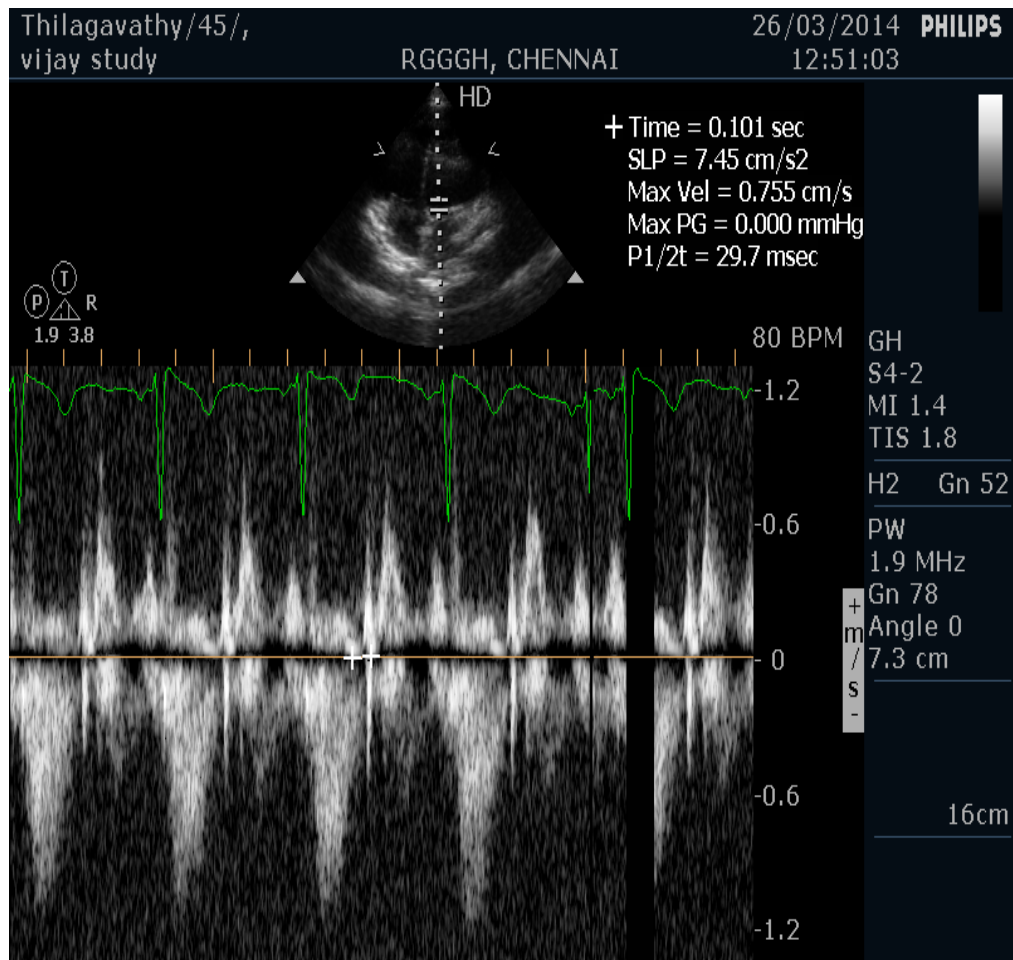


It's clearly evident from this study population that the usual echo parameters were normal and TEI value were begin to increase which shows that the TRI index predict the cardiac outcomes before it can be detected by conventional echo parameters.

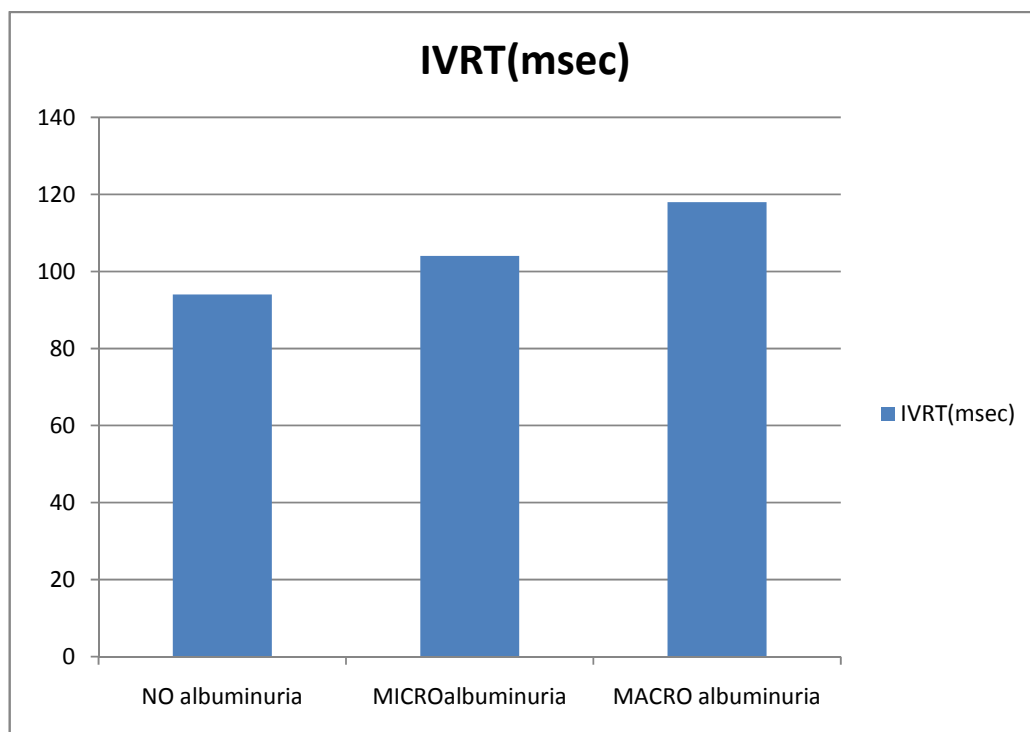
Study proposed that diabetic population will have diastolic malfunction before the systolic function is altered .our study population also shown similar findings and proven this which, clearly demonstrated by Liu al study.

The E wave deceleration time didn't show any significant difference between the 3 study groups (micro Albuminuria 211 ± 84 milli seconds, macro Albuminuria 209 ± 76 milli seconds) ;

Non Albuminuria population (195 ± 78 milli seconds) ($p > 0.1$).

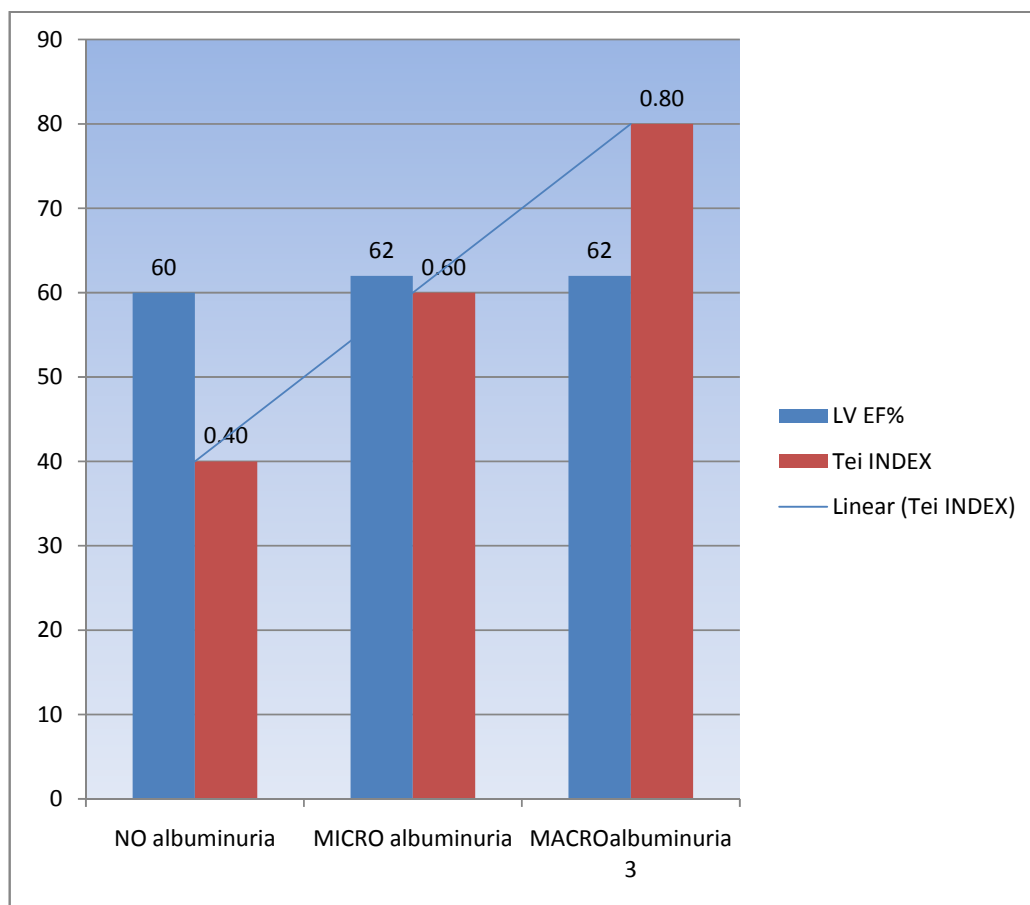


There is stepwise increase in IVRT in 3 groups of study population ($P < 0.004$). Watsc hinger et al study contradicts our findings which means in that study population there is no significant difference in IVRT



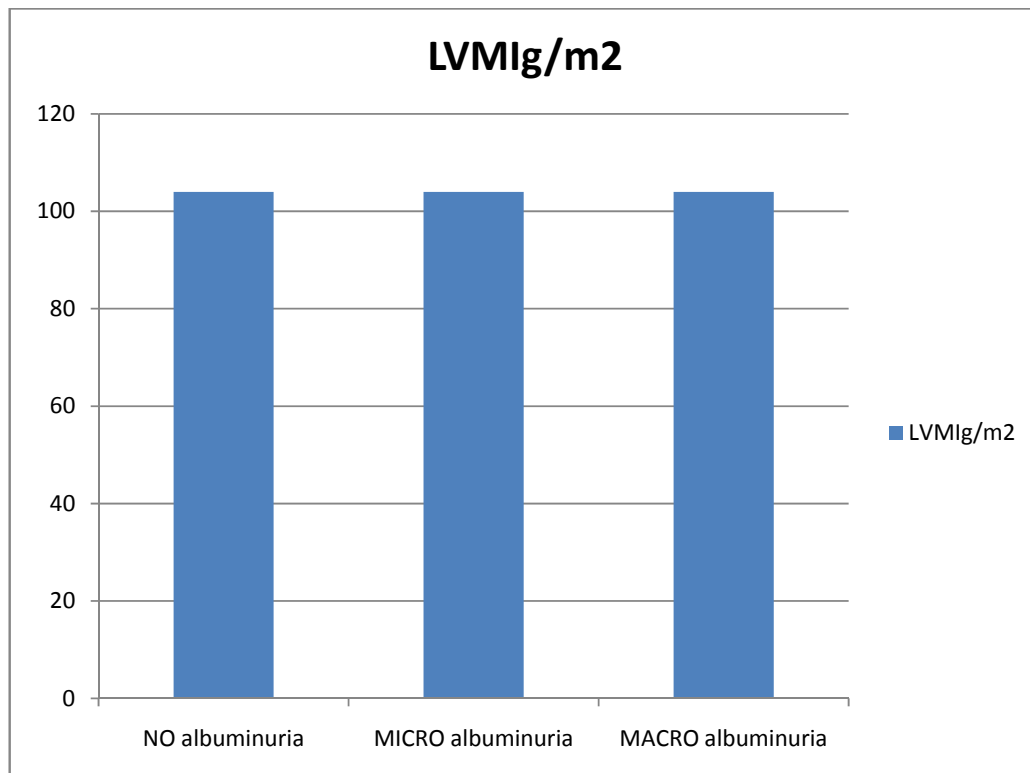
The cardiac function like Ef, RWMA were normal in our study group.

TEI INDEX which reflects the global LV function both systolic and diastolic function were increased guardedly from the increasing degree of urine Albuminuria excretion.



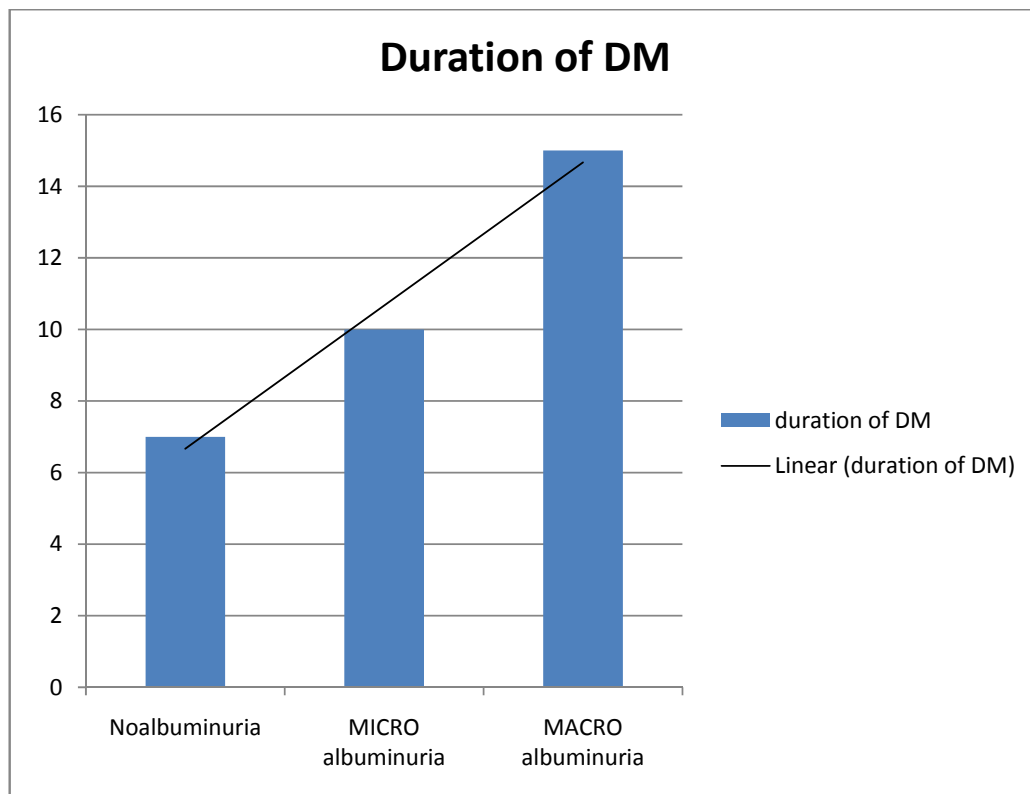
IVCT is influenced by preejection period and it is increased in our study population in graded manner and hence it is suggested that IV CT predicts impaired myocardial performance.

Patients with DM, coronary artery issues, micro-angiopathy, interstitial fibrosis, extra cellular collagen infiltration, calcium transport alterations, and neuro-hormonal modifications, can hinder cardiac function.



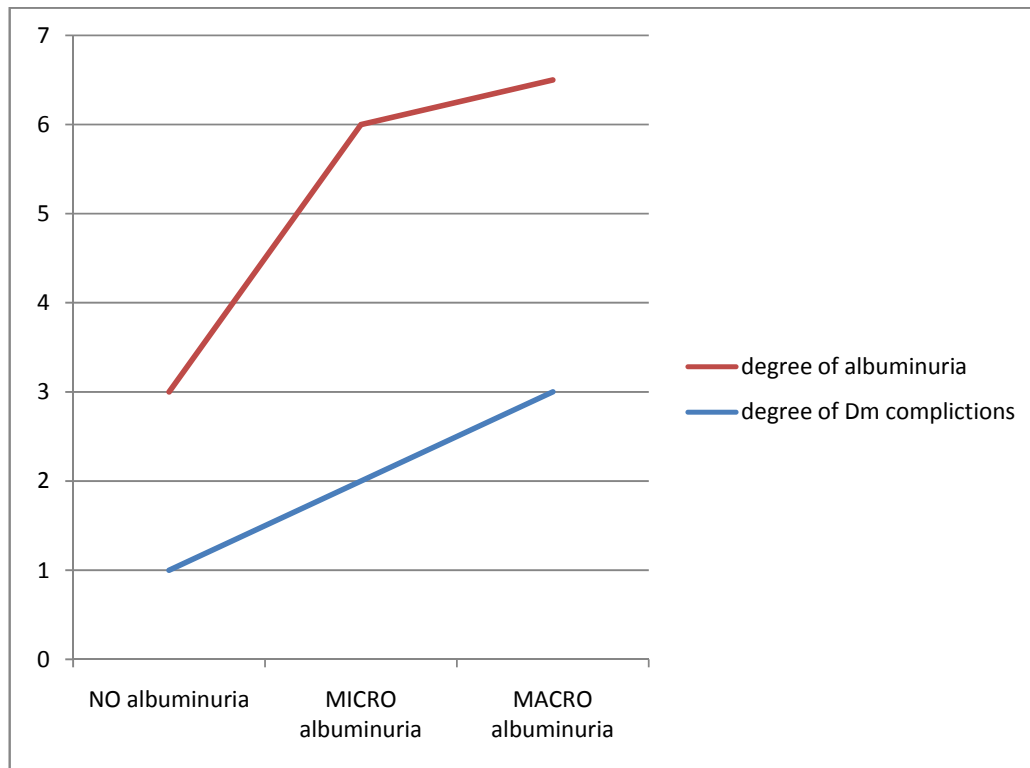
Left ventricular mass index is not altered in our 3 study groups ($p > 0.1$).

Our study also gives evidence for increasing TEI index in diabetic complication patients.



In this current study, the TEI is increase in a graded fashion from the study group ($p<0.03$).

The sub study analysis of our study groups shows tie index is correlated positively with other co morbid factor like creatinine, lipids level.



This graph represents the positive correlation of diabetic complications and duration of diabetes ($p < 0.02$).

By above all the observation, it was shown that TEI INDEX predicts the cardiac dysfunction before it can be detected by conventional echo parameters in diabetic pt with Albuminuria and it helps the diabetic population.

DISCUSSION

Our study was carried out to provide the views on Doppler-derived myocardial performance index which is classically proposed as TEI index in general diabetic patient diabetes mellitus with no Albuminuria as correlated with micro Albuminuria and macro Albuminuria. Our current studies, average value of TEIINDEX were found to be elevated than in the non diabetic population. The average TEI INDEX values were significantly larger than the general population. The TEI value were proportionally elevated from no albuminure pt to micro and macro Albuminuria patients ($p<0.001$)

The study groups have clearly shown that the normal IV

Function parameters like ejection fraction, RWMA were normal and TEI INDEX has shown increased in value and it helps to predict the diabetic cardiovascular dysfunction earlier.

Diabetic population was LV myocardial relaxing abnormality before LV systolic dys function occurs.

The mitral valve inflow velocity ratio was significantly decreasing from the 3 study groups which are shown in different studies.

The study population didn't show any difference in mitral E velocity deceleration time in 3 groups.

The study clearly shown that there is graded increase in IV RT in diabetic patient s with increasing severity of Albuminuria mainly because increase in pre ejection periods.

The Tei index were also shown graded increase in diabetic patients with more number of diabetic years, increasing HBA1C,diabetic complications, and patient taking insulin(reflecting the complications) .

TEI INDEX also increase graded in our study population with increase in serum creatinine, serum lipids.

CONCLUSION

There is clear-cut positive correlation between the LEFT VENTRICULAR TEI INDEX and the degree Albuminuria in diabetics.

LV TEI INDEX can be a useful marker in early detection of cardiac dysfunction in diabetic patients and helps to prognosticate the diabetic population.

LV Tei index is an early parameter to observe the ventricular dysfunction before it can be detected by conventional echocardiography parameters.

This index increases in stepwise pattern in diabetic population depending upon the duration of DM and diabetic complications

LIMITATIONS

The evidence of coronary artery disease cannot be ruled out confidentially because, in this study we used only history, ECG and ECHO for ruling out the CAD and gold standard like coronary angiogram, treadmill test was not done.

The other confounding factors

Like the treatment drugs which the study population are taking can confound the ventricular function cannot be excluded.

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PROFORMA

Name:

Age:

Sex:

Address:

CD No. :

Assesement of doppler derived myocardial performance index in diabetic patients with albuminuria

No albuminuria

Microalbuminuria

Macroalbuminuria

AGE

SEX

BMIA

HYPERTENSION

SMOKING

DURATION OF DM

TYPE OF DM

TOTAL CHOLESTROL

TRIGLYCERIDES

HDL

LDL

SERUM CR

ANTI DIABETIC DRUGS

INSULIN:

OHA

ANTI HYPERTENSIVE

ACEI

ATII BLOCKER

BB

CACHB

ECG : ISCHAEMIC CHANGES ±

CHEST X-RAY PA VIEW : CARDIOMEGALY ±

ECHOCARDIOGRAPHIC ASSESSMENT

Name:

Age:

Sex:

Echocardiography parameters:

M-mode:

LV: EDD- ESD- EF-
FS-

THICKNESS: IVS,PW

Mitral valve:

Aortic valve:

LVOT diameter:

LA:

Tricuspid valve:

Pulmonary valve:

2 D and DOPPLER HEMODYNAMIC ASSESSMENT:

Mitral inflow:

E-wave: Peak velocity- DT-

A-wave: Peak velocity- TVI-

E/A ratio:

ET :

TISSUE DOPPLER

Mitral annular: E' velocity- A' velocity-

E/E' ratio: MASV

TASV

E PROPOGATION VELOCITY

IVRT:

IVCT:

Tei INDEX : $IVCT+IVRT/ET$

Tricuspid inflow:

E-wave: Peak velocity-

DT-

A-wave: Peak velocity-

TVI-

PATIENT CONSENT FORM

Study Details :Assessment of Left Ventricular doppler derived myocardial performance index in diabetic patients with albuminuria

Patient may check (✓) these boxes.

PARTICIPANT NAME:

DATE:

AGE:

SEX:

I.P.NO. :

The details of the study have been provided to me in writing and explained to me in my own language.

I confirm that I have understood the purpose of the above study. I have the opportunity to ask the question and all my questions and doubts have been answered to my complete satisfaction.

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason, without my legal rights being affected.

I understand that investigator, the institution, regulatory authorities and the ethical committee will not need my permission to look at my health records both in respect to the 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 this study.

I hereby consent to undergo complete physical examination, and diagnostic tests including hematological, biochemical, radiological and urine examinations

I have been given an information sheet giving details of the study.

I hereby consent to participate in the above study

Signature of the Participant

INFORMATION SHEET

- We are conducting a study of the "**Assesement of doppler derived myocardial performance index in diabetic patients with albuminuria** "at theDepartment of Cardiology ,Rajiv Gandhi Govt. General Hospital, Chennai.
- The purpose of this study is to analyse the association of Doppler derived myocardial performance index in diabetic patients with albuminuria.
- The privacy of the patients 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 this 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 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 the investigator

Signature of the participant

Originality
GradeMark
PeerMark

ASSESSMENT OF DOPPLER DERIVED MYOCARDIAL PERFORMANCE INDEX IN

BY: R111512, D.M. CARDIOLOGY, VILVASEKARAN B. RAJASIEKARANAIKAI



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Match Overview



1
ASSESSMENT OF DOPPLER DERIVED MYOCARDIAL PERFORMANCE INDEX IN DIABETIC PATIENTS WITH ALBUMINURIA

2
Dissertation submitted to

3
THE TAMIL NADU DR. MGR. MEDICAL UNIVERSITY


4
In partial fulfillment of the requirements for the award of the degree of

5
D.M. CARDIOLOGY

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BRANCH II - CARDIOLOGY

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MASTER CHART

SLNO	Name	Age	Gender	Wt	Ht	BMI	ACR(mcg)	CHO	HDL	LDL	VLDL	SMOKING	DURATION OF DMMVBL	HBA1C	SERUM CREATININE	ANTI DIABETIC DRUGS(OHA/INSULIN)	hypertension	IVS thickness (cm)	PW thickness (cm)	LVSD (cm)	LVDD (cm)	EF (%)	E/A ratio	E DT (ms)	IVRT (ms)	IVCT (ms)	Tei INDEX	ET(msec)
1	Aanbalagan	65	1	53	152	22.94	250	120	24	79	17	Y	8.0	8.5	0.8	OHA	Y	1.0	0.9	2.9	4.7	65	1.0	196	94	41	0.5	173
2	Bashir	44	1	57	150	25.33	600	142	30	100	12	Y	9	9.1	0.9	insulin	Y	1.2	1.21	2.8	4.5	64	0.8	212	104	48	0.6	182
3	Elumalai	49	1	62	172	20.96	25	165	34	104	27	N	9.5	9.4	1.4	OHA	N	1.3	1.3	2.6	4.4	62	0.7	215	105	52	0.7	180
4	Aruldoss	59	1	66	171	22.57	37	165	33	111	21	N	9.0	7.9	2.8	Insulin	Y	1.9	2.2	3.0	4.9	69	1.2	222	114	56	0.9	198
5	Bakthavatchalam	37	1	67	169	23.46	368	224	49	121	54	N	11	8.9	1.0	OHA	N	0.6	0.7	2.4	4.5	67	0.9	209	100	41	0.4	160
6	Subramani	57	1	53	158	21.23	900	224	50	145	29	N	7.0	10	0.6	OHA	N	1.1	1.1	3.2	4.2	58	0.8	211	90	39	0.44	187
7	Srinivasan	57	1	66	170	22.84	400	142	28	94	20	N	7.1	7.0	1.1	OHa	N	1.1	1.0	3.6	4.6	62	1.2	209	103	44	0.41	189
8	Indira	51	2	50	166	18.14	23	357	46	124	28	N	8.0	6.3	0.8	OHA	Y	0.9	1.0	3.3	4.7	67	1.6	212	102	40	0.44	150
9	Gunasekar	45	1	50	145	23.78	21	327	65	234	28	Y	9.0	8.5	0.8	OHA	Y	1.0	0.9	2.9	4.7	65	1.0	196	94	41	0.5	173
10	ganesh	53	1	55	169	19.26	400	181	36	132	13	N	11	9.1	0.9	insulin	N	1.2	1.21	2.8	4.5	64	0.8	212	104	48	0.6	182
11	faizer ali	48	1	54	162	20.58	23	216	44	130	42	N	7.0	9.4	1.4	OHA	Y	1.3	1.3	2.6	4.4	62	0.7	215	105	52	0.7	180
12	ellappan	48	1	85	170	29.41	21	219	44	143	32	N	8.0	7.9	2.8	Insulin	Y	1.9	2.2	3.0	4.9	69	1.2	222	114	56	0.9	198
13	Ekambaram	41	1	89	182	26.87	30	203	30	95	78	N	9	8.9	1.0	OHA	Y	0.6	0.7	2.4	4.5	67	0.9	209	100	41	0.4	160
14	Dhanasekar	53	1	70	176	22.60	1	242	49	142	51	N	9.5	10	0.6	OHA	Y	1.1	1.1	3.2	4.2	58	0.8	211	90	39	0.44	187
15	Baskaran	23	1	66	164	24.54	849	188	39	116	33	Y	7.1	7.0	1.1	OHa	N	1.1	1.0	3.6	4.6	62	1.2	209	103	44	0.41	189
16	Arumugam	28	1	54	153	23.07	157	172	36	117	19	Y	8.0	6.3	0.8	OHA	N	0.9	1.0	3.3	4.7	67	1.6	212	102	40	0.55	150
17	arthikani	36	2	55	151	24.12	190	247	50	157	40	N	9.0	8.5	0.8	OHA	N	1.0	0.9	2.9	4.7	65	1.0	196	94	41	0.5	173
18	Antony	59	1	63	174	20.81	456	278	57	190	31	N	11	9.1	0.9	insulin	Y	1.2	1.21	2.8	4.5	64	0.8	212	104	48	0.6	182
19	Alli	44	2	78	172	26.37	243	358	46	135	35	N	7.0	9.4	1.4	OHA	y	1.3	1.3	2.6	4.4	62	0.7	215	105	52	0.7	180
20	Ahmed basha	40	1	65	167	23.31	221	253	51	150	52	Y	5.6	7.9	2.8	Insulin	Y	1.9	2.2	3.0	4.9	69	1.2	222	114	56	0.9	198
21	Ahmad Ullakhan	58	1	62	165	22.77	21	210	38	148	24	N	8.0	8.9	1.0	OHA	Y	0.6	0.7	2.4	4.5	67	0.9	209	100	41	0.4	160

22	Abdhul Razak	45	1	54	162	20.58	23	157	34	84	39	N	6.3	10	0.6	OHA	N	1.1	1.1	3.2	4.2	58	0.8	211	90	39	0.44	187
23	abdul	49	1	57	160	22.27	234	159	32	73	54	Y	9.0	7.0	1.1	OHa	Y	1.1	1.0	3.6	4.6	62	1.2	209	103	44	0.41	189
24	Arumugam	44	1	75	176	24.21	235	205	41	126	38	N	11	6.3	0.8	OHA	N	0.9	1.0	3.3	4.7	67	1.6	212	102	40	0.41	150
25	Periyasamy	45	1	41	159	16.22	250	184	37	131	16	N	7.0	8.5	0.8	OHA	N	1.0	0.9	2.9	4.7	65	1.0	196	94	41	0.5	173
26	Pasupathi	60	1	80	160	31.25	600	317	63	207	47	N	5.5	9.1	0.9	insulin	N	1.2	1.21	2.8	4.5	64	0.8	212	104	48	0.6	182
27	Panner Selvam	58	1	56	170	19.38	25	245	49	133	63	N	8.0	9.4	1.4	OHA	Y	1.3	1.3	2.6	4.4	62	0.7	215	105	52	0.7	180
29	Pahurdeen	65	1	39	160	15.23	37	178	38	114	26	Y	9	7.9	2.8	Insulin	Y	1.9	2.2	3.0	4.9	69	1.2	222	114	56	0.9	198
30	Nagaraj	60	1	66	180	20.37	368	202	40	125	37	N	9.5	8.9	1.0	OHA	N	0.6	0.7	2.4	4.5	67	0.9	209	100	41	0.4	160
31	Murugan	44	1	57	150	25.33	900	142	30	100	12	Y	9.0	10	0.6	OHA	Y	1.1	1.1	3.2	4.2	58	0.8	211	90	39	0.44	187
32	Murali	60	1	68	170	23.53	400	310	62	209	39	N	11	7.0	1.1	OHa	Y	1.1	1.0	3.6	4.6	62	1.2	209	103	44	0.41	189
33	Vijayan	52	1	51	174	16.85	23	292	58	217	17	Y	7.0	6.3	0.8	OHA	Y	0.9	1.0	3.3	4.7	67	1.6	212	102	40	0.6	150
34	Vijayakumari	52	2	55	180	16.98	21	258	45	135	22	N	5.2	8.5	0.8	OHA	Y	1.0	0.9	2.9	4.7	65	1.0	196	94	41	0.5	173
35	Vijay	60	1	71	153	30.33	400	193	39	115	39	N	8.0	9.1	0.9	insulin	N	1.2	1.21	2.8	4.5	64	0.8	212	104	48	0.6	182
36	Veeramuthu	50	1	66	176	21.31	23	238	49	157	23	Y	9	9.4	1.4	OHA	N	1.3	1.3	2.6	4.4	62	0.7	215	105	52	0.7	180
37	Thulasidoss	67	1	56	169	19.61	21	234	45	171	18	N	9.5	7.9	1.9	OHA	N	1.1	1.1	3.2	4.2	58	0.8	211	90	39	0.44	187
38	Thangamani	55	1	49	153	20.93	30	155	31	98	26	Y	6.7	7.9	2.8	Insulin	Y	1.9	2.2	3.0	4.9	69	1.2	222	114	56	0.9	198
39	Ravichndran	54	1	62	165	22.77	1	210	38	148	24	N	9.0	8.9	1.0	OHA	Y	0.6	0.7	2.4	4.5	67	0.9	209	100	41	0.4	160
40	Tajum Begam	40	2	70	170	24.20	849	264	53	182	29	N	11	10	0.6	OHA	N	1.1	1.1	3.2	4.2	58	0.8	211	90	39	0.44	187
41	Rathakrishnan	45	1	76	159	30.06	157	222	44	144	34	N	7.0	7.0	1.1	OHa	Y	1.1	1.0	3.6	4.6	62	1.2	209	103	44	0.41	189
42	Anif	50	1	56	160	21.88	190	135	28	129	22	N	9.0	6.3	0.8	OHA	N	0.9	1.0	3.3	4.7	67	1.6	212	102	40		150
43	allathiya	48	2	61	176	19.69	456	276	55	141	80	N	11	6.4	0.9	OHA	N	1.1	1.1	3.2	4.2	58	0.8	211	90	39	0.44	187
44	Kesavan	42	1	59	170	20.42	243	138	20	86	32	N	7.0	8.5	0.8	OHA	N	1.0	0.9	2.9	4.7	65	1.0	196	94	41	0.5	173
45	Selvaraj	50	1	51	157	20.69	221	156	32	82	42	Y	8.0	9.1	0.9	insulin	Y	1.2	1.21	2.8	4.5	64	0.8	212	104	48	0.6	182
46	Xavier	58	1	73	173	24.39	21	140	28	92	20	N	9	9.4	1.4	OHA	Y	1.3	1.3	2.6	4.4	62	0.7	215	105	52	0.7	180
47	Stephen	40	1	70	160	27.34	23	217	44	130	43	N	9.5	7.9	2.8	Insulin	N	1.9	2.2	3.0	4.9	69	1.2	222	114	56	0.9	198
48	Shanthi	60	2	50	143	24.45	234	240	50	162	28	N	9.0	8.9	1.0	OHA	Y	0.6	0.7	2.4	4.5	67	0.9	209	100	41	0.4	160
49	Shanmugam	45	1	63	167	22.59	235	190	49	104	37	N	11	10	0.6	OHA	Y	1.1	1.1	3.2	4.2	58	0.8	211	90	39	0.44	187
50	Shankar	63	1	57	158	22.83	250	147	30	98	19	Y	7.0	8.5	0.8	OHA	Y	1.0	0.9	2.9	4.7	65	1.0	196	94	41	0.5	173

51	S.Rajan	66	1	55	166	19.96	600	212	43	151	18	N	7.1	9.1	0.9	insulin	Y	1.2	1.21	2.8	4.5	64	0.8	212	104	48	0.6	182
52	Ravi	61	1	60	168	21.26	25	136	41	71	24	Y	8.0	9.4	1.4	OHA	N	1.3	1.3	2.6	4.4	62	0.7	215	105	52	0.7	180
53	Ramu	39	1	86	186	24.86	37	196	40	125	31	Y	9.0	7.0	1.1	OHa	N	1.1	1.0	3.6	4.6	62	1.2	209	103	44	0.41	189
54	Santhanam	66	1	54	150	24.00	368	321	45	168	45	N	11	6.3	0.8	OHA	N	0.9	1.0	3.3	4.7	67	1.6	212	102	40	0.6	150
55	Ravindran	48	1	72	171	24.62	900	167	34	95	38	Y	7.0	8.5	0.8	OHA	Y	1.0	0.9	2.9	4.7	65	1.0	196	94	41	0.5	173
56	Mary	43	2	78	162	29.72	400	152	30	106	16	N	7.1	9.1	0.9	insulin	Y	1.2	1.21	2.8	4.5	64	0.8	212	104	48	0.6	182
57	Jayaraman	45	1	52	165	19.10	23	253	51	160	42	N	8.0	9.4	1.4	OHA	N	1.3	1.3	2.6	4.4	62	0.7	215	105	52	0.7	180
58	Kanniyammal	53	2	64	139	33.10	21	173	37	97	39	N	8.0	7.9	2.8	Insulin	Y	1.9	2.2	3.0	4.9	69	1.2	222	114	56	0.9	198
59	KUMAR	47	1	60	165	22.00	400	176	36	90	50	Y	9	8.9	1.0	OHA	N	0.6	0.7	2.4	4.5	67	0.9	209	100	41	0.4	160
60	Lalitha	48	2	70	165	25.71	23	253	51	150	52	N	9.5	10	0.6	OHA	N	1.1	1.1	3.2	4.2	58	0.8	211	90	39	0.44	187
61	Lalitha	60	2	60	151	26.30	21	177	36	92	49	N	6.1	8.5	0.8	OHA	N	1.0	0.9	2.9	4.7	65	1.0	196	94	41	0.5	173
62	Kousalya	41	2	67	169	23.46	30	224	49	121	54	N	9	9.1	0.9	insulin	Y	1.2	1.21	2.8	4.5	64	0.8	212	104	48	0.6	182
63	Kamakshi	47	2	50	169	17.51	1	198	40	136	22	N	6.1	9.4	1.4	OHA	Y	1.3	1.3	2.6	4.4	62	0.7	215	105	52	0.7	180
64	Ibrahim	46	1	65	166	23.60	849	134	43.5	68	39	N	9	7.0	1.1	OHa	N	1.1	1.0	3.6	4.6	62	1.2	209	103	44	0.41	189
65	gopi	47	1	62	153	26.49	157	230	48	131	51	Y	8.0	6.3	0.8	OHA	Y	0.9	1.0	3.3	4.7	67	1.6	212	102	40	0.6	150
66	Ganesan	49	1	56	159	22.10	190	171	36	112	23	N	9	8.5	0.8	OHA	Y	1.0	0.9	2.9	4.7	65	1.0	196	94	41	0.5	173
67	Eswaran	58	1	70	157	28.30	456	132	28	70	34	Y	9.5	9.1	0.9	insulin	Y	1.2	1.21	2.8	4.5	64	0.8	212	104	48	0.6	182
68	Balaraman	54	1	82	174	27.10	243	269	55	183	31	N	7.2	9.4	1.4	OHA	Y	1.3	1.3	2.6	4.4	62	0.7	215	105	52	0.7	180
69	Ramaiah	60	1	51	155	21.23	221	108	25	58	25	Y	7.9	2.8	1.1	Insulin	N	1.9	2.2	3.0	4.9	69	1.2	222	114	56	0.9	198
70	Vijaykumar	60	1	67	152	29.00	21	177	34	110	33	Y	8.9	1.0	0.8	OHA	N	0.6	0.7	2.4	4.5	67	0.9	209	100	41	0.4	160
71	Vijayakumar	48	1	55	167	19.70	23	106	22	53	31	N	10	0.6	1.1	OHA	N	1.1	1.1	3.2	4.2	58	0.8	211	90	39	0.44	187
72	Venkatachalapat	45	1	61	180	18.83	234	185	37	124	24	N	6.1	0.8	0.8	OHA	Y	1.1	1.0	3.6	4.6	62	1.2	209	103	44	0.41	189
73	Varadan	49	1	68	160	26.00	235	234	48	145	41	N	9	8.5	0.8	OHA	Y	1.0	0.9	2.9	4.7	65	1.0	196	94	41	0.5	173
74	Thiagaraj	60	1	50	153	21.40	250	142	30	90	22	Y	7.1	9.1	0.9	insulin	N	1.2	1.21	2.8	4.5	64	0.8	212	104	48	0.6	182
75	Subramani	60	1	58	164	21.56	600	180	36	123	21	N	8.0	9.4	1.4	OHA	Y	1.3	1.3	2.6	4.4	62	0.7	215	105	52	0.7	180
76	Sumathy	42	2	68	157	27.60	25	163	33	116	14	N	9	7.0	1.1	OHa	N	1.1	1.0	3.6	4.6	62	1.2	209	103	44	0.41	189
77	Ramu	57	1	50	145	23.78	37	327	65	234	28	N	9.5	6.3	0.8	OHA	N	0.9	1.0	3.3	4.7	67	1.6	212	102	40	0.6	150
78	Raji	32	1	80	160	31.23	368	271	46	196	29	Y	6.1	7.0	1.1	OHa	N	1.1	1.0	3.6	4.6	62	1.2	209	103	44	0.41	189

79	Hussain	55	1	70	167	25.10	900	240	48	119	73	N	9	6.3	0.8	OHA	Y	0.9	1.0	3.3	4.7	67	1.6	212	102	40	0.6	150
80	Nagaraj	38	1	83	171	28.42	400	210	26	160	24	N	6.3	8.5	0.8	OHA	Y	1.0	0.9	2.9	4.7	65	1.0	196	94	41	0.5	173
81	James	52	1	65	167	23.30	23	258	32	120	20	Y	6.1	9.1	0.9	insulin	N	1.2	1.21	2.8	4.5	64	0.8	212	104	48	0.6	182
82	Joseph	50	1	58	169	20.28	21	270	37	203	30	Y	9	9.4	1.4	OHA	Y	1.3	1.3	2.6	4.4	62	0.7	215	105	52	0.7	180
83	Baskaran	67	1	72	174	23.70	400	318	40	244	34	Y	6.0	7.0	1.1	OHa	Y	1.1	1.0	3.6	4.6	62	1.2	209	103	44	0.41	189
84	Bagvathi	55	1	75	162	27.98	23	158	30	102	26	N	6.1	6.3	0.8	OHA	Y	0.9	1.0	3.3	4.7	67	1.6	212	102	40	0.6	150
85	Devi Bai	45	2	60	172	23.47	21	187	35	125	30	N	9	7.0	1.1	OHa	Y	1.1	1.0	3.6	4.6	62	1.2	209	103	44	0.41	189
86	Duraisamy	37	1	72	164	26.00	30	393	42	286	65	N	8.0	6.3	0.8	OHA	N	0.9	1.0	3.3	4.7	67	1.6	212	102	40	0.6	150
87	Mohammed Ghouse	54	1	60	161	23.00	1	210	50	126	34	Y	9	8.5	0.8	OHA	N	1.0	0.9	2.9	4.7	65	1.0	196	94	41	0.5	173
88	Pappammal	47	2	71	155	29.58	849	178	32	122	24	N	9.5	9.1	0.9	insulin	N	1.2	1.21	2.8	4.5	64	0.8	212	104	48	0.6	182
89	Rajendran	50	1	76	167	27.21	157	248	49	162	37	N	6.1	9.4	1.4	OHA	Y	1.3	1.3	2.6	4.4	62	0.7	215	105	52	0.7	180
90	Sheik Mohideen	52	1	58	165	21.32	190	270	40	208	22	N	9	7.0	1.1	OHa	Y	1.1	1.0	3.6	4.6	62	1.2	209	103	44	0.60.41	189
91	Sugumaran	38	1	77	163	28.95	456	215	50	140	25	Y	6.2	6.3	0.8	OHA	N	0.9	1.0	3.3	4.7	67	1.6	212	102	40		150
92	Vijaya	51	2	81	167	29.03	243	214	46	140	28	N	6.1	8.5	0.8	OHA	Y	1.0	0.9	2.9	4.7	65	1.0	196	94	41	0.5	173
93	Velu	51	1	75	151	26.60	221	272	48	191	33	N	9	9.1	0.9	insulin	N	1.2	1.21	2.8	4.5	64	0.8	212	104	48	0.6	182
94	Venkatesh	50	1	64	152	27.00	21	217	41	150	26	N	8.0	9.4	1.4	OHA	N	1.3	1.3	2.6	4.4	62	0.7	215	105	52	0.7	180
95	Velu	33	1	70	168	24.82	23	193	42	118	31	Yy	9	7.0	1.1	OHa	N	1.1	1.0	3.6	4.6	62	1.2	209	103	44	0.41	189
96	Ramu	65	1	60	166	21.76	234	226	52	134	40	Y	9.5	6.3	0.8	OHA	Y	0.9	1.0	3.3	4.7	67	1.6	212	102	40	0.6	150
97	Sakunthala	38	2	56	146	26.29	235	264	47	232	32	Y	6.1	8.5	0.8	OHA	Y	1.0	0.9	2.9	4.7	65	1.0	196	94	41	0.5	173
98	Pandiyan	37	1	62	149	27.93	250	328	49	243	36	N	9	9.1	0.9	insulin	N	1.2	1.21	2.8	4.5	64	0.8	212	104	48	0.6	182
99	Paramisivam	45	1	75	175	24.50	600	189	48	106	37	N	6.1	9.4	1.4	OHA	Y	1.3	1.3	2.6	4.4	62	0.7	215	105	52	0.7	180
100	Rajendran	31	1	56	168	19.86	25	186	42	116	28	Y	9	7.9	2.8	Insulin	Y	1.9	2.2	3.0	4.9	69	1.2	222	114	56	0.9	198
101	Rajendran	58	1	68	162	25.95	37	278	40	210	28	N	9.5	8.9	1.0	OHA	Y	0.6	0.7	2.4	4.5	67	0.9	209	100	41	0.4	160
102	Mohammed	36	1	75	175	24.50	368	189	48	106	37	N	8.1	10	0.6	OHA	Y	1.1	1.1	3.2	4.2	58	0.8	211	90	39	0.44	187