Egyptian Journal of Radiology and Nuclear Medicine

RESEARCH Open Access

MRI evaluation by T1 mapping of the post-myocardial infarction left ventricular thrombus

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Abstract

Background Left ventricular thrombus is a post-myocardial infarct complication. It is composed of a fibrinous composition that evolves over time, being assessable by MRI.

Objectives The objective of this study is to evaluate the post-myocardial infarction left ventricular thrombus on cardiac MRI by measuring its T1 mapping value, thus to determine the age of thrombus.

Methods This observational retrospective study was performed on all patients scheduled for 3.0 Tesla cardiac MRI post-myocardial infarction on our institution from January 2015 to December 2022. Thirty-five patients with a left ventricular thrombus that may be measurable on T1 mapping sequence were included. They were separated in two groups based on the duration between the infarct and the MRI—less than three months: group A and more than three months: group B. T1 mapping value was measured for all thrombi.

Results T1 of thrombi was 1098 ± 61 ms in group A and 1316 ± 75 ms in group B, $p < 10^{-4}$. T1 of the myocardium was 1224 ± 73 ms in group A and 1254 ± 48 ms in group B, $p = 0.139$. T1 of the blood pool was 1934 ± 137 ms in group A and 2008 ± 124 ms in group B, $p = 0.135$.

Conclusions Recent thrombi had shorter mapping T1 than old thrombi.

Keywords Cardiac thrombus, Magnetic resonance imaging, Cardiac imaging sequence

Background

Left ventricular thrombus remains a key component of the burden of myocardial infarction [1], because of the occurrence of embolic events leading to ischemic strokes. The most considerable risk occurs during the first weeks after the infarct. However, the potential of cerebral emboli persists in the large population of patients with chronic left ventricular dysfunction. As follows, an accurate detection and analysis affects clinical outcomes and therapeutic management as thrombus provides a rationale for anticoagulation. Moreover, thrombus retains a dynamic nature of development and resolution [2, 3] which requires an active monitoring of the anticoagulation to balance risks of embolization versus bleeding [4].

Comparative studies have demonstrated that CMR is currently the optimal imaging modality for diagnosis of left ventricular thrombus [5–8]. It maintains a sensitivity of 82–88% and specificity approaching 100% compared to surgical and/or pathological confirmation [5]. This imaging modality is based on intrinsic and morphological characteristics rather than anatomical appearance alone. These characteristics conditioned by principal components of thrombi may also add value to differentiate between recent and old thrombi. It may be important because the risk of systemic emboli is highest during the

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first three months following acute myocardial infarction [9].

Histologically, a thrombus represents a layered conglomeration of fibrin and platelets in variable amounts, often containing degenerated RBC and WBC. Recent thrombi are characterized by RBC- and platelet-rich regions. Older thrombi predominantly contain RBC and fibrin. Taken simultaneously, these findings indicate that thrombus composition changes and affects T1 longitudinal relaxation time. T1 mapping sequences are instantly available that can accurately quantify T1 for thrombus. Studies are investigating T1 to see if differences can be managed to help differentiate between recent and old thrombus [10]. This may be potential to estimate the likely course of medical condition.

In this study, we measured the T1 mapping of left ventricular thrombus post-myocardial infarction. The prime objective was to evaluate if T1 relaxometry might be useful for the staging of left ventricular thrombus and allow a good estimation of the prognosis.

Methods

This observational retrospective study of patients scheduled for routine CMR was performed in accordance with the ethics rules of our institution.

MR imaging

MRI examination was performed on a 3.0 T imaging system (Siemens MAGNETOM Vida) equipped with a dedicated 18 channels cardiac coil. The CMR imaging protocol included was standardized for cardiac ischemic pathologies, with the following sequences: T2 TRUFI single shot in axial plane, SSFP cine sequences acquired in the short axis, post-injection (using CLARISCAN 0.5 mmol/ml, at a dose of 0.4 ml/kg, GE Healthcare, manufactured in Norway) sequences including perfusion sequences in short axis, 4-chamber and 3-chamber axis (early after bolus injection), SSFP cine sequence acquired in the 4-chamber axis and 3-chamber axis as an equivalent of early and delayed gadolinium enhancement up to 10 min after bolus injection (inversion-recovery sequences and PSIR sequences) in the short axis, 3-chamber axis and 4-chamber axis. Regarding the T1 mapping sequences, the native T1 MOLLI sequence was obtained by the Siemens T1 mapping package before injection of Gadolinium. We also proceeded to post-contrast T1 Mapping sequences 15 min after injection. T1 mapping sequences were acquired in the short axis and 4-chamber axis. The reported values of T1 relaxation times on T1 mapping pre-contrast sequences were measured in regions of interest in left ventricular thrombus, non-ischemic myocardium and left ventricular blood pool. A drawn region of interest of the thrombus was obtained on the plane where the thrombus was seen or bigger. All other measurements were performed on two imaging planes and then averaged. The image analysis and post-treatment were performed using syngo.via (Client 5.1, Siemens, Germany).

Population study

From January 2015 to December 2022, all patients referred to our department for a 3.0 T CMR were screened to participate in the study. Patients for which a left ventricular thrombus was seen at the time of the examination were enrolled. Inclusion criteria were patients older than 18 years old, history of acute coronary syndrome. Exclusion criteria were the classical contraindications to MRI, agitation, non-visualization of the left ventricular thrombus on T1 mapping images obtained, history of coronary bypass, coronary stent, heart valve disease or cardiac rhythm disorders justifying anticoagulation, overload diseases with heart damage, and pregnant women. Thirty-five patients were included. The diagnosis of thrombus was based on the clinical context, on the MRI aspect of the mass and on the regression during anticoagulant therapy. Based on the clinical context, thrombi were classified according to the time elapsed between the coronary syndrome and the CMR into recent (less than 3 months) and old (equal or more than 3 months).

Statistical analysis

Statistical analyses were performed using Stata 14.2. Quantitative continuous variables were expressed as mean ± standard deviation. A nonparametric test of Mann and Whitney (continuous variables) was performed to determine differences between the two groups. P values < 0.05 were considered statistically significant.

Results

Patient characteristics

Thirty-five patients with left ventricular thrombus seen on T1 mapping images were included (9 women, 26 men, mean age = 54.9 ± 13.4 years). The mean delay between acute coronary syndrome and the CMR was 3.3 ± 3.5 months. Twenty-three patients had a recent thrombus (5 women, 18 men, mean age = 53.7 ± 14.2 years) while 12 patients had an old one (4 women, 8 men, mean age = 57.2 ± 12.0 years). The mean delay between acute coronary syndrome and the CMR was 1.30 ± 0.47 months in the recent thrombus group, and 7.08 ± 3.65 months in the old thrombus group (Table 1). All thrombi were located in the left ventricle and all the patients had a context of ischemic heart

Table 1 Patients features (age, sex, duration between inferct and MRI)

Patients	Recent thrombus	Old thrombus	Total
n	23	12	35
Sex ratio (F:M)	5:18	4:8	9:26
Mean age (years)	53.7 ± 14.2	57.2 ± 12.0	54.9 ± 13.4
Mean delay (months)	1.30 ± 0.47	7.08 ± 3.65	3.3 ± 3.5

disease. Also, we observed 4 embolic events in the population. Three of them (1 renal infarction, 1 acute lower limb ischemia, 1 stroke) occurred in the recent thrombus group, and 1 (spleno-renal infarction) occurred in the old thrombus group.

CMR findings

The T1 relaxation time of thrombi in the whole population was 1173 ± 123.67 ms vs 1234 ± 66.34 ms for myocardium and 1959 ± 135.73 ms for blood pool. Recent thrombi had a T1 relaxation time significantly shorter than old thrombi (1098.04 ± 61.27 ms vs 1316.25 ± 75.05 ms, $p < 10^{-4}$). T1 relaxation time of the myocardium or blood pool was not significantly different between groups (Table 2).

Discussion

It has been speculated that the left ventricular thrombus plays a positive role in the acutely infarcted myocardium, by offering mechanical support to the infarcted myocardium and therefore protecting against left ventricular rupture [9]. Still, thromboembolic events caused by thrombi can be devastating, and the diagnosis remains important for the prevention of embolic events as well as establishing the need for anticoagulation.

The risk of thromboembolism is most closely related to thrombus mobility and protrusion as described on imaging [5]. It is also highest during the first three months following acute myocardial infarction [9]. Therefore, a distinction between recent and old thrombi remains crucial.

Three histologic characteristics are essential to analyze the MR aspect of the left ventricular thrombus: 1—a thrombus is heterogeneous in composition; 2—thrombus compositions change over time; and 3—fibrin is a major contributor to thrombus.

A thrombus is not a clot [11]. A clot is formed in a static blood, and it contains RBCs, WBCs, and platelets dispersed randomly inside a network of fibrin. A thrombus is developed in the circulating blood and is composed of a head and a queue [11]. The thrombus head consists of a mass of aggregated platelets with many WBCs, few RBCs, and only small quantity of fibrin. The thrombus queue structure, consisted essentially of fibrin enclosing the RBCs, resemble to that of a clot. This turns evident

Table 2 T1 mapping of thrombus, myocardium, blood

n	Recent thrombus	Old thrombus	*P* value
T1 thrombus	1098.04 ± 61.27	1316.25 ± 75.05	$< 10^{-4}$
T1 myocardium	1223.91 ± 73.11	1253.75 ± 47.68	0.139
T1 blood pool	1933.70 ± 137.17	2008.33 ± 123.77	0.135

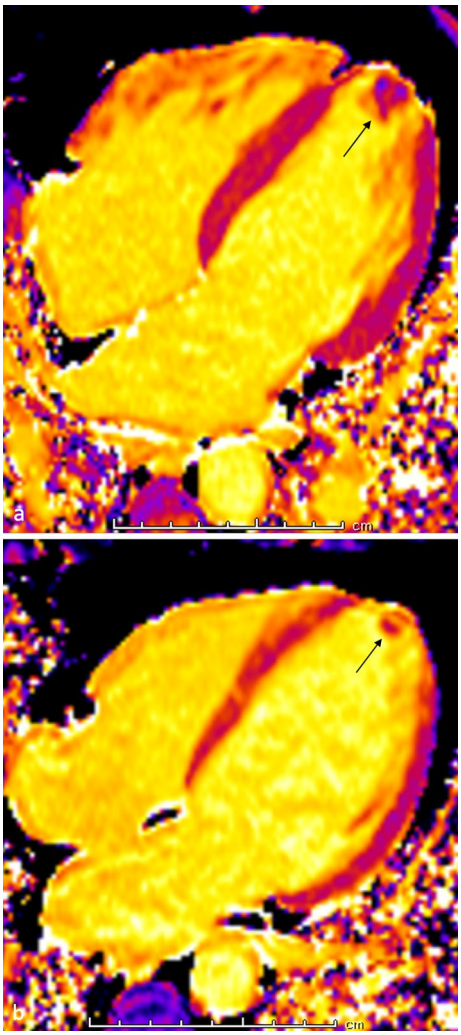

Fig. 1 Heterogeneity of LV thrombus—T1 mapping sequence. **a** Old heterogeneous apical LV thrombus (arrow) in 66-year-old male patient with STEMI on left coronary artery 4 months ago. T1 value measured at 1370 ms. **b** Recent heterogeneous apical LV thrombus (arrow) in 48-year-old male patient with STEMI on left coronary artery 1 month ago. T1 value measured at 1075 ms

that thrombi are heterogeneous in composition and consequently in appearance (Fig. 1).

Thrombi surfaces contain more fibrin and platelets, and fewer RBCs, than inner parts [12]. Inner parts of thrombi are rich in polyhedrocytes [12–14]. Polyhedrocytes are compressed RBCs together into a polyhedral structure [12] by forces generated by platelets pulling on fibrin fibers that lead to a clot contraction. Over time, thrombi showed more compact fibrin network [15] with an increased polyhedrocyte formation. Taken together, these findings indicate that thrombus compositions change over time. Recent thrombi are characterized by RBC- and platelet-rich regions. Older thrombi contain mainly RBCs and fibrin. But overall, thrombi obtained from myocardial infarction are composed of mainly fibrin [3].

Fibrin is a major contributor to thrombi. It polymerizes into a network of fibers [16], stabilizing blood clots. It aligns into continuous 2D films forming a protective layer across the surface of clots [17]. Combined with the findings that fibrin content varies in thrombi, these data indicate an important role for fibrin in thrombus characteristics, influencing stability, embolization, and breakdown.

T1 mapping can detect a variety of myocardial pathologies, where it shows increased values [7, 18, 19], among which the fibrous tissue [20]. For example, a papillary fibroelastoma, composed mainly of avascular fibroelastic tissue, shows a very long T1 relaxometry [10] (Fig. 2).

T1 signal characteristics of a thrombus are largely described as related to the evolution of hemoglobin properties. Increased methemoglobin content in the recent thrombus results in shortening of T1. With time, an old thrombus is depleted of water and cell debris containing methemoglobin are replaced by fibrous tissue, responsible for a longer T1. Thus, MR imaging of the thrombus is also based on fibrin composition.

Investigations were made looking into comparisons of the T1 time of myocardium with that of thrombus to see if differences can be used to help distinguish them. Because thrombi are avascular, T1 times are long and similar to those of unperfused fibrotic myocardium [21]. Other investigations were made looking into comparisons of the T1 time of the thrombi to differentiate them by age [10, 22, 23]. T1 values showed a significant difference between recent (shorter T1) (Fig. 3) and old (longer T1) (Fig. 4) thrombi. Our results seem to be consistent with previous reports from the literature in terms of

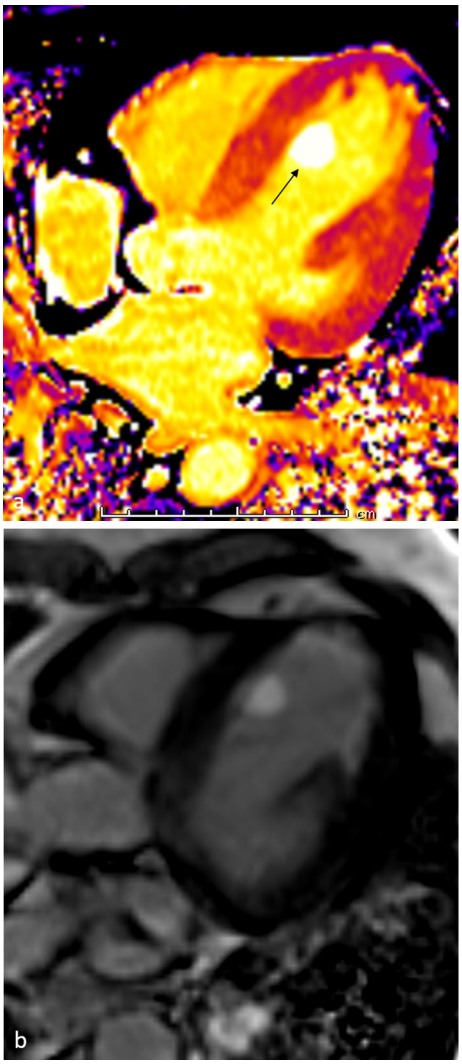

Fig. 2 Cardiac fibroelastoma. Shining intracavity left ventricular fibroelastoma (arrow in **a**) in 76-year-old male patient due to its long T1 mapping. T1 value measured at 2468 ms. Note the enhancement (**b**) oppositely to the thrombus

signal intensity on T1-weighted sequences. Indeed, it is known that MRI thrombi properties depend on their age and content [24]. Most of the literature reviews [25] of cardiac thrombi classify them according to their age as recent or old. Thus, we may be able to differentiate between recent and old thrombi. T1 mapping may allow us to highlight the difference between recent and old thrombi, as recent thrombi had a significantly shorter T1.

In addition to finding a significant difference in T1 values between recent and old thrombus, the values we

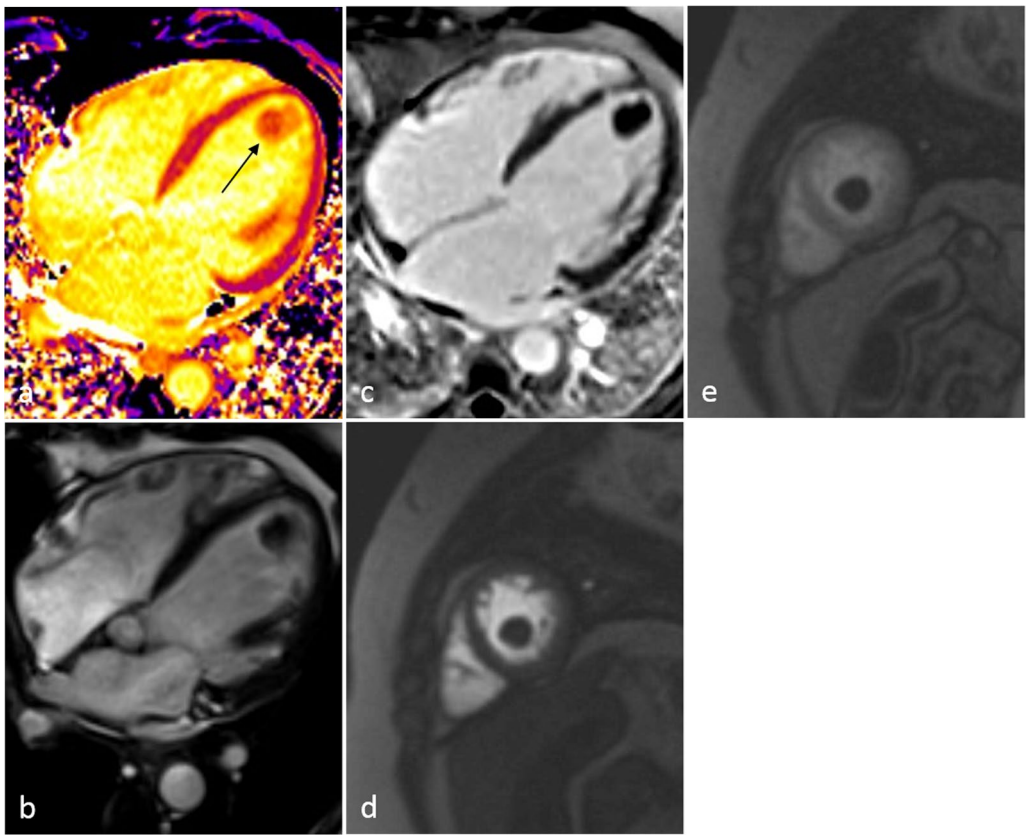

Fig. 3 Recent LV thrombus. Low T1 mapping of recent LV thrombus in T1 mapping (arrow in **a**), cine (**b**), psir (**c**), and dynamic enhancement (early (**d**) and late (**e**) phase) sequences in a 45-year-old female patient with STEMI on left coronary artery 1 month ago. T1 value measured at 1020 ms

found agree with the T1 threshold set as 1150 ± 75 ms on 3.0 Tesla MRI [25].

Despite the promising results, the current study had some limitations, among which its retrospective study design, its single-center nature, its limited number of patients. Besides, there are limits to the treatment of the thrombus using CMR T1 mapping. Access to CMR T1 mapping sequences is still limited. Moreover, the measurement of T1 mapping may be difficult for small thrombi (Fig. 5). On top of that, T1 mapping methods are company specific with variable accuracy and precision of relaxation times values between sequences. Therefore, these data should be interpreted with caution as they might not be applicable to all systems and sequences. Nevertheless, our findings could be verified by a more prospective analysis with a multicenter study.

Conclusions

This small observational study showed that T1 mapping CMR sequence could be appropriate to represent a new approach for the age and the maturity of left ventricular thrombus, and be valuable for the management post-myocardial infarction. Despite the small size of the present study, our results could provide complementary information for the recommendations of imaging control and anticoagulation therapy.

Clinical implications

Using T1 mapping sequence represents a new approach for the age and the maturity of left ventricular thrombus. It provides complementary information for the recommendations of imaging control and anticoagulation therapy.

Our results may encourage cardiac imaging specialists to use this sequence to specify the age of the thrombus.

This age criterion is not currently taken into account for the management of left ventricular thrombus, but it could be interesting for clinicians to develop a personalized management of patients with recent or old left ventricular thrombus.

For example, an old adherent thrombus may require a longer duration of anticoagulation and a more sporadic radiological monitoring. Conversely, a recent thrombus may warrant a shorter duration of anticoagulation and a more frequent radiological monitoring.

This remains to be clarified by prospective studies.

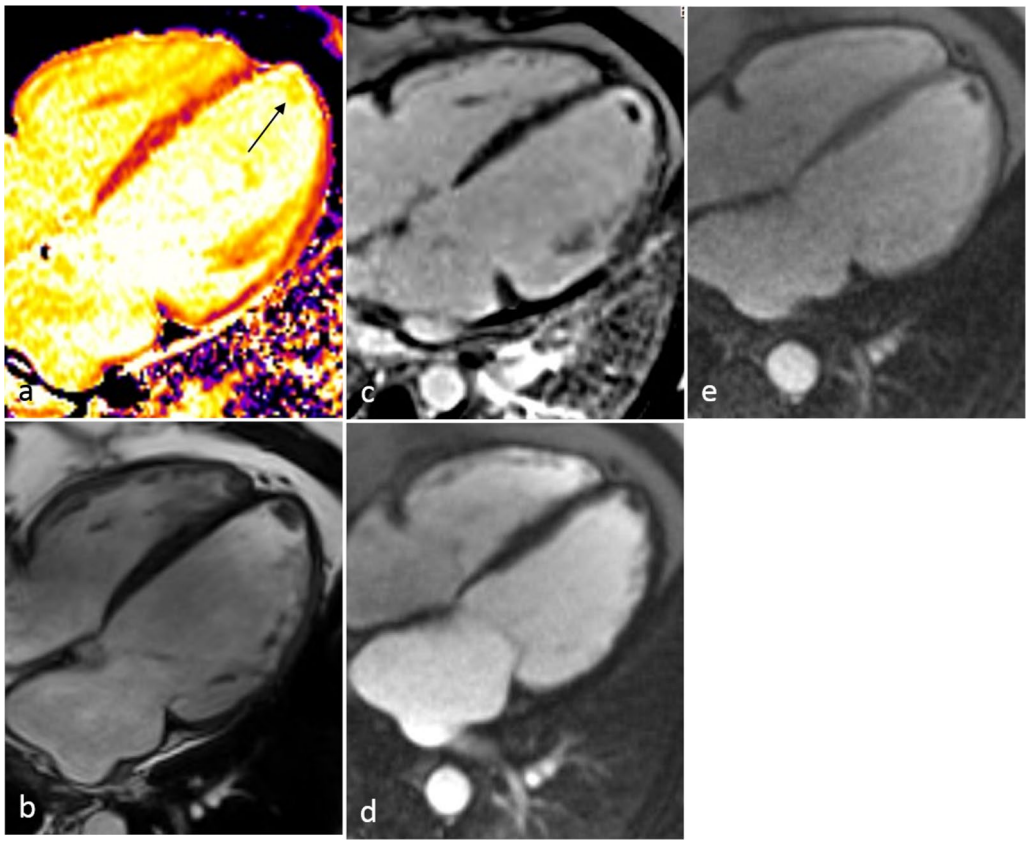

Fig. 4 Old LV thrombus. High T1 mapping of old LV thrombus in T1 mapping (arrow in **a**), cine (**b**), psir (**c**), and dynamic enhancement (early (**d**) and late (**e**) phase) sequences in a 48-year-old female patient with STEMI on left coronary artery 4 months ago. T1 value measured at 1250 ms

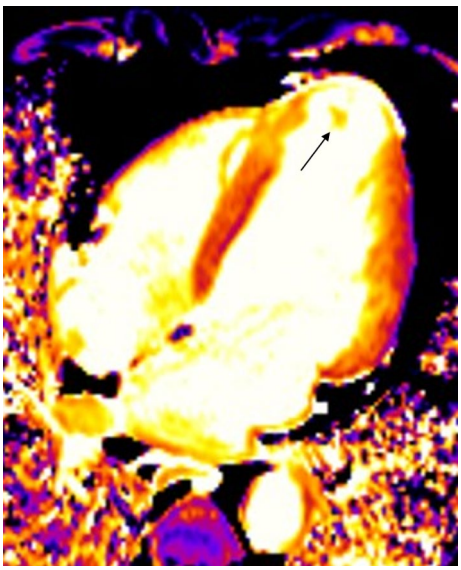

Fig. 5 Size limitation of T1 mapping measurement of LV thrombus. Old laminar apical small LV thrombus (arrow) hardly recognized on T1 mapping sequence in a 72-year-old male patient with STEMI on left coronary artery 6 months ago. Central illustration: Thrombus T1 mapping leads to thrombus age. T1 mapping value of LV thrombus increases with age as its fibrous components grow in

Abbreviations

CMR	Cardiac magnetic resonance
MOLLI	Modified Look-Locker inversion recovery
MRI	Magnetic resonance imaging
PSIR	Phase-sensitive inversion recovery
RBC	Red blood cells
SSFP	Steady-state free precession
TRUFI	True fast imaging with steady-state free precession
WBC	White blood cells

Acknowledgements
Not applicable.

Author contributions
SAK wrote the entire manuscript. SW made corrections. LB made corrections. LB prepared the figures and helped in collecting data. TL collected data and participated in writing the entire manuscript.

Funding
Not applicable.

Availability of data and materials
All data are available in the archiving systems "PACS" and "Log On" of our institute.

Declarations

Ethics approval and consent to participate
This observational retrospective study of patients scheduled for routine CMR was performed in accordance with the ethics rules of our institution.

Consent for publication
Not applicable.

Competing interests
No relationships with industry and no competing interests.

Received: 14 September 2023 Accepted: 28 November 2023
Published online: 04 December 2023

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