ORIGINAL ARTICLE

Impact of Low LDL-C on Steroidogenesis in Diabetic Patients Receiving Statin Treatment

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Objective: Steroidogenesis is associated with low-density lipoprotein (LDL) metabolism. The objective was to compare basal cortisol levels in statin-treated diabetic patients who had LDL-cholesterol (LDL-C) levels of less than 1.8 mmol/L (70 mg/dL) or 1.8 mmol/L or more.

Materials and Methods: Seventy-six statin-treated diabetic patients were divided into two groups of 38 patients each: low LDL-C (less than 1.8 mmol/L) and high LDL-C (1.8 mmol/L or more). Basal cortisol levels were measured, and adrenocorticotropic hormone (ACTH) stimulation tests were performed.

Results: The low LDL-C group had insignificantly higher basal cortisol levels. There were no significant differences in cortisol levels between the two groups at 20, 30, and 40 minutes during the 1-µg ACTH stimulation tests, nor among differing levels of statin intensity. There were also no significant differences in the proportion of patients with abnormal ACTH stimulation test results between low and high LDL-C groups.

Conclusion: The present data indicate that low LDL-C does not compromise steroidogenesis in diabetic patients receiving statin treatment. Aggressive LDL-C lowering in diabetic patients should be encouraged as indicated.

Keywords: Cortisol; Statin; Low LDL-C; Diabetes; ACTH

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Atherosclerotic cardiovascular diseases (ASCVD) remain the leading cause of morbidity and mortality worldwide. Epidemiological, clinical, and genetic studies have established the key role of low-density lipoprotein cholesterol (LDL-C) in atherosclerosis[1]; thus, all dyslipidemia management guidelines have LDL-C as the primary target of cholesterol-lowering therapies. The 2018 American Heart Association/American College of Cardiology (AHA/ACC) guideline on blood cholesterol management states that very high-risk patients with clinical ASCVD should receive high-intensity statin therapy to achieve 50% or more LDL-C reductions toward LDL-C levels of 1.8 mmol/L or less[2]. However, the 2019 and 2025 European Society of Cardiology/European Atherosclerosis Society (ESC/EAS) dyslipidemia management guidelines suggest a more aggressive LDL-C reduction. For primary or secondary prevention in very high-risk patients, e.g., having diabetes mellitus (DM) with target organ damage or at least three major risk factors, an LDL-C reduction 50% or more from baseline with an LDL-C goal of less than 1.4 mmol/L is recommended using high-intensity statins as first-line medications[3,4].

Cholesterol is the precursor for all adrenal and gonadal steroidogenesis, and its principal sources derive from LDL-C circulation as well as adrenal and gonadal de novo cholesterol synthesis. A previous study showed that 80 mg atorvastatin decreased all adrenal and gonadal steroids, whereas 10 mg ezetimibe combined with 10 mg atorvastatin had no impact on cortisol levels. In that study, no adrenocorticotropic hormone (ACTH) stimulation test was performed. Nevertheless, cortisol levels decreased after high-intensity atorvastatin treatment but remained within normal limits[5]. This may indicate that while a similar lowering of LDL can occur from using different lipid-lowering regimens,

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the effects on steroid hormone synthesis may vary in different contexts.

The primary goal of this study was to investigate whether statin-treated diabetic patients with low LDL-C levels would show any cortisol synthesis effects, and whether such alterations were detectable using the ACTH stimulation test, in comparison with patients who had high LDL-C levels. In addition, the secondary goal was to assess the influence of statin intensity on basal cortisol levels.

Materials and Methods

Patient population

Between October 2018 and January 2019, 76 patients were enrolled at Thammasat University Hospital, Pathum Thani, Thailand. Eligible participants were adults (aged 18 years or older) with type 2 diabetes mellitus (T2DM) who had been on a stable statin regimen for at least three months prior to enrollment and demonstrated stable LDL-C levels, defined as a variation of less than 30% compared with previous LDL-C measurements.

Patients with a history of steroid use for longer than six months prior, previous use of steroidogenesis inhibitors such as ketoconazole or etomidate, or oral contraceptive pills; or diagnosed with adrenal insufficiency, Cushing's syndrome, renal insufficiency with estimated GFR of less than 30 mL/minute/1.73 m^2, cirrhosis, severe hypoalbuminemia with serum albumin of less than 2.5 g/dL, or any acute illnesses; or having abnormal thyroid function tests were excluded. All patients provided written informed consent. The study was approved by the Human Research Ethics Committee of Thammasat University No. 1 (Faculty of Medicine) (MTU-EC-IM-2-074/61).

Study design

This was a cross-sectional study. At enrollment, baseline characteristics and lipid-lowering treatments were recorded, and laboratory assessments were performed, including total cholesterol (TC), LDL-C, high-density lipoprotein cholesterol (HDL-C), triglycerides (TG), fasting blood sugar (FBS), hemoglobin A1c (HbA1c), creatinine, albumin, and basal cortisol levels measured between 8:00 and 9:00 a.m. after an overnight fast. Statin intensity was defined by using the reduction percentage of LDL-C levels from baseline values: low (less than 30%), moderate (30% to 49%), and high (50% or more)[2]. Patients were divided into two groups, with 38 patients in each group. The low LDL-C group had LDL-C of less than 1.8 mmol/L, and the high LDL-C group had LDL-C of 1.8 mmol/L or more, depending on the previous LDL-C levels and LDL-C levels on the date of enrollment.

ACTH stimulation test

All patients had a 1-µg ACTH stimulation test immediately following the baseline blood tests. The 1-µg ACTH stimulation test was used to determine adrenal response. A 250-µg synthetic ACTH was diluted with 50 mL of 0.9% sodium chloride. Each 1 cc of solution contained 5 µg ACTH, and the prepared solution was preserved at 4°C. Blood samples for basal cortisol levels were obtained in a silent and bright room after five minutes of the patient resting alone. Samples were always drawn between 8 and 9 hours after an overnight fast. Afterwards, 1-µg synthetic ACTH was administered. To measure plasma cortisol levels, blood samples were drawn from a cannula inserted into an arm vein at 20, 30, and 40 minutes after ACTH administration, and cortisol concentrations were analyzed immediately. If the patient had a peak cortisol level after the 1-µg ACTH stimulation test, defined as less than 500 nmol/L (18 µg/dL), an abnormal response would be diagnosed, and the patient would then undergo the 250-µg ACTH stimulation test to confirm adrenal insufficiency at the next visit.

Laboratory analysis

The Siemens enzymatic method (Siemens Dimension RxL Max) was used to determine LDL-C (intra-assay coefficient of variation of 2.22% to 2.57%), HDL-C, TC, and TG levels. Cortisol levels were analyzed with a Roche Cobas 6000 analyzer using electrochemiluminescence (intra-assay coefficient of variation of 1.4%).

Statistical analysis

At least 76 patients with 38 patients per group were required to provide a power of 80% in detecting different mean cortisol levels between the low and high LDL-C groups at an alpha level of 0.05. All analyses were performed using Stata Statistical Software, version 15.1 (StataCorp LLC, College Station, TX, USA). Continuous data was presented as mean ± standard deviation (SD) or median (interquartile range, IQR). Categorical data were presented as percentages. Comparisons of age, body mass index (BMI), duration of diabetes, FBS, HbA1c, TC, TG, HDL-C, basal cortisol, and cortisol increment after ACTH stimulation between the

Table 1. Characteristics of participants

	LDL-C <1.8 mmol/L (n=38)	LDL-C ≥1.8 mmol/L (n=38)	p-value
Age (years); mean±SD	61.4±10.3	58.8±10.7	0.286
Male; n (%)	22 (57.9)	20 (52.6)	0.645
BMI (kg/m^2); mean±SD	27.0±5.0	27.8±4.5	0.458
Duration of DM (years); mean±SD	9.5±6.2	5.5±4.0	0.001
Established cardiovascular diseases; n (%)			
Coronary artery disease	0 (0.0)	1 (2.6)	1.000
Cerebrovascular accident	1 (2.6)	2 (5.3)	1.000
Hypertension; n (%)	30 (79.0)	30 (79.0)	1.000
Medication at enrollment; n (%)			
Antidiabetic drugs			
• Metformin	38 (100)	35 (92.11)	0.240
• Sulfonylurea	16 (42.1)	22 (57.9)	0.169
• Pioglitazone	15 (39.5)	15 (39.5)	1.000
• Insulin	7 (18.4)	5 (13.2)	0.529
• DPP-4 inhibitors	6 (15.8)	0 (0.0)	0.025
• SGLT2 inhibitors	3 (7.9)	0 (0.0)	0.240
• GLP-1RA	3 (7.9)	1 (2.6)	0.615
Statins			
• Low-intensity	5 (13.1)	2 (5.3)	0.263
• Moderate-intensity	22 (57.9)	19 (50.0)	
• High-intensity	11 (29.0)	17 (44.7)	
Fasting blood sugar (mmol/L); mean±SD	7.11±1.31	7.08±1.95	0.935
HbA1c (%); mean±SD	6.98±0.87	7.30±1.16	0.174
Total cholesterol (mmol/L); mean±SD	3.30±0.67	5.15±0.84	<0.001
Triglyceride (mmol/L); median (25th, 75th percentile)	1.08 (0.67, 1.34)	1.59 (1.19, 1.98)	<0.001
LDL-C (mmol/L); mean±SD	1.38±0.27	2.94±0.71	<0.001
HDL-C (mmol/L); mean±SD	1.38±0.40	1.42±0.34	0.614

ACEI/ARB=angiotensin converting enzyme inhibitors/angiotensin II receptor blocker; BMI=body mass index; DPP-4=dipeptidyl peptidase-4; GLP-1RA=glucagon-like peptide-1 receptor agonist; HbA1c=hemoglobin A1c; HDL-C=high-density lipoprotein cholesterol; LDL-C=low-density lipoprotein cholesterol; SGLT2=sodium-glucose cotransport protein 2; SD=standard deviation

low and high LDL-C groups were performed using independent t-tests. Gender, comorbidities, smoking habits, medications at time of enrollment, and results of the 1-µg ACTH stimulation tests were compared using Chi-square. Associations between LDL-C and basal cortisol levels were examined using simple correlation. ANOVA was used to compare statin intensity groups and basal cortisol levels. All p-values were two-sided, with a level of 0.05 considered to be statistically significant.

Results

In both the low and high LDL-C groups, participants were similar with respect to age, gender, BMI, comorbidities, types of antidiabetic drugs, statins, and glycemic levels (Table 1). The low LDL-C group had a mean LDL-C of 1.38±0.27 mmol/L, whereas the high LDL-C group had a mean LDL-C of 2.94±0.71 mmol/L (p<0.001).

Table 2 shows basal cortisol levels and 1-µg ACTH stimulation test results. The low LDL-C group had insignificantly higher basal cortisol levels. With regard to the ACTH stimulation test, there were no significant differences between cortisol levels at 20, 30, and 40 minutes. Interestingly, differences between basal cortisol and the cortisol levels at each time point (cortisol increment) were significantly higher in the high LDL-C group than those in the lower LDL-C group; however, any abnormal ACTH stimulation test results were not significantly different between these two groups (10.53% in the low LDL-C group versus 5.26% the high LDL-C group, p=0.674). Five out of six patients with abnormal responses to the 1-µg ACTH stimulation test had 250-µg ACTH stimulation tests performed later. Only one patient in the high LDL-C group had a suboptimal response to this latter test, a peak cortisol level of 455.5 nmol/L, but without clinical signs of adrenal insufficiency.

Table 2. Basal cortisol levels and results of 1-μg ACTH stimulation test

	LDL-C <1.8 mmol/L (n=38)	LDL-C ≥1.8 mmol/L (n=38)	p-value
Basal cortisol levels (nmol/L); mean±SD	332.16±107.04	321.68±120.28	0.686
Cortisol levels (nmol/L); mean±SD			
At 20 minutes	569.97±105.39	618.52±115.04	0.059
At 30 minutes	589.28±111.73	639.77±122.21	0.064
At 40 minutes	566.38±111.18	614.66±117.52	0.073
Cortisol increment[a] (nmol/L); mean±SD			
At 20 minutes	237.81±92.97	296.85±127.18	0.023
At 30 minutes	256.84±94.08	317.81±141.80	0.030
At 40 minutes	234.22±104.56	292.98±128.84	0.039
Abnormal 1-μg ACTH stimulation test; n (%)	4 (10.53)	2 (5.26)	0.674

ACTH=adrenocorticotropic hormone; LDL-C=low-density lipoprotein cholesterol; SD=standard deviation
(a) Differences between basal cortisol and timepoint cortisol levels

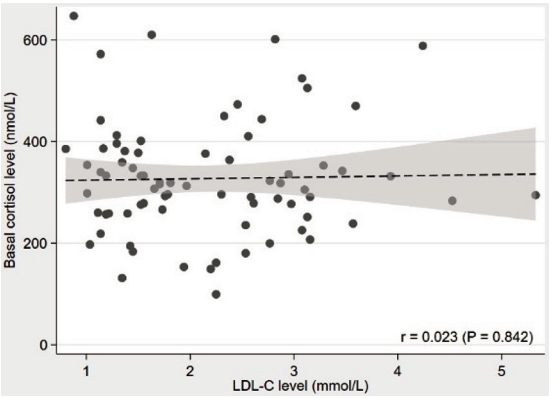

Figure 1. Relationship between LDL-C levels and basal cortisol levels; grey shadows 95% CI. LDL-C levels did not correlate with basal cortisol levels (r=0.023, p=0.842).

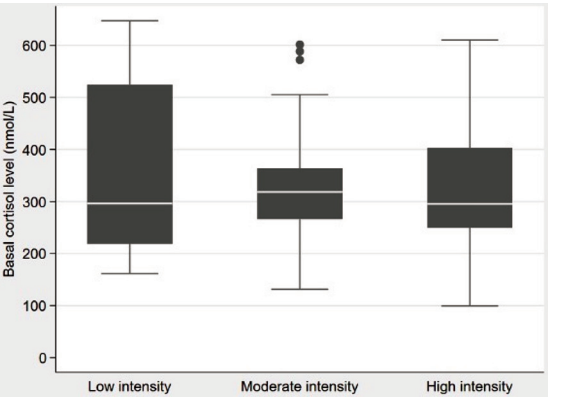

Figure 2. Effects of different intensity statins on basal cortisol levels. No significant differences in basal cortisol levels were evident within low-, moderate-, and high-intensity statin groups (p=0.694).

Relationships between LDL-C and basal cortisol levels are given in Figure 1. LDL-C levels did not correlate with basal cortisol levels (r=0.023, p=0.842); moreover, no significant differences in basal cortisol levels were evident within low-, moderate-, and high-intensity statin groups (p=0.694) (Figure 2).

Discussion

The present study is the first investigating both basal and stimulated cortisol functions in diabetic patients receiving statins with LDL-C levels of less than 1.8 mmol/L. The main finding showed there were no significant differences in basal cortisol levels between participants on statins with LDL-C of less than 1.8 mmol/L and LDL-C of 1.8 mmol/L or more. LDL-C levels did not seem to correlate with basal cortisol levels, and statin-treated diabetic patients with low LDL-C levels displayed no increases in adrenal insufficiency. There were also no significant differences in basal cortisol levels within the three different statin intensity groups.

It is generally accepted that cholesterol typically arises from circulating LDL-C[6]. As it is the precursor for adrenal steroidogenesis, cholesterol uptake occurs by specific cell surface LDL receptors present on adrenal tissue[7]. LDL is then internalized via receptor-mediated endocytosis[8], and the resulting vesicles fuse with lysozymes; free cholesterol is produced after hydrolysis and used for steroid hormone synthesis. However, it is obvious that this cannot be the sole source of adrenal cholesterol, as patients with defective LDL receptors with familial hypercholesterolemia and patients with abetalipoproteinemia who have very low or undetectable circulating LDL-C levels still maintain normal basal steroid synthesis.

Cholesterol can also be generated through a de novo pathway within the adrenal cortex from acetyl coenzyme A (CoA), controlled by 3-hydroxy-

3-methylglutaryl-CoA (HMG-CoA) reductase, the rate-limiting enzyme in cholesterol synthesis, and inhibited by statins[9,10]. Basal adrenal steroid synthesis is likely maintained due to this increased compensatory de novo cholesterol synthesis in patients with abetalipoproteinemia[11] and patients with familial hypercholesterolemia[12]. In contrast, adequate concentrations of LDL will suppress HMG-CoA reductase. There is additional evidence that the adrenal gland can utilize HDL-C after uptake through the putative HDL receptor, scavenger receptor class B type 1 (SR-B1)[13].

The effects of statins on steroid hormone synthesis have long been questioned since statins have the ability to inhibit both hepatic and adrenal de novo cholesterol synthesis. From previous studies in patients with familial hypercholesterolemia or polygenic hypercholesterolemia, there appeared to be no effects from simvastatin 10 to 80 mg/day for various durations (from eight weeks to two years) on basal ACTH, including any response of cortisol to the ACTH stimulation test, compared to baselines or control groups[14-18]. On a note, though, studies have mentioned an increase in basal ACTH and lower peak cortisol after the ACTH stimulation test. These findings would suggest there may be some impairment of adrenocortical reserve by simvastatin[19].

Previous studies observing non-diabetic patients with hypercholesterolemia and coronary artery disease have found that mean LDL-C levels in high-dose statin or ezetimibe-added on statin treatment groups were less than 1.8 mmol/L, at about 1.5 mmol/L. These studies showed there were no effects of treatments on basal cortisol and ACTH levels in those with low LDL-C levels, including any response of cortisol to the ACTH stimulation test, when compared to baselines or control groups[20-22]. Further research in diabetic patients demonstrated that moderate-intensity atorvastatin (20 mg/day) had no effect on basal cortisol levels when mean LDL-C levels were above 1.8 mmol/L[23]. Another study in diabetic patients reported that high-intensity atorvastatin (80 mg/day) was associated with lower basal cortisol levels compared to moderate-intensity atorvastatin (10 mg/day) combined with ezetimibe. However, these levels remained within the normal range. Mean LDL-C levels were below 1.8 mmol/L in both treatment arms[5]. It should be noted that ACTH stimulation tests were not performed in either of the last two studies[5,23].

The present data demonstrated there were no significant differences in basal cortisol levels and abnormal ACTH stimulation test results between diabetic patients on statins with LDL-C of less than 1.8 mmol/L and LDL-C of 1.8 mmol/L or more, regardless of statin intensity. However, the number of patients with abnormal 1-µg ACTH stimulation tests in the low LDL-C group was twice that in the high LDL-C group, although this difference was not statistically significant. Only one patient in the high LDL-C group had an abnormal 250-µg ACTH stimulation test result. This was without clinical signs of adrenal insufficiency. The results indicate that there is no increase in adrenal insufficiency in statin-treated diabetic patients with low LDL-C levels, consistent with previous studies. The reason for the sufficient adrenal steroidogenesis in the low LDL-C group may be that those participants had normal HDL-C levels, similar to the high LDL-C group. There is also the potential of in vivo adrenal synthesis.

Research on proprotein convertase subtilisin kexin type 9 (PCSK9) inhibitors demonstrated that there was no evidence of hypoadrenalism (decreased cortisol, increased ACTH, or a decreased cortisol to ACTH ratio) in evolocumab-treated patients, even at very low LDL-C levels (less than 1 mmol/L)[24]. This result was similar to a study on alirocumab[25]. These findings suggest that adrenal steroidogenesis is not critically dependent on LDL-mediated cholesterol delivery and that alternative pathways, such as endogenous synthesis and HDL-mediated delivery, can supply adequate cholesterol for steroid synthesis.

It is important to point out that the present study investigated both basal and stimulated cortisol functions by using the ACTH stimulation test in diabetic patients receiving statins, as few studies in non-diabetic patients examined the ACTH stimulation test in participants with LDL-C levels of less than 1.8 mmol/L[5,14,15,17-21,23]. However, limitations must be addressed, such as this cross-sectional study design did not help determine cause and effect. In addition, the definition of stable LDL-C in the present study was based on only a single measurement of LDL-C levels prior to enrollment. This may not represent a persistently low LDL-C level over an extended period. Future research could be improved by considering a longer duration of stable LDL-C.

Conclusion

The present study showed that there were no significant differences in basal cortisol levels between diabetic patients on statins with high or low LDL-C levels. Furthermore, LDL-C levels did not appear

to correlate with basal cortisol levels, and statin-treated diabetic patients with low LDL-C levels demonstrated no increases in adrenal insufficiency. These results confirm that diabetic patients with LDL-C levels of less than 1.8 mmol/L can safely receive statins without notable compromised adrenal steroidogenesis. Alternative pathways could potentially compensate for low levels of LDL-C to preserve normal adrenal steroidogenesis.

What is already known about this topic?

Cholesterol is the precursor for all adrenal steroidogenesis, and its principal sources derive from LDL-C circulation.

What does this study add?

Low LDL-C does not compromise steroidogenesis in diabetic patients receiving statin treatment.

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Authors' contributions

PA, TT, and AB designed the study. PA and TT conducted the study. PA, TT, AB, and PT drafted the manuscript. PA and PT analyzed the data. All authors reviewed and approved the final manuscript.

Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Ethics approval and consent to participate

The study was approved by the Human Research Ethics Committee of Thammasat University (Faculty of Medicine) (MTU-EC-IM-2-074/61) and was performed in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards (number of COA: 120/2561). Written informed consent was obtained from all participants prior to their inclusion in the study.

Clinical trial registration

Not applicable.

Use of artificial intelligence

No artificial intelligence tools were used to generate this manuscript.

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Conflicts of interest

The authors declare no conflict of interest.

References

1. Ference BA, Ginsberg HN, Graham I, Ray KK, Packard CJ, Bruckert E, et al. Low-density lipoproteins cause atherosclerotic cardiovascular disease. 1. Evidence from genetic, epidemiologic, and clinical studies. A consensus statement from the European Atherosclerosis Society Consensus Panel. Eur Heart J 2017;38:2459-72.
2. Grundy SM, Stone NJ, Bailey AL, Beam C, Birtcher KK, Blumenthal RS, et al. 2018 AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA Guideline on the management of blood cholesterol: executive summary: A report of the American College of Cardiology/American Heart Association Task Force on clinical practice guidelines. Circulation 2019;139:e1046-81.
3. Mach F, Baigent C, Catapano AL, Koskinas KC, Casula M, Badimon L, et al. 2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk. Eur Heart J 2020;41:111-88.
4. Mach F, Koskinas KC, Roeters van Lennep JE, Tokgözoğlu L, Badimon L, Baigent C, et al. 2025 Focused update of the 2019 ESC/EAS Guidelines for the management of dyslipidaemias. Eur Heart J 2025;46:4359-78.
5. Kanat M, Serin E, Tunckale A, Yildiz O, Sahin S, Bolayirli M, et al. A multi-center, open label, crossover designed prospective study evaluating the effects of lipid lowering treatment on steroid synthesis in patients with Type 2 diabetes (MODEST Study). J Endocrinol Invest 2009;32:852-6.
6. Gwynne JT, Strauss JF 3rd. The role of lipoproteins in steroidogenesis and cholesterol metabolism in steroidogenic glands. Endocr Rev 1982;3:299-329.
7. Faust JR, Goldstein JL, Brown MS. Receptor-mediated uptake of low density lipoprotein and utilization of its cholesterol for steroid synthesis in cultured mouse adrenal cells. J Biol Chem 1977;252:4861-71.
8. Goldstein JL, Anderson RG, Brown MS. Coated pits, coated vesicles, and receptor-mediated endocytosis. Nature 1979;279:679-85.
9. Arlt W, Stewart PM. Adrenal corticosteroid biosynthesis, metabolism, and action. Endocrinol

Metab Clin North Am 2005;34:293-313, viii.
10. Borkowski AJ, Levin S, Delcroix C, Mahler A, Verhas V. Blood cholesterol and hydrocortisone production in man: quantitative aspects of the utilization of circulating cholesterol by the adrenals at rest and under adrenocorticotropin stimulation. J Clin Invest 1967;46:797-811.
11. Illingworth DR, Kenny TA, Orwoll ES. Adrenal function in heterozygous and homozygous hypobetalipoproteinemia. J Clin Endocrinol Metab 1982;54:27-33.
12. Illingworth DR, Lees AM, Lees RS. Adrenal cortical function in homozygous familial hypercholesterolemia. Metabolism 1983;32:1045-52.
13. Landschulz KT, Pathak RK, Rigotti A, Krieger M, Hobbs HH. Regulation of scavenger receptor, class B, type I, a high density lipoprotein receptor, in liver and steroidogenic tissues of the rat. J Clin Invest 1996;98:984-95.
14. Mol MJ, Stalenhoef AF, Stuyt PM, Hermus AR, Demacker PN, Van 'T Laar A. Effects of inhibition of cholesterol synthesis by simvastatin on the production of adrenocortical steroid hormones and ACTH. Clin Endocrinol (Oxf) 1989;31:679-89.
15. Prihoda JS, Pappu AS, Smith FE, Illingworth DR. The influence of simvastatin on adrenal corticosteroid production and urinary mevalonate during adrenocorticotropin stimulation in patients with heterozygous familial hypercholesterolemia. J Clin Endocrinol Metab 1991;72:567-74.
16. Bernini GP, Argenio GF, Gasperi M, Vivaldi MS, Franchi F, Salvetti A. Effects of long-term simvastatin treatment on testicular and adrenal steroidogenesis in hypercholesterolemic patients. J Endocrinol Invest 1994;17:227-33.
17. Dobs AS, Schrott H, Davidson MH, Bays H, Stein EA, Kush D, et al. Effects of high-dose simvastatin on adrenal and gonadal steroidogenesis in men with hypercholesterolemia. Metabolism 2000;49:1234-8.
18. Travia D, Tosi F, Negri C, Faccini G, Moghetti P, Muggeo M. Sustained therapy with 3-hydroxy-3-methylglutaryl-coenzyme-A reductase inhibitors does not impair steroidogenesis by adrenals and gonads. J Clin Endocrinol Metab 1995;80:836-40.
19. Mol MJ, Stalenhoef AF. Adrenocortical function in patients on simvastatin. Lancet 1990;335:412-3.
20. Krysiak R, Okopien B. The effect of aggressive rosuvastatin treatment on steroid hormone production in men with coronary artery disease. Basic Clin Pharmacol Toxicol 2014;114:330-5.
21. Krysiak R, Kowalska B, Żmuda W, Okopień B. The effect of ezetimibe-statin combination on steroid hormone production in men with coronary artery disease and low cholesterol levels. Pharmacol Rep 2015;67:305-9.
22. Sezer K, Emral R, Corapcioglu D, Gen R, Akbay E. Effect of very low LDL-cholesterol on cortisol synthesis. J Endocrinol Invest 2008;31:1075-8.
23. Santini SA, Carrozza C, Lulli P, Zuppi C, CarloTonolo G, Musumeci S. Atorvastatin treatment does not affect gonadal and adrenal hormones in type 2 diabetes patients with mild to moderate hypercholesterolemia. J Atheroscler Thromb 2003;10:160-4.
24. Blom DJ, Djedjos CS, Monsalvo ML, Bridges I, Wasserman SM, Scott R, et al. Effects of evolocumab on vitamin E and steroid hormone levels: Results from the 52-week, phase 3, double-blind, randomized, placebo-controlled DESCARTES study. Circ Res 2015;117:731-41.
25. Robinson JG, Rosenson RS, Farnier M, Chaudhari U, Sasiela WJ, Merlet L, et al. Safety of very low low-density lipoprotein cholesterol levels with alirocumab: Pooled data from randomized trials. J Am Coll Cardiol 2017;69:471-82.