Role of Cardiac MRI Including LGE, T1 and T2 Mapping in the Assessment of Cardiac Involvement in Patients of Nonspecific Aorto-arteritis: A Prospective Study

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Abstract

Objective   Nonspecific aorto-arteritis (NSAA) may involve the myocardium in the form of edema and fibrosis. We conducted this study to investigate role of cardiac MRI including late gadolinium enhancement (LGE), T1 and T2 mapping in the assessment of cardiac involvement in NSAA.

Methods and Materials   Over the period between 2016 and 2019, 36 patients with NSAA presenting with uncontrolled hypertension, left ventricular dysfunction, congestive cardiac failure, or tachyarrhythmia were included in the study. We also had 16 voluntary control patients for providing normal T1 and T2 mapping values.

Results   The average age of patients was 27.1 years and the majority were females. MRI is more sensitive than echocardiography in the detection of LV dysfunction and RWMA. Out of 36 patients, 10 (27.8%) had LGE. The most common pattern of midmyocardial enhancement was present in 5 out of 10 patients. Five (13.8%) patients show mid-myocardial enhancement, followed by epicardial enhancement, which was seen in four (11.11%) patients. The values of post-gad T1 mapping values were significantly lower than pre-gad T1 mapping values. At a cut-off global native T1 mapping value of 1019 milliseconds had the sensitivity of 83.3% and specificity of 81.2% in detecting an abnormal T1 map. No significant association of MRI contrast enhancement with elevated ESR and CRP levels. There was no significant relation of myocardial T2 mapping values between NSAA and control groups.

Keywords
► cardiac MRI
► nonspecific aorto-arteritis
► T1 mapping
► T2 mapping

Conclusion   Quantitative tissue characterization in the myocardium with native T1 mapping values help in the detection of cardiac involvement in patients with NSAA. T1 mapping may provide incremental value in the assessment of myocardial involvement in NSAA in addition to LGE imaging.

Introduction

Nonspecific aorto-arteritis (NSAA) is a chronic inflammatory panarteritis, predominantly involving the aorta and its branches.[1,2] It is also called as Takayasu arteritis. It usually occurs in young patients, most commonly in third and fourth decades of the life. It predominantly affects young women and is associated with premature atherosclerosis. Initial inflammatory phase is followed by late vascular ischemic changes.[1–3]

Uncontrolled hypertension is one of the frequent complications in NSAA, which may be caused secondary to renal artery stenosis and may lead to congestive cardiac failure. Congestive cardiac failure may have multifactorial etiology. It is predominantly caused by obstructive aorto-arteritis or systemic hypertension. Talwar et al showed NSAA may commonly affect the myocardium and found 45 of 54 patients demonstrated some histologic abnormality in their study.[4] Myocardial dysfunction may be associated with hypertension, mitral regurgitation, or aortic regurgitation. Myocarditis may occur in the active stage of NSAA. Talwar et al demonstrated myocarditis by endo-myocardial biopsies in 8 out of 11 active TA patients.[5] This may also cause congestive heart failure. Coronary artery involvement may also be seen in NSAA. Aortic regurgitation is other unusual complication of NSAA, which may cause congestive heart failure in these patients. Aortic aneurysms and dissections may also be seen in NSAA patients.[6]

Echocardiography helps in the screening and evaluation of arterial lesions, especially aortic arch vessel lesions in patients of NSAA. It may also evaluate left ventricular dysfunction, left ventricular hypertrophy, aortic regurgitation, pulmonary hypertension, etc.[7] However, early left ventricular dysfunction and myocardial involvement by NSAA may be difficult to appreciate on echocardiography.

Cardiac MRI with gadolinium contrast helps in assessing myocardial involvement in the form of fibrosis or edema. The presence of myocardial fibrosis implies significant cardiac disease. Left ventricular hypertrophy occurs secondary to increased systemic afterload or increased vascular stiffness in NSAA. Myocardial scarring may be seen in 27% of NSAA patients. Late gadolinium enhancement (LGE) is used for the identification of subendocardial scarring or fibrosis or in the mid-LV wall.[1–3]

Cardiac MRI with LGE can evaluate the myocardial involvement and cardiac function. Myocardial fibrosis may be regional or diffuse, based on the chronicity of the disease. Cardiac MRI LGE may detect patchy, nonspecific patterns of fibrosis, but cannot quantify it. Also, cardiac MRI with LGE may not detect myocardialfibrosis when the fibrosis is diffuse with no definite scar formation. Recent advances in cardiac MRI sequences such as T1 mapping may quantitatively detect and measure diffuse myocardial fibrosis.[5,8] Myocardial T2 mapping can reliably identify and quantify inflammatory edema in myocarditis. Myocardial inflammation leads to increased T2 and T1 relaxation times. These can be pixel wise quantified by myocardial T1 and T2 mapping techniques and offer better diagnostic performance in comparison to conventional T2-weighted sequence. Thus, T1 and T2 mapping MRI techniques can be used for quantitative tissue characterization of the myocardium.

NSAA may involve the myocardium in the form of edema and fibrosis. Cardiac MRI including LGE, T1 and T2 mapping can detect the myocardial inflammation or fibrosis in NSAA. There is a significant paucity of the literature on the LGE, T1 and T2 mapping for the assessment of myocardial involvement in the patients of NSAA. The present study has been designed to address these lacunae.

Materials and Methods

This study was conducted after obtaining institute ethics committee approval. We conducted a prospective study between 2016 and July 2019 to assess if cardiac MRI can evaluate myocardial involvement using LGE, T1 and T2 mapping in patients of NSAA. Patients of NSAA presenting with uncontrolled hypertension, left ventricular dysfunction, congestive cardiac failure, or tachyarrhythmia were included in the study. NSAA was diagnosed using ACR (American College of Rheumatology) criteria for the diagnosis of NSAA. NSAA disease activity was assessed using NIH (National Institutes of Health) criteria. Those patients who didn't give consent to be included in the study, having contrast allergy and contraindication to MRI examination were excluded from the study. We also performed cardiac MRI in consecutive voluntary control subjects for providing normal T1 and T2 mapping values.

Methodology

All cardiac MRI examinations were performed on 1.5 Tesla MRI scanner (Siemens Aera, Erlangen, Germany) with dedicated cardiac coil. Cardiac MRI protocol included localizer sequences; cine sequences obtained in short axis, vertical long axis and horizontal long axis view; T1W double inversion and T2W triple inversion sequences in the short axis/horizontal long axis view. MR angiography images were obtained after injecting 0.01 mL/kg of gadoterate meglumine (DOTAREM). Post-contrast scans were acquired in phase sensitive inversion recovery (PSIR) sequences in short axis and horizontal long axis after 5 and 15 minutes. Post contrast three-dimensional (3D) volumetric interpolated breath-hold (VIBE) sequence of chest was obtained.

T1 and T2 mapping sequences were acquired in ventricular short-axis sections at basal, mid, and apical LV levels. T1 mapping sequence was done using the modified Look-Locker inversion recovery sequence using the MOLLI, 3(3)3(3)5 method. T2 mapping sequence was performed using Siemens Tx-mapping package. Post gad T1 mapping sequence was obtained after 10 minutes of contrast administration. Pixel-wise T1 and T2 mapping analysis was performed for all myocardial segments.

Echocardiography was done to assess cardiac function. The interval between the ECHO and CMR was less than 2 weeks. Laboratory findings such as C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) were collected in all patients. Disease activity (based on NIH criteria) was correlated with cardiovascular MRI findings as well as ESR and CRP values.

MRI Analysis

Cine TRUFISP cardiac images were used for the evaluation of functions of the left and right ventricles. The syngo.via cardiac workstation calculated biventricular cardiac functions values. The LGE images were analyzed visually for identifying the contrast-enhancing segments. The distribution of LGE was interpreted in a 17-segment AHA model.

Native T1 and T2 mapping values were analyzed using the pixel-wise region of interest (ROI) according to cardiac segments of the AHA model. The maximum values obtained in the particular segment were selected for the evaluation of T1 and T2 mapping. A total of 16 cardiac segments were included. Segment 17 (cardiac apex) was not analyzed due to significant cardiac motion artifacts. The cardiac global T1 mapping, T2 mapping, and post gad T1 mapping were calculated at basal, mid and apical LV. Septal T1 mapping, T2 mapping, and post gad T1 mapping values were also calculated at basal, mid, and apical LV levels. All MRI images were processed and reviewed by two radiologists having at least 15 years of experience in cardiovascular imaging.

Statistical Analysis

All the data were analyzed by statistical software (SPSS version 24.0 or STATA version 14) for the evaluation of cardiac involvement on cardiac MRI in NSAA patients. MRI was used for the assessment of myocardial involvement in patients of NSAA. All data were summarized as mean ± standard deviation (SD) or number and percentage as required for the variables. Data were evaluated for normality using the Kolmogorov–Smirnov test. The t-test was utilized to compare the parametric values, whereas the Mann–Whitney U-test was used for nonparametric values wherever required. The Chi-square/Fisher's exact test was utilized for comparison of categorical data. A p-value < 0.05 was considered significant for the study.

Results

Over the period between 2016 and 2019, 36 patients with NSAA presenting to the All India Institute of Medical Sciences, Delhi, India were included. Informed consent was taken

Table 1 Patient demographics

Characteristic	Patient cohort	Control group
Total (*n*)	36	16
Age in years (mean ± SD)	27.1 ± 14.0	24.2 ± 8.2
Males	10 (27.8%)	11 (68.75%)
Females	26 (72.2%)	5 (31.3%)
HTN	25 (69.4%)	–
DM	1 (2.7%)	–
Smoker	0	–

from all patients. A total of 36 patients, who fulfilled our inclusion criteria underwent contrast-enhanced cardiac MRI with T1 and T2 mapping sequences. Cardiac MRI was also performed in 16 control groups for providing normal T1 and T2 mapping values. The average age of NSAA patients was 27.1 years and the majority were females. Patient demographics are outlined in ►**Table 1**.

Clinical Symptoms

Frequent clinical symptoms in NSAA patients were limb pulse difference (83.3%), followed by hypertension (67.7%). Twenty-two patients (61.1%) also complained of dyspnea. The mean systolic and diastolic blood pressures were 153.6 ± 34.1 mm Hg and 90.0 ± 17.3 mm Hg, respectively. Increased ESR levels were found in 17 patients (47.2%) and increased CRP levels in 16 patients (44.4%). The average CRP and ESR levels were 9.1 ± 19.4 mg/L and 26.1 ± 16.2 mm/h, respectively.

Out of 36 patients, 11 (30.6%) patients were put on steroid therapy. Five patients (13.9%) were given immunosuppressant drug regimen. Twenty-five (69.4%) hypertensive patients had received antihypertensive treatment.

Cardiac MRI Findings

Cardiac MRI was performed in all 36 patients of NSAA. LV systolic dysfunction was seen in 15 patients. Regional wall motion abnormality (RWMA), LV wall thinning, and bi-atrial dilatation were seen in two patients each. LV dilatation and hypertrophy were seen in 8 and 5 patients, respectively. LV hypokinesia and RWMA were seen in 15 and 2 patients, respectively. Two patients demonstrated bi-atrial dilatation. Right ventricular systolic dysfunction seen in seven patients. Aortic regurgitation was seen in nine patients.

Association between Echocardiography and MRI Findings

Our analysis showed a significant correlation between echocardiography and MRI in detecting LV systolic dysfunction, LV dilation, and aortic regurgitation. MRI is more sensitive in the detection of LV dysfunction/global hypokinesia and RWMA. Both the modalities were equally sensitive in the detection of LV dilatation, bi-atrial dilatation, and aortic regurgitation.

The sensitivity and specificity of echocardiography in comparison to MRI is as follows: for LV dysfunction was

Table 2 Patterns of LGE on MRI ($n = 36$)

LGE	Number (%)
Present	10 (27.8)
Midmyocardial	5 (13.8)
Epicardial	4 (11.1)
Subendocardial	1 (2.8)
Diffuse (mid-to-epicardial)	1 (2.8)

76.9 and 73.9%; for regional wall motion abnormality was 50 and 97.1%; and for aortic regurgitation 100 and 90%, respectively.

We compared the cardiac ejection fraction (EF) and volumes in NSAA patients with normal controls, which showed no statistically significant difference in the LV functions between the two groups.

Late Gadolinium Enhancement

LGE was observed in 10 (27.8%) out of 36 patients. Midmyocardial LGE was common, which was seen in 5 out of 10 patients (►**Table 2**). Epicardial enhancement was seen in four patients. Sub-endocardial enhancement and diffuse patchy enhancement were seen in one patient each.

We analyzed the association between LV ejection fraction (LVEF) and the presence of myocardial LGE and found no significant association ($p = 0.375$). No statistically significant
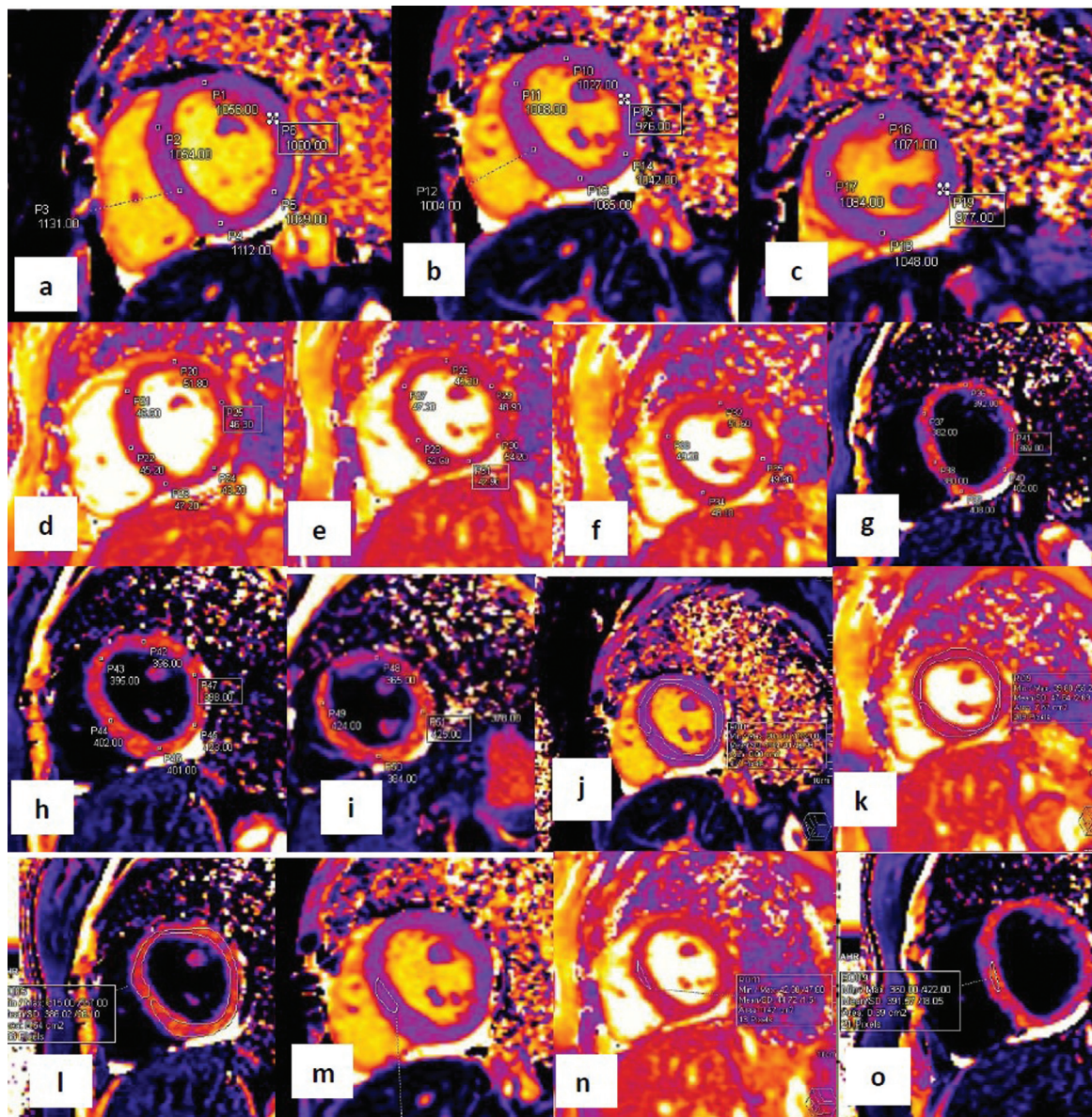

Fig. 1 MRI T1 and T2 mapping value calculations in a 17-year-old female patient with NSAA. The myocardial pre-gadolinium T1 mapping (**a–c**), T2 mapping (**d–f**), and post-gadolinium T1 mapping (**g–i**) mapping values were calculated using the pixel-wise ROI method in all segments in basal, mid, and apical LV levels. The global (**j–l**) and septal (**m–o**) T1 and T2 mapping values were also calculated in the basal, mid, and apical LV levels. T1 mapping values were in the range of 976 to 1131 ms. T2 mapping values were in the range of 42.9 to 51.8 ms. Post-gadolinium T1 mapping values were in the range of 380 to 423 ms.
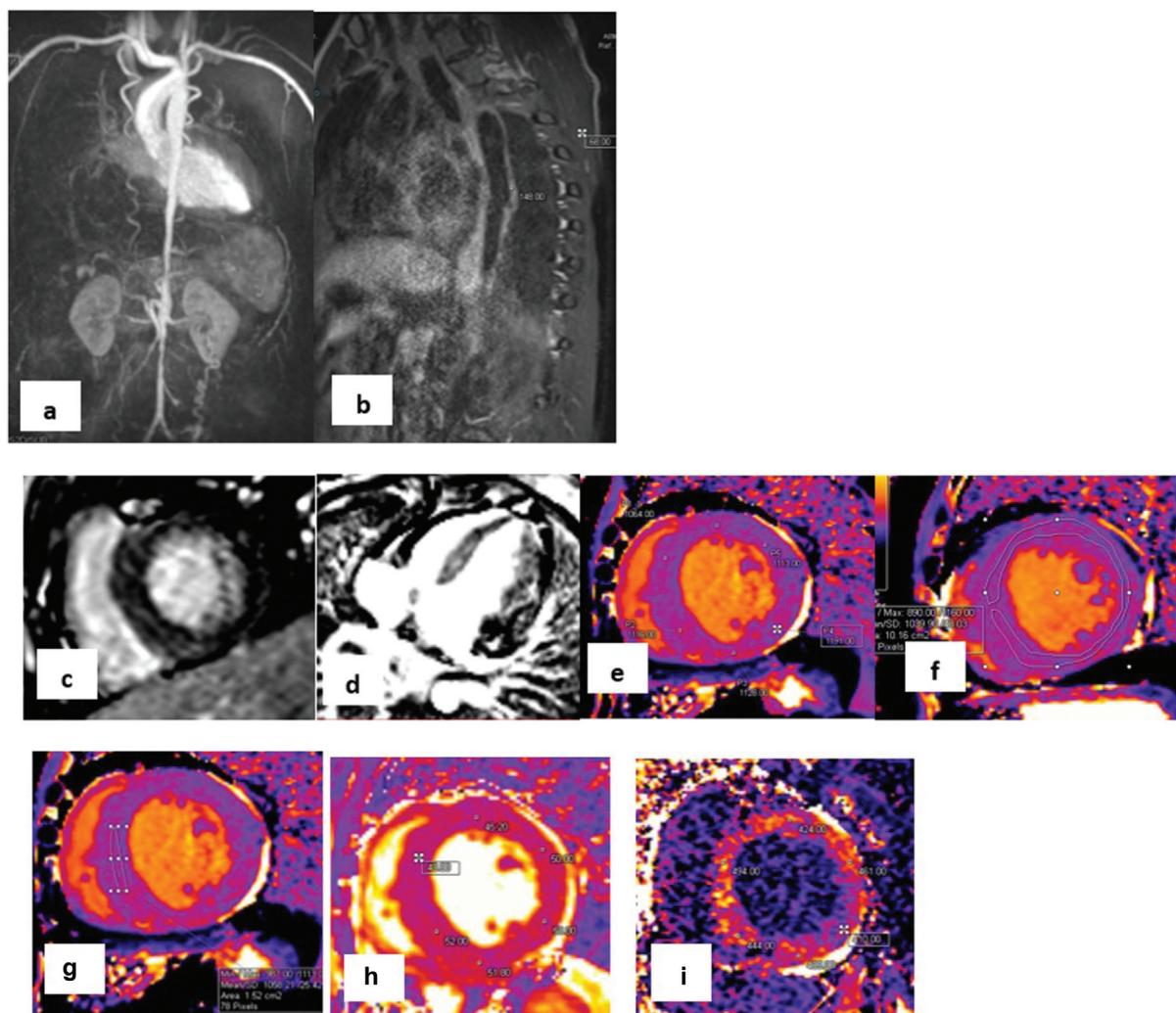
Fig. 2 MRI in a 27-year-old male patient with NSAA. MRA (**a, b**) shows diffuse disease of the thoraco-abdominal aorta with significant stenosis of the supraceliac aorta with wall thickening. There is concentric left ventricular hypertrophy with patchy areas of mid-myocardial LGE (**c, d**). Corresponding areas show increased T1 (1116 ms) and T2 (58 ms) mapping values (**e, f, h, i**). Post gad t1 mapping values are in the range of 410 to 494 ms (**j**).

association of cardiac MRI LGE with ESR ($p = 0.317$) and CRP levels ($p = 0.153$) was found.

T1 Mapping

We analyzed segment-wise T1 mapping values in patients with NSAA (►**Figs. 1–6**). When we compared the NSAA patients ($N = 36$) with the controls ($N = 16$), we observed significantly increased values of T1 mapping values in all segments (except global apical segment) as shown in ►**Table 3**. Global apical segment T1 mapping values were not reliable due to excessive mobility of apical segments.

A total of 42 segments were found to be involved on the basis of LGE and compared with 42 normal segments of healthy controls. The values of post-gadolinium T1 mapping in NSAA patients were significantly higher (1123 ± 42.5 millisec) from pre-gadolinium T1 mapping in control segments (972.2 ± 11.1 millisec). The *p*- value was 0.000.

Pre-gadolinium T1 mapping values in segments with NSAA were compared with the normal segments in healthy

controls. ROC curve analysis was done, which resulted in an area under the curve (AUC) of 0.892. A cut-off value of 1019 milliseconds was obtained for detecting abnormal T1 map and it had the sensitivity and specificity of 83.3% and 81.2%, respectively. We analyzed the T1 map values in LGE-negative NSAA patients using this cut-off value. Increased T1 map values were found in 400 segments out of a total of 576 segments of 36 NSAA patients. Hence, it was found that the myocardium of NSAA patients might show increased T1 map values even if LGE was not seen.

The value of T1 mapping was also calculated after giving gadolinium and it was compared with the pre-gadolinium T1 value. The values of post-gadolinium T1 values were significantly decreased from pre-gadolinium T1 mapping values in all segments.

T2 Mapping

We also analyzed segment-wise T2 mapping values in patients with NSAA (►**Figs. 1,2,3,4,5,6**). When we compared
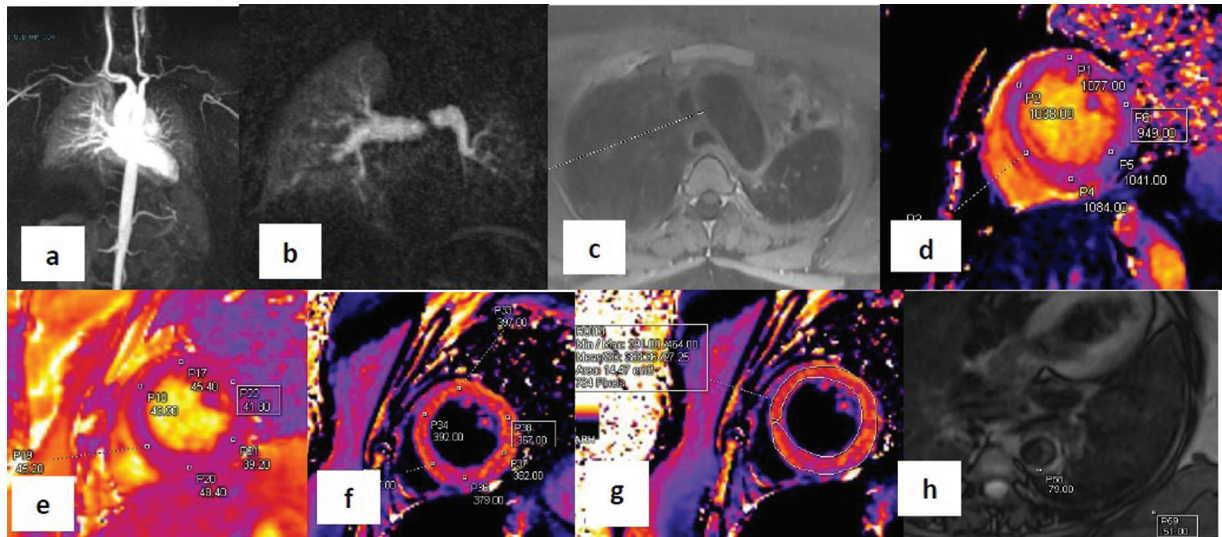
Fig. 3 MRI in a 33-year-old female patient with NSAA. MRA (**a, b**) show long segment occlusion of bilateral subclavian arteries with the involvement of the left upper lobe pulmonary segmental branches causing lung infarcts. Diffuse circumferential wall thickening of the aortic arch (**c**). Cardiac T1 mapping values are in the range of 949 to 1084 ms (**d**). T2 mapping values were in the range of 41.8 to 48.4 ms (**e**). Post gad T1 mapping values were in the range of 362 to 397 ms (**f, g**). The ratio of T2 signal intensity of the vessel wall to the skeletal muscle was 1.54 (**h**).
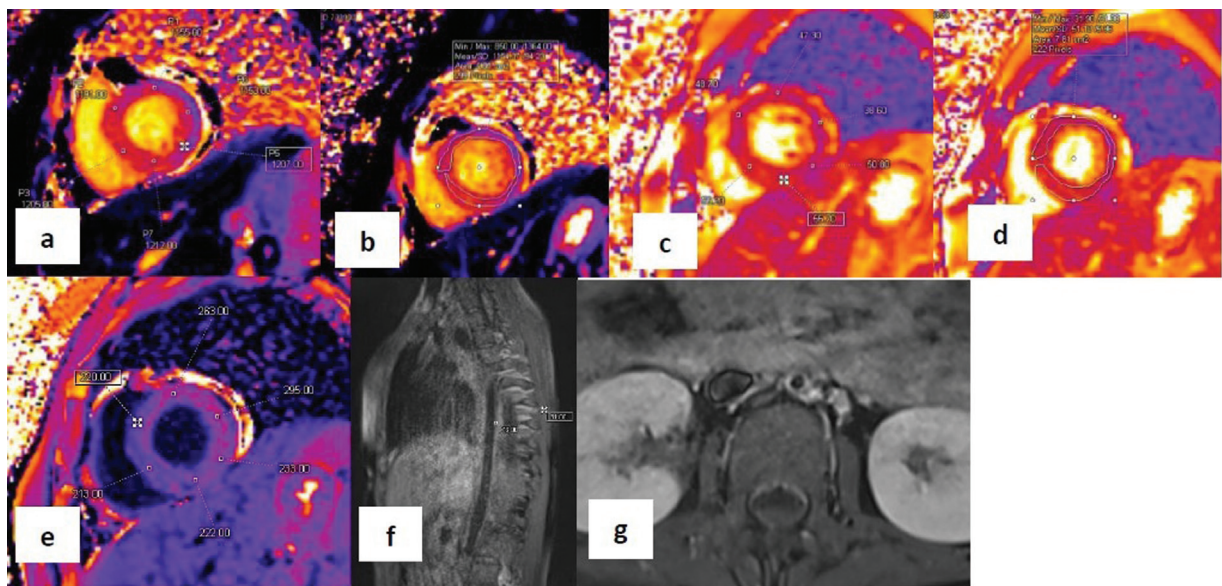

Fig. 4 MRI in a 25-year-old female patient with active NSAA. Diffuse mid-to-epicardial regions in basal to apical LVs show increased native T1 mapping values (**a, b**). T2 mapping values were within the normal range (**c, d**). Decreased post gad T1 mapping values (**e**) seen in involved segments. The patient also had significantly elevated ESR and CRP values. MRA shows abdominal aortic involvement (**f**) with enhancing wall thickening (**g**).

the T2 mapping values in NSAA patients ($N = 36$) with the controls ($N = 16$), we observed no significant relation between the two groups as shown in ►**Table 4**.

Discussion

Cardiovascular MRI provides not only morphological assessment of cardiovascular system but also assesses the myocardial involvement in patients with NSAA. Myocardial inflammation has relatively had worse prognosis in the outcome of patients with NSAA. The gold standard for the detection of myocardial inflammation or fibrosis is endomyocardial biopsy, which is invasive in nature and has its own limitations in the form of procedure-related complications and sampling errors. Cardiac MR imaging is the investigation of choice for the non-invasive identification of myocardial fibrosis or inflammation. Late gadolinium-enhanced MR imaging technique has been traditionally used to evaluate fibrosis with a high degree of confidence with minimal scar depicted is ~1 cm^3.

Latest MRI sequences in the form of T1 and T2 mapping have additionally contributed to the estimation of
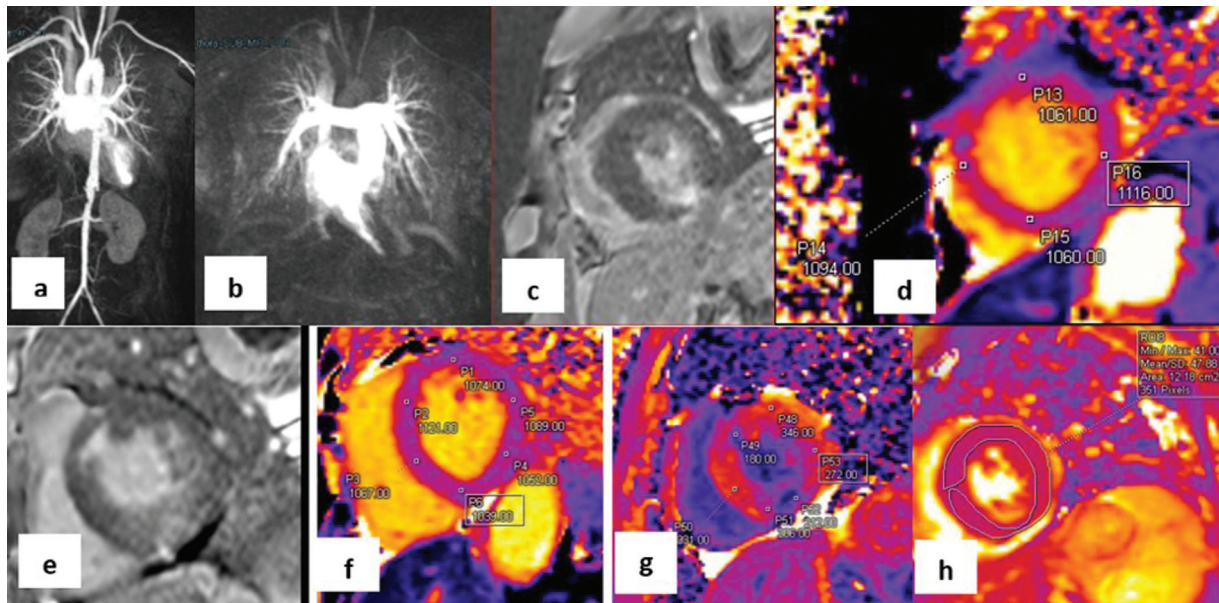
Fig. 5 MRI in a 25-year-old female patient with NSAA and myocardial involvement. MRA shows diffuse disease with intimal irregularity and ulcerations in the juxta-diaphragmatic aorta (**a**). Pulmonary arteries were normal (**b**). Post-contrast MRI phase-sensitive inversion recovery images show patchy mid myocardial LGE (**c, e**), corresponding areas (**d, f–h**) show increased T1 mapping values (1121 ms) and show decreased post gad T1 mapping values (180 ms and 212 ms), as compared with uninvolved segments (306–346 ms).
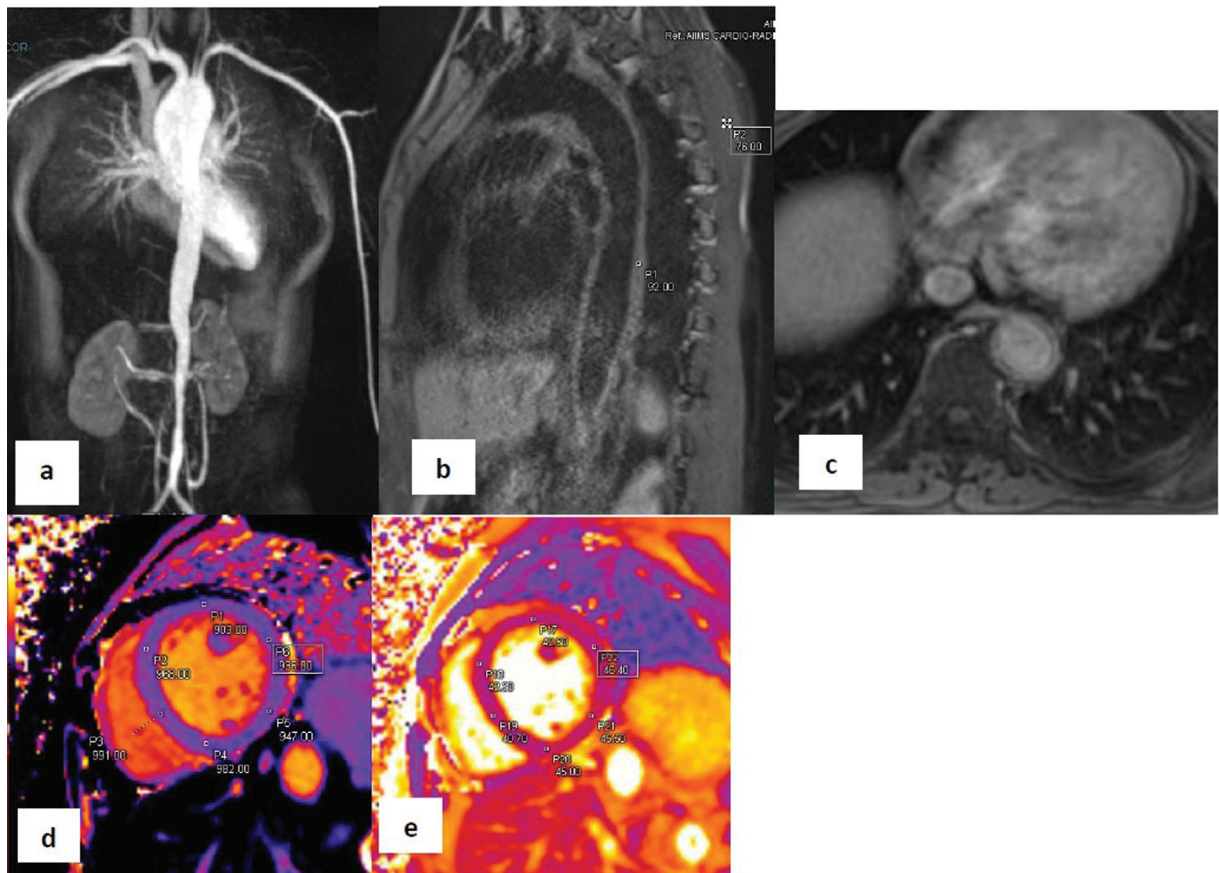

Fig. 6 MRI in a 17-year-old female patient with active NSAA and vessel wall enhancement. MRA shows diffuse disease of the thoracoabdominal aorta with enhancing circumferential wall thickening (**a–c**). Diffuse stenosis of the supra to infrarenal abdominal aorta. Myocardial T1 and T2 mapping values were in the range of 903 to 991 ms and 40.7 to 46.4 ms, respectively (**d, e**).

Table 3 Native (pre-gadolinium) T1 mapping values in control and NSAA cases

Cardiac segments	Control T1 map (N = 16) Mean (± SD) in millisec	Cases T1 map (N = 36) Mean (± SD) in millisec	p-Value
Segment 1	981.1 ± 51.8	1030.6 ± 55.1	0.004
Segment 2	994.3 ± 54.7	1063.6 ± 49.7	0.000
Segment 3	976.9 ± 38.5	1051.5 ± 66.0	0.000
Segment 4	973.6 ± 45.6	1042.1 ± 58.7	0.0000
Segment 5	976.6 ± 57.1	1051.7 ± 63.1	0.0000
Segment 6	965.8 ± 50.4	1038.8 ± 69.7	0.0000
Segment 7	975.6 ± 46.8	1014.94 ± 52.0	0.011
Segment 8	974.9 ± 56.9	1029.6 ± 57.4	0.003
Segment 9	975.1 ± 43.8	1059.8 ± 65.7	0.0000
Segment 10	974.7 ± 57.9	1057.8 ± 63.1	0.0000
Segment 11	958.3 ± 61.2	1037.3 ± 62.5	0.0000
Segment 12	974.8 ± 56.1	1033.6 ± 57.0	0.002
Segment 13	971.8 ± 42.9	1030.1 ± 45.7	0.000
Segment 14	966.2 ± 56.8	1037.6 ± 46.3	0.0000
Segment 15	982.2 ± 66.4	1045.3 ± 59.9	0.003
Segment 16	953.1 ± 54.3	1046.8 ± 58.7	0.0000
Global basal	986.9 ± 39.4	1053.8 ± 44.2	0.0000
Global mid	987.4 ± 46.9	1049.68 ± 41.2	0.0000
Global Apical	989.2 ± 50.8	1027.67 ± 161.	0.202
Septum basal	986.1 ± 48.8	1044. ± 53.4	0.001
Septum mid	977.9 ± 60.0	1050.9 ± 53.3	0.0000
Septum apical	977.9 ± 60.0	1042.5 ± 45.8	0.001

myocardial fibrosis or edema. T1 mapping has been advocated to identify the reversible stage of interstitial fibrosis, which is not identified by late gadolinium-enhanced MR imaging sequences. Recent studies have illustrated the additive value of T1 and T2 mapping sequences for the identification of myocardial fibrosis and edema in vasculitis patients.[5,8,9]

We had included only those patients with NSAA having suspected cardiac involvement so that the select subset of patients was included in the assessment of the cardiac MRI parameters. The mean age of our patient cohort was 27.1 years, which was similar to other studies of NSAA, suggesting that our patient cohort was within this range. The disease was found more commonly in females as compared with males in our study (F: M = 2.6:1) as endorsed by other studies in the literature.[1–4]

NSAA patients demonstrated frequent symptoms of difference in pulses in the limbs (83.3%), hypertension (69.4%), and dyspnea (61.7%). Serum ESR and CRP levels were elevated in 17 (47.2%) and 16 (44.4%) patients each. There was no statistically significant association between serum ESR/ CRP levels and LGE. Increased ESR or CRP values could not be correlated with disease activity.

Out of 36 NSAA patients in our study, 8 patients had dilated chambers, 5 patients had left ventricular hypertro-

phy, and 2 patients had RWMA on cardiac MRI. Fifteen patients demonstrated LV systolic dysfunction and 7 patients showed RV systolic dysfunction. Though cardiac MRI better detected aortic regurgitation and RWMA, the main evident advantage of MRI is the demonstration of LGE. Cardiac MRI demonstrated the mid-to-epicardial LGE in 5 out of the 10 patients; 4 patients had epicardial enhancement, and 1 patient had diffuse patchy enhancement. Most commonly LGE is seen in the mid-to-epicardial region in cardiac involvement suggesting myocarditis.

Myocardial tissue characterization can be done using T1 mapping. As cardiac involvement in NSAA is often not uniform and may involve certain segments, we analyzed segment-wise T1 and T2 mapping values for the estimation of cardiac involvement. However, assessing global T1 mapping values may be better for the assessment of diffuse myocardial involvement. Sometimes, myocardial fibrosis may not show LGE but show increased T1 map values as found in some of our patients in our study.

We had compared the T1 mapping values in the involved segments in patients with cardiac involvement in NSAA with the values from normal segments in healthy controls. The cut-off of 1019 milliseconds of myocardial T1 map values for the identification of abnormal segments in our study had the

Table 4 Segment-wise comparison of T2 mapping in cases and controls

Cardiac segments	Control T2 map (N = 16) Mean (± SD) in millisec	Case T2 map (N = 36) Mean(± SD) in millisec	*p*-Value
Segment 1	45.2 ± 2.7	45.6 ± 3.5	0.687
Segment 2	47.8 ± 4.7	46.2 ± 4.2	0.245
Segment 3	45.8 ± 3.7	46.3 ± 4.2	0.737
Segment 4	45.2 ± 3.5	47.1 ± 4.3	0.125
Segment 5	46.5 ± 4.1	47.9 ± 5.1	0.281
Segment 6	44.9 ± 2.4	46.3 ± 5.4	0.227
Segment 7	45.2 ± 3.1	47.6 ± 4.5	0.028
Segment 8	46.7 ± 3.4	46.9 ± 4.2	0.888
Segment 9	45.5 ± 3.8	47.7 ± 4.7	0.079
Segment 10	46.3 ± 3.0	46.8 ± 3.5	0.597
Segment 11	44.8 ± 4.3	47.1 ± 5.3	0.105
Segment 12	45.9 ± 4.4	46.5 ± 5.8	0.657
Segment 13	45.2 ± 3.1	46.7 ± 3.9	0.149
Segment 14	46.8 ± 4.3	46.8 ± 4.3	0.771
Segment 15	44.4 ± 2.1	45.9 ± 3.6	0.073
Segment 16	44.1 ± 3.5	46.8 ± 5.3	0.034
Global basal	47.9 ± 2.2	48.2 ± 3.5	0.727
Global mid	48.0 ± 2.7	48.1 ± 3.4	0.875
Global apical	48.1 ± 2.8	48.5 ± 3.1	0.594
Septum basal	45.9 ± 2.6	45.8 ± 4.1	0.925
Septum mid	46.2 ± 2.7	46.3 ± 3.7	0.922
Septum apical	46.5 ± 2.9	46.4 ± 3.9	0.938

sensitivity and specificity of 83.3 and 81.2%, respectively. Abnormal T1 mapping values were found even when no LGE was noted in the involved segments. Hence, we propose that T1 mapping values may have additive values to LGE in the diagnosis of cardiac involvement in NSAA. This cut-off value may also depend on various factors such as the MRI technique used, cardiac cycle, and strength of the magnetic field and hence cannot be generalized.

A few of the previous studies have analyzed the role of MRI in patients with NSAA. Keenan et al found that cardiovascular MRI demonstrated vessel wall thickening and assessed the ventricular function and myocardial scarring.[10] Eshet et al observed no significant association between clinical activity and MRI signs of activity and stated that MRI has limited role in long-term follow-up when reactivation of disease is suspected.[11] John et al concluded that MRI signs of disease activity are associated with ITAS 2010, CRP, and ESR levels.[12] Jiang et al found that active NSAA had more stenosis in the left subclavian artery than the inactive disease. MRI scoring system of arterial stenosis, wall thickness, and contrast enhancement could help in disease activity.[13] Papa et al concluded that the MR contrast agent, i.e., gadofosveset trisodium could help in differentiating active from the inactive disease.[14] However, no previous studies have analyzed the additive role of cardiac T1

and T2 mapping in patients of NSAA. To the best of our understanding, this is the first study in the literature that assessed the role of cardiac MRI LGE, T1 and T2 mapping in NSAA patients.

Greulich et al found that ANCA (antineutrophil cytoplasmic antibody)-associated vasculitides patients showed elevated myocardial T1, T2, and ECV values.[9] This advantage of elevated native T1 mapping values in the detection of involved segments could be utilized in the interpretation of non-contrast MRI scan, whenever contrast could not be given due to deranged renal parameters. We found that post-gadolinium T1 mapping values were lesser compared with the pre-gadolinium T1 mapping in the involved myocardial segments of NSAA. However, there was no significant changes in the pre- and post-gadolinium T1 mapping values in control subjects. As T2 mapping can be used to quantify myocardial edema or active inflammation, any inflammation or active disease may show increased T2 map values. However, we could not find any significant difference in T2 mapping values in NSAA and control subjects. Our study highlights the additive value of T1 mapping in identifying abnormal myocardium in patients with NSAA.

There were certain limitations of the study. The sample size of the study was small as we followed strict inclusion

criteria. A strong recommendation could not be made due to lesser number of patients ($n = 36$). There were unavoidable time lags in obtaining different investigations, which could have resulted in different findings varying with disease activity. Another major limitation in our study was the lack of histopathological correlation for the assessment of myocardial involvement in NSAA. LGE and elevated T1 values may well represent fibrosis due to constant afterload in hypertensive patients or myocarditis due to NSAA. We could not definitely attribute LGE or elevated T1 mapping values to myocardial involvement due to NSAA.

We accept that the study was conducted in a single center with a small sample patient size and may not perform equally at other institutions with different practices. However, our study can act as a prototype for further research in the assessment of cardiac involvement in NSAA patients and may impact the management. A large randomized controlled study is needed to confirm our findings.

Conclusion

Cardiac MRI using LGE, cardiac T1 and T2 mapping provide the additive role for the evaluation of myocardial involvement in patients with NSAA. Myocardial involvement was seen in 27.7% of patients with evidence of LGE. The raised ESR and CRP values showed no significant association with LGE. Myocardial LGE may represent myocarditis with active disease. Quantitative myocardial tissue characterization utilizing T1 and T2 mapping values help in the identification of cardiac involvement in NSAA. T1 mapping may identify those myocardial areas, which do not demonstrate LGE.

Conflict of Interests
None declared.

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