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Lipid Profile of Patients with Thyroid Function Disorders: A Literature Review

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ABSTRACT

Thyroid disorders are conditions caused by excessive/insufficient secretion of thyroid hormones and enlargement of the thyroid gland. Thyroid hormones (TH) are necessary for normal development and regulate metabolism in adults. Thyroid hormone dysfunction can also be influenced by lipid homeostasis imbalance. If left untreated, thyroid disorders can cause complications that can affect the patient's quality of life. The population and sample in this study included patients with thyroid dysfunction, namely hypothyroidism and hyperthyroidism. This literature review will examine whether there is a relationship between thyroid dysfunction and the lipid profile of patients, particularly TC, TG, HDL, and LDL.

Keywords: FT4 and TSH serum level; blood lipid profile; hypothyroidism; hyperthyroidism

INTRODUCTION

Thyroid disorders are among the most underdiagnosed and overlooked medical conditions. The lack of general knowledge among patients is also quite concerning. Thyroid disorders are conditions caused by excessive/insufficient secretion of thyroid hormones and enlargement of the thyroid gland. These disorders have been reported in more than 110 countries, with 1.6 billion people at risk. Thyroid hormone dysfunction can also be influenced by lipid homeostasis imbalance. If left untreated, thyroid disorders can cause complications that can impact patients' quality of life [1]. The most common risk factors for thyroid disease are iodine deficiency or excess (61.3%), followed by pregnancy and the postpartum period (61.1%) and radiation exposure (57.4%). The most commonly reported symptoms are fatigue (81.7%), followed by neck lumps (70.6%) and feeling cold and weight gain (68.9%) [1].

Thyroid hormones affect almost every organ system in the body, including the heart, central nervous system, autonomic nervous system, bones, digestive tract, and metabolism [2]. Although TH affects almost all organs, the liver is one of the most important targets and the center of metabolic homeostasis. The liver plays an important role in lipid metabolism, including lipid acquisition, lipid storage, and lipid consumption. When this homeostasis is disrupted, disease can occur [3].

Dyslipidemia associated with hypothyroidism causes intrahepatic fat accumulation, leading to nonalcoholic fatty liver disease (NAFLD), which results in the development of hepatic insulin resistance. The prevalence of NAFLD in the Western world is increasing, and evidence of its association with hypothyroidism is growing [4]. Meanwhile, in hyperthyroidism, there is an increase in HMG-CoA reductase activity, but TC, LDL-C, ApoB, and Lp(a) levels tend to decrease in patients with clinical and subclinical hyperthyroidism. Hyperthyroidism is not only a significant cause of acquired hypobetalipoproteinemia, but it can also be an underlying cause of unexpected increases in lipid profiles in patients with hyperlipidemia [5].

This literature review aims to provide an overview of the relationship between lipid profile and thyroid dysfunction. The review focuses on the physiology of thyroid dysfunction and its relations to lipid metabolism.

REVIEW CONTENT

1. Thyroid Gland

- *Physiology*

In its anatomical position, the thyroid gland is located posterior to the sternothyroid and sternohyoid muscles, enveloping the cricoid cartilage and tracheal ring. Its position is lower than the thyroid cartilage of the larynx, usually corresponding to the level of the C5-T1 vertebrae [6]. The thyroid gland produces approximately 90% of inactive thyroid hormone, or thyroxine (T4), and 10% of active thyroid hormone, or triiodothyronine (T3). Inactive thyroid hormone is peripherally converted into active thyroid hormone or inactive alternative thyroid hormone [7].

Thyroid hormone regulation begins in the hypothalamus [2]. The hypothalamus releases thyroid-releasing hormone (TRH), which stimulates the anterior pituitary thyrotrophs to secrete thyroid-stimulating hormone (TSH). The anterior pituitary releases TSH and stimulates thyroid follicular cells to release thyroxine, T4 (80%), and triiodothyronine or T3 (20%). Thyroid hormone synthesis depends on the availability of iodide, TSH stimulation, and tyrosine residues in thyroglobulin (Tg). When T4 is released into the circulation, it can be converted to T3 through a process called deiodination. T4 and T3 then provide negative feedback on TSH levels: high T3/T4 levels decrease TSH, and low T3/T4 levels increase TSH from the anterior pituitary [8].

2. Thyroid Hormone Disorder

The thyroid gland synthesizes and releases thyroid hormones that greatly affect the Basal Metabolic Rate (BMR) and protein synthesis. In addition, these hormones are also very important for the neurocognitive development of children and adolescents and for maintaining normal physiological function in adults. Thyroid disorders are conditions caused by excessive or insufficient secretion of thyroid hormones, and can lead to enlargement of the thyroid gland. Thyroid disorders can be primary (directly related to the gland itself) or secondary (thyroid dysfunction due to other factors). Thyroid disorders are one of the most common medical conditions worldwide, with iodine deficiency being the leading cause of thyroid disorders. The clinical symptoms of thyroid disorders depend largely on the type of disorder and can affect different body systems. Additionally, because most symptoms are nonspecific, thyroid disorders can easily be overlooked or confused with other medical conditions [1]. In general, hypothyroidism and hyperthyroidism are the two most common endocrine system disorders worldwide [9].

- *Hyperthyroidism*

Hyperthyroidism is a common thyroid disorder with various underlying etiologies. This disease is characterized by excessive production of thyroid hormones. Hyperthyroidism can be clinical or subclinical. Clinical hyperthyroidism is defined as low or suppressed thyroid-stimulating hormone (TSH) levels with high triiodothyronine (T3) levels and/or high thyroxine (T4) levels [10].

- *Hypothyroidism*

Thyroid hormones control lipid metabolism by exerting specific effects on the liver and adipose tissue in a coordinated manner. Various thyroid gland diseases can cause hypothyroidism [4]. Hypothyroidism refers to a common pathological condition resulting from a deficiency of thyroid hormones. If left untreated, this condition can lead to serious health consequences and ultimately death. Due to the wide variation in clinical presentation and the lack of specificity of symptoms in general, the definition of hypothyroidism is dominated by biochemistry [11].

3. Lipid Metabolism

The liver plays an important role in lipid metabolism, including lipid acquisition, lipid storage, and lipid consumption. Lipids metabolized in the liver can be broadly divided into two types: fatty acids and cholesterol. Two main sources of fatty acids are sent to the liver for metabolism. The first is chylomicrons, which originate in the intestine after the digestion and absorption of lipids from food intake. The second is fatty acids released from adipose tissue through lipolysis. In addition to the absorption of fatty acids from extrahepatic sources, de novo lipogenesis (DNL) also occurs in the liver when excess glucose accumulates. After absorption or synthesis by the liver, fatty acids can be used for energy production through β-oxidation in mitochondria or peroxisomes, triglyceride (TG) synthesis and storage in lipid droplets, or TG secretion into the circulation in the form of VLDL as a source of cholesterol for peripheral tissues. Cholesterol is another type of lipid that is metabolized primarily in the liver. About 30% of cholesterol comes from dietary intake, while 70% comes from de novo synthesis, mainly in the liver. The fate of cholesterol in the liver includes conversion to bile acids, packaging in lipoproteins, and secretion into the bloodstream, and use as a component of plasma membranes [3].

- *The Effect of TH on Lipid Metabolism*

Thyroid hormones induce 3-hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) reductase, which is the first step in cholesterol biosynthesis. In addition, triiodothyronine (T3) enhances LDL receptor regulation by controlling LDL receptor gene activation. This T3-mediated gene activation is carried out by direct binding of T3 to specific thyroid hormone response elements (TREs). Furthermore, T3 controls sterol regulatory element-binding protein-2 (SREBP-2), which in turn regulates LDL receptor gene expression. T3 has also been associated with protecting LDL from oxidation [5].

Thyroid hormones can affect HDL metabolism by increasing the activity of cholesteryl ester transfer protein (CETP), which exchanges cholesteryl esters from HDL2 to very low-density lipoproteins (VLDL) and TG in the opposite direction. In addition, thyroid hormones stimulate lipoprotein lipase (LPL), which catabolizes TG-rich lipoproteins, and hepatic lipase (HL), which hydrolyzes HDL2 into HDL3 and contributes to the conversion of intermediate-density lipoproteins (IDL) into LDL and, in turn, LDL into small-density LDL (sdLDL). Another effect of T3 is increased regulation of apolipoprotein AV (ApoAV), which plays a major role in TG regulation. Indeed, increased ApoAV levels have been associated with decreased TG levels. Proposed mechanisms for this effect include decreased hepatic VLDL-TG production and increased plasma LPL levels and activity, resulting in increased lipoprotein remnant formation due to increased LPL-mediated VLDL-TG lipolysis. In addition, greater clearance of lipoprotein core remnants, caused by increased hepatic uptake due to increased affinity for LDL receptors, has also been associated with ApoAV [5].

4. Thyroid Hormone Disorder and Lipid Profile the Relationship Between Hyperthyroidism and Lipid Profile Levels

Despite an increase in HMG-CoA reductase activity, TC, LDL-C, ApoB, and Lp(a) levels tend to decrease in patients with clinical or subclinical hyperthyroidism. This is due to increased expression of the LDL receptor gene, which results in increased LDL particle catabolism mediated by the LDL receptor. Furthermore, no differences in LDL subfraction distribution were observed between subjects with subclinical or overt hyperthyroidism and euthyroid subjects. Additionally, hyperthyroidism results in increased LDL oxidizability, which is associated with FT4 levels [5].

A decrease in HDL-C levels was also observed in hyperthyroidism, due to increased CETP-mediated transfer of cholesteryl esters from HDL to VLDL and increased HDL2 catabolism mediated by HL. Triglyceride levels remained unchanged. On the other hand, no changes in blood pressure, Lp(a) levels, or hsCRP have been described in hyperthyroid patients [5].

Hyperthyroidism is not only a significant cause of acquired hypobetalipoproteinemia but may also be an underlying cause of unexpected lipid profile abnormalities in patients with hyperlipidemia [5].

- *The Relationship Between Hypothyroidism and Lipid Profile Levels*

Clinical hypothyroidism is defined as high TSH levels combined with low FT4 concentrations [12]. TC and LDL-C levels increase in patients with clinical hypothyroidism. This is due to decreased LDL receptor activity, which results in decreased LDL and IDL catabolism. In addition, decreased LPL activity is found in clinical hypothyroidism, which reduces the clearance of TG-rich lipoproteins (Rizos et al., 2011). Thus, it can be concluded that hypothyroidism causes increased cholesterol absorption in the intestine and decreased LDL cholesterol clearance, leading to higher plasma LDL cholesterol levels and triglyceride accumulation in the liver [4].

Hypothyroid patients may also show increased HDL-C levels, primarily due to increased HDL2 particle concentrations. Additionally, decreased CETP activity results in reduced transfer of cholesterol esters from HDL to VLDL, thereby increasing HDL-C levels [5].

Subclinical hypothyroidism (SH), defined as a clinical state of elevated serum TSH with normal FT4 and FT3 levels, is a disorder that is much more common than overt hypothyroidism. SH is associated with increased TC and LDL-C levels. In addition, several studies have shown that subclinical hypothyroidism dyslipidemia may also be accompanied by increased TG and decreased HDL-C levels [5].

CONCLUSION

There are relations between thyroid hormone disorder, hyperthyroidism, and hyperthyroidism with blood lipid profile. In hyperthyroidism, LDL and TC often decrease, while HDL might decrease. As for TG, there is no significant change. In hypothyroidism, LDL and TC often increase, mainly shown in clinical hypothyroidism, while TG might increase. As for HDL, there is no significant change, but it may also increase.

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