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RESEARCH

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Reduction of myocardial lipid content assessed by H1 magnetic resonance spectroscopy in dyslipidemic patients after statins

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Abstract

Background Dyslipidemia is one of the main modifiable risk factors for cardiovascular diseases, which accounts for one third of total deaths worldwide. Statin is considered the cornerstone therapy for treating dyslipidemic patients. H1 Cardiac magnetic resonance spectroscopy (MRS) is a special non-invasive, non-irradiating method for assessing myocardial lipid content in vivo in both health and disease.

Aim To compare dyslipidemic patients and healthy individuals, and to detect the efficacy of statin on the myocardial lipid content in dyslipidemic patients to detect if there will be changes 6 months after starting statin therapy.

Methods Laboratory lipid profile and myocardial lipid content had been measured by H1 MRS in thirty dyslipidemic patients and fifteen healthy matched age and sex individuals as a control group, then dyslipidemic patients were followed up 6 months after statin therapy at Cardiovascular Medicine and Radiology departments; Mansoura University Hospitals, Dakahlia Governorate, Egypt, during the period from January 2020 to October 2022.

Results A total of thirty dyslipidemic patients were screened for lipid profile, myocardial lipid content by H1 MRS; 56.67% were male, with a mean age of 49 ± 9.19 years, and compared with fifteen healthy matched age and sex individuals as a control group. Laboratory lipid profile, and triglyceride lipid concentration by MRS were significantly higher in dyslipidemic group before initiating statin therapy compared to control group (p value, 0.001, 0.019 respectively). Median LDL levels were 161.10 ± 30.28 mg/dl before the start of statin therapy and were 114.27 ± 48.33 mg/dl after statin therapy ($p < 0.001$). There was a statistically significant reduction in triglyceride lipid concentration in dyslipidemic patients after 6 months of statin therapy: from 0.011 (0.001–0.55 (mmol/l), to 0.0025 (0.001–0.04 mmol/l) with a p value < 0.001.

Conclusions Increased myocardial lipid content as measured by magnetic resonance spectroscopy was demonstrated in dyslipidemic patients in our study that decreased after 6 months of statin therapy.

Keywords MRS, Spectroscopy, Dyslipidemia, Statin

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Background

Dyslipidemia is one of the main modifiable risk factors for cardiovascular diseases, which accounts for one third of total deaths around the world [1]. Dyslipidemia is characterized by an excess of blood lipids, which include total cholesterol (TC), triglycerides (TG), and low-density lipoprotein (LDL), and a decrease in high-density lipoprotein (HDL) in the blood stream. The risk of cardiovascular death in those with dyslipidemia can be significantly reduced with statin therapy and lifestyle modification [2].

Statin therapy is considered the cornerstone therapy for both primary and secondary prevention of cardiovascular ischemic events in dyslipidemic patients [3]. Statins have numerous pleiotropic effects, including decreased platelet activation, decreased coagulation, decreased endothelin, increased endothelial function, increased nitric oxide bioavailability, increased endothelial progenitor cells, decreased reactive oxygen species, decreased immunomodulation, and decreased matrix metalloproteinase [4].

Cardiac magnetic resonance spectroscopy (MRS) is a unique, non-invasive, and non-irradiating imaging modality which can assess cardiac energy metabolism in vivo in health and disease. Different nuclei can be used for the evaluation of metabolism with MRS: 1H, 31P, 13C, and 23Na [5]. Fatty hearts are common in a variety of metabolic diseases, and noninvasive cardiac fat imaging by H1 MRS techniques offers quantitative evaluation of myocardial fat content [5]. H1-MRS has also been validated for quantitative evaluations of the metabolite content of in vivo myocardial tissue [6].

Aim

Our objective was to detect the efficacy of statin on the myocardial lipid content in dyslipidemic patients and to detect if there will be changes 6 months after starting statin therapy.

Methods

Study design

Case–control study followed by prospective follow up study (Quasi experiment).

Study population

Thirty nadir dyslipidemic patients (dyslipidemia group; recently diagnosed before start of statin therapy and lifestyle modification) were engaged in this study from January 2020 to October 2022 before statin drug therapy and followed up for 6 months after therapy. A comparative group of fifteen healthy individuals non-dyslipidemic were also included in the study to compare parameters with dyslipidemic group before and after therapy.

Inclusion criteria for dyslipidemic patients were as follows

Hyperlipidemia: total cholesterol (TC) ≥ 200 mg/dL, Low density lipoprotein (LDL) ≥ 130 mg/dL, and Triglycerides (TG ≥ 150 mg/dL in naive patients with no past history of statin therapy.

Exclusion criteria

Prior use of statins, patients with hypertension, thyroid problems, diabetes mellitus, coronary heart disease, arrhythmia, congenital heart disease, moderate-to-severe valve heart disease, primary cardiomyopathy, thyroid disease, liver dysfunction, and renal dysfunction were excluded from the study. Also patients how are claustrophobic, with implantable cardiac devices, and had breathing difficulties or arrhythmia were excluded from the study.

Clinical and laboratory examinations

For each participant, data on their age, sex, height, weight, systolic and diastolic blood pressure, body mass index (BMI), history of diabetes, smoking, obesity, and drug use were collected.

Laboratory tests

Laboratory tests including TC, TG, LDL-C, and high-density lipoprotein cholesterol (HDL-C), were performed at first presentation before starting statins and after 6 months of follow-up.

Cardiac MRI

CMR machine

1.5-T scanner, Philips, Ingenia, The Netherlands.

Image acquisition

The 1H MRS sequence was built on a single voxel PRESS sequence with a 10 ms TE, a TR of 1500 ms, a frequency of 63.9, and an NSA of 40. The cardiac coil was a 4-element, phased-array cardiac coil. Repeated end-expiratory breath holds were used to collect images. The horizontal long-axis and short-axis views were planned using scout images in the coronal, sagittal, and axial planes. In order to avoid the spectrum being contaminated by signals arising from pericardial fat, the voxel (10, 20, and 30 mm3) for cardiac MRS was subsequently placed in the interventricular septum in the short-axis view. Then lipid signals and signals of other metabolites were processed by the machine software, which drew a curve of different metabolites. The lipid spectrum signal was measured at 1.3 ppm on resonance with suppression of the dominant

Table 1 Socio-demographic characteristics and medical history of the studied cases at start of study

Parameter	
Age (year)	49 ± 9.19
Sex	
Male	17 (56.67%)
Female	13 (43.33%)
Height (m)	1.69 ± 0.05
Weight (kg)	93.3 ± 10.51
BMI (kg/m^2)	32.33 ± 3.08
Smoking	
Yes	7 (23.33%)
No	23 (76.67%)
Diabetes mellitus	
Yes	0 (0%)
No	30 (100%)
Hypertension	
SBP	120 (110–135)
DBP	80 (70–85)

BMI Body mass index, *DBP* Diastolic blood pressure, *SBP* Systolic blood pressure

water signal [7]. The acquisition time was about 5–7 min (Table 1).

Treatment follow up

Patients were followed up for 6 months with a focus on lifestyle modification and compliance with treatment. After therapy, laboratory tests and MRS were performed.

Ethical consideration (consent)

Approval of the institutional research board (IRB) and the medical ethics committee in Mansoura University was obtained (MD.19.04.170). All included patients signed an informed consent.

Statistical analysis

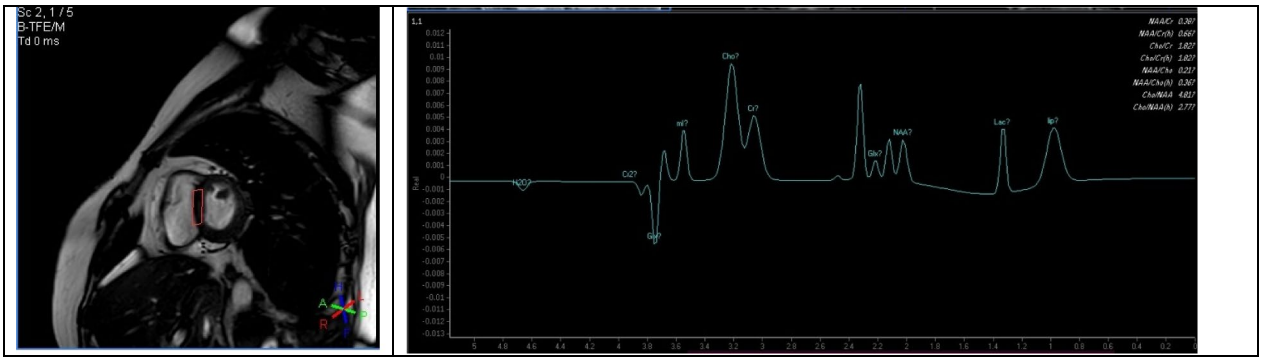
Data were analyzed with SPSS version 26. The normality of data was first tested with one-sample Shapiro–Wilk test. Qualitative data were described using number and percent. Continuous variables were presented as mean ± SD (standard deviation) for parametric data and median, minimum and maximum for non-parametric data. Variables before and after statin therapy were compared by paired t-test for normally distributed data and Wilcoxon signed rank test for non-normally distributed data, McNemar test was used to compare categorical data (Fig. 1).

For comparison between dyslipidimic cases and control group; independent sample t test was used for normally distributed data, while Mann Whitney test was used for non-normally distributed data, and Fischer's exact test or Chi-square test for categorical data. (SPSS Inc., Chicago, Illinois, USA). Level of significance: For all above mentioned statistical tests done, the threshold of significance is fixed at 5% level (p value). The results were considered significant when the probability of error is less than 5% ($p < 0.05$).

Discussion

Dyslipidemia is characterized by an excess of blood lipids, which include cholesterol, triglycerides, and low-density lipoprotein, and a decrease in high-density lipoprotein in the blood stream. In Dyslipidemic patients, excess fat can be present in myocardial tissue and may have an adverse effect on left ventricular (LV) performance [2].

Many animal studies provide the idea that myocardial steatosis can cause LV dysfunction. They theorize



(a) **(b)**

Fig. 1 1H MR spectrum from the myocardium. **a** The left panel displays the voxel positioning in an end-systolic short-axis view. **b** The right panel depicts the MR spectra (mean spectrum in blue). Curve of metabolites and lipid signal by magnetic resonance spectroscopy, lipid signal was detected at 1.3 ppm

that myocardial accumulation of hazardous fatty acid intermediates results in cellular damage, apoptosis and additional fibrosis, leading to contractile LV dysfunction and some ^{1}H-MRS human studies report similar associations [8].

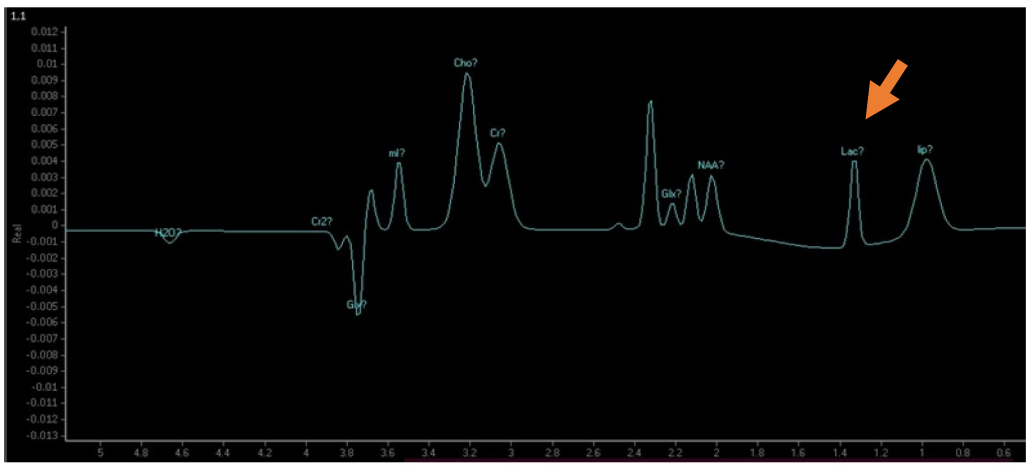
Statin treatment in association with physical exercise can substantially reduce the cardiovascular mortality risk of dyslipidemic individuals. [4]. Statin therapy is not only a medication for treating dyslipidemia; it also has numerous pleiotropic beneficial effects, all on a cellular or molecular basis, but there is a lack of evidence about metabolic cardiac lipid changes in patients receiving statin therapy [9].

The formation of myocardial TG is faithfully dependent on myocardial lipid metabolism. The accumulation of myocardial TG is primarily related to the supply of the fatty acids and mitochondrial energy-producing efficacy. Moreover, myocardial lipid metabolism is regulated by a complex balance between the stream of fatty acids to the heart, challenging energy substrates, energy demand and supply of oxygen to the heart. Therefore, it is chief to evaluate the myocardial TG content using 1 H-MRS non-invasively [10].

There is some degree of intramyocardial fat in normal myocardium as well as in a number of cardiomyopathies. Fat–water separated imaging in the heart by MRI is a sensitive method for detecting intramyocardial fat. Non-invasive cardiac fat imaging by H1 magnetic resonance spectroscopy (MRS) techniques offers quantitative evaluation of myocardial fat content [11].

Our research aimed to find out whether dyslipidemic patients had high myocardial lipid content as determined by magnetic resonance spectroscopy in comparison with healthy individuals and effect of statin in reducing lipid content after 6 months of effective therapy in dyslipidemic patients (Fig. 2).

(a) MRS of dyslipidemic patient before start of statin therapy



(b) shows MRS of dyslipidemic patient after statin therapy.

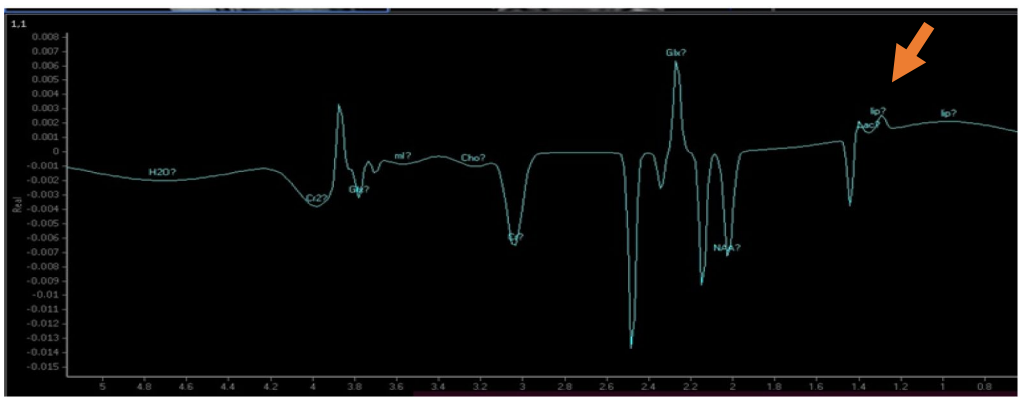


Fig. 2 In the (**a**), we noticed increase in myocardial lipid concentration, while in (**b**), we noticed decrease in myocardial lipid concentration

This study included 30 patients: 17 males (56.67%) and 13 females (43.33%). The age ranged from 33 to 63 years, with an average of 49 9.19 years. Also, the study included 15 healthy controls of matched age and sex with dyslipidemic cases (Table 2). The main finding in this study is that there was a statistically significant difference in myocardial lipid content in dyslipidemic patients when compared with control before statin therapy (Table 2). Furthermore, after treating dyslipedimic patients with statin, TG content was statistically insignificant different when compared with control (Table 3). Also, a statistically significant reduction of myocardial lipid content in dyslipidemic was observed 6 months after statin drug therapy was observed (Table 4).

To our knowledge, this study is the first to measure the changes in myocardial triglyceride (TG) content in dyslipidemic patients without additional risk factors using magnetic resonance spectroscopy (MRS), after statin therapy. Some previous studies used MRS to evaluate TG content in metabolic syndrome and type 2 diabetic patients however these studies didn't perform follow up after treatment [12]. Other studies compared TG myocardial content after nutritional interventions [13] or exercise training [14] but not medical therapy as we did.

When comparing dyslipidemic patients before statin and controls there were statistically significant higher levels as regard body weight, BMI, total cholesterol, LDL, serum TG (p 0.001) and cardiac lipid content (p 0.019) (Table 2). These findings are consistent with previous studies in which there were increased TG myocardial content in patients with metabolic syndrome (which includes obesity and dyslipidemia) when compared with normal individuals [12, 15].

Our findings are in harmony with in vivo MRS studies which demonstrated that triglycerides were detectable in the hearts of even extremely slim individuals and were elevated in overweight and obese individuals. [16, 17].

When comparing laboratory lipid levels in dyslipidemic patients after statin and control, there was no statistically significant difference (Table 3). This reflects the effect of

Table 2 Demographic parameters, laboratory findings, TG lipid concentration in dyslipidemic patients before statin therapy and control group

Parameter	Dyslipidemic patients before start of statin	Control		*p* value
Demographic parameters				
Age (years)	49 ± 9.1	45.9 ± 4.7	$t = 1.2$	0.15
Sex				
Male	17 (56.7%)	7 (46.7%)	$\chi^2 = 0.4$	0.75
Female	13 (43.3%)	8 (53.3%)		
Weight (kg)	93.3 ± 10.5	79.1 ± 11.9	$t = 4.1$	**< 0.001***
BMI (kg/m^2)	32.3 ± 3.1	26.6 ± 2.0	$t = 6.5$	**< 0.001***
Smoking				
Yes	7(23.3%)	2(13.3%)	FET	0.7
No	23(76.7%)	13(86.7%)		
Diabetes mellitus				
Yes	0	0		
No	30(100%)	15(100%)		
Hypertension				
SBP	120 (110–135)	120 (110–130)	$Z = -0.5$	0.6
DBP	80 (70–85)	80 (70–85)	$Z = -0.2$	0.8
Laboratory investigations				
TC (mg/dL)	265.6 ± 36.8	180.6 ± 11.8	$t = 8.7$	**< 0.001***
LDL (mg/dL)	157.0 (119.0–258.0)	112.9 (86.0–128.0)	$Z = -5.1$	**< 0.001***
HDL (mg/dL)	46.5 (6.0–70.0)	52.0 (34.0–63.0)	$Z = -1.0$	0.3
TG (mg/dL)	166 (73.0–788.0)	96.5 (40.0–139.0)	$Z = -3.6$	**< 0.001***
Magnetic resonance spectroscopy				
TG lipid concentration (mmol/l)	0.011 (0.001: 0.55)	0.003 (0.001: 0.15)	$Z = -2.35$	**0.019***

The values presented in bold are significant by refrence value

HDL, High-density lipoprotein cholesterol; LDL, Low-density lipoprotein cholesterol; TC, Total cholesterol; TG, Triglycerides; FET, Fisher's Exact Test; *t*, Independent *t* test; χ^2, Chi square test; *Z*, Mann Whitney test

*p value < 0.05: significant

Table 3 Demographic parameters, laboratory findings, TG lipid concentration in dyslipidemic patients after statin therapy and control group

Parameter	Dyslipidemic patients after start of statin	Control		*p* value
Demographic parameters				
Age (years)	49 ± 9.2	45.9 ± 4.7	$t = 1.2$	0.15
Sex				
Male	17 (56.67%)	7(46.7%)	$\chi^2 = 0.4$	0.75
Female	13 (43.33%)	8(53.3%)		
Weight (kg)	87.5 (63–117)	84 (58–93)	$Z = -1.7$	0.09
BMI (kg/m^2)	30.7 ± 4.6	26.6 ± 2.0	$t = 4.16$	**< 0.001***
Smoking				
Yes	1	2	FET	0.25
No	29	13		
Diabetes mellitus				
Yes	4 (13.3%)	0	FET	0.28
No	26 (86.7%)	15		
Hypertension				
SBP	120 (110–160)	120 (110–130)	$Z = -0.71$	0.48
DBP	80 (70–100)	80 (70–85)	$Z = -0.30$	0.76
Laboratory investigations				
TC (mg/dL)	198.8 ± 48.9	180.6 ± 11.8	$t = 1.83$	0.08
LDL (mg/dL)	100.5 (38:265)	112.9 (86.0: 128.0)	$Z = -0.28$	0.78
HDL (mg/dL)	47.7 ± 12.4	49.7 ± 8.4	$t = -0.56$	0.58
TG (mg/dL)	110.5 (57–318)	96.5 (40.0–139.0)	$Z = -1.36$	0.17
Magnetic resonance spectroscopy				
TG lipid concentration (mmol/l)	0.002 (0.001: 0.04)	0.003 (0.001: 0.15)	−0.92	0.35

The values presented in bold are significant by refrence value

BMI, Body mass index; HDL, High-density lipoprotein cholesterol; LDL, Low-density lipoprotein cholesterol; TC, Total cholesterol; TG, Triglycerides; FET, Fisher's exact test, *t*, Independent *t* test; χ^2, Chi square test; Z, Mann Whitney test

**p* value < 0.05: significant

statin therapy on reducing serum lipid levels as demonstrated in different studies [4, 18, 19]

Our study showed that, regarding MRS, there was a statistically non-significant difference in myocardial lipid content in dyslipidemic patients 6 months after statin therapy life style modifications and control cases with (*p* value 0.35) (Table 3). We hypothesis that dyslipidemic patients suffered from high myocardial lipid content detected by MRS which decreased after 6 months of statin therapy which results in decrease serum lipid levels and subsequent decrease in lipid contents in myocardium. This finding was not studied before, only previous studies detect changes after nutritional interventions [13], exercise training [14], and studies performed on animals [20] but not after statin medical therapy like us.

As we compared baseline data to 6 months after lifestyle modification and statin therapy, we found a statistically significant difference in weight, body mass index (BMI), and lipid levels in dyslipidemic patients (Table 4). This result confirms the efficacy of statin therapy in reducing total lipids, particularly low-density lipoprotein (LDL).

Also, our study showed that, regarding MRS, there was a statistically significant difference in myocardial lipid content in dyslipidemic patients before statin therapy and 6 months after statin therapy with lifestyle modifications (*p* 0.001) (Table 4). This result is compatible with the study done by Zlobine et al., who studied that dyslipidemia increases the risk for cardiovascular disease, and it has been suggested that alterations in myocardial lipid metabolism are included in the pathophysiology and development of ventricular dysfunction [21].

Fatty acids are absorbed through the intestines and deposited as triglycerides in adipocytes under normal physiological conditions, with very little accumulation in non-adipose tissues like the heart. Nevertheless, increased myocardial fatty acid delivery and uptake are caused by the simultaneous dyslipidemia and increased dietary fatty acid intake. This causes steatosis, or the

Table 4 Demographic parameters, laboratory findings, TG lipid concentration in dyslipidemic patients before and after statin therapy

Parameter	Before statin	After statin		*p* value
Demographic parameters				
Weight (kg)	93.30 ± 10.51	88.73 ± 14.69	$t = 2.66$	0.012
BMI (kg/m^2)	32.33 ± 3.08	30.74 ± 4.58	$t = 2.91$	**0.007***
Smoking				
Yes	7 (23.33%)	1 (3.3%)		**0.03***
No	23 (76.67%)	29 (96.7%)		
Diabetes mellitus				
Yes	0 (0%)	4 (13.3%)		0.125
No	30 (100%)	26 (86.7%)		
Hypertension				
SBP	120 (110–135)	120 (110–160)	$t = 0.74$	0.46
DBP	80 (70–85)	80 (70–100)	$t = 0.1$	0.68
Laboratory investigations				
TC (mg/dL)	265.63 ± 36.75	197.80 ± 48.85	$t = 6.63$	**< 0.001***
LDL (mg/dL)	157.0 (119–258)	100.5 (38–265)	$Z = -3.56$	**< 0.001***
HDL (mg/dL)	46. 0 (6–70)	49.5 (14–68)	$Z = -0.606$	0.545
TG (mg/dL)	166 (73–788)	110.5 (57–318)	$Z = -2.63$	**0.009**
Magnetic resonance spectroscopy				
TG lipid concentration (mmol/l)	0.011 (0.001–0.55)	0.0025 (0.001–0.04)	$Z = -3.92$	**< 0.001***

The values presented in bold are significant by refrence value

HDL, High-density lipoprotein cholesterol; LDL, Low-density lipoprotein cholesterol; TC, Total cholesterol; TG, Triglycerides, *t*, Paired *t* test, McNemar test; *Z*, Wilcoxon Signed Ranks test

**p* value < 0.05: significant

accumulation of intracellular triglycerides within the cytoplasm of myocytes [22].

Our result also goes in line with a study done by Korosoglou et al., who stated that myocardial steatosis may represent an early marker of subclinical myocardial dysfunction irrespective of myocardial perfusion reserve [23].

This is also in agreement with Ruberg FL, who stated that myocardial lipid content may be used as a biomarker to expect the development of cardiac dysfunction in dyslipidemic patients and may serve as a measurable target for intervention before the development of overt cardiac dysfunction [24]. Sai et al. said that left ventricular end-systolic volume was found to be an independent factor of the myocardial TG content as measured by H1 MRS, which may be useful for assessing non-invasively the associations between the myocardial TG content and cardiac morphology [11].

In contrary to our finding, a study by McGavock et al. showed that there was no association between myocardial lipid accumulation and LV diastolic function [25].

We studied the correlation between TG content by MRS in dyslipidemic patients before therapy with other clinical and laboratory lipid levels. There was only a statistically significant positive correlation with weight (r 0.66, p 0.004, Table 5), while no other statistically significant correlations were detected. This finding was also observed in a study by Kankaanpaa et al. who found elevated level of myocardial TG in obese compared to lean subjects [17]. However, Rial et al. [26] and Liu et al. [27], revealed that BMI was related with TG.

Table 5 Correlation between TG lipid content by MRS before starting statin with demographic, clinical and laboratory lipid levels

	r	*p* value
Age	0.20	0.44
Sex	0.14	0.56
Weight	**0.66**	**0.004****
BMI	0.43	0.084
Total cholesterol	0.41	0.11
LDL	0.19	0.47
HDL	0.026	0.92
TG	0.243	0.37

The values presented in bold are significant by refrence value

BMI, Body mass index; HDL, High-density lipoprotein cholesterol; LDL, Low-density lipoprotein cholesterol; TC, Total cholesterol; TG, Triglycerides; *r*, Pearson correlation, Spearman correlation for sex

** *p* value < 0.05: significant

On the other hand, Szczepaniak et al. reported a highly significant correlation of myocardial TG content and biochemical assay of lipids (r 0.9 $p < 0.001$), but this result was obtained from experimental rats [28]. When studying on humans, they found a non-statistically significant correlation with BMI (p 0.07) [28].

Noted that despite positive findings of the present research, the following restrictions must be taken into account:

- First, our study's small sample size (30 cases). A large number of patients have to be studied to confirm our findings. The small number in our study is due to including dyslipidemic patients without other risk factors, which makes the selection difficult due to the common associations between dyslipidemia and other risk factors. Also the study was started at the COVID-19 pandemic era which limits and restricts patients' hospital admission, visits, participation in elective research, and follow up.
- Second, most of the newly joined patients were middle-aged. So, the results cannot be generalized to the extremes of age groups in the adult population.
- Third, the short-term duration of our study's follow-up means further data on the relationship between statin therapy and myocardial lipid content may not be obtained.
- Fourth, it was a single-center, nonrandomized study. However, we were careful to include dyslipidemic patients without other risk factors. Our findings need to be supported by large-scale, multi-center clinical studies.

Conclusions

In our study, dyslipidemic patients had increased myocardial lipid content as measured by magnetic resonance spectroscopy, which decreased after 6 months of statin therapy.

Abbreviations

TC	Total cholesterol
TG	Triglycerides
LDL	Low-density lipoprotein
HDL	High-density lipoprotein
MRS	Cardiac magnetic resonance spectroscopy
BMI	Body mass index
LV	Left ventricle
LVESV	Left ventricular end-systolic volume
MRI	Magnetic resonance imaging
CMR	Cardiac magnetic resonance

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Author contributions

Eslam Elsayed analyzed and interpreted the patient data regarding the cardiac manifestation and follow up, Hanaa Mahmoud was a major contributor in writing the manuscript, Hend Gamal analyzed and follow image acquisition, both prof Hesham and prof Abdelrazek were supervisors of work. All authors read and approved the final manuscript.

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Availability of data and materials

The datasets used and or analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

Approval of the institutional research board (IRB) and the medical ethics committee in Mansoura University was obtained (MD.19.04.170). All included patients signed an informed consent.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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