Al-Azhar International Medical Journal

Volume 5 | Issue 1 Article 47

2024
Section: Cardiovascular

Changes in Myocardial Native T1 &T2 after Dobutamine stress (A Noncontrast CMR Study)

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How to Cite This Article

khalaf, Hussein Nasser Mohammed Abdallah; Sharaf, Abd El Rahman Abd El Gawad; Mahrous, Mary Rabea; and Abdelfattah, Ashraf Alamir (2024) "Changes in Myocardial Native T1 &T2 after Dobutamine stress (A Noncontrast CMR Study)," *Al-Azhar International Medical Journal*: Vol. 5: Iss. 1, Article 47.
DOI: https://doi.org/10.58675/2682-339X.2189

ORIGINAL ARTICLE

Changes in Myocardial Native T1 and T2 after Dobutamine Stress: A Non-contrast Cardiac Magnetic Resonance Study

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Abstract

Background: **Cardiac MRI mapping is a novel technique that allows for the measurement of myocardial T1 and T2 values, which reflect its intrinsic tissue properties.**

Patients and methods: **In 10 healthy individuals and 15 patients with coronary artery disease, T1 and T2 mapping values were measured on 16 segments of each case at rest and stress. Also, T1 and T2 reactivity, such as variations in T1 and T2 at rest and poststress values, were calculated.**

Results: **After stress, native T1 was elevated in healthy individuals and ischemic patients. A statistically significant difference was found ($P = 0.005$) in T1 reactivity, with the median being 3.847 % in the control and 3.539 % in the cases. In recovery, the T1 values decreased in both groups, yet the median T1 recovery in control was 1002.6 ms compared with 1016.7 ms in cases, which was statistically significant ($P = 0.001$). The best T1 reactivity cutoff value for predicting flow-limiting stenosis was 1.17 %; it had a specificity of 94.37 %, sensitivity of 66.25 %, negative predictive value of 84.80 %, positive predictive value of 85.50 %, and an excellent area under the curve of 0.763 with statistical significance ($P = 0.001$).**

Data conclusion: **Incorporating T1 mapping into the dobutamine stress cardiac magnetic resonance study can increase test sensitivity and specificity to detect ischemia using T1 reactivity and T1 recovery values.**

Keywords: Assessment of ischemia, Dobutamine stress, Noncontrast cardiac magnetic resonance, Significant coronary artery disease, T1 map, T1 stress

1. Introduction

Ischemic heart disease (IHD) is one of the major causes of mortality in the world. Interestingly, IHD mortality rates have markedly decreased in developed countries compared with the early 1980s due to preventive measures. So, early identification of the early stages of the disease is an important challenge for decreasing mortality and morbidity.[1] Over the last 10 years, technological advances have added new noninvasive imaging techniques to the armamentarium of diagnostic and risk stratification techniques for IHD.[2] One of these new tools is cardiovascular magnetic resonance (CMR) tissue mapping. Each tissue, such as the myocardium, has a specific range of typical T1 values according to its normal composition, with a significant deviation denoting pathology. Native (noncontrast) T1 and T2 values are elevated with increased myocardial water content in cases of acute events such as infarction, acute myocarditis, and Takotsubo cardiomyopathy. By measuring T1 and T2 mapping values for each segment, we provide a quantitative value that reflects the intracellular and extracellular myocardial environments.[3] The idea behind using CMR native T1 mapping in stress tests is based on the fact that it can provide information about myocardial blood volume changes without using contrast.[4] So, in this

Accepted 29 August 2023.
Available online 23 September 2024

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https://doi.org/10.58675/2682-339X.2189

study, we intended to add T1 and T2 mapping to the dobutamine stress test in the coronary artery disease (CAD) population and examine its value.

Therefore, using ICA (Invasive Coronary Angiography) as the gold standard, we set out to compare the changes in myocardial native T1 and T2 values at rest and following dobutamine stress as a potential objective, quantitative imaging marker of ischemia to SPECT (Single Photon Emission Computed Tomography).

2. Patients and methods

We enrolled 25 patients in the study, yet two patients could not afford to complete the recovery phase due to agonizing chest pain. Ten healthy adult volunteers (age 22–38 years, four men) with no history of medical illness were prospectively enrolled.

In all, 15 patients with CAD (age 38–64 years, 10 men) undergoing dobutamine stress CMR with rest and stress T1 and T2 mapping within 60 days of SPECT/MPI with no coronary angiography intervention before, followed by coronary angiography with FFR (Fractional Flow Reserve).

On a 1.5 T scanner (Siemens Magnetom Aera; Siemens Medical Systems, Erlangen, Germany), all individuals were scanned. Our Institutional Review Board gave its approval to the research protocol. From each participant, written informed consent was obtained.

2.1. Inclusion criteria

Ten healthy adult individuals were prospectively enlisted, none of them had a history of cardiovascular illness or heavy endurance exercise. Furthermore, we looked at 15 CAD patients, who had been clinically referred for dobutamine stress and/or rest SPECT/MPI (Myocardial Perfusion Imaging) to assess their chest discomfort. Within 60 days of the SPECT/MPI, patients will receive dobutamine stress and/or rest CMR T1 and T2 mapping with no internal intervention. Our Institutional Review Board gave its approval to the research protocol. From each participant, written informed consent was obtained.

2.2. Exclusion criteria

Patients with severe arterial hypertension (210/120 MMHG), aortic stenosis of significance (peak gradient, 50 mmHg), hypertrophic obstructive cardiomyopathy, cardiac arrhythmias (including atrial fibrillation), unstable angina pectoris, congestive heart failure, myocarditis, endocarditis, and pericarditis are contraindications to CMR as are metallic implants, pacemakers, even if updated standards permit imaging in some circumstances, claustrophobia, contrast allergy, MR-incompatible prosthetic heart valves, and body weight (certain weight restrictions for MRI tables).

2.3. Operation design

A history was performed, and each risk factor for CAD will be assessed such as hypertension, diabetes, cholesterol, dyslipidemia, smoking, and BMI. Personal family history is also important to identify the genetic predisposition.

Clinical examination: all participants had thorough medical examinations, which included evaluations of their overall health and vital indicators including blood pressure and heart rate. Analysis of the location, kind, start, duration, and site of referred pain in chest discomfort was made.

Laboratory investigations: all patients will have their whole blood count and serum creatinine.

Standard 12-lead ECG was done for all patients and healthy volunteers.

Echocardiography: full documented 2D echo done for CAD patients by Phillips (Village of Reedsville, Municipal Building, USA) echo.

Every patient be evaluated (EF, RWMA, valve assessment) by Doppler M-mode and eye-balling.

SPECT and thalium study: Patients with CAD undergoing evaluation for ischemia.

2.4. Dobutamine cardiac magnetic resonance

Dobutamine CMR displays the procedure for healthy participants, following image localization; individuals will go through native T1 and T2 mapping, rest cine CMR, dobutamine stress induction, and three further native T1 and T2 mapping scans. Targeted peak stress was determined by a heart rate of at least 0.85 % of the predictive heart rate (220 age), age in years, followed by a half-dose dobutamine withdrawal period, recovery stage, and T1 and T2 recovery mapping.

Dose of dobutamine stress: 10 mU/kg and uptitrated up to 40 mU/kg.

Before the scan, all patients stopped using beta-blockers for 48 h. An ECG was taken as a baseline. A peripheral infusion pump in the control room was used to give dobutamine (1 mg/ml concentration). Blood pressure and heart rate were monitored at rest and then every 2 min while dobutamine was being infused.

2.5. Data analysis

According to the normal American Heart Association paradigm, the basal, mid, and apical LV SAX images were split into 6, 6, and 4 segments, respectively.[5]

T1 and T2 mapping values were measured on 16 segments of each case in rest, peak stress, and recovery by two observers. T1 and T2 reactivity were also calculated as the differences in T1 rest, T1 poststress, T2 rest, and T2 poststress values, respectively:

$$\Delta T1 = \frac{\Delta T1 \text{ post stress} - \Delta T1 \text{ rest}}{\Delta T1 \text{ rest}} \text{x}100.$$

$$\Delta T2 = \frac{\Delta T2 \text{ post stress} - \Delta T2 \text{ rest}}{\Delta T2 \text{ rest}} \text{x}100.$$

T1 and T2 reactivity measurements of all cases were assessed by two observers, while a random sample of six patients with CAD was assessed twice on two separate days with a washout period of 10 days to evaluate interobserver and intraobserver reproducibility.

With symptom-limited treadmill exercise stress, technetium-99 m sestamibi was used to conduct stress and/or rest SPECT-MPI in all CAD patients. Experienced observers (A.E.) assessed the myocardial segments during rest and stress images, respectively. Myocardial ischemia was defined as any reversible defect and infarcted myocardium was defined as any permanent defect. Angiographic analysis: quantitative coronary angiography was performed in 15 cases by two operators blinded to SPECT and CMR data. Each myocardial segment was assigned to a coronary artery territory according to the American Heart Association 17-segment model standard criteria. We labeled the 16 segments either ischemic if they were along the territory of significant coronary stenoses or normal remote if there were no significant proximal stenotic lesions.

3. Results

Twenty-five patients who arrived at the National Heart Institute directly, without being transferred from another cardiac institution, were included in the research. As a result, practically all of the patients had the same intervention method, increasing the homogeneity of the research group. Data on baseline clinical traits are summarized in this table dividing the research into two groups: controls and cases. There were 60 % women and 40 % men in the control group. The SD was 9.41, and the mean age was 30 of the control participants. The younger and older ages that were given were 22 and 38 years, respectively. Some participants are risk-free and in good health.

In all, 33.3 % of cases were women, while 66.7 % were men.

The SD was 9.59, while the mean age of the ischemic patients was 57 years. The youngest and oldest ages represented were 38 and 64 years, respectively. All patients had concomitant conditions, with 86.7 % of patients having hypertension and just 13.3 % presenting without hypertension, according to the patient's risk profile. In addition, 66.7 % of patients had diabetes compared with 33.3 % of nondiabetic individuals. While 53.3 % of patients denied smoking, 46.7 % of patients had smoking behaviors ranging from light to excessive. All of the group's patients had a history of IHD in the past, either with prior coronary intervention or while receiving ongoing medical care without intervention. In addition, every patient in this group had dyslipidemia.

3.1. SPECT and invasive coronary radiograph angiography

Regarding reliability between SPECT and cardiac angiography among the studied cases ($n = 240$ segments), 136 segments were found to be normal by both SPECT and cardiac angiography (56.7 %), and 43 were found to be ischemic by both SPECT and cardiac angiography (17.9 %; $P = 74.6$ %) While 24 segments found to be ischemic by SPECT were normal in cardiac angiography and 37 segments found to be ischemic by cardiac angiography were normal in SPECT (a discrepancy of 61 segments), so $P = 25$ % to find the degree of agreement between SPECT and cardiac angiography. Using Cohen's Kappa interrater agreement coefficient, the results show moderate reliability ($K = 0.5$ with significance, $P < 0.001$; Tables 1 and 2).

Regarding the ability of SPECT to detect ischemia compared with the gold standard of cardiac angiography and FFR, it shows 53.75 % sensitivity with 85 % specificity. The positive predictive value (PPV) of SPECT was 64.18 %, and the negative predictive value (NPV) was 78.61 %, with an accuracy of 74.58 %. With a P value of less than 0.01, the area under the receiver-operating characteristic curve was statistically substantial at 0.694 (Table 3).

As regards the native T1 mapping values in healthy volunteers compared with cases with CAD, there is no substantial difference between the rest of the native T1 mapping values, with a mean of 1018.78 ms in healthy volunteers and 1024.54 ms in cases with a P value of 0.087. T1 mapping values showed an increase in both cases and healthy volunteers under stress, with no significant difference

Table 1. Comparison between control and case regarding baseline clinical characteristics.

Basic characteristics	Control ($N = 10$)	Case ($N = 15$)	Test	P value (significance)
	n (%)	n (%)		
Sex				
Male	4 (40)	10 (66.7)	1.732[a]	0.241
Female	6 (60)	5 (33.3)		NS
Age (years)				
Mean ± SD	30 ± 9.48	52.66 ± 9.59	−0.750[b]	0.001
Range	22–38	38–64		S
Dyslipidemia				
Absent	10 (100)	0		
Present	0	15 (100)		
Diabetes mellitus				
Absent	10 (100)	5 (33.3)		
Present	0	10 (66.7)		
Hypertension				
Absent	10 (100)	2 (13.3)		
Present	0	13 (86.7)		
Family history				
Absent	10 (100)	8 (53.3)		
Present	0	7 (46.7)		
Smoking				
Absent	10 (100)	8 (53.3)		
Present	0	7 (46.7)		

[a] chi square test.
[b] Mann Whitney U test.

Table 2. Agreement statistics between SPECT and cardiac angiography among the studied cases (N = 240 segments).

SPECT	Cardiac angiography [n (%)]		Total	
	Normal	Ischemic		
Normal	136 (56.7)	37 (15.4)	173 (72.1)	
Ischemic	24 (10)	43 (17.9)	67 (27.9)	
Total	160 (66.7)	80 (33.3)	240 (100)	
	All segments ($N = 240$)	P value[a]	K	P value
	n (%)	Sig.	95 % CI	Sig.
Concordant	179 (74.6)	0.124	0.404	<0.001
Ischemic/ischemic	43 (17.9)	NS	0.281–0.527	HS
Normal/normal	136 (56.7)			
Discordant	61 (25.4)			
Ischemic/normal	24 (10)			
Normal/ischemic	37 (15.4)			

[a] McNemars test.

Table 3. Diagnostic performance of SPECT in the diagnosis of cardiac ischemic among the studied cases (N = 240 segments).

	Cardiac angiography [n (%)]		Total
	Normal	Ischemic	
SPECT			
Normal	136 (56.7)	37 (15.4)	173 (72.1)
Ischemic	24 (10)	43 (17.9)	67 (27.9)
Total	160 (66.7)	80 (33.3)	240 (100)
SPECT			
SN (95 % CI)	53.75 % (42.24–64.97)		
SP (95 % CI)	85 % (78.51–90.15)		
PPV (95 % CI)	64.18 % (54.04–73.19)		
NPV (95 % CI)	78.61 % (74.21–82.45)		
Accuracy (95 % CI)	74.58 % (68.58–79.97)		
AUROC (95 % CI)	0.694 (0.631–5.460)		
P value (significance)	<0.001 (HS)		

AUROC, area under receiver-operating characteristic curve; NPV, negative predictive value; PPV, positive predictive value.

($P = 0.782$). Stress T1 mapping values in healthy volunteers were 1061.81 and 1060.51 ms in cases. While there is a significant difference of P value of 0.005 between healthy volunteers and cases in T1 reactivity, T1 reactivity in healthy participants is 4.22 and 3.51 in cases. T1 mapping significantly drops in healthy cases, with a significant difference of P value less than 0.001. These changes reflect changes in myocardial blood flow during stress, so T1 could be used as an indicator of myocardial blood flow (Table 4).

As regards the native T2 mapping values in healthy volunteers compared with cases with CAD, there is no significant difference between the resting native T2 mapping value with a mean of 52.25 ms in healthy volunteers and 52.29 ms in cases with a P value of 0.402. T2 mapping values showed an increase in both cases and healthy volunteers at stress, with no significant difference ($P = 0.729$). Stress T2 mapping values in healthy volunteers were 55.32 ± 3.94 and 55.54 ms in cases, while there is a significant difference of P value of 0.010 between healthy volunteers and cases in T2 reactivity. T2 reactivity in healthy participants is 5.94 and 7.050 ms in cases. Also, T2 mapping during recovery showed a significant difference between healthy participants and cases, with a P value of 0.027 (Table 5).

As regards the native T1 mapping values in cases compared with coronary angiography on a per-vessel basis, there is a significant difference between the stress T1 mapping value with a mean of 1071.704 ms in normal segments and 1038.143 ms in ischemic segments with a P value of 0.001. Also, there is a significant difference in T1 reactivity in normal segments (4.3622 ms) and 1.828 ms in ischemic segments ($P < 0.001$), confirming that T1 could be used as a strong surrogate indicator for changes in ischemic segments (Table 6).

As regards the T2 mapping values in cases compared with coronary angiography, there is no significant difference between rest, stress T2, recovery T2 mapping values, or T2 reactivity between normal and ischemic segments (Table 7 and Fig. 1).

According to receiver-operating characteristic curve analysis, the stress T1 MA P value less than 1049 ms with a confidence interval (CI) of 95 % showed 71.25 % sensitivity, 73.75 % specificity, 83.7 % NPV, and 57.6 % PPV with an accuracy of about 73 % and a highly significant P value of 0.001 (Table 8).

Compared to SPECT in the diagnosis of ischemia, which showed 53.75 % sensitivity, 85 % specificity, 78.61 % NPV, and 64.18 % PPV, with an accuracy of about 74.58 % and a high significant P value of 0.001.

The interclass coefficients for interobserver and intraobserver measurements of T1 values were 0.85 and 0.97, respectively (Table 8).

On analyzing the diagnostic accuracy of ΔT1 MAP first, it had 66.25 % sensitivity and 94.37 % specificity compared with angiography. The PPV and NPV of T1 MAP first were 85.5 and 84.8 %, respectively. The area under the receiver-operating characteristic curve was 0.763, and it was statistically substantial ($P < 0.001$) (Table 9).

Table 4. Comparison between control and case regarding cardiac magnetic resonance–T1 MAPP.

CMR–T1 MAPP	Control ($N = 160$)	Case ($N = 240$)	Test	P value (significance)
T1 rest (MS)				
Mean ± SD	1018.78 ± 38.74	1024.54 ± 37.69	−1.713[a]	0.087
Median	1018.70	1026.55		NS
Range	905–1112.30	903–1146		
T1 stress (MS)				
Mean ± SD	1061.81 ± 42.76	1060.51 ± 47.88	0.276[b]	0.782
Median	1064.40	1059		NS
Range	932.80–1187	934.30–1206.30		
T1 Δ				
Mean ± SD	4.228 ± 1.791	3.518 ± 3.214	−2.786[a]	0.005
Median	3.847	3.539		S
Range	1.102–11.786	−5.492–18.176		
T1 recovery (MS)	$N = 160$	$N = 192$[c]		
Mean ± SD	997.06 ± 38.49	1014.46 ± 40.47	−3.741[a]	<0.001
Median	1002.60	1016.70		
Range	853–1075.30	908.20–1109.80		

CMR, cardiac magnetic resonance.
[a] Mann Whitney U test.
[b] Independent samples students T test.
[c] Refers to 3 patients not wait until recovery period so their segments were excluded.

Table 5. Comparison between control and case regarding cardiac magnetic resonance–T2 MAP.

CMR–T2 MAPP	Control ($N = 144$)	Case ($N = 240$)	Test	P value (significance)
T2 rest (MS)				
Mean ± SD	52.25 ± 3.95	52.29 ± 5.39	−0.839[a]	0.402
Median	51.75	51.30		NS
Range	42.80–64.60	39.80–67.10		
T2 stress (MS)				
Mean ± SD	55.32 ± 3.94	55.54 ± 5.59	−0.347[a]	0.729
Median	55.20	55.10		NS
Range	45.20–66.50	40.30–69.40		
T2 Δ				
Mean ± SD	5.944 ± 2.828	7.050 ± 9.108	−2.580[a]	0.010
Median	5.582	6.858		S
Range	0.984–16.942	−25.232–70.60		
T2 recovery (MS)	$N = 144$	$N = 192$[b]		
Mean ± SD	53.11 ± 4.28	52.40 ± 5.32	−2.215[a]	0.027
Median	53.10	51.20		(S)
Range	43.80–66	40.10–66		

CMR, cardiac magnetic resonance.
[a] Mann Whitney U test.
[b] refers to 3 patients not wait until recovery period so their segments were excluded.

4. Discussion

MRI tissue mapping is a novel technique that can reflect tissue composition and also reflect changes such as hyperemia and myocardial blood flow. One of the major advantages of MR-tissue mapping is that it can increase the sensitivity of the CMR stress test to detect ischemia in CAD patients without the need to inject contrast and with a relatively short scan time sequence.

In this investigation, we evaluated the capability of native T1 and T2 mapping during stress and at rest to identify myocardial ischemia or aberrant blood flow following dobutamine stress. This study was conducted on 15 ischemic patients with CAD as well as 10 healthy volunteers studied at rest and with dobutamine stress. A comparison was done between the two groups for segment-based analysis and then with coronary angiography based on territorial vessel-based analysis.

We demonstrated that there was no substantial variation in rest T1 or T2 mapping values between normal and ischemic segments, consistent with Nakamori et al.'s[6] exercise stress study.

This study revealed that the T1 showed elevation during stress with substantial variation between remote and ischemic myocardium in CAD patients, and this is blunted when compared with the control during maximum dobutamine stress, which agrees

Table 6. Comparison between cases with normal cardiac angiography and cases with ischemic cardiac angiography regarding cardiac magnetic resonance–T1 MAPP.

CMR–T1 MAPP	Normal ($N = 160$)	Ischemic ($N = 80$)	Test	P value (significance)
T1 rest (MS)				
Mean ± SD	1026.83 ± 41.14	1019.95 ± 29.32	−1.623[b]	0.105
Median	1032	1022.85		NS
Range	903–1146	925.40–1073.70		
T1 stress (MS)				
Mean ± SD	1071.70 ± 48.83	1038.14 ± 37.10	−5.737[b]	<0.001
Median	1078.10	1033.90		HS
Range	940.30–1206.30	934.30–1131.90		
T1 Δ				
Mean ± SD	4.362 ± 2.526	1.828 ± 3.752	−6.643[b]	<0.001
Median	4.067	0.603		HS
Range	−2.347–15.615	−5.492–18.176		
T1 Recovery (MS)	$N = 131$	$N = 61$		
Mean ± SD	1017.08 ± 40.77	1008.84 ± 39.55	−1.540[b]	0.124
Median	1201.30	1010		NS
Range	908.20–1109.80	924–1105.70		

CMR, cardiac magnetic resonance.

Table 7. Comparison between cases with normal cardiac angiography and cases with ischemic cardiac angiography regarding cardiac magnetic resonance–T2 MAPP.

CMR–T2 MAPP	Normal ($N = 160$)	Ischemic ($N = 80$)	Test	*P* value (significance)
T2 rest (MS)				
Mean ± SD	52.74 ± 5.50	51.38 ± 5.09	−1.805[a]	0.071
Median	51.60	50.55		NS
Range	42.70–67.10	39.80–64.10		
T2 stress (MS)				
Mean ± SD	55.67 ± 5.75	55.28 ± 5.27	−0.575[a]	0.565
Median	55.25	54.75		NS
Range	40.30–69.40	45.50–68.30		
T2 Δ				
Mean ± SD	6.643 ± 9.963	7.865 ± 7.082	−1.389[a]	0.165
Median	6.687	7.308		NS
Range	−25.232–70.600	−12.963–25.377		
T2 recovery (MS)	$N = 131$	$N = 61$		
Mean ± SD	52.34 ± 5.09	52.51 ± 5.85	−0.159[a]	0.874
Median	51.30	51		(NS)
Range	44.30–66	40.10–64.90		

CMR, cardiac magnetic resonance.
[a] Mann Whitney U test.

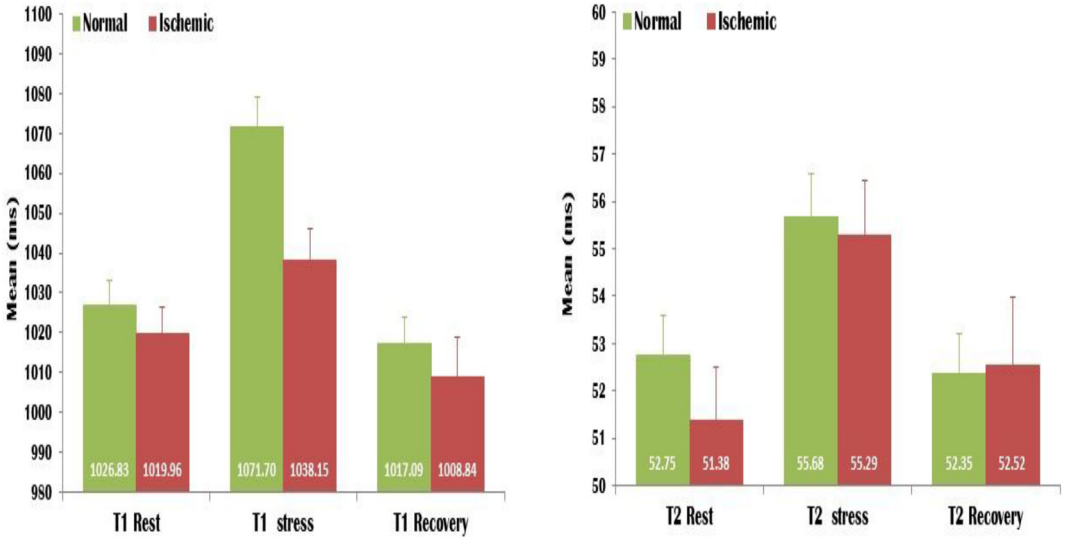

Fig. 1. Relationship between regional native T1 and T2 parameters and coronary angiography among ischemic patients.

Table 8. T1 MAPP stress (ms) as a diagnostic test for cardiac ischemia: receiver-operating characteristic curve analysis.

T1 MAPP stress (ms)	
Cutoff value	≤1049
SN (95 % CI)	71.25 % (60–80.8)
SP (95 % CI)	73.75 % (66.2–80.4)
PPV (95 % CI)	57.60 % (50.3–64.60)
NPV (95 % CI)	83.70 % (78.2–88)
Accuracy (95 % CI)	72.92 % (64.1–80.5)
AUROC (95 % CI)	0.727 (0.666–0.783)
P value (significance)	<0.001 (HS)

AUROC, area under receiver-operating characteristic curve; CI, confidence interval; NPV, negative predictive value; PPV, positive predictive value.

Table 9. T1 MAPP first stress (ms) as a diagnostic test for cardiac ischemia: receiver-operating characteristice curve analysis.

Δ T1 MAPP	
Cutoff value	≤1.175
SN (95 % CI)	66.25 % (54.8–76.4)
SP (95 % CI)	94.37 % (89.6–97.4)
PPV (95 % CI)	85.50 % (75.4–91.9)
NPV (95 % CI)	84.80 % (80.4–88.4)
Accuracy (95 % CI)	85.01 % (78–90.4)
AUROC (95 % CI)	0.763 (0.704–0.815)
P value (significance)	<0.001 (HS)

AUROC, area under receiver-operating characteristic; CI, confidence interval; NPV, negative predictive value; PPV, positive predictive value.

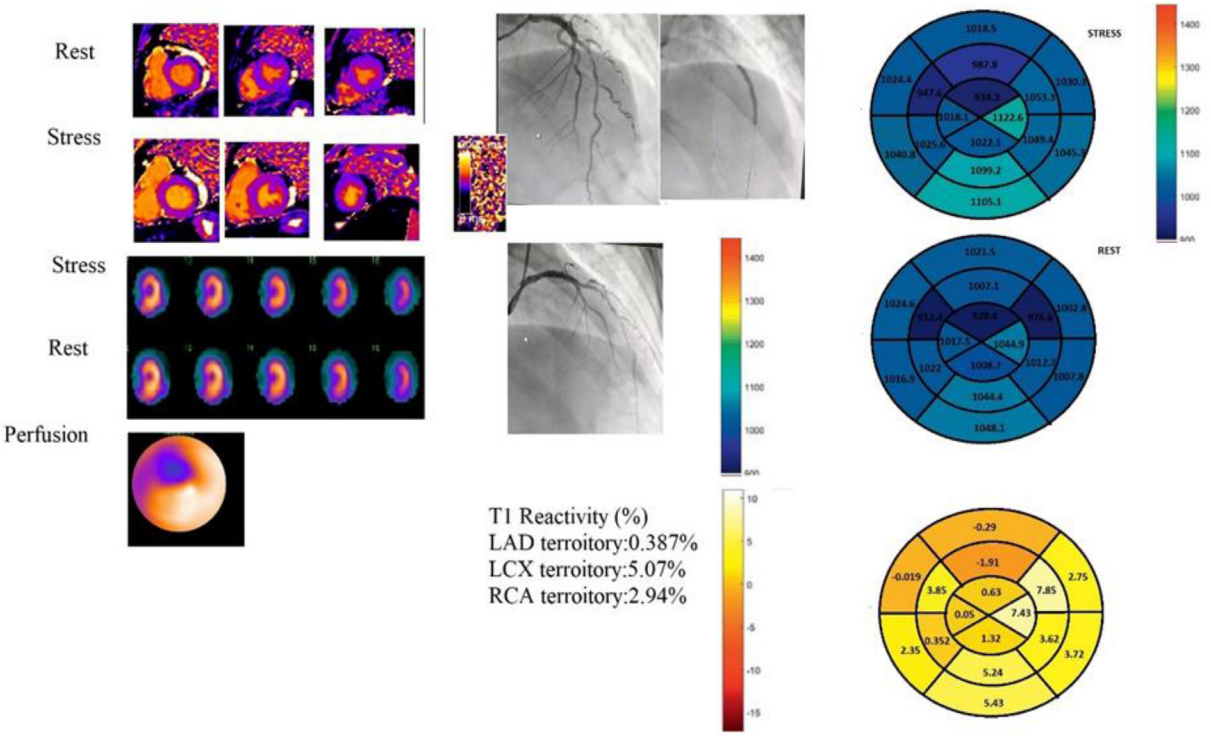

Fig. 2. A case with LAD ischemia.

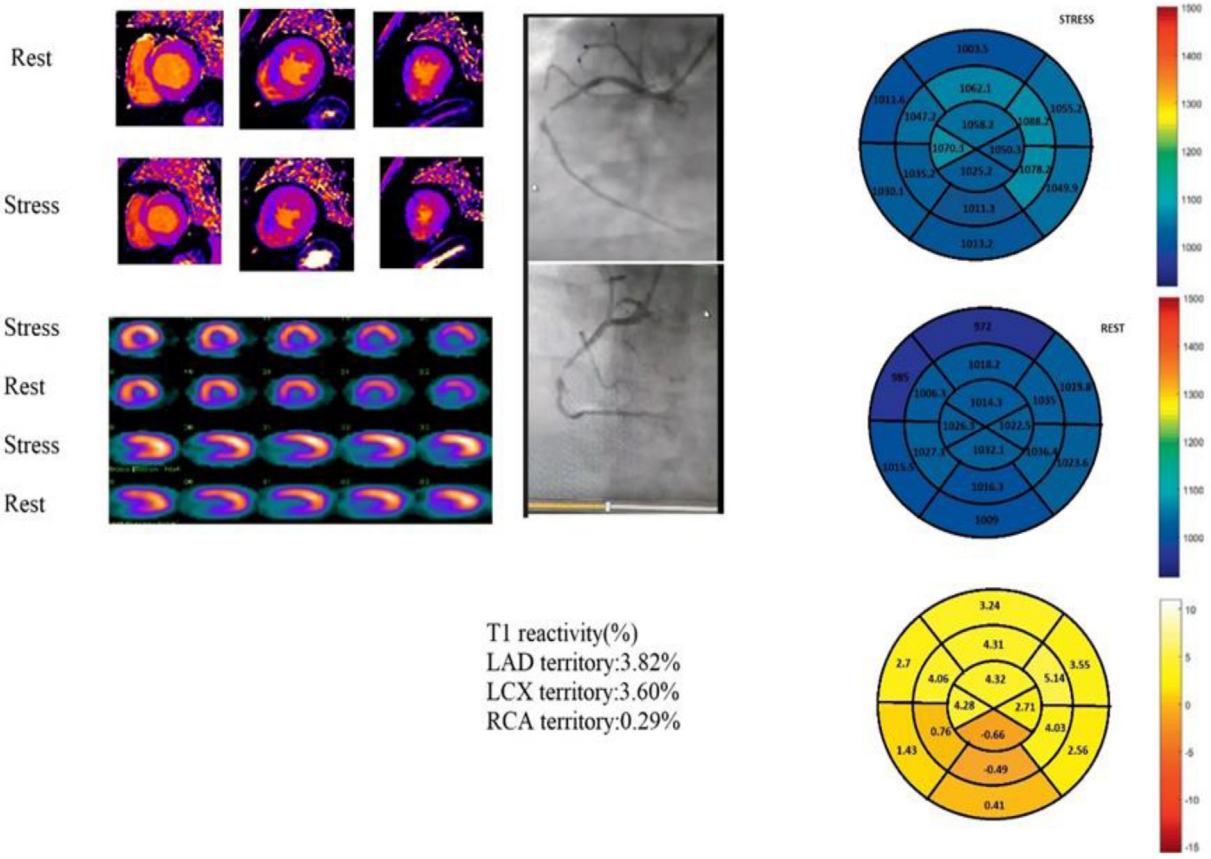

Fig. 3. Acase with RCA ischemia.

with Nakamori and colleagues as well as Liu and collegaues' studies.[7–9]

There is a highly significant difference in T1 reactivity in both groups, which is explained by the presence of flow-limiting stenosis and physiological changes during ischemia. These were consistent with changes in T1 mapping either with exercise or adenosine stress.[7,8]

The cutoff value of T1 reactivity in our research was 1.175 % with 94.37 % specificity and 66.25 %, which was consistent with 1.2 % for mild cases in the Nakamori et al.[6] study.

Incorporating T1 reactivity with a cutoff value of 1.17 % to stress CMR makes stress and rest T1 mapping greater than SPECT/MPI according to the receiver-operating characteristic curve analysis for predicting significant stenosis with sensitivity of 66.25 % (95 % CI, 54.8–76.4), SP of 94.37 % (95 % CI,89.6–97.4), PPV of 85.50 % (95 % CI, 75.4–91.9), NPV of 84.80 % (95 % CI, 80.4–88.4), accuracy of 85.01 % (95%CI, 78–90.4), and an area under receiver-operating characteristic curve of 0.763 (95 % CI, 0.704–0.815).

There is a significant difference in T2 reactivity and T2 recovery values between healthy volunteers and cases; these differences were blunted by being nonsignificant when territorial-based analysis compared with coronary angiography was done. This might be explained by changes in T2 mapping values being more diffuse, impaired micro-circulation in the remote myocardium of ischemic cases, as well as the high sensitivity of the T2 mapping sequence to detect subclinical myocardial edema and the absence of a significant difference in T2 mapping values. So, changes in T2 mapping values could not be used as a good marker for ischemia, and this is consistent with the results of Nakamori et al.[6]

4.1. Representative cases

A 56-year-old male with dyspnea and pain on excretion with no ischemic changes on ECG or echocardiography was referred for noninvasive testing to detect ischemia, stress SPECT showed reversible ischemia in the anterior and anteroseptal walls. A rest stress native T1 map showed decreased T1 reactivity in the basal, anteroseptal, mid anterior, and apex walls. Patient coronary angiography revealed proximal to mid-long significant stenosis in LAD (Left Anterior Descending), so a stent was deployed in the same session (Fig. 2).

A 50-year-old male with typical chest pain on exertion. Resting ECGs show PVCs (Premature Ventricular Contraction), and echos show mild hypokinesia in the inferior wall of a patient undergoing stress trademill test. During the test, the patient suffered agonizing chest pain, and the stress ECG showed nonsustained VT, prompting to stop the test. Thallium imaging showed reversible ischemia in the inferior and inferior septal walls. The T1 reactivity map reveals low T1 reactivity values around 0.29 % in the corresponding myocardial segments along the RCA territory. Coronary angiography revealed single-vessel disease with mid-RCA subtotal occlusion and stenting occurring in the same session (Fig. 3).

4.2. Conclusion

We found that segments with ischemia showed significant changes in myocardial native T1 during dobutamine stress, especially T1 reactivity. Low T1 reactivity with a cutoff value of 1.17 % showed a sensitivity of 66.25 % and a specificity of 94.37 % with an area under the curve of 0.763 for identifying substantial CAD on coronary angiography, likely due to impaired microvascular dilatation that causes a decrease in blood flow during dobutamine stress resulting from significant coronary stenosis. The main advantage of using T1 reactivity is that it can overcome the major disadvantage of the variability of T1 mapping according to the scanner as it calculates a ratio with a cutoff value that can detect ischemia with very high specificity, good sensitivity, PPV, and NPV.

Conflicts of interest

None declared.

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