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Lipid profile abnormalities associated with endocrine disorders

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Abstract

Nearly 30% of patients with lipid profile abnormalities suffer from secondary dyslipidaemias. Endocrine disorders are one of the most important causes of dyslipidaemia. Dyslipidaemia can be observed in the pathologies of a variety of endocrine glands, including the thyroid, the pituitary, the adrenals, and the gonads. The most common endocrinopathy causing dyslipidaemia is hypothyroidism.

In this paper, we review the lipid profile alterations observed in endocrinopathies. We describe changes in classic lipid profile parameters, including total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, and triglycerides. However, we also focus on the influence of endocrine disorders on relatively new cardiovascular markers such as apolipoprotein B, apolipoprotein A1, and lipoprotein(a). While almost all endocrinopathies cause detrimental changes to the lipid profile, hyperthyroidism seems to be a disorder in which lowering of such parameters as total cholesterol, low-density cholesterol, and triglycerides can be observed. Comprehensive screening for endocrine disorders should always be included in the differential diagnostic process of secondary causes of dyslipidaemia. Early detection and treatment of endocrinopathy have a considerable impact on a patient's health. Proper treatment of those disorders plays a crucial role in modifying the cardiovascular risk and improving the lipid profile of those patients. Even though lipid-lowering therapy is usually still needed, in some cases restoration of hormonal balance might be sufficient to normalize the lipid profile abnormalities. **(Endokrynol Pol 2022; 73 (5): 863–871)**

Key words: *dyslipidaemia; endocrine diseases; hyperthyroidism; hypothyroidism, acromegaly; Cushing's disease*

Introduction

Dyslipidaemia often occurs in a spectrum of endocrine disorders. It can be caused by pathologies of the thyroid, the pituitary, the adrenals, or the gonads. Nearly 30% of patients with lipid profile abnormalities suffer from secondary dyslipidaemias, including type 2 diabetes mellitus, alcohol abuse, and several endocrine disorders. Endocrine diseases (excluding diabetes) cause up to 13% of secondary dyslipidaemias [1]. This shows the importance of excluding secondary causes of dyslipidaemia by practitioners. The abnormalities that can be observed in the lipid metabolism in the course of endocrine diseases are very broad and diverse. Thus the diagnosis of endocrinopathy should be primarily based on the clinical symptoms, and the lipid profile results can be regarded as a secondary aid in the diagnostic process [2].

In this paper, we review the lipid profile alternations observed in endocrinopathies. We describe changes in classic lipid profile parameters like total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides (TG). However, we also shed light on the influence of endocrine disorders on relatively new cardiovascular markers, such as apolipoprotein A1 (Apo A1), apolipoprotein B (Apo B), and lipoprotein(a) [Lp(a)]. Apo B is the primary apolipoprotein of atherogenic lipoproteins, including very low-density lipoprotein (VLDL), Lp(a), intermediate-density lipoprotein (IDL), and low-density lipoprotein (LDL). Apo B has 2 major isoforms: Apolipoprotein B-48 (Apo B48), mainly expressed in the intestine, and Apolipoprotein B100 (Apo B-100), which is expressed in the liver [3, 4]. Apo B can be used as a marker of cardiovascular risk, and according to recent data, it is a more reliable indicator of the risk than LDL-C [5]. Apo A1 is the main component in high-density lipoprotein [6], and it is considered to be a factor inversely corelated with the risk of atherosclerosis and cardiovascular disease [7]. Lp(a) is a lipoprotein particle produced in the liver, which constitutes an independent risk factor for cardiovascular disease [8].

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Table 1. *The most common endocrinopathies and their influence on lipid profile [29–96]*

Endocrinopathy	TC	LDL-C	HDL-C	TG
Hyperthyroidism	↓	↓	⇔	↓
Hypothyroidism	↑	↑	⇔/↑	↑
Cushing's syndrome	↑	↑	⇔/↑	↑
Testosterone deficiency	↑	↑	↓	↑
Oestrogen deficiency	⇔	↑	⇔/↓	⇔
Polycystic ovary syndrome	↑	↑	↓	↑

TC — total cholesterol; LDL-C — low-density lipoprotein cholesterol; HDL-C — high-density lipoprotein cholesterol; TG — triglycerides

While almost all endocrinopathies cause detrimental changes in the lipid profile, hyperthyroidism seems to be a disorder in which lowering of such parameters as total cholesterol, low density cholesterol, and triglycerides can be observed. A detailed description of the effect of the most common endocrinopathies on lipid profile parameters follows in the main body of the text below. A short summary of changes in the classical lipid profile parameters can be viewed in Table 1, while the influence of chosen endocrinopathies on Lp(a), Apo B, and Apo A1 is illustrated in Figure 1.

Disorders of the thyroid gland

Hypothyroidism

Based on studies from the USA, Japan, and northern Europe, hypothyroidism occurs in 0.6–12% of women and 1.3–4.0% of men [9]. The risk of hypothyroidism increases with age. Hypothyroidism can be divided into primary, secondary, and tertiary (due to thyroid, pituitary, and hypothalamus dysfunction, respectively). Regardless of aetiology, hypothyroidism is always characterized by retardation of metabolic processes, which induces dyslipidaemia. When left untreated, disturbances in the lipid profile can increase the risk of serious, life-threatening cardiovascular events.

Overt hypothyroidism

According to the National Health and Nutrition Survey, the prevalence of overt hypothyroidism in the world is about 0.3% [10, 11]. Overt hypothyroidism results in increased total cholesterol (TC) and up to 30% elevation of LDL-C [12]. It is caused by the decreased catabolism

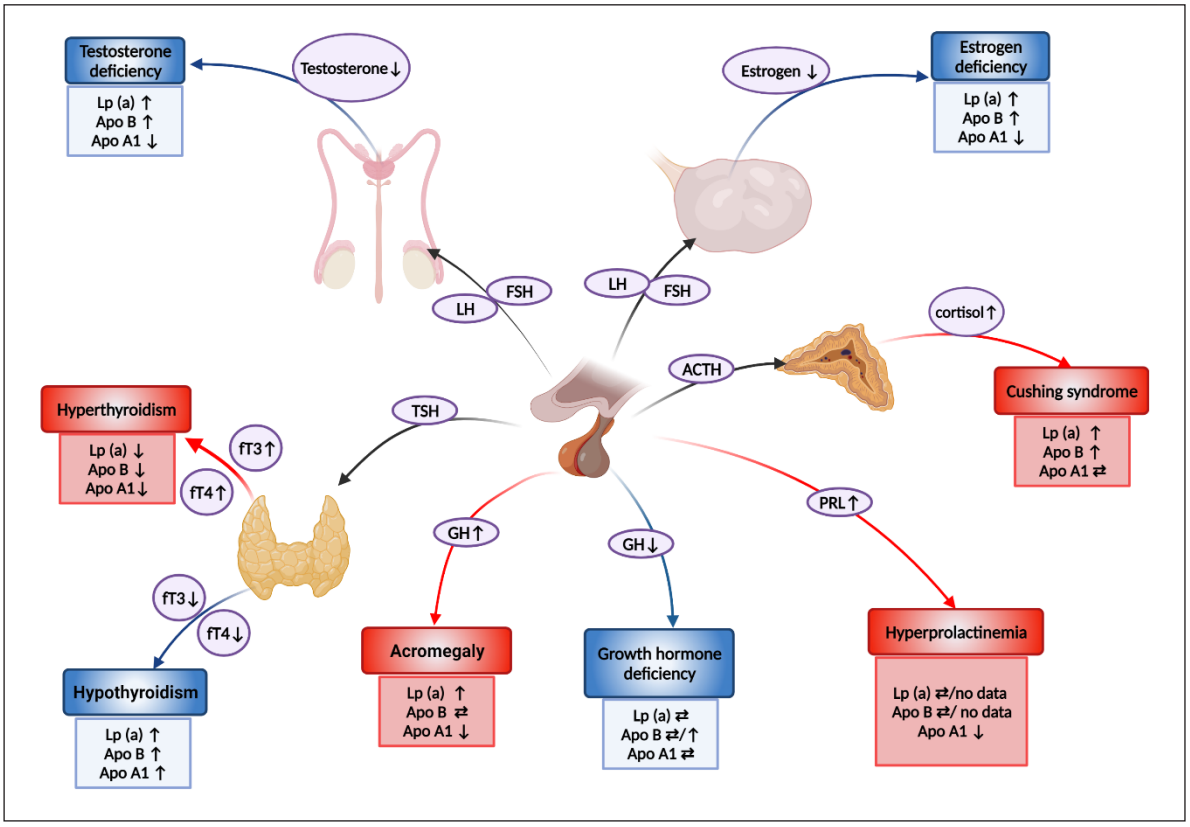

Figure 1. *Influence of selected endocrinopathies on lipoprotein a, apolipoprotein B and apolipoprotein A1 serum concentrations [29–96]. Lp(a) — lipoprotein a; Apo B — apolipoprotein B; Apo A1 — apolipoprotein A1; LH — luteinizing hormone; FSH — follicle-stimulating hormone; ACTH — adrenocorticotropic hormone; GH — growth hormone; TSH — thyroid stimulating hormone; fT3 — free triiodothyronine; fT4 — free thyroxine; PRL — prolactin. Created with BioRender.com*

of intermediate-density lipoprotein cholesterol (IDL-C) and LDL-C, which is related to reduced activity of receptors in the liver. Accumulation of LDL-C occurs due to a lower rate of bile acid synthesis from cholesterol, which is also regulated by thyroid hormones. Increased serum LDL-C levels lead to oxidation of LDL-C particles, which induces pro-inflammatory states, ultimately resulting in the progression of atherosclerosis. Goldberg et al. showed that hypothyroidism is related to an increase in Apo B levels, which increases intestinal absorption of cholesterol. Overt hypothyroidism slows the HDL-C metabolism, but the overall effect on HDL-C levels is relatively small and of uncertain clinical significance [13]. However, thyroid hormones seem to have effect on HDL-C particle size. They promote the synthesis of the smaller HDL2 particles, which convey more cardiovascular protection than the larger HDL3 particles. This is because in hypothyroidism the serum concentration of Apo A1 increases (they are a part of HDL2) and serum concentration of Apo A2 does not change (they are a part of HDL3) [14].

Other lipid profile disturbances include increased TG levels due to reduced lipoprotein lipase activity and increased serum concentration of Lp(a), which is one of the major factors increasing the cardiovascular risk [15].

Certain pathological findings like hypertension, increased arteries stiffness, endothelial dysfunction, increased homocysteine, uric acid, and phosphate levels tend to co-exist with hypothyroidism. Their occurrence further increases cardiovascular disease-related morbidity [16]. One of the major factors contributing to premature atherosclerosis might be increased levels of Apo B48, which are seen in both overt and subclinical hypothyroidism. Fortunately, they decrease after starting treatment with levothyroxine, and they correlate with the decrease in TSH [17, 18]. Thyroid hormone replacement therapy has an overall beneficial effect on the lipid profile and results in a decrease in TC, LDL-C, and Apo B, Lp(a). Apo A1, HDL-C, and TG levels do not change with treatment [16].

Lp(a) serum concentration, as some research suggests, might be partially modulated in a thyroid-related mechanism and continue to fall for many months after commencing levothyroxine therapy [19, 20].

It is important to rule out hypothyroidism in every patient with dyslipidaemia prior to commencing statin therapy. Starting statin therapy in overt hypothyroidism can increase the risk of muscle injury. The first line of treating dyslipidaemia in a hypothyroid patient is levothyroxine substitution therapy. After achieving euthyroidism, it is prudent to re-evaluate the lipid profile, and if dyslipidaemia persists despite levothyroxine treatment, then statin therapy should be commenced.

Subclinical hypothyroidism

Subclinical hypothyroidism is characterized by elevated TSH, normal free triiodothyronine (fT3) and free thyroxin (fT4) levels, and an asymptomatic or mildly symptomatic course. Subclinical hypothyroidism affects about 4.3–9% of the population, more commonly women, and its prevalence increases with age [16, 21]. The lipid profile disturbances comprise a rise in TC, LDL-C, Apo B, Lp(a), and TG. However, some studies did not find any changes in Lp(a) levels. Apo A1 and HDL-C levels do not seem to be affected in this disorder. Subclinical hypothyroidism is characterized by less evident changes in the lipid profile. The magnitude of dyslipidaemia is correlated with TSH levels [12, 22, 23]. Free thyroid hormones are negatively correlated with Apo B and Lp(a) serum concentrations in patients with subclinical hypothyroidism [24]. To date, there is much controversy regarding the treatment of subclinical hypothyroidism. Outcomes of studies are equivocal. Some studies confirm the positive effect of levothyroxine in lowering TC and LDL-C levels. No change in Lp(a), Apo A1, and Apo B serum concentrations has been observed after commencing levothyroxine therapy [22, 23, 25]. However, this kind of therapy does not significantly improve quality of life or reduce cardiovascular morbidity or improve survival [25]. The decision about starting levothyroxine therapy should be individualized based on the patient's clinical state, comorbidities, and risk of adverse events. Patients with higher TSH levels benefit more from levothyroxine therapy. A clear positive impact of levothyroxine on the abnormal lipid profile is seen in patients with TSH above 10 mIU/L [26].

It is also worth noting that, aside from a potentially positive effect on the lipid profile, levothyroxine treatment seems to improve both diastolic and systolic cardiac function, which are impaired in patients with subclinical hypothyroidism [25]. If normalization of lipid profile is not achieved after rendering the patient euthyroid, a lipid-lowering therapy (e.g. statins) should be applied.

Hyperthyroidism

The prevalence of hyperthyroidism in a population with adequate iodine intake is about 0.2-1.3% [27]. Women are affected more commonly than men [28]. Lipid profile changes observed in this clinical entity are different from those observed in hypothyroidism.

Overt hyperthyroidism

In the course of hyperthyroidism, lowering of LDL-C and TC levels is observed. In recent years, emerging evidence also suggests that the disease is associated with reduced levels of HDL-C, Apo A1, Apo B, and Lp(a).

Not only quantitative but also qualitative changes of various lipoproteins have been reported, e.g. increased LDL-C oxidation reflected by high levels of markers of lipid peroxidation [29].

Treatment with antithyroid drugs increases total cholesterol, LDL-C, HDL-C, Apo B, and Lp(a) but does not affect triglyceride levels [30]. It is worth mentioning that no difference in LDL-C particle size, which is associated with several cardiovascular risk factors, was found between hyperthyroid (including subclinical hyperthyroidism) and euthyroid individuals. Hyperthyroidism is also found to be an important reason for acquired hypobetalipoproteinaemia [31]. Even though patients with hyperthyroidism manifest beneficial changes in the lipid profile, restoration of euthyroidism removes this effect [16]. A low VLDL-C level (especially accompanied by symptoms of hyperthyroidism) should prompt clinicians to include this endocrinopathy in the process of differential diagnosis [32].

Subclinical hyperthyroidism

The effect of subclinical hyperthyroidism on the lipid profile is ambiguous. There are studies that show normal levels of LDL-C, TC, and TG in patients with subclinical hyperthyroidism. Nonetheless, other studies show a tendency to reduced levels of those parameters, despite the increased activity of HMG-CoA reductase. This might be a result of increased expression of genes coding the LDL-C receptor [16]. HDL-C levels are reduced in patients with subclinical hyperthyroidism due to increased cholesteryl ester transfer protein activity and hepatic lipase activity. The effect of subclinical hyperthyroidism treatment on the lipid profile is yet still not well established. It might be similar but smaller in magnitude because it is observed in overt hyperthyroidism; however, to date, only a few studies regarding this matter have been performed and more data are needed [16, 29].

Pituitary disorders

The pituitary gland consists of 3 parts (the anterior-glandular, intermediate, and posterior-neurohypophysis). The anterior lobe comprises somatotropic, corticotropic, thyrotropic, gonadotropic, and lactotrophic cells, which synthesize various hormones. The pituitary controls multiple vital organs, including the thyroid, the adrenal glands, and the gonads. It also controls such processes as growth, reproduction, and response to stress.

Hyperprolactinaemia

Prolactin is a polypeptide responsible for the growth of the mammary glands, and the commencing and sustaining of lactation in women. It can also influence the menstruation cycle [33].

Hyperprolactinaemia contributes to atherogenic dyslipidaemia by affecting various processes of lipid and glucose metabolism [34]. In hyperprolactinaemia, a rise in most lipid fractions, including TC and LDL-C, can be observed. HDL-C levels tend to be reduced, while TG stays unchanged or becomes slightly elevated. Patients with prolactin-secreting pituitary tumours were found to have increased waist circumference, BMI, fasting glucose, insulin resistance, TG, and Apo B/Apo A1 ratio and decreased Apo A1, compared to healthy subjects [35, 36]. Hyperprolactinaemia was also found to be associated with increased carotid artery intima media thickness and endothelial disfunction measured as flow-mediated dilatation. It is, however, still not clear if it is caused by a direct effect of prolactin or by promotion of atherogenic factors [35]. Men with prolactinoma are at higher risk of cardiovascular disease incidents. Interestingly, no such association was observed in females [36]. Macroprolactin is a non-active isoform of prolactin. One small study from Poland has shown that young women (20–45 years of age) with macroprolactinaemia have increased plasma levels of high-sensitivity C-reactive protein (hsCRP), higher glucose levels in the 2nd hours of the oral glucose tolerance test, higher insulin resistance (measured as HOMA1-IR), and reduced serum concentrations of HDL-C cholesterol and 25-hydroxyvitamin D. The mechanism of these abnormalities might be dissociation of the large macroprolactin complexes and the release of prolactin monomers in the peripheral tissue or the chronical stimulation of prolactin receptors by macroprolactin (which has a weak affinity to this receptor) [37].

Hyperprolactinaemia can be treated with dopamine agonists (e.g. bromocriptine, cabergoline). Treatment results mainly in total cholesterol and LDL-C level reduction [38].

Growth hormone deficiency

Growth hormone (GH) induces the production of somatomedins, also called insulin-like growth factors (IGF), in the liver [39]. Those molecules have a growth-stimulating effect on many cells, most notably on osteocytes and chondrocytes, which results in bone growth [40]. Growth hormone also plays a role in glucose metabolism by inducing glycogenolysis in the liver and stimulating endogenous insulin production from the pancreas [41]. In the adipose tissue, GH increases lipolysis and inhibits lipogenesis. Thus, GH deficiency can lead to dyslipidaemia. In GH deficiency, the levels of TC, LDL-C, and TG are increased, while HDL-C is reduced and Lp(a) levels are unaffected. However, some studies on the paediatric population show also

increased Lp(a) and Apo B levels compared to healthy individuals, while others show no difference in baseline serum lipid values [42]. Abnormal very low-density VLDL Apo B100 metabolism has also been reported in those patients. This involves an increased hepatic secretion rate of VLDL Apo B100 and reduced VLDL Apo B100 catabolism [43]. VLDL are precursors of intermediate-density lipoprotein (IDL) and LDL. Abnormalities in their metabolism, especially increased hepatic production, have been linked to premature atherosclerosis [44, 45]. Patients with GH deficiency tend to have increased blood pressure, which, together with the aforementioned lipid profile disturbances, leads to increased cardiovascular risk [46]. Adult patients with GH deficiency suffer from metabolic syndrome twice as often as healthy subjects [47].

Proper GH supplementation lowers TC and LDL-C levels. The effect on HDL-C and TG serum concentrations remains equivocal. Unfortunately, in adult patients, this effect is less evident than in children, and atherogenic dyslipidaemia persists despite treatment [48,49]. There is also a risk of a treatment-induced rise in Lp(a) [50, 51]. That is why treatment with hypolipidemic drugs should be considered according to the estimated cardiovascular risk. Replacement therapy in children leads to several improvements in the lipid profile. Some studies have shown a beneficial impact of GH supplementation on Apo A1 levels [52], while other found a decrease in LDL, IDL, non-HDL, and Apo B levels [53]. While the studies agree that GH supplementation has a beneficial impact on the lipid profile, the results regarding specific lipoprotein concentrations are not very consistent. The impact of GH supplementation on the lipid profile and its potential long-term benefits require further observation.

Acromegaly

Studies on dyslipidaemia in acromegaly deliver ambiguous results. Acromegalic patients tend to have normal total cholesterol and Apo B, either normal or increased LDL, increased TG and Lp(a), and decreased Apo A1 and HDL-C [54-56]. Increased TG and decreased HDL-C are the most common findings in acromegaly. An elevated TG level is a result of increased synthesis of fatty acids and reduced activity of lipoprotein lipase [57]. Lower HDL-C is a result of decreased activity of the lecithin-cholesterol acyl-transferase, which takes part in the process of free cholesterol esterification into HDL [58]. The aforementioned lipid profile disturbances further accelerate the progression of atherosclerosis, which is one of the factors responsible for increased cardiovascular risk [59]. The increased cardiovascular risk might be also related to altered physical properties of LDL. Acromegalic patients tend to have smaller and more dense LDL-C particles, in comparison with controls [60].

Lipid profile improvement is achieved through proper control of the disease. Thus, the best results are observed after successful surgical treatment. However, using somatostatin analogues also leads to a significant reduction in TG level as well as a rise in HDL-C. Furthermore, LDL-C and Lp(a) serum concentrations can also decrease [55, 61]. After cardiovascular risk estimation, additional cholesterol-lowering treatment should be considered (e.g. statin or fibrates - a priori to introducing fibrates, gallstones must be excluded).

Adrenal disorders

The adrenal glands consist of the outer cortex and the inner medulla. The cortex of the adrenals secretes mineralocorticoids, glucocorticoids, and androgens, while the medulla produces catecholamines (adrenaline, noradrenaline, dopamine).

Cushing's syndrome

Nearly 52% of patients with hypercortisolism suffer from secondary lipid abnormalities [62]. Excess of glucocorticoids causes an increase in serum TC, LDL-C, VLDL-C, and TG levels [63]. Regarding other lipoproteins, hypercortisolaemia causes a rise in Lp(a) levels and an increase in Apo B levels, while the effect on Apo A1 remains equivocal. The scale of changes observed in the lipid profile depends on the magnitude of hypercortisolaemia [64]. Typical clinical features of Cushing's syndrome include central obesity with characteristic moon facies, buffalo hump, thick, purple striae, and easy bruising [65, 66]. An increased amount of adipose tissue, especially visceral fat, increases the risk of metabolic syndrome occurrence. Most clinical features and laboratory abnormalities (e.g. reduction in TG and LDL-C levels and increase in HDL-C) can be reverted by the introduction of proper treatment. Not all changes are, however, entirely reversible; e.g. in a prospective study of 25 patients with Cushing's syndrome, a significant reduction of LDL-C serum concentration was observed one year after cortisol level normalization. However, the LDL-C level was still higher compared to a healthy control group [67]. Thus, even after successful treatment of hypercortisolaemia, a lipid-lowering therapy may be needed.

Exogenous glucocorticoids, which patients take because of their immunosuppressive and anti-inflammatory properties, also have a similar, detrimental effect on the lipid profile. The observed effect is dose dependent [68].

Gonadal disorders

Testosterone deficiency

Many studies have shown that testosterone deficiency is associated with HDL-C and Apo A1 serum concentrations. Patients with low testosterone tend to have lower HDL-C and Apo A1, and higher total cholesterol, LDL-C, Apo B, Lp(a), and TG levels. A strong negative correlation between testosterone and total cholesterol and triglyceride levels has been observed. The tendency to develop dyslipidaemia related to low testosterone levels was especially pronounced in young and middle-aged men [69]. Because of the aforementioned changes in the lipid profile, patients with testosterone deficiency are more susceptible to developing metabolic syndrome. It is, however, important to note that low testosterone serum concentration is not always the primary cause of metabolic syndrome. An important factor is obesity, which not only leads to the development of metabolic syndrome but also reduces testosterone serum concentration. Losing weight can normalize testosterone levels [70–73]. Testosterone replacement therapy generally has a beneficial impact on the quality of life and glucose metabolism, but its influence on the lipid profile is less prominent. Endogenous testosterone levels are associated with positive changes in the lipid profile, which include an increase in HDL-C and a decrease in total cholesterol and TG. However, exogenous testosterone administration, even in the form of testosterone replacement therapy, has been associated with a decrease in HDL-C and in some studies also in LDL-C and total cholesterol. However, a significant fall in HDL-C has been observed mainly with the use of supraphysiological testosterone doses. A negligible reduction in TC and TG levels (less than 5%) can be observed during proper testosterone replacement therapy [72, 74, 75]. In contrast to the effect on HDL-C, improvements in other markers of cardiovascular diseases have been observed after commencing testosterone replacement therapy. This might suggest that this kind of therapy could reduce cardiovascular disease-related morbidity, but data from large, long-term, placebo-controlled trials are still missing [76]. Interestingly, some studies have shown that intramuscular testosterone administration reduced Lp(a) levels in healthy men, but not in hypogonadal men [77]. The current opinion is that testosterone replacement therapy does not increase but also does not decrease cardiovascular disease risk.

Oestrogen deficiency

The lipid profile of women in the premenopausal period is more beneficial when compared to men of the same age. The main difference is the HDL-C level, which is about 10 mg/dL higher in women. LDL-C and non-HDL cholesterol, and TG levels are slightly lower in women than in men, while the LDL-C particle size is larger in women [78]. Some of those changes can already be seen during adolescence [78]. Postmenopausal women compared to premenopausal females have increased LDL-C, TC, TG, VLDL, and Apo B, and decreased Apo A1 levels [79, 80]. Moreover, a shift towards smaller LDL-C particles in the postmenopausal period can be observed. Even though only a few studies have compared Lp(a) levels in pre- and postmenopausal women, most of them reported an increase in Lp(a) [81]. HDL-C levels tend to stay stable; however, some studies showed a subtle decline in its serum concentration. It has been observed that such changes are more prominent and occur faster during surgical menopause. An increase in the percentage of body fat and a decrease in insulin sensitivity are observed during menopause. Those changes further exacerbate disturbances in lipid metabolism [78, 82, 83]. After menopause, the prevalence of cardiovascular diseases among women significantly increases. The influence of oestrogen therapy on the lipid profile has been studied extensively. Studies have shown that oestrogen supplementation raises HDL-C by 5–15% and lowers LDL-C by 5–20%. However, oestrogen supplementation, mainly through the oral route, leads to a rise in triglyceride levels, especially in patients with genetic or acquired metabolism abnormalities. The addition of progestogens can ameliorate this effect. Oestrogens also cause an increase in Apo A1, a decrease in Apo B, and a decrease in Lp(a) by 20–25%. Transdermal oestrogen administration has a less significant effect on the lipid profile. In particular, the effect on triglycerides serum concentrations is greatly reduced, which is very important in patients at risk of hypertriglyceridaemia. The weaker effect of transdermal oestrogen preparations is probably related to the lack of the "first pass" effect through the liver [84–87]. Despite the influence of oestrogens and progestogens on the lipid profile, hormone replacement therapy does not reduce cardiovascular risk and therefore is not recommended for prevention of cardiovascular disease [88].

Polycystic ovary syndrome (PCOS)

Women with PCOS suffer from atherogenic dyslipidaemia [89]. A decrease in HDL-C level and an increase in TG, non-HDL, and LDL-C levels can be observed. The LDL-C fraction is represented mainly by small, dense LDL-C [90, 91]. TG and non-HDL-C serum concentrations can increase two-fold compared to a group of healthy individuals, while HDL-C levels can be up to 60% lower. Women with PCOS also tend to have higher Lp(a) serum concentrations and lower

Apo B levels than healthy females [90, 92]. Elevated Lp(a) levels might occur in women with PCOS but otherwise a normal lipid profile; thus, Lp(a) might be used as an additional tool to assess the cardiovascular risk in this population of women [90]. The unfavourable lipid profile probably has a multifactorial aetiology, including androgen excess, lower oestrogen serum concentrations, obesity, unfavourable adipose tissue distribution, insulin resistance, impaired glucose tolerance, and impaired fasting glycaemia [91, 93, 94]. It is recommended that proper lifestyle changes are introduced and physical activity is increased to reduce weight and correct lipid profile abnormalities. Treatment with metformin may lead to further benefits [95]. If there are additional cardiovascular risk factors, lipid-lowering therapy should be considered. It is, however, prudent to remember that statin therapy may worsen glucose sensitivity in patients with PCOS [96].

Conclusions

Endocrine disorders are often associated with various abnormalities in the lipid profile. Thus, they should always be included in the differential diagnosis of secondary causes of dyslipidaemia. Proper treatment of those disorders plays a crucial role in modifying the cardiovascular risk and improving the lipid profile of those patients. Although, usually, a lipid-lowering therapy is still needed, in some cases restoration of hormonal balance might be sufficient to normalize the lipid profile abnormalities.

Conflict of interest

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