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RESEARCH

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Highlights on the link between vitamin D and lipid panel in Egyptian multiple sclerosis patients

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Abstract

Background Diversity of risk factors, namely, vitamin D and lipid panel abnormalities, are connected to multiple sclerosis (MS) etiology and may possess an influential role on disease course. In a cross-sectional study, we correlated the demographic, clinical and radiological characteristics of 111 relapsing–remitting MS (RRMS) patients with their serum levels of vitamin D and lipid profile to evaluate the consequences of their abnormalities on disease activity and/or its progression.

Results In the study group, the mean serum level of vitamin D was 18.93 ± 9.85 ng/mL, over 80% had insufficient level (< 30 ng/mL) and significantly lower in females ($P = 0.011$). Insufficient vitamin D significantly associated with high relapse frequency ($P = 0.005$). Measurement the direction of this association showed that each 1 ng/mL increase in vitamin D was correlated with both decrease in annualized relapse rate (ARR) of 0.02 relapse/year ($P = 0.017$) and with decrease in number of relapses during last 2 years of 0.02 relapse ($P = 0.045$). Analysis of serum lipid panel showed a direct link between higher levels of TC and LDL to increased total number of relapses ($P < 0.001$ and 0.003, respectively) and EDSS ($P = 0.001$ and 0.022), also between higher TG and EDSS ($P = 0.001$). This link became indirect between HDL and both total number of relapse and EDSS ($P = 0.001$ and 0.001). Radiologically, positively linked confluent brain lesion to elevated TC and TG levels ($P = 0.001$ and 0.002, respectively) and cord lesions to elevated TC ($P = 0.007$). Longer disease duration positively associated with all lipids-related variables. As a direct effect on lipid metabolism, each 1 ng/mL increase in vitamin D was associated with reduction in serum TC of 1.48 mg/dL ($P = 0.002$) and rise in HDL of 0.35 mg/dL ($P = 0.028$).

Conclusions Management of vitamin D insufficiency may decrease risk of higher ARR and the same for dyslipidemia in reduction of disability and confluent brain T2 lesion. Increasing vitamin D was positively correlated with HDL but negatively with TC.

Keywords Vitamin D, Lipids, Multiple sclerosis, Egyptian

Background

Many environmental risk factors were accused to be involved in the etiology of MS. Modifying these risk factor may help in reduction of disease activity and slowing its progression, one of them is vitamin D deficiency. Adequate vitamin D was found to reduce disease risk and activity also have a key role in lipid metabolism. Researchers found that vitamin D insufficiency could

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affect lipid metabolism negatively and associated with obesity as it accumulates in the fatty tissue instead of being converted into active 1, 25 (OH) D3 [1]. Studies emphasized its protective role and stated that the likelihood of developing metabolic dyslipidemia is much less with adequate levels of vitamin D as to its deficiency [2].

The precise explanation for the inverse correlation between 25 vitamin D serum levels and LDL cholesterol and TC is still unknown. Studies propose that vitamin D receptors (VDRs) balance lipid serum levels through adjusting bile acid synthesis. VDRs produce its effect genomically indirectly through suppression of the expression of the Cyp7α1 gene that codes for CYP7A1, the rate-limiting enzyme in bile acid synthesis, thus causing cholesterol levels to increase [3].

Lipid panel abnormalities are well-known vascular risk to buildup atherosclerotic changes. The presence of dyslipidemia as verified in more than 8000 patients that were analyzed retrospectively and it was shown that the risk for disability progression in MS is much higher with vascular comorbidities related to dyslipidemia [4].

Methods

This was a cross-sectional, hospital-based study. Based on the 2010 McDonald criteria [5], 111 patients with a diagnosis of RRMS were selected and enrolled from MS unit at Ain Shams University Hospitals after consenting to join in this study in the period between June 2019 to June 2020. Their ages range between 18 and 50 years. We excluded patients with other neurological diseases or concomitant medical illness as diabetes and hypertension, patients on vitamin D supplementation or lipid lowering drugs and patients with history of relapse during 3 months previous to the study.

Each patient has demographic data, detailed medical history including; sex, age of disease onset, duration of the illness, total number of relapses, relapses during last 2 years and disease-modifying drugs (DMDs). Expanded Disability Status Scale (EDSS) score [6] was used to examine neurological disability among patients done by certified EDSS rater. We assessed total lesion load, black holes and enhanced lesions in baseline MRI brain and cervical spine. For serum assessment of vitamin D and lipid panel including LDL, HDL, Cholesterol and Triglycerides; 4 mL of venous blood were taken from patients and placed in gel containing vacutainers. They were allowed to clot for 30 min. Vacutainers were centrifuged for 5 min and serum was separated. Lipid profile was immediately analyzed, while 200 uL of serum were aliquoted and frozen at -20˚C for further analysis of vitamin D.

Vitamin D was tested by Cobas e411 (Roche) hitachi high technologies corporation 1–24–14 Nishi-shimbashi Minato-Ku Tokyo 105–8717 Jaban and according to the US National Kidney Foundation; 25-hydroxyvitamin D level < 30 ng/mL is defined insufficient or deficient as the preferred level is ≥ 30 ng/mL.

TC, TG, LDL and HDL were done by Beckman Coulter, Inc,250 S. Kraemer Blvd., Brea, CA 92821 USA AU480 autoanalyzer. According to the National Cholesterol Education Program (NCEP); TC level < 200 mg/dl (desirable), 200–239 mg/dL (borderline to high) and ≥ 240 mg/dL (high). TG level < 150 mg/dL (desirable), 150–199 mg/dL (borderline high) and ≥ 200 mg/dL (high). LDL level < 100 mg/dL (optimal), 100 – 129 mg/dL (near optimal), 130–159 mg/dL (borderline high) and ≥ 160 mg/dL (high). HDL level > 40 mg/dL is considered to be the desirable level.

Statistical methods

Analysis was performed using SPSS (Statistical Package for Social Sciences) software version 22.0(IBM SPSS Statistics for Windows, Version 22.0, IBM Corp., Armonk, NY).

Statistical methods, descriptive data were performed using the mean, standard deviation, median, minimum and maximum ranges for quantitative data, while number and percentage for qualitative data. In quantitative variables, for comparison between two groups unpaired Student *T* test was used, while in the same group, ANOVA tests were used for difference analysis among different times.

Inferential analysis for qualitative data was done using Chi-square test for independent variables and differences between proportions, while Pearson correlation for numerical normally distributed data.

Correlation coefficient was used for measurement of linear correlation between two numeric variables in one group. Significance (S) of data was determined when $P \leq 0.05$.

Results

This study included 111 patients with definite RRMS, predominating female gender (76.58%), with mean age 31.39 ± 7.20 years at time of presentation and 26.78 ± 6.81 years at disease onset and mean duration of illness 4.70 ± 4.00 years. The mean relapse rates (RR) were 3.40 ± 2.28 for total number of relapses and 1.60 ± 0.80 relapses for the last 2 years. The median of annualized relapse rate (ARR) was 1 [interquartile range (IQR) (0.6–1.3)]. Degree of disability measured by EDSS was 2.5 with IQR (1–3.5) that was considered mild (EDSS ≤ 3) in majority of patients (71.17%) (Table 1).

vitamin D analysis exhibited mean serum level of 18.93 ± 9.85 ng/mL. Most of patients (81.08%) had insufficient level (< 30 ng/mL) of serum vitamin D level. This

Table 1 Demographic characteristics of the patients

Variable	*n*=111
Gender	
Male	26 (23.42%)
Female	85 (76.58%)
Age	
Mean ± SD (Range)	31.39 ± 7.20 (19–49)
Duration of illness in years	
Mean ± SD (Range)	4.70 ± 4.00. (1–20)
Total number of relapses	
Mean ± SD (Range)	3.40 ± 2.28 (1–14)
Number of relapses during the last 2 years	
Mean ± SD (Range)	1.60 ± 0.80 (0–4)
ARR	
Median (IQR)	1 (0.6–1.3)
EDSS	
Median (IQR)	2.5 (1–3.5)
EDSS	
Mild disability (EDSS ≤ 3)	79 (71.17%)
Moderate-to-severe disability; EDSS > 3	32 (28.83%)

ARR annualized relapse rate, *EDSS* Expanded Disability Status Scale, *SD* standard deviations, *IQR* inter-quartile range

insufficiency was statistically significant in females ($P=0.011$) (Tables 2 and 3).

The lipid profile showed that mean levels of TC, TG, LDL and HDL were 204.9 ± 50.9 mg/dL, 105.4 ± 44.6 mg/dL, 122.2 ± 38.8 mg/dL and 56.2 ± 16.6 mg/dL, respectively. Abnormal values were observed more for high TC in 24 patients (21.662%), On the other hand, 20 patients (18.01%) have low HDL level (Table 2).

The defensive role against disease activity was declared from both ARR and number of relapses during last 2 years that were statistically significantly lower in sufficiency group compared to insufficiency group (P = 0.005 and 0.043, respectively) (Table 3 and Figs. 1 and 2). Moreover, this significance continued to exist after the linear regression analysis as each 1 ng/mL increase in vitamin D was associated with decrease in ARR of 0.02 relapse/year with 95% confidence interval (CI) was − 0.03 to − 0.003 and $P=0.017$ (Fig. 3) and with decrease in number of relapses during last 2 years of 0.02 relapse with 95% CI was − 0.03 to − 0.00 and $P=0.045$ (Fig. 4).

On the other side, we found no significant correlation between serum vitamin D level and age, age at onset of illness, its duration, EDSS nor MRI finding, including T2 lesion load, black holes and enhanced lesions ($P>0.05$) (Tables 4 and 5).

As regards the lipid profile, patients with higher levels of TC, TG and LDL were observed to have a significant correlation with longer disease duration ($P=0.038$, 0.025 and 0.01, respectively). Patients with higher total number of relapses showed higher levels of serum TC, LDL with a significant correlation ($P<0.001$ and 0.003, respectively) and lower levels of HDL ($P=0.003$). This correlation remains significant only with TC after linear regression analysis as each 1 mg/dL increase in TC was associated with increase in total number of relapses of 0.03 relapse (95% CI 0.01–0.05 and $P=0.007$) (Table 6 and Fig. 5).

Table 2 Vitamin D and lipid profile levels

Variable	*n*=111
Vitamin D, ng/mL	18.93 ± 9.85 (4–41)
Mean ± SD (range)	
Insufficient vitamin D level (<30 ng/mL)	90 (72 females–18 males) (81.08%)
Sufficient vitamin D level (≥ 30 ng/mL).	21 (13 females–8 males) (18.92%)
Total Cholesterol, mg	
Mean ± SD (Range)	204.9 ± 50.9 (119–442)
Patients with High TC level (≥240 mg/dL),	24 (21.62%)
TG, mg/dL	
Mean ± SD (Range)	105.4 ± 44.6 (39–329)
Patients with High TG level (≥200 mg/dL),	4 (0.04%)
HDL, mg/dL	
Mean ± SD (Range)	65.2 ± 16.6 (31–99)
Patients with low HDL level (< 40 mg/dL)	20 (18.01%)
LDL, mg/dL	
Mean ± SD (Range)	122.2 ± 38.8 (37–326)
Patients with High LDL level (≥160 mg/dL)	12 (10.81%)

HDL high density lipoprotein, *HS* highly significant, *TC* total cholesterol, *TG* tri-glycerides, *SD* standard deviations

Table 3 Relation between S. vitamin D level and gender, duration and number of relapses during last 2 years

Variable	Vit D		*T* test	
	Mean + SD	Mean + SD	*T*	*P* value
Gender	Female	Male		
	17.635 ± 9.723	23.192 ± 9.204	− 2.581	0.011 HS
	Insufficient (n=90)	Sufficient (*n*=21)		
Number of relapse during last 2 years	1.678 + 0.819	1.286 + 0.644	2.049	0.043
Duration	4.378 + 3.976	6.095 + 3.910	1.788	0.077

HS highly significant

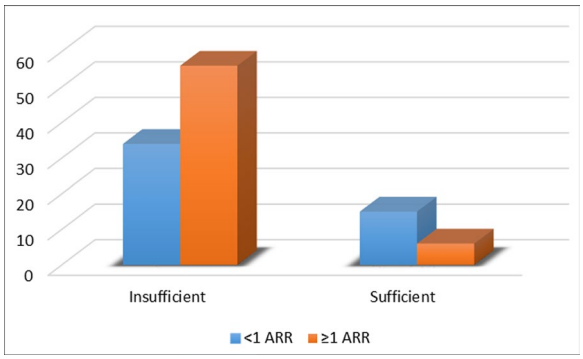

Fig. 1 Relation between S. vitamin D level and relapse frequency (Chi-square)

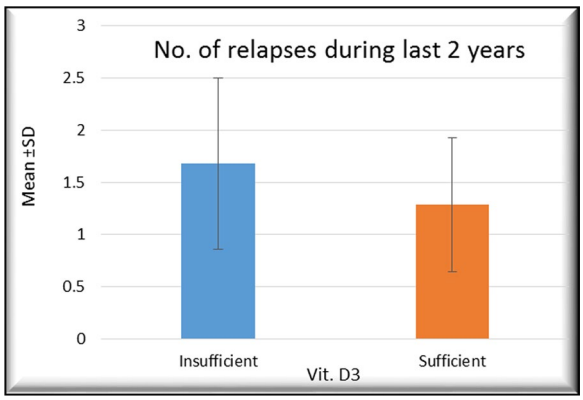

Fig. 2 Relation between S. vitamin D level and number of relapses during last 2 years (*T* test)

In the same context, number of relapses during last 2 years showed a positive significant correlation with TC and LDL, while a negative significant correlation with HDL ($P<0.001$ for each) (Fig. 6). However, there was no significant association with age, age at onset of illness nor ARR and abnormal lipid profile.

Regarding to the degree of disability, in moderate-to-severe group, TC and TG levels were statistically significantly higher ($P=0.001$ for each), while HDL levels were statistically significantly lower ($P<0.001$) compared to mild group (Fig. 7).

Regarding to T2 lesion load, patients with confluent lesions have significant statistical higher levels of TC and TG (P = 0.001 and 0.002, respectively) (Table 7). Number of cord lesions also showed a highly significant correlation positively with TC only with P = 0.007. We found no significant relation between lipid profile and enhanced lesions.

Concerning serum vitamin D level in connection with lipid profile, results found out negative link between vitamin D and TC levels ($P=0.002$) and positive link between vitamin D and HDL levels ($P=0.028$). The direction of this correlation remains significant after the linear regression analysis either negative as each 1 ng/mL increase in vitamin D was associated with reduction in TC of 1.48 mg/dL (95% confidence interval (CI) was − 2.42 to − 0.54 and $P=0.002$) or positive as each 1 ng/mL increase in vitamin D was associated with increase in HDL of 0.35 mg/dL (95% CI was 0.04 to − 0.66 and $P=0.028$) (Figs. 8 and 9).

Discussion

Many disease-triggering factors were assumed to stimulate disease susceptibility, of them abnormalities of both serum vitamin D and lipid panel. Both presumably may have substantial impact on MS activity and disease progression. Therefore, this study positively verified this assumption. Results showed a predominant female gender with female: male ratio 3.26:1 and mean age 31.39 ± 7.20 years which was compatible with global studies.

Both genders had a mean serum level of vitamin D 18.93 ± 9.85 ng/mL, where substantial number of patients had insufficient vitamin D level (81.01%), a significant lower levels detected in females (P = 0.011). This was in accordance with what Thouvenot and his colleagues [7] have found in their study, where the mean level of serum vitamin D was 20 ± 11.9 ng/mL and also with an Egyptian study by Zamzam and her colleagues (2019) [8] that revealed insufficient vitamin D levels in 88.4% of patients. Despite the fact that Egypt is blessed by abundant sunshine almost always throughout the year, this can be attributed to decreased consumption of vitamin D rich diet along with more indoor activities in Egyptian females where both MS and vitamin D deficiency predominate.

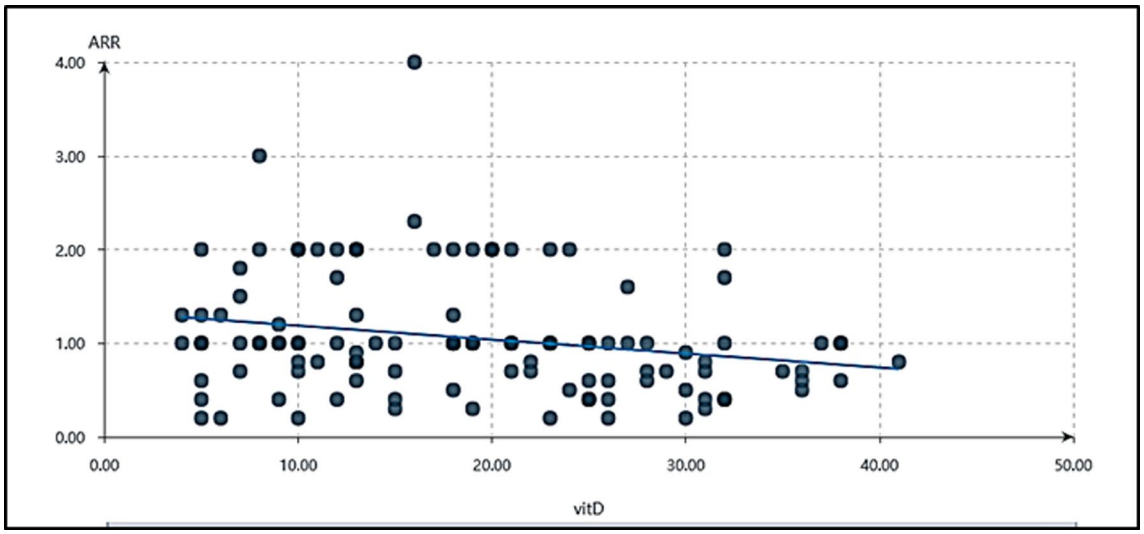

Fig. 3 Scatter plot of the correlation between S. vitamin D level and ARR (linear regression)

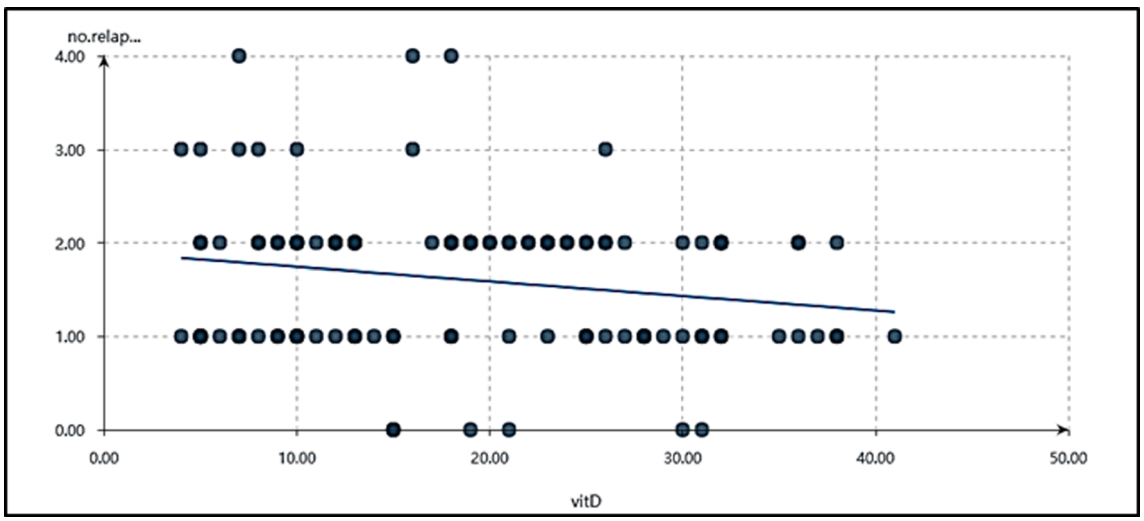

Fig. 4 Scatter plot of the correlation between S. vitamin D level and number of relapse in last 2 years (linear regression)

Table 4 Correlation between S. vitamin D level and clinical data of the patients (linear correlation coefficient)

	Vit D3	
	R	***P* value**
Age	0.078	0.413
Age of onset	− 0.047	0.625
EDSS	− 0.156	0.103

EDSS: Expanded Disability Status Scale

Moreover, low serum levels of vitamin D influence disease activity being linked to significant higher relapse rate (total relapses, last 2 years and ARR). This was consistent with the study of Laursen and her colleagues [9 and 10]. The association between vitamin D and MS has some molecular interpretations and anti-inflammatory effect. Vitamin D counter the inflammation in the CNS by reducing access of autoimmune T-cell to the CNS, facilitating its elimination and T-regulatory cells induction,

Table 5 Correlation between S. vitamin D level and MRI of the patients (Chi-square)

Number of MRI brain T2 lesions	Vit D3						Chi-square	
	Insufficient		Sufficient		Total			
	n	Percent	*n*	Percent	*N*	Percent	X^2	*P* value
1–5 Lesions	16	17.78	2	9.52	18	16.22	1.464	0.691
6–10 Lesions	22	24.44	7	33.33	29	26.13		
Numerous	40	44.44	10	47.62	50	45.05		
Confluent	12	13.33	2	9.52	14	12.61		
Total	90	100.00	21	100.00	111	100.00		

MRI magnetic resonance image

Table 6 Correlation between lipid profile and clinical data of the patients (linear correlation coefficient)

Correlations								
	Cholesterol		TG		HDL		LDL	
	r	*P* value	*R*	*P* value	*r*	*P* value	*r*	*P* value
Duration of illness	0.197	0.038	0.212	0.025	− 0.138	0.148	0.243	0.010
Total number of relapses	0.373	<0.001	0.047	0.625	− 0.279	0.003	0.280	0.003
Age	0.086	0.367	0.112	0.242	− 0.106	0.268	0.132	0.167
Age of onset	− 0.029	0.766	− 0.012	0.897	− 0.105	0.274	− 0.008	0.937
ARR	− 0.029	0.765	− 0.137	0.151	0.029	0.762	− 0.057	0.556

HDL high density lipoprotein, *LDL* low density lipoprotein, *TC* total cholesterol, *TG* tri-glycerides -

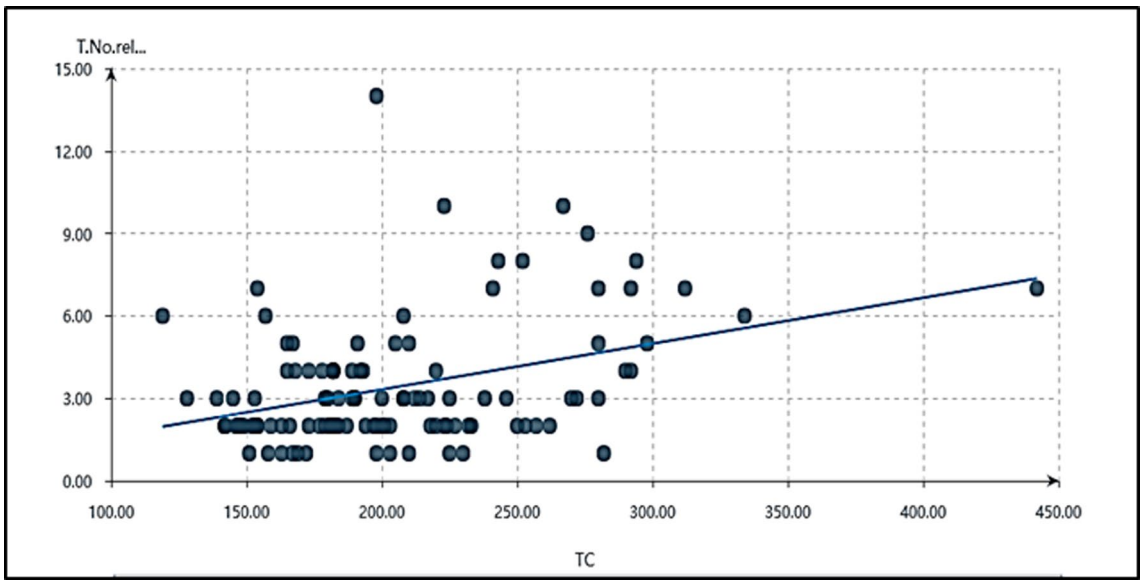

Fig. 5 Scatter plot of the correlation between total cholesterol and total number of relapses (linear regression)

which preserve immunological resilience [11]. Furthermore, the active metabolite 1,25(OH)2 D3 suppress MHC II-antigens expression, decrease the inflammatory T cell cytokines, promote regulatory T cells formation instead of the T-helper 17 [12].

Association between lipid panel and/or its abnormalities and disease characteristics was detected to degree of disability, duration of illness, relapse rate and MRI brain lesion load.

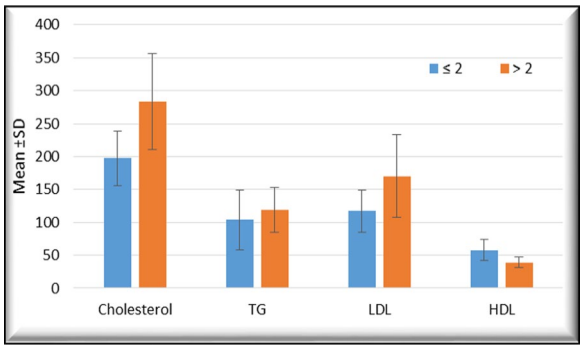

Fig. 6 Relation between lipid profile and number of relapses during last 2 years (*T* test)

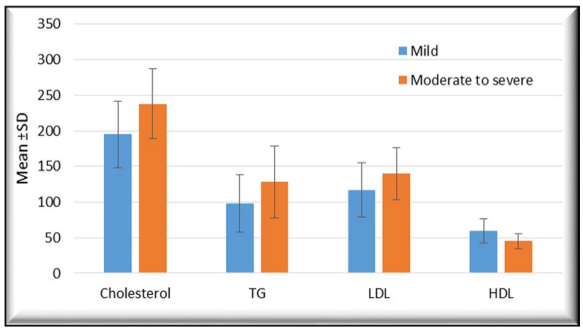

Fig. 7 Relation between lipid profile and degree of disability (*T* test)

Patients with moderate-to-severe disability have higher levels of TC and TG ($P=0.001$ for each of them) and lower levels of HDL ($P<0.001$). Although it became non-significant after linear regression analysis. It was in accordance with what Tettey and his colleagues [13] reported in their study as they showed that patients who had a 2 mmol/L higher baseline TC, LDL and non-HDL, had on average a 0.61 ($P=0.006$), 0.54 ($P=0.037$) and 0.59 (P = 0.003) worse EDSS scores, respectively. This alliance could denote reverse causality, since physical inactivity advocates for increased disability along with adverse lipid profile.

Longer duration of illness positively correlated with high serum level of TC ($P=0.038$), TG ($P=0.025$) and LDL ($P=0.01$) in this study. This was conflicting with the study of Weinstock-Guttman and his colleagues (2011) [14] that declared that disease duration is not related to lipid profile. This can be attributed to longer disease duration which was 12.8 ± 10 years.

This study noted that the correlation between total number of relapses and lipid profile remains statistically significant after the linear regression analysis only with TC as we found that each 1 mg/dL increase in TC was connected to increase in total number of relapses of 0.03 relapse (95% CI 0.01–0.05 and $P=0.007$). This was incompatible with the study of Tettey and his colleagues [13] which concluded that the lipid profile was not involved with the relapse risk. The pro-inflammatory and thrombogenic measures subsequent to dyslipidemia could allegedly aid in disease progression by means of various mechanisms at the blood brain barrier, one of them through promoting leukocyte recruitment and boosting endothelial dysfunction. In this study, the brain T2 lesion load was associated significantly with TC and TG levels ($P=0.001$ and 0.002, respectively) as cases with confluent MRI lesions have higher levels of TC and TG, while there was no significant correlation with HDL or LDL. This was inconsistent with the study of Weinstock-Guttman and his colleagues [14] that reported that T2 lesion volume were not related to any of lipid profile variables. This can be elucidated by the role of activated microglia and infiltrating macrophages in handling lipid during the early stages of MS plaque development. HDL to the contrary, is well-known for its antioxidant characteristics and its role in inverting cholesterol transportation. Thus it may have a protective influence [13].

We identified a significant relation between lipid profile and DMD, since the patients on Fingolimod had higher levels of TC and LDL ($P<0.001$ and 0.012, respectively) and lower levels of HDL ($P<0.001$) compared to patients on Interferon. Fingolimod could be held responsible for causing dyslipidemia or due to the

Table 7 Correlation between lipid profile and no. of MRI brain T2 lesions

Number of brain T2 lesion						
	1–5 Lesions	6–10 Lesions	Numerous	Confluent	ANOVA	
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	*F*	*P* value
Cholesterol	195.33 ± 41.32	186.65 ± 55.30	206.18 ± 46.90	250.85 ± 41.33	5.969	0.001
TG	86.27 ± 34.63	96.34 ± 37.00	107.26 ± 39.77	141.92 ± 64.64	5.205	0.002
HDL	59.55 ± 15.93	53.27 ± 16.88	56.14 ± 16.01	58.07 ± 19.47	0.598	0.617
LDL	114.83 ± 24.78	111.17 ± 47.98	127.16 ± 38.61	136.50 ± 26.69	1.950	0.126

Anova analysis of variance, *HDL* high density lipoprotein, *LDL* low density lipoprotein, *TC* total cholesterol, *TG* tri-glycerides

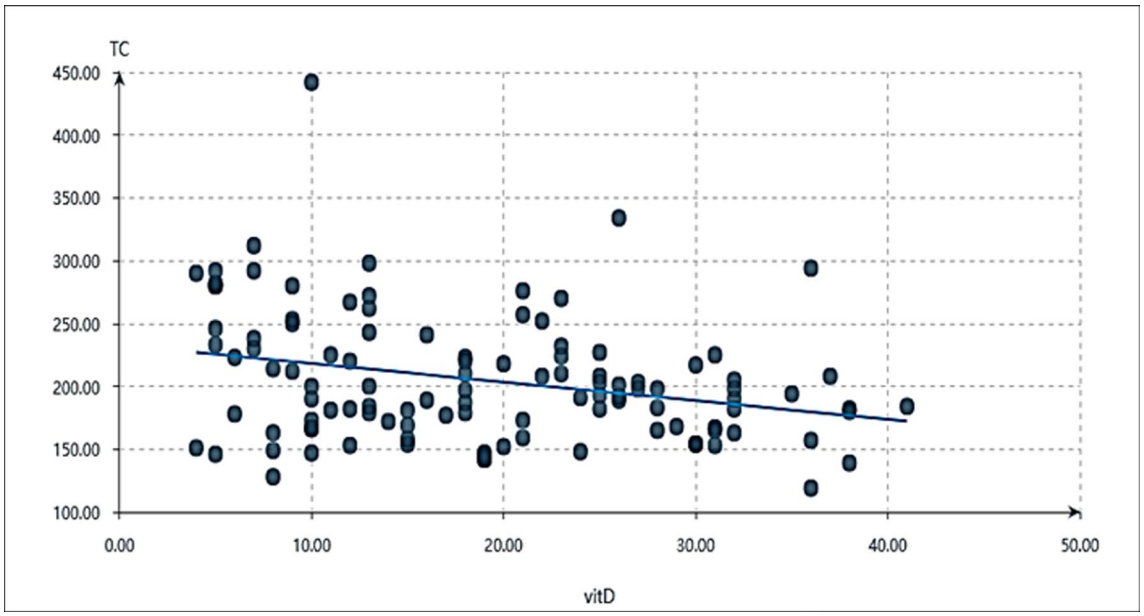

Fig. 8 Scatter plot of the correlation between S. vitamin D level and total cholesterol (linear regression)

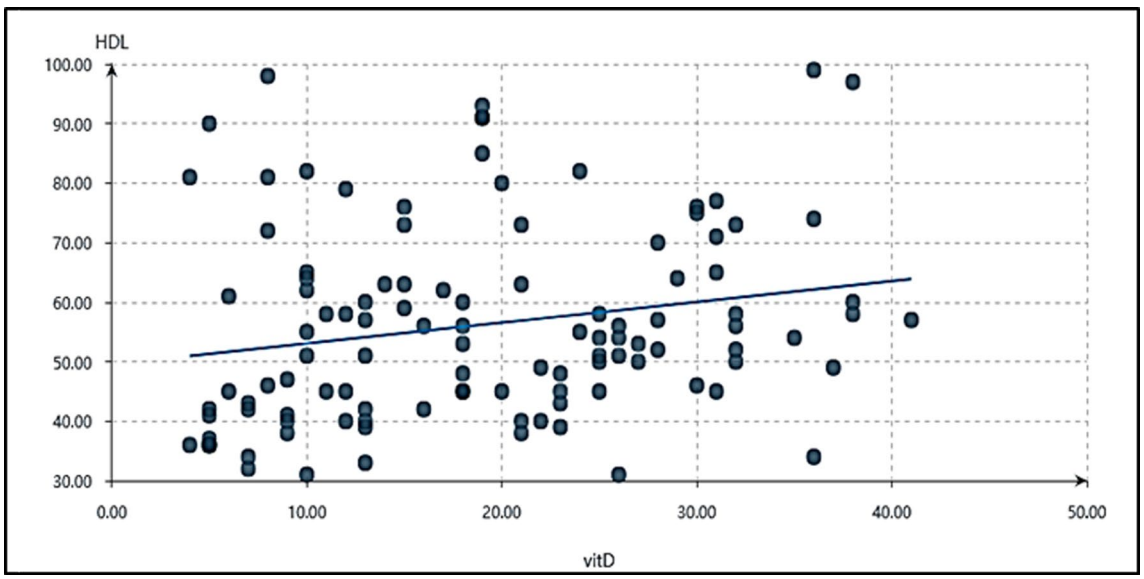

Fig. 9 Scatter plot of the correlation between S. vitamin D level and HDL (linear regression)

fact that 35.5% of patients receiving Fingolimod ($n=31$) have confluent T2 lesions in MRI brain which is already linked significantly to TC. This was in accordance with the study of Klingerberg and his colleagues [15] conducted on ApoE-deficient mice which received Fingolimod for 12 weeks and subsequently showed that there was a 2.4 fold increase in TC levels with a considerable elevation of the VLDL, while the TG levels were maintained with no changes.

The influence of serum vitamin D concentration on lipid metabolism in this study revealed indirect link to TC levels since we recognized that each 1 ng/mL increase in vitamin D was coupled with reduction in TC of 1.48 mg/dL with 95% CI was − 2.42 to − 0.54 and $P=0.002$. This was conforming with the study of Sriram and his colleagues [3] which noted that each 1 ng/mL vitamin D elevation was linked with reduction in TC of 1.38 mg/dL with 95% CI was − 2.63 to − 0.14 and

$P=0.03$. While there was a positive significant correlation between vitamin D and HDL levels ($P=0.028$) and this was in agreement with the study done by Ge and his colleagues [16] that declared a direct link to HDL levels ($P<0.001$).

Calcium absorption was recognized to be regulated by vitamin D. It has been proposed that the more the level of serum calcium, the less hepatic TG formation and intestinal absorption of fatty acid. Thus, vitamin D insufficiency may influence lipid absorption through calcium level. Moreover, studies have claimed that vitamin D level is capable of altering insulin sensitivity through its effect on β islet-cell function which is definitely impactful on lipid metabolism [16].

Conclusion

A considerable number of Egyptian MS patients have vitamin D deficiency and dyslipidemia. As a consequence, their abnormalities were associated with higher annualized relapse rate with both but higher disability scores and load of brain T2 lesions with dyslipidemia only. Increasing vitamin D serum level was positively linked to higher HDL and negatively with TC. Incorporation of these modifiable risk factors into management plan could prevent disease exacerbation.

Abbreviations

ANOVA	Analysis of variance
ARR	Annualized relapse rate
CI	Confidence interval
DMD	Disease-modifying drugs
EDSS	Expanded disability status scale
HDL	High density lipoprotein
HS	Highly significant
LDL	Low density lipoprotein
MHC	Major histocompatibility complex
MRI	Magnetic resonance imaging
MS	Multiple sclerosis
NCEP	National Cholesterol Education Program
NS	Non-significant
RRMS	Relapsing–remitting multiple sclerosis
S	Significant
SD	Standard deviation
TC	Total cholesterol
TG	Tri-glycerides
VDR	Vitamin D receptor

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Author contributions

MS and JM collect the data and analyse it including statistical analysis. MA analysed the data, drafted the manuscript for intellectual content and was a major contributor in writing the manuscript. AA and SS analysed the data and drafted the manuscript for intellectual content, and all authors read and approved the final manuscript.

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We did not receive fund for this project.

Availability of data and materials

The data sets generated during and/or analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

This manuscript was approved from the local Ethical Committee of Faculty of medicine, Ain shams university FWA 000017585 date 25/3/2023. The faculty of medicine, Ain shams university research ethics committee certifies that this observation research of minimal risk extracted from a master thesis, is approved from ethical point of view. However, it was not eligible for review by the committee before its initiation according to faculty authorities' decision in 2019. All patients signed consents by themselves to use their tests in research purposes.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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