Original Article

Technetium-99m Methoxyisobutyl Isonitrile Stress MPI in Suspected Coronary Artery Disease Patients: A Prospective Study to Evaluate Clinical Significance of Adenosine-induced ECG Changes

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Abstract

Purpose: The purpose of this study is to evaluate the adenosine pharmacological stress-induced electrocardiogram (ECG) changes and their association with stress-induced ischemic defects on myocardial perfusion scintigraphy (MPS) in the evaluation of coronary artery disease (CAD) and to evaluate event-free survival among patients with positive and negative ECG/MPS image findings. **Methods:** A total of 100 patients were examined using stress MPS from March 2020 to August 2021. Stress-induced ECG changes during adenosine infusion were evaluated. The summed stress score (SSS) was evaluated to identify ischemic defects in myocardium. Association of stress ECG changes and scintigraphic results was evaluated. **Results:** Out of 100 patients, stress ECG changes during adenosine infusion were seen among 34 patients, whereas 66 patients had normal ECG findings. Positive stress MPS findings with SSS >3 were seen in 22 patients, whereas 78 patients had SSS ≤3. There was no agreement between stress ECG changes and MPS findings with Cohen's kappa coefficient (κ) = −0.023, whereas there was mild agreement between stress ECG changes and SSS >7 with κ = 0.105. Median follow-up of 11 months showed more events among patients with positive ECG changes than negative ECG changes. **Conclusion:** Adenosine, pharmacological stress is safe to use, but few patients might experience some minor and transient symptoms. Adenosine may induce ECG changes in patients with or without positive MPS findings. Patients with concordant positive findings need aggressive cardiac care, whereas patients with small or no defects on MPS need close monitoring.

Keywords: *Adenosine, coronary artery disease, myocardial perfusion scintigraphy, stress ECG, Technetium-99m methoxyisobutyl isonitrile*

Introduction

Cardiovascular disease (CVD) became the leading cause of mortality at the turn of the century. Globally, CVD has led to 17.9 million deaths in 2019.[1] According to the Registrar General of India, 23% of total and 32% of adult deaths were due to coronary heart disease (CHD) in 2010–13. In contrast to developed countries, where mortality from CHD is decreasing, it is increasing in developing countries which was attributed to industrialization, urbanization, and related lifestyle changes.[2]

The ischemic cascade of myocardium is initiated by imbalance in oxygen supply and demand, followed by wall motion abnormalities and electrocardiography (ECG) changes. Evaluation of coronary artery disease (CAD) should be done with basic first-line testing including biochemical tests, ECG, and rest echocardiography to rule out possible other disorders such as hypertrophy or valvular disorders and also stratify patients in clinical pretest probability. Noninvasive imaging modalities such as single-photon emission computed tomography (SPECT) and myocardial perfusion scintigraphy (MPS) is performed in subsequent risk-stratified patients before invasive coronary angiography.

ECG changes during treadmill test and dobutamine stress in the evaluation of CAD were established, as stress causes an increased metabolic oxygen demand and thus ST/T wave abnormality. These changes are considered significant in patients who harbor hemodynamically significant CAD, adding to the interpretation of MPS findings.[3] Similarly, during adenosine

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pharmacological stress, ECG changes may occur, but their association with CAD is not considered significant since adenosine causes direct vasodilatation without increase in metabolic oxygen demand. Few studies have documented positive association between ECG and MPS findings and stated that these ECG changes have some significance and they are not just an observation of random event.

Hence, the aim of the current study is to evaluate the adenosine pharmacological stress-induced ECG changes and their association with stress-induced ischemic defects on MPS in the evaluation of CAD.

Methods

It is a prospective analytical study conducted in suspected CAD patients. A total of 100 patients from March 2020 to August 2021 were recruited for the study.

Study population

Patients above 18 years of age with clinical suspicion of CAD with typical chest pain, atypical chest pain with normal/abnormal ECG, echo, and willing to participate in the study were included in the study. Patients with contraindication for stress/adenosine stress as per ASNC guidelines were excluded from the study.[4] Pregnant and lactating women were also excluded from the study.

All the patients were explained about the procedure, purpose, benefits, and side effects of the study. The scintigraphic study was performed with 6 h of fasting. Patients were advised to remove any metal objects and articles to avoid any artifacts.

Study protocol

Same day stress and rest protocol was adopted for the study. Continuous ECG monitoring with 12 leads was performed, and vital signs (heart rate [HR] and blood pressure [BP]) were recorded at regular intervals throughout the stress and recovery phase. Continuous infusion of adenosine at the rate of 140 µg/kg/min was given for the patient over a period of 6 min. Early termination of adenosine infusion was done in case of (1) severe hypotension, (2) development of symptomatic/persistent II or III degree atrioventricular block, (3) significant arrhythmia/wheeze, (4) sustained supraventricular or ventricular tachycardia, (5) moderate-to-severe angina pectoris, (6) signs of poor perfusion (pallor and cyanosis), and (7) patient request to terminate the test.

ECG changes were considered positive if horizontal or downsloping ST depression of >1.0 mm, 80 mm after J point, upsloping ST depression >1.5 mm, 80 mm after J point, ST segment elevation more than 1.0 mm, and 80 mm after J point.[5] HR, systolic BP (SBP), and diastolic BP (DBP) before and after adenosine infusion were calculated.

Radiopharmaceutical preparation

MPS was performed using technetium-99m (Tc-99m) Methoxyisobutyl isonitrile (MIBI). Tc-99m MIBI was prepared by mixing freshly eluted sodium pertechnetate from molybdenum–technetium generator into cold kit and boiling them for 10 min at 95°C –100°C. It was allowed to cool to room temperature. Then, one-fourth of the total dose (8–12 mCi) of Tc-99m MIBI was injected intravenously 3 min after adenosine for stress imaging, and three-fourth of the total dose (16–24 mCi) was given for rest imaging, with a gap of 3 h for stress-rest imaging. In patients, when stress MPS shows normal findings, rest MPS study was avoided.

Image acquisition

Both stress and rest SPECT imaging was done using a dual-head SPECT Gamma camera Symbia E (Siemens) with 90° configuration using a low-energy high-resolution collimator. Patients were positioned supine, with both arms raised; a step-and-shoot acquisition in counterclockwise rotation in a noncircular orbit with 32 stops separated by 6° was used. Rotation was from right antero-oblique position to left postero-oblique position. SPECT used an acquisition protocol with a Matrix of 64 × 64. ECG gating for the acquisition of cardiac function was used.

Image processing and analysis

Data was reconstructed using filtered back projection with Butterworth filter with cutoff frequency of 0.5 cycles/cm. 4DM SPECT Software was used for the quantification of data and polar map regeneration. Semi-quantitative visual interpretation of SPECT-MPS was performed using short-axis, horizontal long–axis, and vertical long-axis slices, dividing the heart into 17-segment model. Computer-generated summed stress score (SSS) and summed difference score (SDS) were used for quantitative analysis. Images were evaluated for areas of decreased radiopharmaceutical concentration in the stress and rest images and changes in regional count density in gated data were recorded.

Each segment was scored based on the tracer uptake as 0 – normal; (1) mildly reduced; (2) moderately reduced; (3) severely reduced; and (4) absent tracer uptake in rest and stress images. A SSS was obtained by adding the scores of the 17 segments of the stress images. Perfusion defect was classified according to SSS as following: SSS <4: no significant perfusion defect. SSS = 4–7, low risk; SSS = 8–12, intermediate risk; and SSS >12, high risk. Ischemic tissue showing reversibility was assessed by SDS as follows: mild ischemia = 0–2; moderate ischemia = 3–7; and severe ischemia >7.

Follow-up

In the median follow-up duration of 11 months (range: 6–20 months), the patients were categorized to have no

event (asymptomatic), soft event (chest pain resolved with nitrate), hard event (hospitalization/intervention), and death due to a cardiac cause.

Statistical analysis

All data obtained, were recorded on a predesigned pro forma and managed using Microsoft Excel worksheet. All the entries were double checked for any possible error. Statistical analysis was done by entering all the variables into the Statistical Package of Social Sciences (SPSS) software versions 21.0 M/s International Business Machines (IBM, Armonk, New York, United States of America.) for data analysis. Normality of distribution was assessed using the Shapiro–Wilk test. Continuous variables such as age, baseline and peak HR, and change in HR were expressed as mean and standard deviation, baseline and peak SBP, DBP, and change in SBP and DBP were expressed as median with interquartile range.

Frequency and percentages were computed for gender, risk factors (hypertension, diabetes, smoking, and dyslipidemia), and myocardial perfusion imaging (MPI) categories. Cohen's Kappa coefficient (κ) was used to measure the association between ECG changes and MPS results. Event free survival was assessed using the Kaplan–Meyer survival curve and difference between the groups were analysed using log rank test. $P < 0.05$ was considered statistically significant.

Results

Demographic profile

A total of 100 patients were included in the study. Sixty-one were female and 39 were male, with a mean age of 57.3 ± 10.9 years ranging between 31 and 84 years. 5/100 patients were clinically asymptomatic, whereas 68/95 (71.5%) symptomatic patients had exertional dyspnea. Sixty-eight (68%) patients were hypertensive and 50 (50%) patients had type 2 diabetes mellitus [Table 1].

Adenosine stress-induced clinical profile

The mean HR at rest was 81 ± 15 bpm which increased significantly over 41.5% to 114 ± 18 bpm at peak stress ($P < 0.001$). The median DBP at rest of 80 mmHg with interquartile range of 20 showed significant fall ($P < 0.005$) [Table 2].

62/100 patients (62%) experienced one or the other symptoms under adenosine infusion, whereas 38 patients (38%) remained asymptomatic. Most common symptom among the patients was chest pain (43), followed by flushing/sweating (26) [Table 3]. None of the patients had severe symptom warranting aminophylline administration. Among various risk factors, obese patients (31/39, 79%) experienced adenosine stress-induced symptoms frequently ($P < 0.05$). The mean age of patients who experienced adenosine stress-induced symptoms was 54.67 ± 10.6 years, and the mean age of asymptomatic patients was 58.6 ± 10.6 years with significant difference ($P = 0.038$).

34/100 patients had stress-induced ST segment changes, of whom 8 were male and 26 were female patients ($P = 0.022$). No significant association was seen between stress-induced ST segment changes and risk factors.

Association of clinical profile with stress ECG changes

There was no agreement between stress ECG changes and adenosine-induced symptoms with $\kappa = -0.067$ [Table 4].

Table 1: Demographic profile of the patients

	n	
Gender		
Male	39	
Female	61	
Age, mean±SD (range)	57.32±10.9 (31–84)	
	Yes	**No**
Symptoms at presentation	95	5
Diabetes mellitus	50	50
Hypertension	68	32

SD: Standard deviation

Table 2: Changes in vitals between rest and peak adenosine stress

Parameters	Rest	Peak	*P*
HR (bpm)	81±15	114±18	<0.001
SBP (mm of Hg)	140 (130–150)	140 (130–150)	0.904
DBP (mm of Hg)	80 (70–90)	80 (70–80)	0.012

HR: Heart rate, SBP: Systolic blood pressure, DBP: Diastolic blood pressure

Table 3: Symptom evaluation at peak adenosine stress

Symptom	Male (*n*=39), *n* (%)	Female (*n*=61), *n* (%)	Total (*n*=100), *n* (%)	*P*
Flushing and sweating	11 (28.2)	15 (24.5)	26 (26)	0.687
SOB	4 (10.2)	13 (21.3)	17 (17)	0.257
Chest pain	14 (35.8)	29 (47.5)	43 (43)	0.251
Headache	5 (12.8)	8 (13.1)	13 (13)	0.965
Palpitation	4 (10.2)	6 (9.8)	10 (10)	0.945
Throat and neck discomfort	3 (7.69)	4 (6.5)	7 (7)	0.828
Total	24 (61.5)	38 (62.2)	62 (62)	0.939

SOB: Shortness of breath

Table 4: Association between postadenosine symptoms and electrocardiogram changes at peak stress

ECG changes	Symptoms		Total
	Present	Absent	
Present	13	21	34
Absent	30	36	66
Total	43	57	100

ECG: Electrocardiogram

Similarly, there was no agreement between stress ECG changes and chest pain during adenosine infusion with κ = −0.05.

Scintigraphic data

On evaluation of scintigraphic data, 22/100 patients had perfusion defect with SSS >3, whereas rest 78/100 patients had no significant perfusion defect (62/78 with SSS = 0, 16/78 with SSS 1–3). 14/39 (35.8%) male patients and 8/61 (13.1%) female patients had perfusion defect with statistical significant difference between the two genders (P = 0.007) [Table 5].

On evaluation of SDS data, 16/100 patients had SDS ≥3, whereas 84/100 patients had SDS <3. Of those 16 patients with significant ischemic burden, 3/16 patients had SDS ≥7, whereas the remaining 13/16 patients had SDS <7.

Image and ECG data

Patients were divided into four groups based on their poststress ECG and image findings as follows:

1. Concordant positive – Stress ECG +ve/image +ve
2. Concordant negative – Stress ECG −ve/image −ve
3. Discordant image +ve – Stress ECG −ve/image +ve
4. Discordant ECG +ve – Stress ECG +ve/image −ve.

Based on the above division, most of the patients were concordant negative (51/100, 21 males and 30 females). Seven were concordant positive (4 males and 3 females), 15 were discordant image positive (10 males and 5 females), and 27 were discordant ECG positive (4 males and 23 females) [Table 6].

Further evaluation showed there is a significant difference between the two genders among four groups (P > 0.05). Discordant ECG-positive findings were seen more among female (23, 37.7%), and discordant image-positive findings were seen more among male patients (10, 25.64%). There is no agreement between image findings and stress ECG changes with κ: −0.023 with 58% agreement (κ < 0 considered as no agreement) [Table 6].

However, when the cutoff for positive image findings was kept at SSS >7 instead of 3, there was slight agreement between image findings and stress ECG changes with κ: 0.105 with 67% agreement (SSS >7) [Table 6].

Similar evaluation with SDS ≥3 as image-positive finding showed slight agreement with κ = 0.028 with 62% agreement.

Follow-up

A median follow-up of 11 months ranging from 6 to 20 months showed more events among positive ECG and positive image group than negative ECG and negative image groups, respectively [Figures 1 and 2].

Subsequently, event-free survival analysis of four groups showed a significant decrease in trend for survival function (P = 0.007) with gradually decreased from concordant negative to discordant ECG +ve to discordant image +ve to concordant-positive group [Figure 3].

Discussion

Stress MPS works under the principle of coronary flow reserve. Physical stress by treadmill testing is widely accepted modality of stress performed in the evaluation

Table 5: Perfusion defect among male and female patients

	Male	Female	Total
Perfusion defect (+)	14	8	22
Perfusion defect (−)	25	53	78
Total	39	61	100

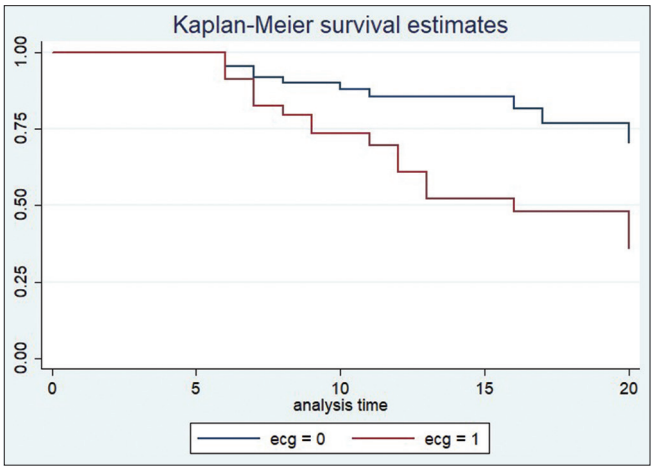

Figure 1: Kaplan–Meier event-free survival curve between electrocardiogram (ECG) +ve and ECG −ve group (0 = −ve, 1 = +ve). Log-rank *p* test showed statistical significant difference with *P* = 0.005

Table 6: Distribution of patients among concordant and discordant electrocardiogram/myocardial perfusion scintigraphy Image findings with positive myocardial perfusion scintigraphy image finding cut off at summed stress score >3 and summed stress score >7

Groups	SSS >3		SSS >7	
	Male (39), *n* (%)	Female (61), *n* (%)	Male (39), *n* (%)	Female (61), *n* (%)
ECG (+)/image (+)	4 (10.25)	3 (4.91)	4 (10.25)	1 (1.63)
ECG (−)/image (−)	21 (53.84)	30 (49.18)	28 (71.8)	34 (55.74)
ECG (−)/image (+)	10 (25.64)	5 (8.19)	3 (7.7)	1 (1.63)
ECG (+)/image (−)	4 (10.25)	23 (37.7)	4 (10.25)	25 (41)

SSS: Summed stress score, ECG: Electrocardiogram

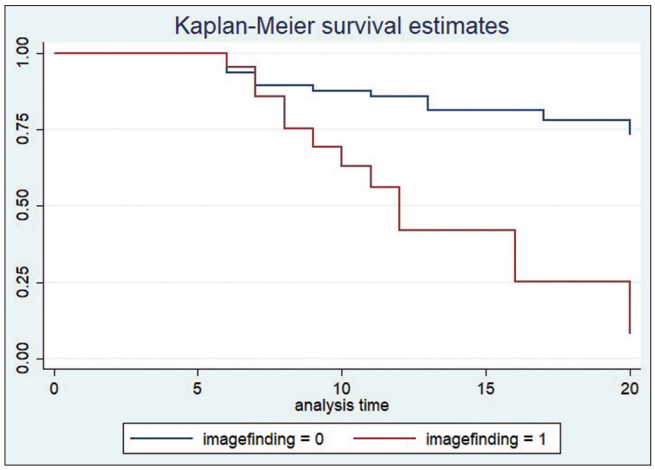

Figure 2: Kaplan–Meier event-free survival curve between myocardial perfusion scintigraphy (MPS) image positive and MPS image negative (0 = negative, 1 = positive). Log-rank *P* test showed statistical significant difference with *P* < 0.05

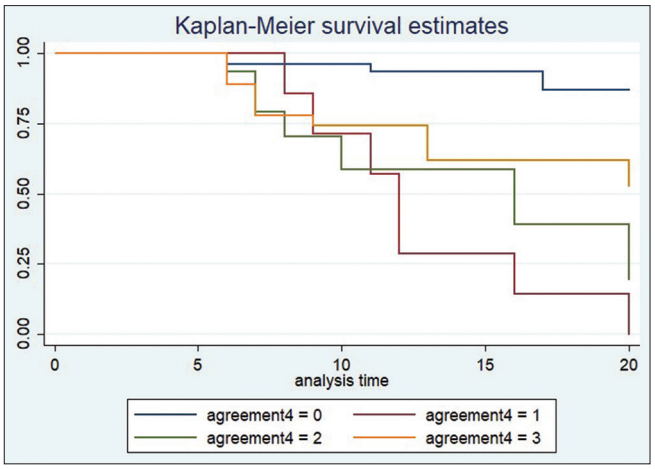

Figure 3: Kaplan–Meier event-free survival curve between groups with concordant and discordant image/ electrocardiogram (ECG) findings. 0 = concordant negative, 1 = concordant positive, 2 = discordant image positive, 3 = discordant ECG positive. Log-rank *P* test showed statistical significant difference with *P* < 0.05

of CAD. Alternate stress with pharmacological agents includes vasodilators such as dipyridamole, adenosine, and inotropic–chronotropic drug agent like dobutamine. Lee *et al*. (1994) from Philadelphia studied biokinetics of Thallium-201 in 15 normal subjects, who underwent the study four times each time under, exercise, dobutamine, dipyridamole, and adenosine. Despite patients experiencing short-lived adverse events more frequently with adenosine (83%) than dobutamine (73%), more subjects preferred adenosine (60%) than dobutamine (13%) and more subjects reported dobutamine to be the most intolerable (67%) than adenosine (13%).[6] However, the effects of adenosine-induced ECG changes and its significance in CAD evaluation are debatable.

In our study, a total of 100 patients underwent adenosine stress MPI with suspected CAD. The mean age of the patients was 57.32 ± 10.9 years, with male-to-female ratio of 1:1.56. Vasodilator stress under adenosine infusion acts through the activation of A2A receptors present in coronary vessels.[4] In our current study, postadenosine infusion showed statistically significant rise in HR from mean resting HR of 81 ± 15 bpm to peak HR of 114 ± 18 bpm with $P < 0.001$. There was a statistically significant fall in DBP between rest (mean: 81 ± 9 mmHg) and peak adenosine stress (mean: 78 ± 9 mmHg), but no significant difference in SBP between rest (139 ± 17 mmHg) and stress (139 ± 15 mmHg). Similar results were documented in Adenoscan multicenter trial registry, with significant rise in HR (72.6 ± 13.7–86.3 ± 27.3) and fall in DBP (79.9 11.9–71.9 ± 14.2 mmHg).[7] Rongen *et al*. from Canada evaluated the effect of adenosine on HR variability in humans through two series of experiments with intravenous and intrabrachial adenosine infusion and concluded that adenosine-induced decreased parasympathetic and increased cardiac sympathetic tone to be the reason for rise in HR.[8]

In our study, 62 out of 100 patients (62%) experienced one or the other symptom postadenosine infusion, out of which 24 were male and 38 were female. In Adenoscan multicenter trial registry, 81.1% patients experienced symptoms during adenosine infusion.[7] The majority of the patients had chest pain (43%) as the most common symptom postadenosine infusion, followed by flushing and sweating (26%). Adenoscan multicenter trial registry observed flushing to be the common adverse event (36.5%), followed by shortness of breath (SOB) (35.2%) with chest pain being the third common adverse event (34.6%). Similarly, Yıldırım Poyraz *et al*. documented that flushing (31.3%) as the most common symptom experienced postadenosine, followed by dyspnea (27.9%) and chest pain (23.1%).[9]

We observed adenosine-induced clinical symptoms more among obese patients and younger age group with significant difference ($P < 0.05$). There was no significant difference in postadenosine clinical symptoms between the two genders. Adenoscan multicenter trial registry documented similar findings with adenosine-induced clinical symptoms more among overweight, female gender, and younger age group.[7]

On evaluation of scintigraphic image findings, 22 patients had SSS >3, whereas 76 had SSS ≤3. 35.8% male patients had abnormal MPS findings, whereas 13.1% of female patients had perfusion defect, suggestive of CAD. The results of our study showing male preponderance of CAD were in accordance with other studies published by Battula *et al*. from SVIMS, Tirupati, and Sriharibabu *et al*. from GSL Hospital, Andhra Pradesh.[10,11] The cause of this variation in the occurrence of CAD among males is due to high-risk factor exposure and low physiological and hormonal protection as compared to female population.

Post adenosine stress ECG changes and MPS image findings showed 58% concordance, and 42% discordance [Concordance includes ECG +ve Image +ve [Figure 4] and ECG -ve, Image -ve [Figure 5]; Discordance includes ECG +ve, Image -ve [Figure 6] and ECG -ve and Image -ve]. Positive ECg and negative Image findings were seen in 27 patients (4 were male and 23 were female). Taywade *et al.* retrospectively evaluated 117 patients with suspected CAD for the prevalence of adenosine ECG changes and its association with myocardial perfusion image findings. 9/117 patients had discordant image findings and 6 of those were female [Representative patient shown in Figure 6]. This discordant-positive ECG changes could be due to digoxin-like vasoconstrictor effect of estrogen during stress.[12]

There was no agreement between postadenosine ECG changes and myocardial perfusion image findings with Cohen's kappa coefficient: −0.023. However, when cutoff for positive image findings was kept at SSS >7 instead of 3, there was a slight agreement with Cohen's kappa coefficient: 0.105. Similarly, there was a slight agreement with Cohen's kappa coefficient: 0.028 between post adenosine ECG changes and ischemic burden on myocardial perfusion image based on SDS. Thus, ECG changes show a good association with image findings with larger perfusion defects and significant reversibility. Taywade *et al*. showed moderate strength of association between adenosine ECG changes and myocardial perfusion image findings.[12]

In our current study, cardiac events were seen more among patients with positive ECG changes than with normal ECG with worsening event-free survival among positive ECG group ($P < 0.05$) [Figure 1]. Similarly, Azemi *et al*. evaluated ECG changes during vasodilator SPECT myocardial perfusion imaging in diagnostic cohort of 622 patients along with angiographic data and prognostic cohort of 3566 patients with a mean follow-up of 2.4 ± 1.5 years and showed that the likelihood of significant CAD increased in those with positive ECG changes and abnormal MPS in comparison to angiography (Gold standard). Cardiac events were observed more among patients with ECG changes

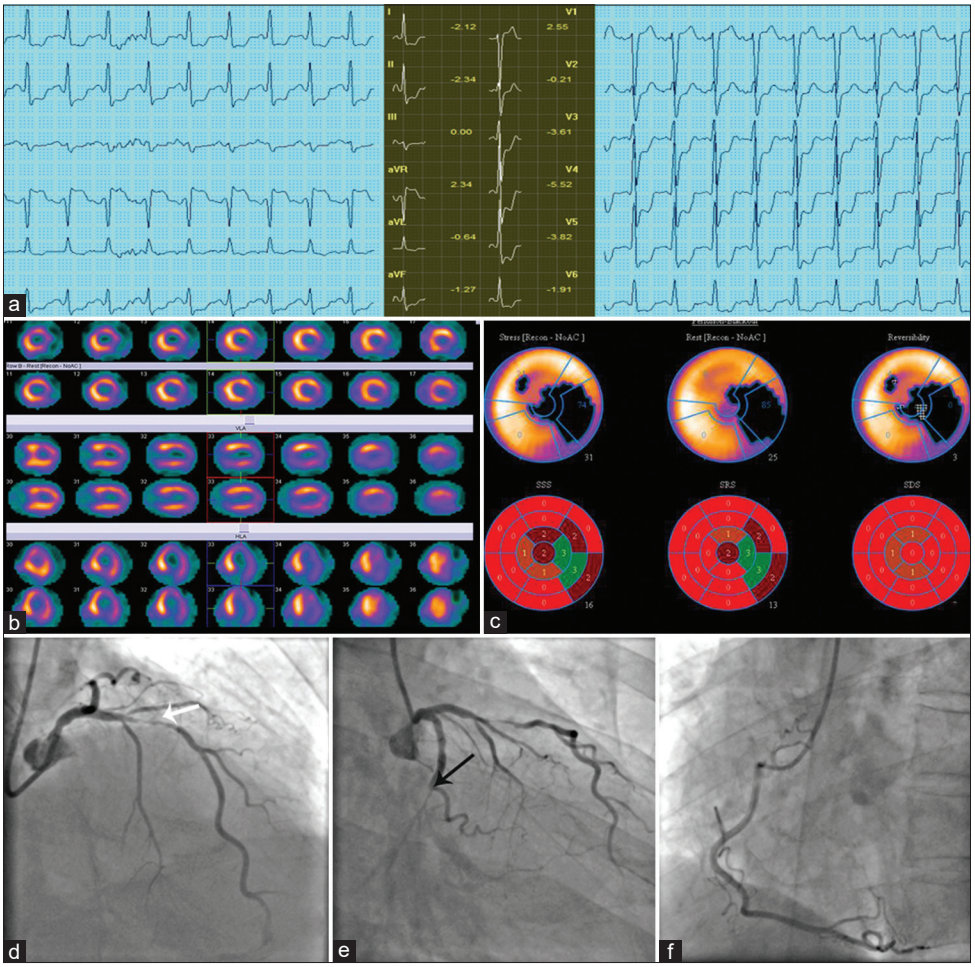

Figure 4: Representative image of a patient showing positive electrocardiogram (ECG) changes with 2.5 mm ST depression in leads I and II, V3–5 (a). Fixed perfusion defect with mild improvement at rest images involving apex and lateral wall of left ventricular myocardium in short-axis, vertical long-axis, and horizontal long-axis images (b) with summed stress score = 16 and summed difference score = 3 (c). Patient subsequently underwent angiogram which showed significant stenosis of mid left anterior descending (LAD) artery (white arrow) (d), significant stenosis of distal left circumflex (LCx) artery (Black arrow) (e), and normal right coronary artery (RCA) (f)

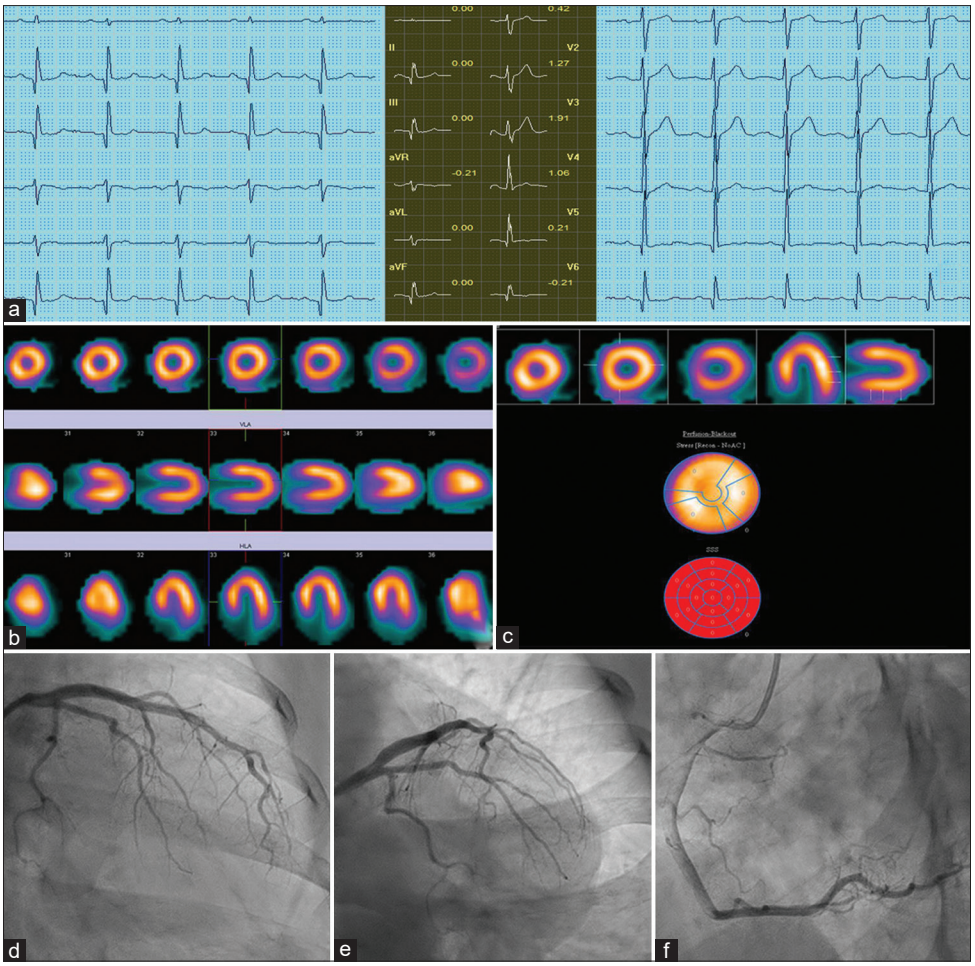

Figure 5: Representative of a patient showing no significant electrocardiogram (ECG) changes during peak adenosine stress (a), Normal perfusion of left ventricular myocardium during stress study (b) with summed stress score = 0 (c). Patient underwent coronary angiography showing normal coronaries (d-f)

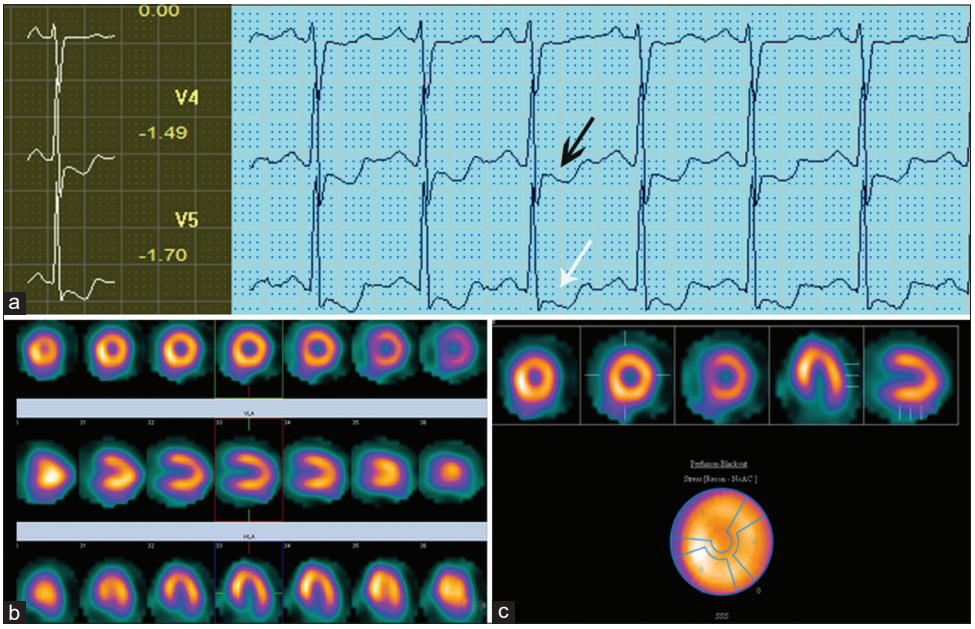

Figure 6: Representative image of a patient with positive electrocardiogram (ECG) change with 1.5 mm ST segment depression in lead V4 (black arrow) and V5 (White arrow) leads during peak adenosine stress (a), Normal perfusion of left ventricular myocardium during stress (b), with summed stress score = 0 (c)

than without changes (10% vs. 7%). However, incidence of cardiac events in any SSS category is not significantly different between those with or without ECG changes.[13]

In our study, there is significant statistical difference in event-free survival among the four groups with concordant/discordant findings with $P < 0.05$ with worsening trend from concordant negative to discordant ECG positive to discordant image positive to concordant-positive groups. Thus, ECG-positive changes show worsening event-free survival among image negative and positive groups.

Conclusion

Adenosine is a tolerable pharmacological stress agent with no severe adverse events. However, few patients experienced minor clinical symptoms such as chest pain, breathlessness, and flushing/sweating with more common occurrence in obese and younger age group. Concordant-positive ECG and image findings are seen frequently with larger perfusion defects. Discordant-positive ECG changes (ECG+/Image−) are seen more among female gender probably due to hormone fluctuation. Patients with concordant positive findings in ECG/image findings need aggressive management, whereas discordant-positive ECG/negative image findings needs close follow-up with good prognosis.

Limitations

- Small sample size
- Angiographic correlation of scintigraphic-positive patients is not performed for all the patients.

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Conflicts of interest

There are no conflicts of interest.

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