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Impact of Vitamin D Deficiency on the Efficacy of Atorvastatin in Iraqi Patients with Dyslipidemia

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Abstract

Dyslipidemia is a distribution of lipid levels in the blood, which may involve elevated cholesterol and triglycerides. The response of dyslipidemia to statin therapy is diverse, ranging from 5-70% and this may be related to vitamin D3 deficiency. A study aims to assess the effect of vitamin D3 deficiency on the therapeutic response to atorvastatin. The cohort study involved 243 dyslipidemic patients who received different doses of atorvastatin according to the 2018 American Heart Association/American College of Cardiology cholesterol guidelines. The serum lipid profile level, vitamin D3, and atherogenic index were measured. After three months of atorvastatin therapy, responded patients exhibited a significant reduction in total cholesterol, LDL-cholesterol, and atherogenic index, and a significant increase in HDL-cholesterol compared with non-responded patients ($P < 0.05$). Serum vitamin D3 levels were significantly lower in non-responded patients, and vitamin D3 deficiency was significantly associated with reduced atorvastatin efficacy (95% CI: 0.759–0.849; $P < 0.05$). In conclusion, Vitamin D3 deficiency significantly reduces the effectiveness of atorvastatin in Iraqi patients with dyslipidemia, as reflect by an elevated atherogenic index
Keywords: Atherogenic index, Atorvastatin, Cholesterol, Dyslipidemia, Vitamin D3.

Introduction

Dyslipidemia is a distribution of fats in the blood, involving elevated total cholesterol (TC) and triglycerides (TG). This condition is linked to disruption in the transport of cholesterol CHOL and TG. Impairment in lipoprotein production or function can lead to increased levels of undesired CHOL (TC, TG, low-density lipoprotein-cholesterol (LDL-C), very low-density lipoprotein-cholesterol (VLDL-C)) and decreased levels of helpful CHOL (high-density lipoprotein-cholesterol (HDL-C)). The development of dyslipidemia is influenced by genetics, diet, lifestyle, and underlying health conditions [(1)]. Elevated CHOL can have serious consequences, with 4 million deaths in 2023 attributed to high LDL-C [(2)]. As a predisposing factor for cerebrovascular and cardiovascular diseases, it often contributes to both morbidity and mortality [(3)]. Vitamin D3 is crucial in the control of lipid metabolism, including modifying the lipid metabolism imbalance associated with the inflammatory pathway, affecting the metabolism of TC, TG, and LDL-c by promoting bile salt synthesis, regulating genes to maintain CHOL balance, and inhibiting the activity of lecithin-cholesterol acyltransferase [(4)]. The response of dyslipidemia to statin therapy varies widely, from 5% to 70% [(5)]. This variability may be related to several factors, including racial ancestry, pharmacokinetic parameters of statins, enzymes involved in cholesterol biosynthesis, and the lipoprotein metabolic pathway [(6)].

Vitamin D3 not only maintains bone function but also improves insulin resistance, inhibits inappropriate activity of the renin-angiotensin pathway, supports immunity, and provides anti-inflammatory effects [(7)]. Numerous studies have demonstrated a link between vitamin D3 insufficiency and the onset of dyslipidemia. Consequently, sustaining optimal vitamin D3 levels may aid in lowering LDL-C levels and elevating HDL-C levels [(8, 9)]. Vitamin D3 acts by inhibiting the rate-limiting step, suppressing 3-hydroxy-3-methylglutaryl coenzyme A reductase (HMG-CoA), which is required for cholesterol biosynthesis and considered a pharmacological

target of statins[10, 11]. A deficiency of vitamin D3 significantly enhances the expression of HMG- CoA by upregulating sterol regulatory element-binding protein. Vitamin D3 may also play an essential role in reducing serum TG. It does so by decreasing hepatocyte TG production or secretion, possibly by influencing hepatocellular calcium [12]. Additionally, vitamin D3 may indirectly suppress parathyroid hormone levels, which in turn promotes peripheral removal of TG by enhancing lipolytic activity [13]. This study aims to evaluate how vitamin D3 deficiency might affect the therapeutic response to atorvastatin ATO in Iraqi patients with dyslipidemia, potentially through these outlined mechanisms.

Patients and Methods

Patients

Patients with dyslipidemia of both genders between the ages of 35 and 71 years old were chosen due to dyslipidemia screening, cardiovascular risk assessment, and pharmacologic intervention commonly occurring in these patients and diagnosed based on the 2018 AHA/ACC Cholesterol guideline [14]. These patients were randomly selected from the outpatient setting of the Cardiology Department and enrolled in this study after obtaining informed consent. Random selection was done to avoid selection bias and ensure that the study population was representative of the outpatient dyslipidemia cohort. During their participation, they received medical care and advice. The patients were included and received ATO therapy of different doses based on the roles of selected guidelines as follows:

- If the patient suffers from initial hypercholesterolemia (LDL-C ≥ 190 mg/dL), a high-intensity dose (40–80 mg/day) of ATO is initiated.
- If a patient aged 40–75, with or without diabetes and/or at least two risk factors (such as family history, hypertension, chronic kidney disease, women-related disorders like premature menopause, inflammatory disorders like rheumatoid arthritis or psoriasis, obesity, or smoking) for cardiovascular events and LDL-C ≥ 130 mg/dL, an intermediate dose (20 mg) of ATO once daily is started.
- If a patient aged 40–75 without diabetes and fewer than two risk factors for CVE, and with LDL-C ≥ 100 mg/dL, a low dose (10 mg) of ATO is started once daily.

Exclusion criteria were as follows: prior intolerance or hypersensitivity to statins, hepatic impairment, glycosylated hemoglobin (HbA1c) ≥ 10% because this level reflects poorly controlled diabetes with significant metabolic instability, which could substantially influence lipid metabolism, uncontrolled hypertension, gastrointestinal disorders affecting absorption, heavy alcohol intake or drug abuse, pregnancy or lactation, secondary hyperlipoproteinemia due to uncontrolled hypothyroidism, severe conditions such as cancer, recent coronary events or interventions (coronary angioplasty, bypass grafting, myocardial infarction, or unstable angina within three weeks before assessment). In addition, patients using vitamin D3 supplements or medications known to cause myopathy or liver dysfunction, such as cyclosporin, erythromycin, nefazodone, fluvoxamine, and other potent hepatic enzyme inhibitors, were also excluded.

Study design

The prospective cohort study was conducted from October 2023 to February 2024, with samples collected from the cardiology center at Imam Al Hussein Hospital in Kerbala Province, Iraq. To determine the study sample size, Fisher's formula [15] was used, based on a known target population of 653 patients with dyslipidemia, as reported by the cardiology department of Imam Al-Hussein Medical City. Subsequently, 243 patients were assessed individually using a questionnaire to obtain demographic data. To evaluate the intervention, the lipid profile was measured before the start of ATO and after 3 months of ATO use. Patients were then classified as responders or non-responders based on specific criteria aligned with AHA/ACC cholesterol guidelines. Responders were patients whose LDL-C levels, after 3 months of ATO therapy, fell below 100 mg/dL if they did not have CVE, or below 70 mg/dL for those aged 40–75 years with or without diabetes mellitus and/or with two or more CVE risk factors. Non-responders were patients whose LDL-C levels remained above these respective thresholds. Additionally, atherogenic risk was determined using the atherogenic or Castelli index (TC/HDL ratio): values ≥ 4.5 for men or ≥ 4 for women indicated increased risk and low ATO efficacy [16]. Based on these LDL-C and index criteria, each patient with dyslipidemia was ultimately designated as a responder or non-responder.

Assessment of body mass index

The body mass index (BMI) is calculated by measuring a patient's weight and height. BMI is specifically estimated by dividing body weight in kilograms (Kg) by the square of body height in meters (m). Based on the resulting value, a patient is considered obese if their BMI is 30 or greater.

Blood collection

Following an 8-hour fast, approximately 3 mL of blood was withdrawn from all patients and placed in a gel tube to obtain serum for assessment of the vitamin D3 and lipid profile before being treated with ATO, and blood was collected again after the use of ATO to assess the lipid profile.

Lipid profile and vitamin D3 assessment

The lipid profile levels (TC, TG, and HDL-C) in serum were measured using a single differentiated biochemical testing device, a Fujifilm Dri-Chem NX500 Apparatus and Kits (Germany). The values of LDL-C and VLDL-C could be identified based on the values of TC, TG, and HDL according to the Friedewald equation [17].

$$\mathbf{LDL-C\ (mg/dl)\ = TC(mg/dl)\ -\ HDL-C(mg/dl)} - \frac{\boldsymbol{TG}}{\mathbf{5}}\boldsymbol{(mg/dl)}$$

Following lipid profile analysis, the VLDL-C value can be estimated by dividing the TG value by 5 [18]. The serum level of vitamin D3 was then assessed using the Ichroma biotechnology technique with the Ichroma vitamin D3 kit and apparatus (Boditech Med Inc., Korea). Vitamin D3 level was classified according to its serum level as follows: deficiency if its serum level is <20 ng/dl, insufficient if its serum level is 20 – 30 ng/dl, and normal if its serum level is >30 ng/dl [19].

Statistical analysis

The Statistical Package for the Social Sciences (SPSS 26) was used for statistical analysis. Numerical data were represented by the mean and standard deviation (Mean ± SD) and assessed for normality using the Shapiro–Wilk test; an independent sample t-test was then applied to compare means between groups. Non-numerical data were represented by numbers and percentages and analyzed with the Chi-square test to compare group distributions. The binomial logistic regression assessed whether vitamin D3 deficiency was associated with low efficacy of atorvastatin. A P value < 0.05 was considered statistically significant.

Results and Discussion

Demographic data

Demographic data for participants, including age, gender, BMI, ATO dose strength, and CVE risk factors, were assessed. The mean ages for responders and non-responders were similar (54.68 ± 0.882 and 55 ± 0.796, respectively; P > 0.05). Males comprised 60% of responders and 59% of non-responders, with no significant difference between the groups (P > 0.05). Among patients with dyslipidemia who responded to ATO, about 69 % had a family history of the disorder. Smoking rates were comparable in both groups (48%). Obesity was significantly higher in non-responders (67.7%, P < 0.05) versus responders (38.1%). Hypertension was more common in non-responders (90.8%) than in responders (67.3%, P < 0.05). The distribution of CVE was present in 56.6% of non-responders versus 21.1% of responders (P < 0.05). Women-related disorders (medical conditions affected female reproductive system, including the uterus, ovaries, and menstruation function) appeared in 17 (15%) responders and 24 (18.5%) non-responders. Inflammatory disorders (status characterized by chronic activation of the inflammatory pathway, such as rheumatoid arthritis, inflammatory bowel disease, systemic lupus erythematosus, and chronic pelvic inflammatory disease) were more frequent in non-responders (26.9%) than in responders (6.2%, P < 0.05). Diabetes mellitus affected 46 (40.7%) responders and 66 (50.8%) non-responders. Chronic kidney disease was rare, found in 6 (4.6%) non-responders and 4 (3.5%) responders. The different ATO strengths were prescribed as follows: 10 mg (33 responders, 30 non-responders), 20 mg (54 responders, 63 non-responders), and 40 mg (26 responders, 37 non-responders), as shown in Table 1.

Table 1. Demographic data of dyslipidemic patients.

Variables			Dyslipidemic patients		*P*-value
			Responded (n=113)	Non-responded (n=130)	
Age (y)			54.68 ± 9.38	55 ± 9.08	0.788
Gender		Male	68 (60.2%)	77 (59.2%)	0.881
		Female	45 (39.8%)	53 (40.8%)	
BMI			31.61 ± 7.66	34.44 ± 7.13	0.003
Risk factors	Family history	No	35 (31%)	41 (31.5%)	0.925
		Yes	78 (69%)	89 (68.5%)	
	Smoking	No	58 (51.3%)	69 (53.1%)	0.785
		Yes	55 (48.7%)	61 (46.9%)	
	Obesity	No	70 (61.9%)	42 (32.3%)	<0.001*
		Yes	43 (38.1%)	88 (67.7%)	
	Hypertension	No	37 (32.7%)	12 (9.2%)	<0.001*
		Yes	76 (67.3%)	118 (90.8%)	
	CVE	No	89 (78.8%)	55 (42.3%)	<0.001*
		Yes	24 (21.2%)	75 (57.7%)	
	Women-related disorder	No	96 (85%)	106 (81.5%)	0.478

		Yes	17 (15%)	24 (18.5%)	
	Inflammatory disorder	No	106 (93.8%)	95 (73.1%)	<0.001*
		Yes	7 (6.2%)	35 (26.9%)	
	Diabetes mellitus	No	67 (59.3%)	64 (49.2%)	0.117
		Yes	46 (40.7%)	66 (50.8%)	
	Chronic kidney disease	No	109 (96.5%)	124 (95.4%)	0.674
		Yes	4 (3.5%)	6 (4.6%)	
ATO dose strengths		10 mg	33 (29.2%)	30 (23 %)	0.455
		20 mg	54 (47.8%)	63 (48.5%)	
		40 mg	26 (23%)	37 (28.5%)	

Independent t-test and Chi-square test, two-sided P-value <0.05, data present as mean ± SD and No (%), Women-related disorders (medical conditions affected female reproductive system, including uterus, ovaries, and menstruation function), Inflammatory disorders (status characterized by chronic activation inflammatory pathway, such as rheumatoid arthritis, inflammatory bowel disease, systemic lupus erythematosus, and chronic pelvic inflammatory disease), *: Significant effect (*P*<0.05) between the study groups.

Effect of vitamin D3 on lipid profile and efficacy of atorvastatin

The serum lipid profile was significantly improved after ATO use in dyslipidemic patients, as shown in Figure 1, at P < 0.05. For clarity, responded patients are defined as those who achieved a clinically meaningful reduction in TC and LDL-C levels, while non-responded patients did not reach such thresholds after ATO use. The serum level of bad lipid profile, including TC and LDL-C, was significantly reduced, and the serum level of HDL-C was significantly elevated in responded patients in comparison with non-responded patients at P <0.05. The atherogenic index was significantly higher in non-respondent patients compared to respondent patients at P < 0.05. The serum level of vitamin D3 was significantly lower in non-responded patients compared to responded patients at P <0.05. There was a significant association between vitamin D3 deficiency (95% CI 0.759 – 0.849), atherogenic index (95% CI 14.898 – 249.643), and a decrease in the efficacy of ATO at P <0.05, as explained in Tables 2 and 3. The very large odds ratio (OR) for the atherogenic index indicates a strong association with the vitamin D3 deficiency and non-response to ATO, proposing that patients with high atherogenic index values and vitamin D3 deficiency were at significantly higher risk for CVE.

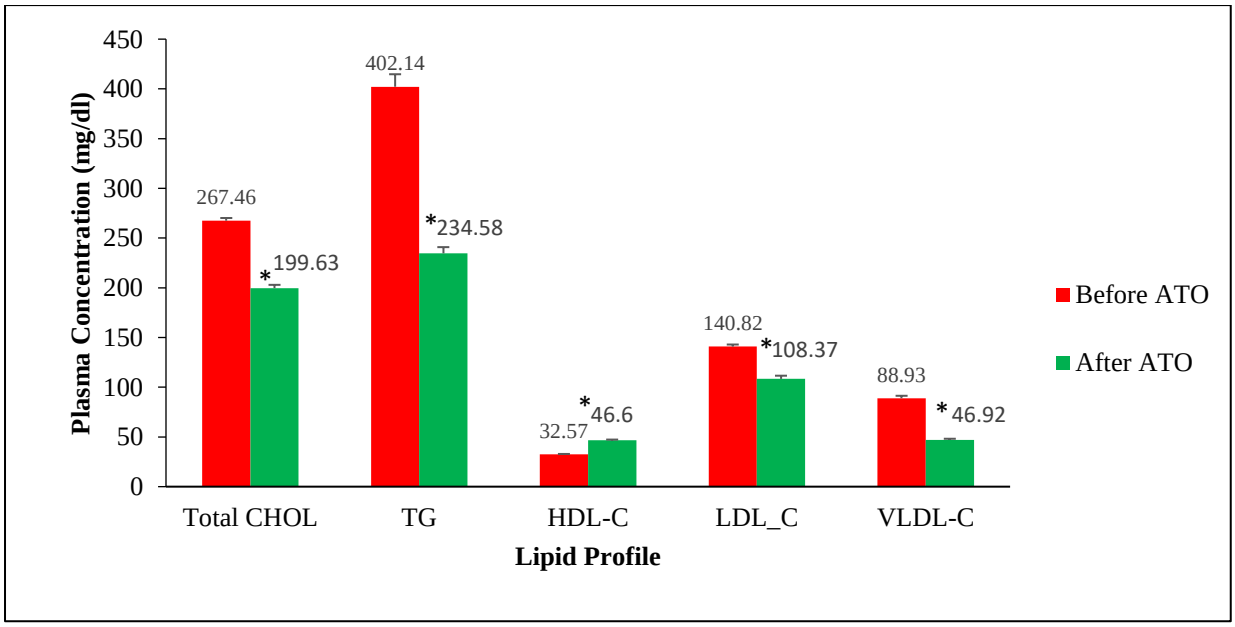

Figure 1. The serum level of lipid profile before and after ATO therapy, ATO: atorvastatin, *: Significant effect (P<0.05) between before and after using ATO.

Table 2. The serum level of lipid profile, vitamin D3, and atherogenic index of dyslipidemic patients treated with ATO.

Variables		Dyslipidemic patients		Reference range	*P* – value
		Responded (n=113)	Non-responded (n=130)		
Lipid profile	TC (mg/dl)	172.8 ± 43.89	224.25 ± 38.24	Normal <200 Moderate high 200-239 High ≥ 240	<0.001*
	TG (mg/dl)	246.08 ± 93.54	224.98 ± 92.11	Normal 10 -150 High >150	0.079
	HDL-C (mg/dl)	51.03 ± 11.94	41.81 ± 8.61	Normal >35 High ≥ 60 Low <35	<0.001*
	LDL-C (mg/dl)	77.06 ± 3.47 36.9	137.45 ± 35.68	Optimal <100 Above optimal 100 - 129 Borderline high 130 - 159 High 160 – 189 Very high ≥190	<0.001*
	VLDL-C (mg/dl)	49.22 ± 18.71	45 ± 18.42	Normal <30 High ≥ 30	0.078
Atherogenic index		3.59 ± 0.69	5.28 ± 0.71	For men: Normal <4.5 High risk ≥ 4.5 For women: Normal <4 High risk ≥ 4	<0.001*
Vitamin D3 level (ng/dl)		31.6 ± 8.99	17.17 ± 7.07	Normal >30 Insufficient 20-30 Deficiency <20	<0.001*
Status of vitamin D3	Normal	75 (66.4%)	3 (2.3%)		<0.001*
	Insufficient	30 (26.5%)	48 (36.9%)		
	Deficiency	8 (7.1%)	79 (60.8%)		

Independent t-test and Chi-square test, two-sided P-value <0.05, Data presented as mean ± SD, TC: total cholesterol, TG: triglyceride, HDL-C: high-density lipoprotein-cholesterol, LDL-C: low-density lipoprotein-cholesterol, VLDL-C: very low-density lipoprotein-cholesterol, *: Significant effect between the study groups.

Table 3. The binary logistic regression of predictors of response to ATO.

Variable	B	S.E.	Wald X^2	P -value	OR	95% CI for OR
Vitamin D3 deficiency	-0.153	0.043	12.964	<0.001*	0.858	0.789-0.932
Atherogenic index	4.147	0.717	33.494	<0.001*	63.252	15.528-257.646
Age	0.033	0.031	1.122	0.289	1.033	0.972-1.098
Gender	0.849	0.636	1.783	0.182	2.338	0.672-8.134
BMI	0.061	0.035	3.037	0.081	1.063	0.992-1.139
Constant	-19.614	4.235	21.449	<0.001*	<0.001	

Binary logistic regression, two-sided P-value <0.05, B: Unstandardized Coefficient, S.E.: standard error of B, OR: Odds Ratio, CI: Confidence interval, *: significant effect.

In this study, the serum lipid profile improved significantly after ATO treatment in dyslipidemic patients. Atorvastatin was a more effective, high-intensity statin than simvastatin for reducing LDL-C [20]. At therapeutic doses (10 – 80 mg), ATO could reduce the plasma level of LDL by approximately 40% [21]. This study demonstrated a significant difference in the response of dyslipidemic patients to ATO. The variability in the cholesterol-lowering capacity of ATO may be related to several factors, including genetic variation

and non-adherence, which can lead to an increased incidence of CVE [22].

This study showed that the atherogenic index was significantly higher in dyslipidemic patients who received ATO and suffered from vitamin D3 deficiency, indicating a strong predictor of ATO efficacy. The atherogenic index is frequently used as a better marker to reflect the exact relationship between the effectiveness of treatment and the incidence of atherosclerosis and CVE [23]. A study mentioned that vitamin D3 deficiency was strongly associated with atherogenic dyslipidemia [24]. Another study found that there is a relationship between low serum vitamin D3 level and high serum total CHOL, TG, LDL-C in young adults and the elderly of the Chinese population; consequently, these suggesting the vitamin D3 deficiency is an indicator for increasing atherosclerosis-associated lipoprotein [25].

In this study, the non-responding patients exhibited a significantly lower level of serum vitamin D3 compared to responding patients. Researchers demonstrated that patients with low serum vitamin D3 showed no significant reduction in LDL-C following treatment with rosuvastatin for a sufficient period [26]. In a small population study, when hyperlipidemic patients received a vitamin D3 supplement in addition to being treated with stable doses of statin exhibited a significant reduction in the TC, TG, and LDL-C [27]. A study mentioned that the elevation of vitamin D may quickly reduce the activity of the 7DHC reductase enzyme, thereby enhancing the efficacy of statin drugs [28]. Vitamin D3 plays a vital role in maintaining the normal function of the cardiovascular system, and it has many cardioprotective activities, including anti-inflammatory and antithrombotic properties; thus, it enhances the anti-atherogenic action of ATO [29].

Limitation of study

We acknowledge that the study was conducted at a single tertiary cardiology center, which may limit the generalizability of the findings to other populations or healthcare settings. The period of three months for follow-up may be insufficient to capture long-term lipid-lowering responses. Studies involving a large sample size are needed to get more precise estimates and to further the association of vitamin D3 deficiency, response to ATO, and atherogenic index

Conclusion

This study concludes that the vitamin D3 deficiency significantly reduced the effectiveness of ATO in Iraqi patients with dyslipidemia, as reflected by an elevated atherogenic index

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Conflicts of Interest

The authors declare that they have no conflicts of interest.

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Ethics Statements

The study was approved by the scientific and ethical committee, Kerbala University, College of Pharmacy, with the project being assigned No: 2022HU9 on 3/11/2022, in addition to patient approval.

Author Contribution

AA. designed the study, discussed the results and strategy, and made statistical analysis; **AZ** and **FM** collected data; and **MQ** followed up with the patients and wrote the manuscript

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تأثير نقص فيتامين د٣ على الفعالية العلاجية للأتورفاستاتين في المرضى العراقيين المصابين بعسر شحميات الدم

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الخلاصة

عسر شحميات الدم هو مرض يمتاز بوجود مستويات غير طبيعي من الدهون في الدم، وقد يشمل الكوليسترول والدهون الثلاثية. تتفاوت استجابة المرضى المصابين بعسر شحميات الدم للعلاج بالستاتين، حيث تتراوح بين ٥٪ و٧٠٪، وقد يرتبط ذلك بنقص فيتامين د٣. تهدف هذه الدراسة إلى تقييم تأثير نقص فيتامين د٣ على الاستجابة العلاجية للأتورفاستاتين. شملت الدراسة ٢٤٣ مريضًا يعانون من عسر شحميات الدم تلقوا جرعات مختلفة من الأتورفاستاتين وفقًا لتوصيات جمعية القلب الأمريكية/الكلية الأمريكية لأمراض القلب لعام ٢٠١٨ بشأن الكوليسترول. تم قياس مستوى الدهون وفيتامين د٣ في مصل الدم، ومؤشر تصلب الشرايين. لوحظ انخفاض ملحوظ في مستوى الكوليسترول الكلي وكوليسترول البروتين الدهني منخفض الكثافة، ومؤشر تصلب الشرايين، بينما ارتفع مستوى كوليسترول البروتين الدهني عالي الكثافة بشكل ملحوظ لدى المرضى المستجيبين للعلاج مقارنةً بالمرضى غير المستجيبين ($P < 0.05$). كان مستوى فيتامين د٣ في مصل الدم منخفضًا بشكل ملحوظ لدى المرضى الذين لم يستجيبوا للعلاج مقارنةً بالمرضى الذين استجابوا له ($P < 0.05$). وُجد ارتباطٌ ذو دلالة إحصائية بين نقص فيتامين د٣ (CI 95% 0.759 – 0.849) وانخفاض فعالية الأتورفاستاتين ($P < 0.05$). وخلصت الدراسة إلى أن نقص فيتامين د٣ يُقلل بشكل ملحوظ من فعالية الأتورفاستاتين لدى المرضى العراقيين المصابين بعسر شحميات الدم.

الكلمات المفتاحية: مؤشر تصلب الشرايين، أتورفاستاتين، الكوليسترول، عُسر شحميات الدم، فيتامين د٣.