Study of the association between thyroid dysfunction and serum lipid abnormalities

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Abstract. The prevalence of thyroid dysfunction is increasing, often leading to unfavorable alterations in lipid profiles. Dyslipidemia is a risk factor for cardiovascular disease. The present study aimed to assess the prevalence of thyroid dysfunction and examine its effects on serum lipid profiles among Jordanians. A total of 228 subjects were recruited and divided into two groups: patients with thyroid dysfunction (n=178, mean age=52.6±9.8 years) and a control group (n=50, mean age=51.7±9.2 years). Serum thyroid-stimulating hormone, free thyroxine 4, free triiodothyronine 3, total cholesterol (TC), low-density lipoprotein (LDL), high-density lipoprotein and triglycerides (TG) were measured. Results showed that thyroid dysfunction was diagnosed in 75% of participants, with an increased frequency among females. The prevalence of overt hypothyroidism was 17.4%, subclinical hypothyroidism was 43.8%, overt hyperthyroidism was 18.4% and subclinical hyperthyroidism was 20.4%. There was a significant association between hypothyroidism and elevated TC (>200 mg/dl), LDL (>130 mg/dl) and TG (>200 mg/dl; P<0.05). Among the hypothyroid patients, 48.4% had hypercholesterolemia and 32.3% had hypertriglyceridemia. In conclusion, public screening and education are necessary to combat thyroid dysfunction. There is a notable link between thyroid dysfunction and lipid abnormalities, necessitating regular monitoring for dyslipidemia and cardiovascular disease in affected patients.

Introduction

Disorders of the endocrine system, particularly those affecting the thyroid gland, are widespread globally affecting >200 million people around the world (1-4). Thyroid gland disorders, including both iodine deficiency-related and non-iodine deficiency-related disorders, are increasing (5). Among them, non-iodine deficiency-related thyroid disorders, such as thyroid cancer, have shown the highest increase in occurrence compared with other solid tumors (6). Indeed, except for Sweden, most countries recorded an increased rate of thyroid cancer over the period 1972-2002. It has been reported that thyroid cancer incidence around the world is 3.2 million individuals. There are >560,000 new cases of thyroid cancer reported every year globally (7). Hypothyroidism, a severe iodine deficiency-related thyroid disorder, is also increasing worldwide (8) with 5% of the world's population suffering from this condition (7). Hyperthyroidism is also a common thyroid dysfunction condition with a global prevalence of 0.2-13% (9).

Thyroid hormones are chemical substances produced by the thyroid gland, which is located in the front of the neck and uses iodine to make thyroxine (T4) and triiodothyronine (T3) hormones (10). The thyroid gland is regulated by thyroid-stimulating hormone (TSH), produced by the anterior pituitary gland, which is in turn regulated by thyrotropin-releasing hormone (TRH) synthesized in the hypothalamus (11). Thyroid hormones play key roles in various metabolic activities, reproduction, growth and development (12,13). They are directly and indirectly involved in lipid biosynthesis and degradation (4). T4 is converted to the active form, T3, by 5'-deiodinase type 2 (14).

Thyroid dysfunction is a condition that alters the amount of thyroid hormones secreted. Over-secretion of thyroid hormones leads to hyperthyroidism, while reduced production results in hypothyroidism (15,16). Thyroid dysfunction has diverse effects on other health conditions in humans, such as dyslipidemia (17) and cardiovascular diseases (10,18).

Dyslipidemia is characterized by an imbalance of different circulating lipids, including total cholesterol (TC), triglycerides (TG), low-density lipoprotein (LDL) and high-density lipoprotein (HDL) (19). This pathological condition is a major risk factor for cardiovascular diseases and may result from dietary and genetic factors, as well as lifestyle choices such as smoking and physical inactivity (20).

In the Jordanian population, an Arab nation, the prevalence of thyroid disorders is reportedly high compared with global statistics (16,21). It has been reported that the prevalence of thyroid disorders among Jordanians is 11.9% (21). Nevertheless, the highest prevalence of thyroid disease among adult Americans was observed in non-Hispanic Caucasians at 8.1% (22).

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A meta-analysis study of thyroid dysfunction in Europe showed that the mean prevalence of diagnosed and undiagnosed thyroid dysfunction is 3.82% for hypothyroidism and 0.75% for hyperthyroidism (23). A study from Jordan reported no relationship between the high percentage of hyperlipidemia and levels of serum TSH and free thyroxine 4 (T4) hormone (24). However, studies from Jordan focusing on the association between thyroid dysfunction and lipid profile biomarkers are sparse or entirely missing. Hence, there is a need to intensify research on the association and/or causative relationship between thyroid dysfunction and lipids abnormalities among Jordanians.

Materials and methods

Subjects. All subjects in the present study enrolled voluntarily. The present study recruited 178 patients with thyroid dysfunction (27 males and 151 females; mean age=52.6±9.8 years). All patients were attending the endocrinology clinic at Tohamma Medical Center in Amman, Jordan, during the period between 15 September 2020 and 19 January 2021. A control group of 50 healthy individuals was also enrolled (25 males and 25 females; mean age=51.7±9.2 years).

Information such as age, sex and family history of chronic disease was collected and recorded for all patients. Patient privacy and confidentiality were ensured and the information was used solely for this research, with proper disposal procedures to be followed afterwards.

The present study received approval with a reference number MLS_R_01/06/2020 from the Ethics and Scientific Research Board at Jadara University (Irbid, Jordan). Written informed consent was obtained from all participants involved in the present study after providing a comprehensive explanation of the present study's purpose, procedures and significance. The present study was conducted in accordance with the principles outlined in the Declaration of Helsinki.

Exclusion and inclusion criteria. Patients with thyroid dysfunction, clinically diagnosed by alteration in serum levels of thyroid hormones (subclinical and overt) of both sexes and aged ≥18 years were included in the present study. Subclinical thyroid dysfunction is defined by an abnormal level of serum TSH (the normal level is 0.4-4.2 μIU/ml) and normal levels of serum free triiodothyronine 3 (fT3; 0.4-4.0 μg/ml) and free thyroxine 4 (fT4; 0.5-1.9 ng/ml). Overt thyroid dysfunction is defined by alterations in serum levels of TSH as well as serum levels of fT3 and fT4 simultaneously. None of the participants in the present study was known to have or clinically diagnosed with thyroid cancer. All patients diagnosed with chronic kidney disease, liver diseases, diabetes mellitus, pregnancy, women on oral contraceptives, patients with history of thyroxine and hypolipidemic treatment in the last 90 days or being <18 years old were excluded from the present study.

Male and female healthy participants with normal serum thyroid hormone levels, no history of disease or medication that affect thyroid gland function and serum levels of lipid and aged >18 years were included in the present study.

Blood sampling and sample analysis. Venous blood samples (5-10 ml) were drawn from participants between 8:00 and 10:00 a.m. into labeled blood tubes after a minimum of 14 h of fasting. The blood samples were left to clot for 15 min at room temperature, then centrifuged at 5,000 x g at room temperature for 10 min, after which serum samples were collected and analyzed for lipid profile and thyroid function parameters.

Serum samples analysis was performed at Tohamma Medical Center in Zarqa'a, Jordan. All tests were performed in Teryaq Alrouh medical laboratory. Serum TG, TC and HDL cholesterol concentrations were measured using the BioMaxima BM 200 chemistry auto-analyzer (BioMaxima S.A.). Levels of serum LDL-cholesterol were calculated using the Friedewald formula: LDL cholesterol=(total serum cholesterol)-(HDL cholesterol)-(triglyceride concentration/5) (25).

Levels of serum TSH, fT4 and fT3 were measured using the Cobas e 411 auto-analyzer (Roche Diagnostics GmbH), with corresponding Roche Diagnostics kits (Roche Diagnostics GmbH) used for the analysis of all parameters.

Reference range. Normal values for lipid profile parameters are as follows: TC (150-200 mg/dl), TG (50-200 mg/dl), HDL-Cholesterol (10-60 mg/dl), LDL-Cholesterol (60-160 mg/dl) and the TC/HDL-Cholesterol ratio is 4 (Roche Diagnostics GmbH).

The normal reference range for thyroid parameters according to the kits used were as follows: TSH (0.4-4.2 μIU/ml), fT4 (0.5-1.9 ng/ml) and fT3 (0.4-4.0 μg/ml). Hypothyroidism was clinically defined by TSH ≥4.5 μIU/ml and hyperthyroidism was classified clinically by TSH ≥0.1 μIU/ml (Roche Diagnostics GmbH).

Statistical analysis. All statistical analyses of data were performed using the Statistical Package for the Social Sciences (SPSS) version 20.0 for windows (IBM Corp.) and Microsoft Excel 2010 (Microsoft Corporation). Results are reported as mean ± standard deviation (SD). Differences in mean ± SD values were analyzed for statistical significance using one-way ANOVA test followed by Tukey's post hoc test. $P<0.05$ was considered to indicate a statistically significant difference.

Results

Prevalence of thyroid dysfunction. In the present study, 178 patients (49 males and 129 females) with thyroid disorders were recruited, along with a sex- and age-matched healthy control group of 50 subjects (25 males and 25 females). The mean age of the patient group was 52.6±9.8 years and it was 51.7±9.2 years for the control group. Among patients with overt thyroid disorders, the mean age was 51.4±8.2 years, while for those with subclinical thyroid disorders, it was 53.9±6.4 years.

The overt hypothyroid group was 17.4% of total patients, overt hyperthyroidism was at 17.5% and subclinical hypothyroidism was the most prevalent thyroid disorder at 43.8%, with subclinical hyperthyroidism accounting for 21.3% of total patients. Overt thyroid patients comprised 34.9% of the total, while subclinical thyroid patients comprised 65.1%. Additionally, the present study found that thyroid dysfunction was more common among females (72.5%) compared with males (27.5%) across all forms of thyroid dysfunction (Table I).

Thyroid function analytes. Results of thyroid function tests (Table II) revealed that mean serum TSH levels were

Table I. Age, frequencies and percentages of participants in both sex in controls and each condition of thyroid dysfunction.

Parameter	Controls (%)	Overt hypothyroid frequency (%)	Subclinical hypothyroid frequency (%)	Overt hyperthyroid frequency (%)	Subclinical hyperthyroid frequency (%)
Age, years	51.7	51.1	53.8	51.7	53.9
Male	25 (50)	12 (6.7)	21 (11.8)	9 (5.1)	7 (3.9)
Female	25 (50)	19 (10.7)	57 (32)	22 (12. 4)	31 (17.4)

Table II. TSH, fT4 and fT3 serum levels among subjects.

Serum levels	Control (n=50)	Subclinical thyroid dysfunction (n=116)	Overt thyroid dysfunction (n=62)	P-value[a]
TSH μIU/ml	2.34±0.4	6.88±0.7	35.67±1.3	0.042
fT4 pg/ml	0.97±0.05	0.86±0.06	0.46±0.03	0.031
fT3 ng/ml	3.54±0.09	3.12±0.07	2.63±0.07	0.039

[a]vs. the Control group. Data were analyzed using one-way ANOVA test followed by Tukey's post hoc test. Results are expressed as mean ± SD. P<0.05 was considered to be statistically significant. TSH, thyroid-stimulating hormone; fT4, free thyroxine 4; fT3, serum free triiodothyronine 3.

Table III. Comparison of mean lipid profiles between controls and thyroid dysfunction patients.

Lipid	Control (n=50)	Subclinical thyroid dysfunction (n=116)	P-value[a]	Overt thyroid dysfunction (n=62)	P-value[a]
TC (mg/dl)	172.74±58.60	226.86±48.60	P=0.039	248.78±48.70	P=0.047
TG (mg/dl)	127.74±48.70	165.37±44.20	P=0.040	184.87±61.01	P=0.033
LDL (mg/dl)	98.76±39.80	144.14±60.75	P=0.028	148.17±49.23	P=0.022
HDL (mg/dl)	57.66±28.60	39.86±9.45	P=0.011	37.35±7.94	P=0.030
LDL/HDL	2.95±0.16	3.71±0.78	P=0.540	3.91±0.78	P=0.220

[a]vs. the Control group. Data were analyzed using one-way ANOVA test followed by Tukey's post hoc test. Results are expressed as mean ± SD. P<0.05 was considered to be statistically significant.

significantly higher in the subclinical thyroid group compared with the control group (6.88±0.7 vs. 2.34±0.4 μIU/ml; P<0.05), with even higher levels in the overt thyroid group (35.67±1.3 vs. 2.34±0.4 μIU/ml; P<0.05). Serum levels of fT4 and fT3 were significantly lower in subclinical thyroid patients compared with the corresponding control group (0.86±0.06 pg/ml vs. 0.97±0.05 pg/ml; P<0.05) and (3.12±0.07 ng/ml vs. 3.54±0.09 ng/ml; P<0.05) respectively. In overt thyroid patients, serum levels of fT4 and fT3 were significantly lower compared with the control group (0.46±0.03 pg/ml vs. 0.97±0.05 pg/ml; P<0.05) and (2.63±0.07 ng/ml vs. 3.54±0.09 ng/ml; P<0.05) respectively.

Lipids analytes. Results of lipid assays (Table III) indicated significantly higher mean serum levels of TC, TG and LDL-cholesterol in the patient group. Mean serum TC levels were significantly elevated in the subclinical thyroid group compared with the control group (226.86±48.6.7 vs. 172.74±58.6 mg/dl; P<0.05), with greater levels observed in the overt thyroid group (248.78±48.7 vs. 172.74±58.6 mg/dl; P<0.05). Similar findings were noted for serum TG levels in both subclinical and overt thyroid groups compared with the healthy control group (165.37±44. 2 vs. 127.74±48.7 mg/dl; P<0.05) and (184.87±61.01 vs. 127.74±48.7 mg/dl; P<0.05) respectively. Serum LDL levels were also significantly higher in both groups (144.14±60.75 vs. 98.76±39.8 mg/dl; P<0.05) and (148.17±49.23 vs. 98.76±39.8 mg/dl; P<0.05). Additionally, mean serum HDL-cholesterol levels were significantly lower in the patient group compared with the control group (39.86±9.45 vs. 57.66±28.6 mg/dl; P<0.05) and (37.35±7.94 vs. 57.66±28.6 mg/dl; P<0.05). There was no significant difference in LDL/HDL ratio between patients and the control group.

Discussion

The main finding of the present study was the significant increase in serum TC, TG and LDL-C levels in subjects with thyroid dysfunction, along with a significant decrease in serum

HDL. This significant association between thyroid dysfunction and serum dyslipidemia is reported for the first time in a community-based study in Jordan, to the best of the authors' knowledge. By contrast, the only previous study that investigated the association between the prevalence of dyslipidemia and levels of TSH and T4 hormones among Jordanian patients reported no correlation between these parameters (24). These discrepancies between the two studies may be due to the variations in the geographical districts of the south and middle of Jordan. The genetic factors and environmental conditions of two districts could contribute to the discrepancies as the southern district of the study (24) is classified as a desert area with mainly indigenous Bedouin inhabitants. By contrast, the middle district of the present study is more westernized in life style.

Thyroid hormones promote the activities of two enzymes related to lipid metabolism: lipoprotein lipase (LPL) and hepatic lipase (HL). LPL catabolizes TG-rich lipoproteins, while HL hydrolyzes HDL2 to HDL3 and contributes to the conversion of intermediate-density lipoproteins to LDL and subsequently, LDL to small dense LDL (26,27).

Subclinical hypothyroidism is a pathological condition characterized by normal levels of serum fT3 and fT4, together with an increased level of serum TSH (28). When serum fT3 and fT4 levels decrease and serum TSH levels increase, the pathological condition becomes overt hypothyroidism (29). Consistent with other studies, the current study also demonstrated a higher prevalence of hypothyroidism compared with hyperthyroidism in the present studied population, with subclinical hypothyroidism being more common than overt hypothyroidism. Hypothyroidism is closely linked to lipid metabolism disorders and dyslipidemia, increasing the risk of cardiovascular diseases (30).

There is controversy surrounding lipid levels in subclinical hypothyroidism and its clinical significance (31-33). Manifestations of overt hypothyroidism include serum hypercholesterolemia and an increase in serum LDL due to reduced fractional clearance of LDL. This reduction is due to a decrease in the number of LDL receptor proteins in the liver (34).

The results of the present study indicated a high prevalence of thyroid dysfunction in the Jordanian population, consistent with another Jordanian study by Ajlouni *et al* (21). In the present study, the frequency of hypothyroidism was higher (64%) compared with hyperthyroidism (36%). This agrees with what Ajlouni *et al* (21) found; that there is a preponderance of hypothyroidism over hyperthyroidism as well as female thyroid dysfunction preponderance over male thyroid dysfunction in the Jordanian population. Ajlouni *et al* (21) also reported that 2.9% of the Jordanian population studied were known to have thyroid dysfunction. Markedly, the prevalence of newly discovered primary thyroid dysfunction was 9.0% among Jordanian participants in his study.

The findings of the present study showed an association between thyroid dysfunction and hyperlipidemia among Jordanians. In patients with subclinical and overt thyroid dysfunction, serum TC, TG and LDL-cholesterol concentrations were significantly higher compared with the control group. These results agree with findings from previous studies that reported an association and causative correlation between hypothyroidism and disturbance of lipid profile in the patients. For example, O'Brien *et al* (35) reported an association between hyperlipidemia and both primary and secondary hypothyroidism. Additionally, Ahmed *et al* (36) demonstrated that primary hypothyroidism is significantly correlated with high BMI and serum cholesterol, TG and LDL levels. Moreover, subclinical hypothyroidism was associated with increased serum LDL-C concentration (37).

The association between overt hypothyroidism and hypercholesterolemia has been firmly established; however, the link between subclinical hypothyroidism and hypercholesterolemia remains controversial (38). This controversy may primarily stem from a lack of evidence supporting the association between these two variables. On one hand, subclinical hypothyroidism has not been shown to be associated with abnormalities in serum cholesterol or triglyceride levels (32). On the other hand, subclinical hypothyroidism can potentially contribute to a pro-atherogenic lipid profile (39). Furthermore, consistent with another study (40), the results of the present study showed that serum HDL-cholesterol levels were significantly lower in patients with thyroid dysfunction compared with the control group. The effect of thyroid hormones on serum lipids, a risk factor for cardiovascular disease, is not fully understood and understanding the integration of various thyroid hormone pathways remains a great challenge. Thus, comprehending the mechanisms and interactions of the various thyroid hormones signaling pathways in metabolism will enhance our understanding of the link between dyslipidemia and cardiovascular disease.

Lipid homeostasis in the liver is regulated by the direct actions of thyroid hormones. T3 regulates cholesterol synthesis through several mechanisms. A plausible mechanism for hypercholesterolemia in thyroid dysfunction patients is the increased enzymatic activity of 3-hydrox-3-methylglutaryl-coenzyme A reductase in the cholesterol biosynthesis signaling pathway, as well as the decreased hepatic uptake of cholesterol from the circulation, both mechanisms being upregulated by T3 (41). Additionally, thyroid hormones can increase cholesterol absorption in the intestine by activating the Niemann-Pick C1-like 1 protein in enterocytes (42).

The physiological function of thyroid hormones requires interaction with their receptors (TRs) α and ß in the signaling pathway (43). TR regulates cholesterol metabolism through direct actions on gene expression and cross-talk with other nuclear receptors, including peroxisome proliferator-activated receptor, liver X receptor and bile acid signaling pathways (14).

Serum TG were significantly increased in the thyroid dysfunction patients in the present study. This might be due to decreased activity of lipoprotein lipase, which is responsible for the clearance of triglyceride-rich lipoproteins.

The elevated levels of serum LDL in thyroid dysfunction conditions observed in the present study might be due to the role of thyroid hormones in the expression of LDL receptors and cytochrome P450 7A1, a rate-limiting enzyme in bile acid synthesis. The T3 hormone, in particular, is involved in regulating the gene expression of LDL receptors (44,45). LDL receptor-related proteins, such as LDL receptor-related protein 1, are cell surface glycoprotein signaling receptors that bind and internalize diverse ligands including lipoprotein particles (46). They are expressed in all cell types and, in particular, in hepatocytes. The underlying mechanisms of T3-mediated hypercholesterolemia in the hypothyroidism

dysfunction is that the reduced serum level of T3 inhibit the transcription of the LDL-receptor gene, resulting in decreased cholesterol uptake by hepatocytes and reduction in clearance of circulating lipoproteins, leading to hypercholesterolemia in thyroid dysfunction patients (46). In particular, T3 may affect the expression of sterol regulatory element binding protein 2 transcription factor involved in the transcription of the LDL-receptor gene (47). Additionally, T3 has a protective role in preventing LDL oxidation (48). However, thyroid dysfunction not only increases the number of LDL molecules but also promotes LDL oxidation, thereby increasing the risk of atherosclerosis (49).

Thyroid hormones are involved in HDL metabolism by enhancing the activity of cholesteryl ester transfer protein, a molecule responsible for converting cholesteryl esters from HDL to very low-density lipoproteins (10). This might explain the decrease in serum HDL observed in the current study.

Hyperthyroidism, characterized by excess thyroid hormone, promotes a hypermetabolic state, which reduces cholesterol levels and increases lipolysis. Subclinical hyperthyroidism is marked by normal serum fT3 and fT4 levels and reduced serum TSH (21). Overt hyperthyroidism involves elevated serum fT3 and fT4 levels and reduced serum TSH. The effect of hyperthyroidism did not show a significant impact on dyslipidemia in the present study.

The present study also showed a predominance of thyroid dysfunction in females compared with males. Females in the Jordanian population appear to be more susceptible to thyroid dysfunction, particularly subclinical hypothyroidism. This finding is consistent with reports from different populations around the world (50,51). Women may be more prone to thyroid dysfunction due to the hormonal changes they experience throughout their life cycle, including puberty, the menstrual cycle, pregnancy, childbirth, lactation and menopause (52). The immunological changes during antepartum, pregnancy and the postpartum periods that affect thyroid hormone levels can also increase the frequency of thyroid dysfunction in females (53,54). While the effect of thyroid hormones on female reproduction has been investigated, the effect of female sex hormones on thyroid function and the differences in the frequency of thyroid dysfunction between sexes require further investigation.

The present study has several strengths and limitations. On the one hand, it has the strength that it may be the first to study the association of thyroid dysfunction and dyslipidemia in the Jordanian population. On the other hand, some limitations of the present study included the disproportionate number of female participants compared with male participants, which could bias the results and conclusions. The higher percentage of female patients in the present study is due to the greater susceptibility of females to thyroid diseases compared with males. Hence, there were difficulties in recruiting male patients due to the limited cases in the designated geographical area. Additionally, the age of the subjects was not adjusted in the present study. Further, the present study was conducted in a small population in the middle district of Jordan. It would be a more comprehensive if it involved a larger population sample from the three main districts of Jordan: north, middle and south.

In conclusion, the present study showed that the prevalence of thyroid dysfunction is high among Jordanians. It also demonstrated that the lipid profile is abnormal in thyroid dysfunction conditions. Dyslipidemia is a major risk factor for cardiovascular disease. Therefore, the present study recommended screening high-risk groups for thyroids dysfunction, such as females. Furthermore, educating the public about thyroid dysfunction can help detect thyroid conditions at an early stage for proper intervention. Finally, the present study emphasized the importance of monitoring lipid levels in patients with thyroid dysfunction to prevent or, at the very least, minimize cardiovascular diseases. Supplementing with a full diet of iodine, such as seafood and dairy products helps the thyroid gland to have enough materials to produce hormones and prevent thyroid disease. Thyroid medication including levothyroxine could also enhance the efficacy of hypolipidemic drugs, such as statins, if was prescribed to the patients.

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Availability of data and materials

The data generated in the present study may be requested from the corresponding author.

Authors' contributions

SA and MA were involved in designing the present study. SA was involved in collecting and analyzing the data and writing the manuscript. SA and IA confirm the authenticity of all the raw data. IA was involved in analyzing the data and writing the manuscript. The initial draft of the manuscript was prepared by SA. SA and IA contributed to revising and finalizing the manuscript. MA provided feedback on the final draft of the manuscript. All authors reviewed and approved the final manuscript.

Ethics approval and consent to participate

The present study received approval with a reference number MLS_R_01/06/2020 from the Ethics and Scientific Research Board at Jadara University (Irbid, Jordan). Written informed consent was obtained from all participants involved in the present study after providing a comprehensive explanation of the present study's purpose, procedures and significance. The present study was conducted in accordance with the principles outlined in the Declaration of Helsinki.

Patient consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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References

1. Bayer MF: Effective laboratory evaluation of thyroid status. Med Clin North Am 75: 1-26, 1991.
2. Regmi A, Shah B, Rai BR and Pandeya A: Serum lipid profile in patients with thyroid disorders in central Nepal. Nepal Med Coll J 12: 253-256, 2010.
3. Heuck CC, Kallner A, Kanagasabapathy AS and Riesen W: Diagnosis and monitoring of diseases of the thyroid. World Health Organization, 2000.
4. Pucci E, Chiovato L and Pinchera A: Thyroid and lipid metabolism. Int J Obes Relat Metab Disord 24 (Suppl 2): S109-S112, 2000.
5. WHO: Assessment of iodine deficiency disorders and monitoring their elimination: A guide for programme managers. World Health Organization, 2001.
6. Bilek R, Dvořáková M, Grimmichova T and Jiskra J: Iodine, thyroglobulin and thyroid gland. Physiol Res 69 (Suppl 2): S225-S236, 2020.
7. Moini J, Pereira K and Samsam M: Epidemiology of thyroid disorders. Netherlands, Elsevier, 116-152, 2020.
8. Hatch-McChesney A and Lieberman HR: Iodine and iodine deficiency: A comprehensive review of a re-emerging issue. Nutrients 14: 3474, 2022.
9. Wiersinga WM, Poppe KG and Effraimidis G: Hyperthyroidism: Aetiology, pathogenesis, diagnosis, management, complications, and prognosis. Lancet Diabetes Endocrinol 11: 282-298, 2023.
10. Duntas LH: Thyroid disease and lipids. Thyroid 12: 287-293, 2002.
11. Shahid MA, Ashraf MA and Sharma S: Physiology, thyroid hormone. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing, 2022.
12. Taylor PN, Albrecht D, Scholz A, Gutierrez-Buey G, Lazarus JH, Dayan CM and Okosieme OE: Global epidemiology of hyperthyroidism and hypothyroidism. Nat Rev Endocrinol 14: 301-316, 2018.
13. Liu YY and Brent GA: Thyroid hormone crosstalk with nuclear receptor signaling in metabolic regulation. Trends Endocrinol Metab 21: 166-173, 2010.
14. Mullur R, Liu YY and Brent GA: Thyroid hormone regulation of metabolism. Physiol Rev 94: 355-382, 2014.
15. Ridgway EC: Modern concepts of primary thyroid gland failure. Clin Chem 42: 179-182, 1996.
16. Abu-Helalah M, Alshraideh HA, Al-Sarayreh SA, Al Shawabkeh AHK, Nesheiwat A, Younes N and Al-Hader A: A cross-sectional study to assess the prevalence of adult thyroid dysfunction disorders in Jordan. Thyroid 29: 1052-1059, 2019.
17. Asvold BO, Vatten LJ, Nilsen TIL and Bjøro T: The association between TSH within the reference range and serum lipid concentrations in a population-based study. The HUNT study. Eur J Endocrinol 156: 181-186, 2007.
18. Neves C, Alves M, Medina JL and Delgado JL: Thyroid diseases, dyslipidemia and cardiovascular pathology. Rev Port Cardiol 27: 1211-1236, 2008 (In English, Portuguese).
19. Pappan N and Rehman A: Dyslipidemia. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing, 2022.
20. Krauss RM: Dietary and genetic probes of atherogenic dyslipidemia. Arterioscler Thromb Vasc Biol 25: 2265-2272, 2005.
21. Ajlouni KM, Khawaja N, El-Khateeb M, Batieha A and Farahid O: The prevalence of thyroid dysfunction in Jordan: A national population-based survey. BMC Endocr Disord 22: 253, 2022.
22. Zhang X, Wang X, Hu H, Qu H, Xu Y and Li Q: Prevalence and trends of thyroid disease among adults, 1999-2018. Endocr Pract 29: 875-880, 2023.
23. Garmendia Madariaga A, Santos Palacios S, Guillén-Grima F and Galofré JC: The incidence and prevalence of thyroid dysfunction in Europe: A meta-analysis. J Clin Endocrinol Metab 99: 923-931, 2014.
24. Atrooz OM, Hiresh MN, Dlewan AR, Atrooz MO, Hiresh GN, Alasoufi AM and Atrooz IO: Prevalence of dyslipidemia and the association with levels of TSH and T4 hormones among patients in south region of Jordan. J Med Biochem 42: 706-713, 2023.
25. Friedewald WT, Levy RI and Fredrickson DS: Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin Chem 18: 499-502, 1972.
26. Friis T and Pedersen LR: Serum lipids in hyper- and hypothyroidism before and after treatment. Clin Chim Acta 162: 155-163, 1987.
27. Canaris GJ, Manowitz NR, Mayor G and Ridgway EC: The Colorado thyroid disease prevalence study. Arch Intern Med 160: 526-534, 2000.
28. Cooper DS and Biondi B: Subclinical thyroid disease. Lancet 379: 1142-1154, 2012.
29. Ross DS: Hypothyroidism during pregnancy: clinical manifestations, diagnosis, and treatment. UpToDate Waltham, MA (Accessed 8 Jun 2013), 2013.
30. Wang C and Crapo LM: The epidemiology of thyroid disease and implications for screening. Endocrinol Metab Clin North Am 26: 189-218, 1997.
31. Mansfield BS, Bhana S and Raal FJ: Dyslipidemia in South African patients with hypothyroidism. J Clin Transl Endocrinol 29: 100302, 2022.
32. Hueston WJ and Pearson WS: Subclinical hypothyroidism and the risk of hypercholesterolemia. Ann Fam Med 2: 351-355, 2004.
33. Jayasingh IA and Puthuran P: Subclinical hypothyroidism and the risk of hypercholesterolemia. J Family Med Prim Care 5: 809-816, 2016.
34. Su X, Peng H, Chen X, Wu X and Wang B: Hyperlipidemia and hypothyroidism. Clin Chim Acta 527: 61-70, 2022.
35. O'Brien T, Dinneen SF, O'Brien PC and Palumbo PJ: Hyperlipidemia in patients with primary and secondary hypothyroidism. Mayo Clin Proc 68: 860-866, 1993.
36. Ahmed NU, Kabir MA, Razzak A and Akter S: Relation of hypothyroidism on BMI and dyslipidemia. Med Today 31: 93-97, 2019.
37. Walsh JP, Bremner AP, Bulsara MK, O'leary P, Leedman PJ, Feddema P and Michelangeli V: Thyroid dysfunction and serum lipids: A community-based study. Clin Endocrinol (Oxf) 63: 670-675, 2005.
38. Deschampheleire M, Luyckx FH and Scheen AJ: Thyroid disorders and dyslipidemias. Rev Med Liege 54: 746-750, 1999 (In French).
39. Ineck BA and Ng TMH: Effects of subclinical hypothyroidism and its treatment on serum lipids. Ann Pharmacother 37: 725-730, 2003.
40. Jawzal K, Hami M, Mohammed L and Ibrahiem A: The relationship between thyroid hormones and lipid profile in subclinical hypothyroidism female patients. Baghdad J Biochem Appl Biol Sci 3: 200-209, 2022.
41. Duntas LH and Brenta G: A renewed focus on the association between thyroid hormones and lipid metabolism. Front Endocrinol (Lausanne) 9: 511, 2018.
42. Pearce EN: Update in lipid alterations in subclinical hypothyroidism. J Clin Endocrinol Metab 97: 326-333, 2012.
43. Chen C, Xie Z, Shen Y and Xia SF: The roles of thyroid and thyroid hormone in pancreas: Physiology and pathology. Int J Endocrinol 2018: 2861034, 2018.
44. Lagrost L: Regulation of cholesteryl ester transfer protein (CETP) activity: Review of in vitro and in vivo studies. Biochim Biophys Acta 1215: 209-236, 1994.
45. Faure P, Oziol L, Artur Y and Chomard P: Thyroid hormone (T3) and its acetic derivative (TA3) protect low-density lipoproteins from oxidation by different mechanisms. Biochimie 86: 411-418, 2004.
46. Moon JH, Kim HJ, Kim HM, Choi SH, Lim S, Park YJ, Jang HC and Cha BS: Decreased expression of hepatic low-density lipoprotein receptor-related protein 1 in hypothyroidism: A novel mechanism of atherogenic dyslipidemia in hypothyroidism. Thyroid 23: 1057-1065, 2013.
47. Su X, Chen X, Peng H, Song J, Wang B and Wu X: Novel insights into the pathological development of dyslipidemia in patients with hypothyroidism. Bosn J Basic Med Sci 22: 326-339, 2022.
48. Kuusi T, Saarinen P and Nikkilä EA: Evidence for the role of hepatic endothelial lipase in the metabolism of plasma high density lipoprotein2 in man. Atherosclerosis 36: 589-593, 1980.

49. Baral N, Lamsal M, Koner BC and Koirala S: Thyroid dysfunction in eastern Nepal. Southeast Asian J Trop Med Public Health 33: 638-641, 2002.
50. Olmos RD, de Figueiredo RC, Aquino EM, Lotufo PA and Bensenor IM: Gender, race and socioeconomic influence on diagnosis and treatment of thyroid disorders in the Brazilian longitudinal study of adult health (ELSA-Brasil). Braz J Med Biol Res 48: 751-758, 2015.
51. Santos Palacios S, Llavero Valero M, Brugos-Larumbe A, Díez JJ, Guillén-Grima F and Galofré JC: Prevalence of thyroid dysfunction in a Large Southern European Population. Analysis of modulatory factors. The APNA study. Clin Endocrinol (Oxf) 89: 367-375, 2018.
52. Redmond GP: Thyroid dysfunction and women's reproductive health. Thyroid 14 (Suppl 1): S5-S15, 2004.
53. Weetman AP: Immunity, thyroid function and pregnancy: Molecular mechanisms. Nat Rev Endocrinol 6: 311-318, 2010.
54. Kokandi AA, Parkes AB, Premawardhana LDKE, John R and Lazarus JH: Association of postpartum thyroid dysfunction with antepartum hormonal and immunological changes. J Clin Endocrinol Metab 88: 1126-1132, 2003.