E-ISSN 2250-0944

ISSN 2250-1150

doi: 10.48047/ijprt/15.02.245

UTILITY OF FDG PET SCAN VIABILITY ASSESSMENT OF AKINETIC MYOCARDIAL SEGMENTS ON ECHO FOR PREDICTION OF CONTRACTILITY RECOVERY AT SIX MONTHS AFTER CORONARY ARTERY BYPASS SURGERY: A PROSPECTIVE OBSERVATIONAL STUDY

Imran Akram Khanday[1], Abdul Majid dar[2], Dr Aamir Rashid[3*], Dr Imran Hafeez[4]

[1]Senior Resident, Department of Cardiology, Medanta the Medicity, Gurugram, Haryana.

[2] Senior Resident, Department of Cardiology, SKIMS, Soura.

[3*]Additional Professor, Department of Cardiology SKIMS, Soura.

[4]Professor, Department of Cardiology, SKIMS, Soura.

Corresponding Author: Dr Aamir Rashid

Additional Professor, Department of Cardiology SKIMS, Soura.

Received date: 10-08-2025, Accepted date: 16-08-2025, Date of publication: 23-08-2025

Abstract

Introduction: Heart failure (HF) is a complex clinical syndrome resulting from structural and functional impairment of ventricular filling or ejection of blood. Approximately half of the patients with HF have normal left ventricular function, that is, HF with preserved ejection fraction (HFpEF), the balance have HF with reduced ejection fraction (HFrEF). HFpEF generally is defined as a left ventricular ejection fraction of 50% or greater, whereas HFrEF is defined as an ejection fraction below 40%. Because treatment strategies for treating HF are based on these two categories, these distinctions are crucial.

Materials and Methods: The study was conducted prospectively at Medanta Hospital in the department of Cardiology. All consecutive patients who met all inclusion criteria were enrolled. Informed consent was taken. Detailed history and clinical examination recorded. Heart failure symptoms were graded according to NYHA (1-4) and CCS (0-4) classification before and 3-6 months after CABG. A single operator performed 2D Transthoracic echocardiography to assess regional and global LV function before and after 3-6 months of CABG. Regional wall motion was divided in 16 segments. Coronary angiography was used to assess coronary anatomy. Thallium 201 scan used to study coronary perfusion. F18-Fluorodeoxyglucose (FDG) PET used to assess myocardial viability before CABG on a SIEMENS BIOGRAPH MCT S (64)-3R scanner.

Results: The majority of patients in this study were males (89.1%). Regarding Body Mass Index (BMI), 57.6% of patients had a normal BMI, 37.0% were overweight, and 5.4% were obese. The mean BMI was 24.6 ± 3.0. Hypertension was present in 62% of patients, Diabetes Mellitus in 76.1%, and 47.8% were smokers. A third of the patients had a sedentary lifestyle, and 38% were dyslipidemic. Out of 92 patients, 76.1% were diabetic, and 62% had hypertension. CKD and stroke were each present in 7.6% of patients. The majority of patients were in NYHA class 4 (67.1%), while 32.8% were in NYHA class 3.

Conclusion: Patients with large areas of viable myocardium (>18%) on PET scan and an improvement in >4 akinetic segments show the highest sensitivity and specificity for predicting an improvement in LVEF after CABG.

Key words: Heart failure, PET, myocardium, Coronary angiography, NYHA.

INTRODUCTION

Heart failure (HF) is a complex clinical syndrome resulting from structural and functional impairment of ventricular filling or ejection of blood. Approximately half of the patients with HF have normal left ventricular function, that is, HF with preserved ejection fraction (HFpEF), the balance have HF with reduced ejection fraction (HFrEF). HFpEF generally is defined as a left ventricular ejection fraction of 50% or greater, whereas HFrEF is defined as an ejection fraction below 40%. Because treatment strategies for treating HF are based on these two categories, these distinctions are crucial.[1]

The worldwide prevalence and incidence rates of heart failure (HF) are approaching epidemic proportions, as evidenced by the relentless increase in the number of HF-related hospitalizations, the growing number of HF-attributable deaths, and the spiralling costs associated with the care of patients with HF. Worldwide, HF affects nearly 23 million people. In the United States, the most recent epidemiologic data suggest that 5.1 million Americans 20 years of age and older have HF, and it is estimated that by 2030, the prevalence will increase 25% from current estimates. Estimates of the prevalence of symptomatic HF in the general European population are similar to that in the United States, ranging from 0.4 to 2%. The prevalence of HF rises exponentially with age, and the condition affects 4% to 8% of people older than 65 years of age.[2] Although the relative incidence of HF is lower in women than men for all age groups, women constitute at least half of the cases of HF because of their longer life expectancy, and the overall prevalence of HF is greater in women than in men 80 years of age and older. In the ARIC (Atherosclerosis Risk in Communities) study, funded by the National Institutes of Health (NIH), the age-adjusted incidence of HF was greatest in black men, followed by black women, white men, and white women.[3] The higher incidence of HF in blacks was attributed to the greater levels of atherosclerosis risk factors in this population. Similar findings were observed in the NIH-funded MESA (Multi-Ethnic Study of Atherosclerosis) study, which showed that blacks had the highest risk for development of HF, followed by Hispanic, white, and Chinese Americans. In North America and Europe, the lifetime risk of developing HF is approximately 1 in 5 for a 40-year-old. The overall prevalence of HF is thought to be increasing, in part because current therapies of cardiac disorders, such as myocardial infarction (MI), valvular heart disease, and arrhythmias, are allowing patients to survive longer. Very little is known with respect to the prevalence or risk of developing HF in emerging nations because of the lack of population-based studies in those countries.[4]

Although HF was once thought to arise primarily in the setting of a depressed left ventricular ejection fraction (LVEF), epidemiologic studies have shown that approximately one half of patients who develop HF have a normal or preserved EF (EF > 50%). Accordingly, patients with HF are now broadly categorized as having either (1) HF with a reduced or depressed EF (HFrEF) of 35% or less, also referred to as systolic failure, or (2) HF with a preserved EF (HFpEF) at 50% or greater, also referred to as diastolic failure. HF in patients with an EF in the range 35% to 50% represents a "grey zone"; such patients are likely to have at least mild systolic dysfunction.

Risk factors for the development of HF in men and women include coronary artery disease (CAD), hypertension, diabetes, obesity, and smoking. In a more recent population based case-control study, the population-attributable risk for developing HF was greatest for CAD, followed by diabetes, obesity, hypertension, and smoking. Of interest, sex-based differences in the cause of HF also were noted, with hypertension playing the greatest role in women and coronary disease in men.[5]

Heart failure may be viewed as a progressive disorder that is initiated after an index event either damages the heart muscle, with a resultant loss of functioning cardiac myocytes or, alternatively, disrupts the ability of the myocardium to generate force, thereby preventing the heart from contracting normally. This index event may have an abrupt onset, as in the case of a myocardial infarction; it may have a gradual or insidious onset, as in the case of hemodynamic pressure or volume overloading, or it may be hereditary, as in the case of many of the genetic cardiomyopathies. Regardless of the nature of the inciting event, the feature that is common to each of these index events is that they all, in some manner, produce a decline in pumping capacity of the heart. In most instances, patients will remain asymptomatic or minimally symptomatic after the initial decline in pumping capacity of the heart, or symptoms develop only after the dysfunction has been present for some time. A number of compensatory mechanisms that become activated in the setting of cardiac injury or depressed cardiac output appear to modulate LV function within a physiologic/homeostatic range, such that the patient's functional capacity is preserved or is depressed only minimally. With progression to symptomatic heart failure, however, the sustained activation of neuro-hormonal and cytokine systems leads to a series of end-organ changes within the myocardium referred to collectively as LV remodelling. Later LV remodelling is sufficient to lead to disease progression in heart failure independent of the neuro-hormonal status of the patient.

The term ischemic cardiomyopathy is used to describe the myocardial dysfunction that arises secondary to occlusive or obstructive coronary artery disease. Although ischemic cardiomyopathy was considered the second most common cause (after hypertension) of heart failure in the Framingham Study, ischemic cardiomyopathy is now recognized as the most common cause of heart failure in clinical trials of patients with low LVEF. Ischemic cardiomyopathy can be envisioned as three interrelated pathophysiologic processes: myocardial hibernation, defined as persistent contractile dysfunction at rest, caused by reduced coronary blood flow that can be partially or completely restored to normal by myocardial revascularization, myocardial stunning, wherein the viable myocardium may demonstrate prolonged but reversible post-ischemic contractile dysfunction caused by the generation of oxygen-derived free radicals on reperfusion and by a loss of sensitivity of contractile filaments to calcium; and irreversible myocyte cell death, leading to ventricular remodeling and contractile dysfunction.

The assessment of myocardial viability has become an integrated part of the diagnostic and prognostic work-up of patients with heart failure symptoms attributable to ischemic cardiomyopathy. Many imaging techniques have been proposed over the last 2 decades. These techniques rely on different characteristics of dysfunctional but viable myocardium. [108] The most tested and clinically used techniques include:

- Nuclear imaging by PET (evaluating glucose use with 18F-FDG)
- Nuclear imaging by SPECT (evaluating perfusion, cell membrane integrity, and intactness of mitochondria with 201T1-or 99mTc-labeled agents).
- Echocardiography with dobutamine (to assess contractile reserve).
- Echocardiography with intravenous contrast agents (to assess perfusion).
- MRI with dobutamine (to assess contractile reserve),
- MRI with intravenous contrast agents (to assess scar tissue), and
- CT with intravenous contrast agents (to assess scar tissue).

The initial uptake of 201TI is mainly determined by regional perfusion, whereas sustained uptake, over a longer period of time, depends on cell membrane integrity and thus myocyte viability. Although many protocols are available, the 2 protocols most frequently used are stress-redistribution-reinjection imaging and rest-redistribution imaging. The first protocol provides information on both stress-inducible ischemia and viability, whereas the latter provides information only on viability. Four markers of viability are:

1. Normal 201TI uptake (normal perfusion) at stress
2. Stress defects with redistribution (reversible defects) on 3-to 4-h delayed images.
3. Redistribution in fixed defects on images at redistribution after reinjection or on delayed rest images (frequently a threshold of a 10% increase in tracer uptake is used).
4. Tracer uptake of greater than 50% on redistribution-reinjection images or on delayed rest images.

Over the years, echocardiography has been used extensively for the assessment of myocardial viability. It has been demonstrated that severely thinned walls most likely represent scar tissue. In a study of a large registry, Schinkel et al. demonstrated that segments with LV end-diastolic wall thicknesses of less than 6 mm virtually never exhibited contractile reserve. On the other hand, the majority of segments with relatively well preserved end-diastolic wall thicknesses (≥ 6 mm) had contractile reserve.

A reasonable management strategy for patients who present with heart failure secondary to coronary artery disease (i.e., ischemic cardiomyopathy) includes coronary angiography, especially if patients have any component of angina pectoris. Viability studies may be appropriate for those patients with severe disease and adequate surgical targets. If significant viability (≥25%) is documented, the weight of currently available clinical evidence suggests that CABG may be superior to medical therapy alone in outcome measures of survival and quality of life.

AIMS & OBJECTIVES

Aim:

To study the utility of FDG PET scan viability assessment of akinetic myocardial segments on ECHO for prediction of contractility recovery at six months after CABG.

Objectives:

1. To study the value of FDG PET scan estimated viability on predicting recovery of a left ventricular global function at six months after CABG.
2. To study the association between FDG PET scan estimated viability and postoperative improvement in heart failure symptoms.
3. To study mortality from cardiac causes within 6 months of surgery.
4. To correlate FDG PET estimated scan viability with 2D Transthoracic Echocardiographic viability.

MATERIALS AND METHODS

Study site: Heart Institute, Medanta the Medicity.

Study population: After approval from the institutional ethics committee, consecutive patients found to meet inclusion criteria were enrolled.

Study design: "A prospective, observation study".

Sample size with justification: The objective is to find a cut off in myocardial viability associated with about 75% sensitivity and specificity for predicting a change in functional status. Assuming

95% confidence and ±10% precision, the sample size works out to be 75 patients with a cutoff value more than optimum.

Assuming a proportion of 80% of such patients, the total suggested sample size is 100.

Time frame: February 2016 to February 2017.

Inclusion criteria

1. Patients willing to give informed consent.
2. Patients with chronic heart failure with LVEF ≤ 35%.
3. Patients with multi-vessel coronary artery disease with suitable coronary anatomy for CABG.

Exclusion criteria

1. Patients with acute myocardial infarction within preceding 2 weeks.
2. Patients with organic valvular heart disease requiring valve replacement surgery.
3. Patients with left ventricular aneurysm requiring ventricular remodeling surgery.
4. Patients requiring emergency CABG.

Outcome parameters

1. Change in regional/ global left ventricular function.
2. Change in symptoms.
3. Mortality from cardiac causes after 6 months.
4. Re-hospitalization due to cardiac causes within 6 months.

Methodology:

The study was conducted prospectively at Medanta Hospital in the department of Cardiology. All consecutive patients who met all inclusion criteria were enrolled. Informed consent was taken. Detailed history and clinical examination recorded. Heart failure symptoms were graded according to NYHA (1-4) and CCS (0-4) classification before and 3-6 months after CABG. A single operator performed 2D Transthoracic echocardiography to assess regional and global LV function before and after 3-6 months of CABG. Regional wall motion was divided in 16 segments. Coronary angiography was used to assess coronary anatomy. Thallium 201 scan was used to study coronary perfusion. F18-Fluorodeoxyglucose (FDG) PET was used to assess myocardial viability before CABG on a SIEMENS BIOGRAPH MCT S (64)-3R scanner.

Statistical Analysis Plan:

Quantitative data is presented as means and standard deviation, while qualitative data is presented as absolute numbers and proportions. ROC analysis is used to calculate the optimum cutoff value for viability. Independent Student t-test is used for comparison of outcome parameters, and chi-square test was used for testing associations. A p-value < 0.05 is considered statistically significant. SPSS software Version 20.0 was used.

RESULTS

Table 1: Demographic Profile (N=92)

Demographic profile	Number/Mean ± SD	Percentage (%)
Age (Mean ± SD)	60.3 ± 9.0	
BMI (Mean ± SD)	**24.6 ± 3.0**	
Normal	53	57.6%
Over Weight	34	37.0%
Obese	5	5.4%
Gender		
Female	10	10.9%
Male	82	89.1%

The majority of patients in this study were males (89.1%). Regarding Body Mass Index (BMI), 57.6% of patients had a normal BMI, 37.0% were overweight, and 5.4% were obese. The mean BMI was 24.6 ± 3.0.

Table 2: Distribution of Risk factors (N=92)

Risk Factors	Number	Percentage (%)
HTN	57	62.0%

Diabetes	70	76.1%
Smoking	44	47.8%
Sedentary Life Style	30	32.6%
Dyslipidemia	35	38.0%

Hypertension was present in 62% of patients, Diabetes Mellitus in 76.1%, and 47.8% were smokers. A third of the patients had a sedentary lifestyle, and 38% were dyslipidemic.

Table 3: Distribution of Comorbidities (N=92)

Comorbidities	Number	Percentage (%)
HTN	57	62.0
DM	70	76.1
PAD	1	1.1%
CKD	7	7.6%
Carotid	2	2.2%
Hypothyroid	6	6.5%
Stroke	7	7.6%
Hyperthyroid	1	1.1%

Out of 92 patients, 76.1% were diabetic, and 62% had hypertension. CKD and stroke were each present in 7.6% of patients.

Table 4: Clinical presentation (N=92)

Clinical Presentation	**Number**	**Percentage (%)**
Breathlessness	73	79.3%
Chest Pain	32	34.8%
Palpitation	5	5.4%
Syncope	2	2.1%

79.3% of patients presented with breathlessness, while 34.8% had chest pain.

Table 5: Functional Classification (Pre-surgery)

Distribution of patients with respect to NYHA functional class:

NYHA class 1-4	**Number N=73**	**Percentage (%)**
3	24	32.8
4	49	67.1

NYHA Class: The majority of patients were in NYHA class 4 (67.1%), while 32.8% were in NYHA class 3.

Table 6: Functional Classification (Pre-surgery)

Distribution of patients with respect to CCS grade functional class:

CCS grade 0-4	Number N=32	Percentage (%)
2	20	62.5
3	12	37.5

CCS Grade: Among the 32 patients with chest pain, 20 had grade 2 symptoms and 12 had grade 3 symptoms.

Table 7: Distribution of old MI (N=92)

Previous Myocardial Infarction	Number	Percentage (%)
Yes	15	16.3%
No	77	83.7%

Old myocardial infarction was present in 16.3% of patients.

Table 8: Distribution of drug treatment at admission (N=92)

Medications	**Number**	**Percentage (%)**
Diuretic	62	67.4%
Antiplatelets	83	90.2%
Lipid Lowering Drugs	85	92.4%
Beta Blockers	47	51.1%
Ace Inhibitors/AT antagonist	41	44.6%
Nitrates	0	0.0%
Calcium Channel Blockers	5	0.0%
Amiodarone	9	9.8%

Table 9: Surgical Details (N=92)

Number of grafts	Patient	Percentage (%)
1.00	9	9.8%
2.00	21	22.8%
3.00	46	50.0%
4.00	16	17.4%

50% of patients received three grafts, 22.8% received two grafts, 17.4% received four grafts, and 9.8% received one graft.

Table 10: Regional Wall Motion Abnormality Distribution

	Pre ECHO	Post ECHO
Normal	391	870
Hypokinetic	420	93
Akinetic	661	365

- Out of 661 akinetic segments before surgery, 365 remained akinetic six months after surgery whereas normokinetic segments increased from 391 to 870. Similarly, out of 420 hypokinetic segments, only 93 remained hypokinetic six months after surgery.

Table 12: Change in preoperative Akinetic segment distribution after surgery along 3 coronary vessels

	LAD	LCX	RCA	Overall
Normal	141	32	88	261
Hypokinetic	7	4	12	23
Akinetic	219	56	90	365
Total	367	92	190	649

In this study, Out of 367 akinetic segments in the LAD territory, 219 remained akinetic, 141 became normokinetic, and 7 became hypokinetic after surgery. Out of 92 akinetic segments in the LCX territory, 56 remained akinetic, 32 became normokinetic, and 4 became hypokinetic. Out of 190 akinetic segments in the RCA territory, 90 remained akinetic, 88 became normokinetic, and 12 became hypokinetic.

Table 13: Correlation between Hibernation and Change (improvement) in Akinetic Segments

	Correlation Coefficient (r)	**p-value**
Change in Akinetic Segment	0.383	<0.0001*
Change in LVEF	0.248	0.020*

There was a statistically significant positive correlation between hibernation and improvement in akinetic segments (r=0.383, p<0.0001) and between hibernation and improvement in global LV function (r=0.248, p=0.020).

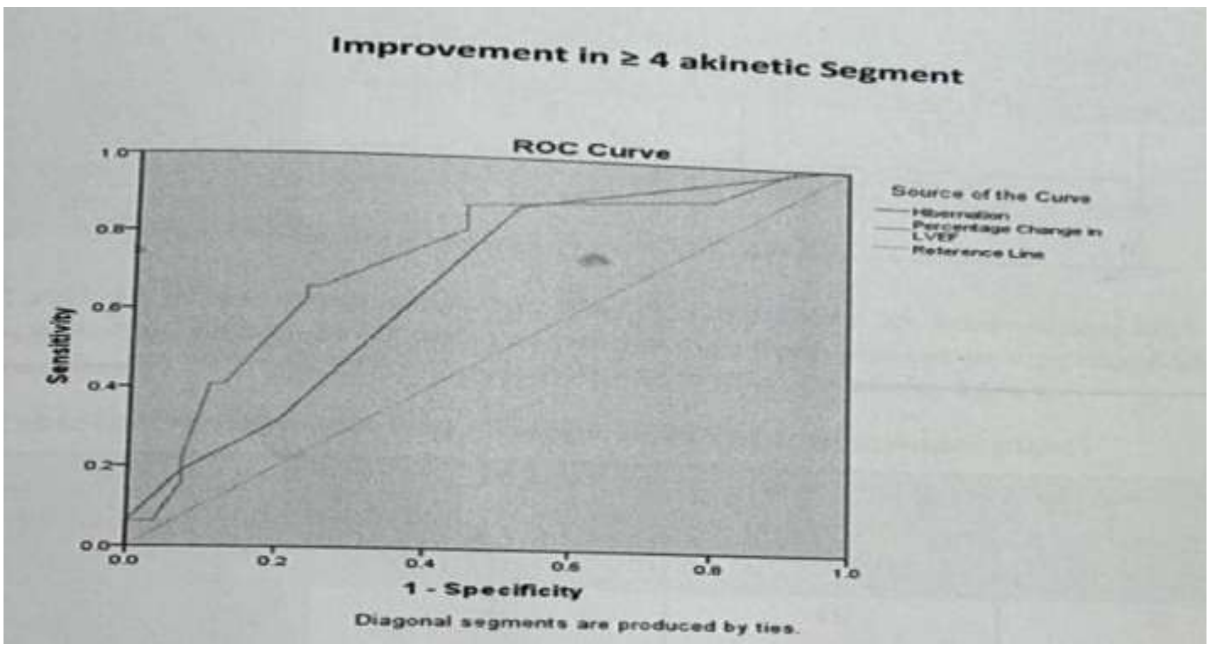

Table 14: Area under ROC Curve and their statistical significance are given below:

Area under ROC Curve

Test result variable (s)	Area	STD. Error	P value	95% CI	
				Lower bound	Upper bound
Hibernation	.681	.057	.005	.568	.793
% change in LVEF	.742	.056	.000	.633	.851

P value < 0.05 statistically significant.

Table 14 showed positive correlation between hibernation and improvement in 4 or more akinetic segments (Area Under the Curve = 0.681, p=0.005). There was also a positive correlation between improvement in global LV function and improvement in 4 or more akinetic segments (Area Under the Curve = 0.742, p=0.000).

Table 15: Cut off values with corresponding sensitivity and specificity are presented below: Cut off points were calculated as minimum of (1- sensitivity) + (1-specificity).

Parameters	**Cutt off point**	**Sensitivity (%)**	**Specificity (%)**
Improvement ≥ 4 akinetic segments			
Hibernation	**17.5**	**87.5**	**47.36**
% change in LVEF	**12.5**	**65.6**	**76.4**

A hibernation area cut-off of 17.5% had a sensitivity of 87.5% and a specificity of 47.36% for predicting improvement in 4 or more akinetic segments. This was associated with an increase in LVEF of 12.5% (sensitivity 65.6% and specificity 76.4%).

Table 16 Distribution and Change in NYHA Functional Class

Pre surgery NYHAA class	Post surgery NYHAA class			
	1	2	3	Total
3	2	19	0	21
4	3	33	9	45
Total	5	52	9	66

	Correlation coefficient (r)	P value
Change in NYHA Functional Class	0.366	0.003

NYHA Class: In this study we found that out of 66 patients who presented with breathlessness 21 were in NYHA class 3 and 45 were in class 4.six months after surgery, 52 improved to class 2 and 5 improved to class 1and 9 improved to class 3. This change was statistically significant (r=0.366, p=0.003).

Table 17: Distribution and Change in CCS Functional grade.

Pre surgery CCS grade	**Post surgery CCS grade**		
	1	**2**	**Total**
2	15	5	20(64.5)
3	4	7	11(35.5)
Total	19 (61.3%)	12 (38.7%)	31

	Correlation coefficient (r)	P value
Change in CCS Functional Class	0.O5	0.788

In this study we found that out of 31 patients who presented with chest pain 20 were in CCS grade 2 and 11 were in CCS grade 3.six months after surgery, 19 improved to grade 1 and 12 improved to grade 2. This change was statistically non- significant (r=0.05, p=0.788).

Mortality and Rehospitalization

There were 5 deaths out of 92 patients (5.4%) within 7 days of surgery. The causes were arrhythmia, cardiac arrest, and perioperative ACS. Four patients were lost to follow up.

At 6 months, 58 patients (69.87%) presented for a routine checkup, while 25 patients were re-admitted. Of those re-admitted, 9 (36.0%) had worsening heart failure, 4(16%) had ACS, 7(28%) had stable angina, and 5 (20%) had non-cardiac symptoms.

Table 18 Distribution of viability along 3 coronary vessels by Echocardiography and FDG PET scan

		PET Scan								
		LAD			LCX			RCA		
		viable	Non viable	total	Viable	Non viable	Total	viable	Non viable	total
ECHO	Viable	174	63	237 (68.7)	37	6	43 (82.7)	84	25	109 (92.4)
	Non viable	71	37	108 (31.3)	5	4	9 (17.3)	9	0	9 (7.6)
	Total	245 (71.0)	100 (29.0)	345	42 (80.8)	10 (19.2)	52	93 (78.8)	25(21.2)	118
Sensitivity		71.0			88.1			90.3		
Specificity		37.0			40.0			10.0		

Table 19: Correlation of Viability between Echocardiography and FDG PET Scan

		Pet scan		
		Overall		
		viable	Non-viable	Total
ECHO	Viable	295	94	389 (75.5%)
	Non-viable	85	41	126 (24.5%)
	total	380 (73.8%)	135 (26.2%)	515 (100%)

Sensitivity	77.6%
Specificity	30.4%

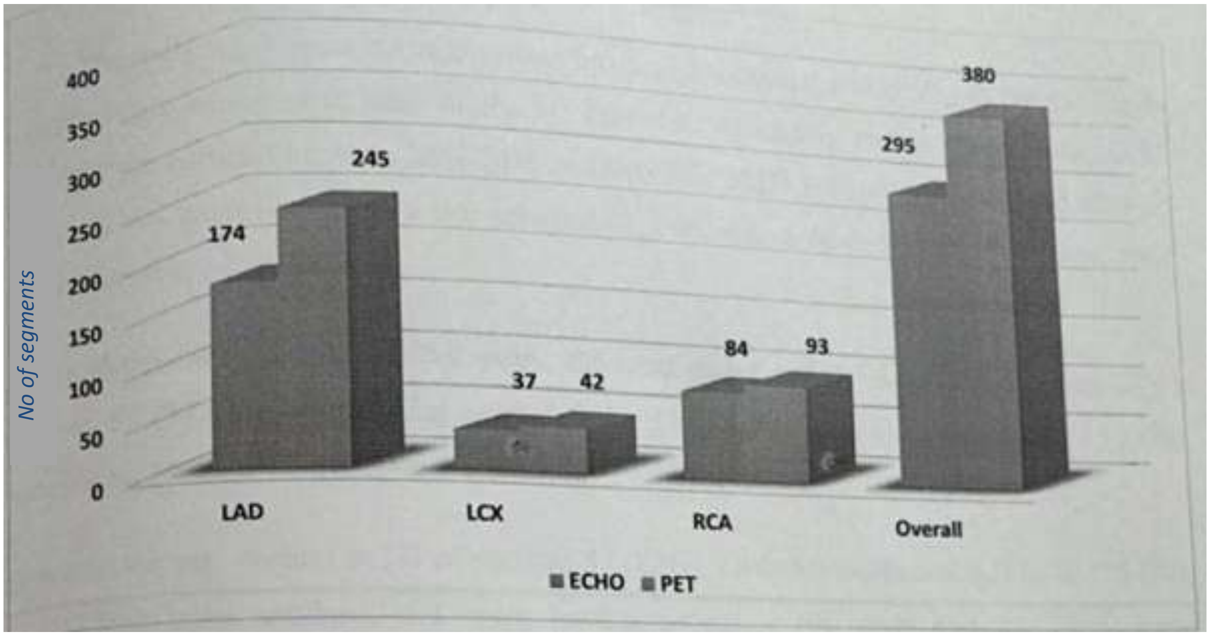

Table 19 and above figure showed sensitivity and specificity of echocardiography in predicting true viable and non-viable segments in comparison to FDG PET Scan. In this study overall out of 380 true viable segments assessed by PET Scan, echocardiography assesses 295 viable segments with sensitivity of 77.6%. Mean-while among 135 true negative segments by PET Scan echcardiography assess only 41 segments with specificity of 30.4%.

DISCUSSION

The study was conducted at the Heart Institute, Medanta the Medicity, Gurgaon. This is a tertiary care centre with state of the art facilities. According to the study protocol, 92 patients were enrolled between JAN 2016 to December 2017. Patients were included after all inclusion and exclusion criteria are satisfied. These were followed up to six months after CABG Surgery.

In our study, mean age was 60±9 years, 82 were males and 10 were females. Out of 92 patients, 53 (57.6%) were having normal BMI, 34 (37.0%) were overweight and 5 (5.4%) were obese. Mean BMI was 24.6±3.0.

Hypertension was present in 2/3 of patients 57 (62%), Diabetes mellitus (DM) in 70 (76.1%), 44 (47.8%) were smokers, 1/3 were having sedentary life style and 35 (38%) were dyslipidemic. CKD was present in 7(7.6%), 7 (7.6%) had stroke and hypothyroidism in 6 (6.5%). Old MI was present in 15 patients (16.3%).[6]

73(79.3%) presented with breathlessness while 32(34.8%) patients had chest pain. 5 patients were having palpitation and syncope history in 2 patients. Majority of patients were in NYHA class 4 (53.3%), while (25%) were in NYHA class 3. Out of 32 patients who had chest pain 20 (21.7%) were having grade 2 symptoms and 12 patients had grade 3 symptoms.[7]

In this study 46(50.0%), out of 92 received three grafts while 21(22.8%) received 2 grafts and 16(17.4%) received 4 grafts and 9 (9.8%) had 1 graft.

In STICH AND STICHES study trial by Velasquez 2016 et al , Median age was 60 (54–68) yr, Female proportion was 12%, Median body-mass index was 27 (24–30). With respect to risk factor distribution, Hyperlipidemia was present in 59%, Hypertension in 59%, Diabetes in 39%, Previous stroke in 8%, Chronic renal insufficiency in 8%. Majority of about 43% were having CCS class 3 and 52% were having NYHA class 2. Most of the results were consistent with this study, except diabetes and NYHA heart failure class. Diabetes is more prevalent in our study. One likely explanation for this finding is prevalence of diabetes in Asians is increasing more as compared to western countries.[8]

Distribution of regional wall motion abnormality in our study

Out of 661 akinetic segments on echo before surgery 365 remained akinetic 6 months after surgery whereas normokinetic segments increased from 391 to 870. Similarly out of 420 hypokinetic segments only 93 remained hypokinetic 6 months after surgery.

Change in Preoperative Akinetic segment distribution after surgery along 3 coronary vessels

	LAD	LCX	RCA	Overall
Normal	141	32	88	261
Hypokinetic	7	4	12	23
Akinetic	219	56	90	365
Total	367	92	190	649

Figure: Distribution of akinetic segments and their change after surgery along 3 coronary vessels. In this study out of 367 akinetic segments in LAD, 219 remained akinetic, 141 and 7 segments became normokinetic and hypokinetic respectively. Similarly out of 92 akinetic segments in LCX, 56 remained akinetic and 32 and 4 became normokinetic and hypokinetic. Out of 190 akinetic segments in RCA, 90 remained akinetic, 88 changed to normokinetic and 12 became hypokinetic.

correlation between Hibernation and Change (improvement) in Akinetic Segments

Parameter	Correlation Coefficient (r)	p-value
Change in Akinetic Segment	0.383	<0.0001*
Change in LVEF	0.248	0.020*

*p-value < 0.05, statistically significant

This shows that in our study, there is a positive correlation between hibernation and change in Akinetic segment (r = 0.383; p-value < 0.0001*). Similarly there is statistically significant correlation between hibernation and improvement in global LV function = 0.248; p = 0.020*.

The area under the ROC curves & their statistical significance are given below:

Test Result Variable(s)	Area	Std. Error	P value	95% CI Lower	95% CI Upper
Hibernation	0.681	0.057	0.005	0.568	0.793
Percentage Change in LVEF	0.742	0.056	0.000	0.633	0.851

p value <0.05, Statistically significant

Above Table shows positive correlation between PET scan hibernation and improvement in 4 or more akinetic segments. There is also positive correlation between improvement in global LV function and improvement in 4 or more akinetic segments.

Cut-off Point

Parameter	Cut-off (%)	Sensitivity (%)	Specificity (%)
Hibernation area (PET)	17.5	87.5	47.36
% Change in LVEF	12.5	65.6	76.4

In this study we found that at a cut off 17.5% PET scan hibernation area there is 87.5% sensitivity (specificity 47.36%) of predicting improvement in 4 or more Akinetic segments with increase in LVEF of 12.5% (sensitivity 65% and specificity 76%).

Jeroen J. Bax et al 2001-In this study, the number of viable segments per patient was directly related to the improvement in LVEF after revascularization (r=0.79, P, 0.01). Receiver operating characteristic curve analysis revealed that the cut off level of four viable segments (representing 31% of the left ventricle) yielded the highest sensitivity and specificity (86% and 92%, respectively) for predicting improvement in LVEF. Furthermore, the presence of four or more viable segments predicted improvement in heart failure symptoms after revascularization, with positive and negative predictive values of 76% and 71%, respectively. Results of our study are consistent with the above study.[9]

Distribution and change in NYHA functional class

Pre-surgery NYHA	Post-surgery NYHA 1	Post-surgery NYHA 2	Post-surgery NYHA 3	Total
3	2	19	0	21
4	3	33	9	45
Total	5 (7.6%)	52 (78.8%)	9 (13.6%)	66

	Correlation coefficient r	P value
Change in Functional NYHA Class	0.366	0.003*

In this study we found that out of 66 patients who presented with breathlessness, 21 were in NYHA class 3 and 45 were in NYHA class 4.Six months after surgery, 52 improved to class 2, 5 to class 1, and 9 improved to class 3. This change is statistically significant.

Distribution and Change in CCS functional grade

Pre-surgery CCS	Post-surgery CCS 1	Post-surgery CCS 2	Total
2	15	5	20 (64.5)
3	4	7	11 (35.5)
Total	19 (61.3)	12 (38.7)	31

	Correlation coefficient r	P value
Change in CCS Class	0.050	0.788

In this study we found that out of 31 patients who presented with chest pain 21 were in ccs grade 2 and 11 were in ccs grade 3. Six months after surgery 19 improved to grade 1 and 12 grade 2. This functional class change is statistically non-significant (r= 0.05), p=0.788 .

Di Carli and colleagues 1995: studied 36 patients with LVEF of 28% by PET imaging. A mismatch of more than 18% was associated with a sensitivity of 76% and a specificity of 78% for predicting a change in functional status after revascularization. Our finding of 17.5% cut off value of hibernation area is consistent with above study in predicting change in global lv function and change in functional class

Mortality:

In our study there were 5 deaths out of 92 (5.4%). Two patients died due to arrhythmia (ventricular tachycardia), two due to cardiac arrest and one with perioperative ACS. All deaths occurred within 7 days of surgery.

LIMITATIONS OF STUDY:

1. The study was a non-randomised, prospective observational study, which has inherent limitations.
2. Follow-up angiography was not performed, so early graft issues could have prevented some viable segments from recovering.

CONLCUSION

Patients with large areas of viable myocardium (>18%) on PET scan and an improvement in >4 akinetic segments show the highest sensitivity and specificity for predicting an improvement in LVEF after CABG.

REFERENCES

1. Brouwers FP, Hillege HL, van Gilst WH, et al: Comparing new onset heart failure with reduced ejection fraction and new onset heart failure with preserved ejection fraction: An epidemiologic perspective. Curr Heart Fail Rep 2012; 9:363.
2. Go AS, Mozaffarian D. Roger VL, et al: Heart disease and stroke statistics 2013 update: A report from the American Heart Association. Circulation 2013: 127:e6.
3. McMurray JJ, Adamopoulos S, Anker SD, et al: ESC guidelines for the diagnosis and treatment of acute and chronic heart failure 2012: The Task Force for the Diagnosis and Treatment of Acute and Chronic Heart Failure 2012 of the European Society of Cardiology. Developed in collaboration with the Heart Failure Association (HFA) of the ESC. Eur Heart J 2012; 33:1787.

4. Loehr LR, Rosamond WD, Chang PP, et al: Heart failure incidence and survival (from the Atherosclerosis Risk in Communities study). Am J Cardiol.2008; 101:1016.

5. Mendez GF, Cowie MR: The epidemiological features of heart failure in developing countries: A review of the literature. Int J Cardiol.2001; 80:213.

6. Dunlay SM, Weston SA, Jacobsen SJ, et al: Risk factors for heart failure: A population-based case-control study. Am J Med.2009; 122:1023.

7. Mann DL, Bristow MR: Mechanisms and models in heart failure: The biomechanical model and beyond. Circulation.2005; 111:2837.

8. Floras JS: Sympathetic nervous system activation in human heart failure: Clinical implications of an updated model. J Am Coll Cardiol.2009; 54:375.

9. Kumar R, Baker KM, Pan J: Activation of the renin-angiotensin system in heart failure. In Mann DL (ed.): Heart Failure: A Companion to Braunwald's Heart Disease. 2nd ed. Philadelphia, Saunders, 2010, pp 134-151.

10. Mann DL: Left ventricular size and shape: Determinants of mechanical signal transduction pathways. Heart Fail Rev 2005; 10:95.