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Physiological and Biochemical Study for women Patients with Hypothyroidism

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By

Rusul Mushtaq Talib Sabti Abboud

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Supervised By

Assist. Prof.Dr. Walaa Salih Hassan Allbn

Prof. Dr.Hussein Jasim Obaid Al-Harbi

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بسم الله الرحمن الرحيم

يَا أَيُّهَا الَّذِينَ آمَنُوا إِذَا قِيلَ لَكُمْ تَفَسَّحُوا فِي الْمَجَالِسِ فَافْسَحُوا يَفْسَحِ اللَّهُ لَكُمْ ۖ وَإِذَا قِيلَ انشُزُوا فَانشُزُوا يَرْفَعِ اللَّهُ الَّذِينَ آمَنُوا مِنكُمْ وَالَّذِينَ أُوتُوا الْعِلْمَ دَرَجَاتٍ ۚ وَاللَّهُ بِمَا تَعْمَلُونَ خَبِيرٌ (١١)

صدق الله العظيم

سورة المجادلة الآية (١١)

Dedication

To the Great Prophet Muhammad, may God bless him and his family and grant them peace..... A source of knowledge and a teacher

The first human

For the five owners of cloak (peace be upon them).... Flags of guidance and piety. To the expected owner of the matter, Imam Mahdi (peace be upon him)....the savior of humanity

To those whom I call upon, may God grant me success, grant me success, and send forth love and tenderness... My parents and my mother are those whom I hold dearest, my blessings and my treasure... my brothers are behind me... my sisters

RUSUL

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RUSUL

Summary

Hypothyroidism is the common clinical condition of thyroid hormone deficiency and, if left untreated, can lead to serious adverse effects such as , the cardiovascular diseases and breast cancer. Thyroid hormone is essential for the normal development of many human tissues and regulates the metabolism of virtually all cells and organs of the human body throughout life. Hypothyroidism considered the second most common disease after diabetes mellitus.

The present study was done in department of Biology, College of Science, University of Babylon. The sample collection and occurred through the period from June to November 2022.the samples of hypothyroidism women were collected from the Imam Al-Sadiq hospital and Merjan Teaching Hospital in Hilla city.

One hundred of blood samples were collected from women female with hypothyroidism. which divided in to two groups: apparently control group 30 samples(with out any chronic disease) and 70 samples collected from patients with Hypothyroidism, then samples were divided to three group according to their ages: From 17-20 years (15 sample); 21 -40 year (20 sample) and more than 40 year (35sample) also the samples can be devided into three groups according to the body mass index (BMX).

The results showed that the statistical analysis of thyroxin (T4), triiodothyronine (T3) and thyroid stimulating hormone (TSH) hormone in hypothyroidism patients compared with AHC group.

The results revealed that significant decrease($p<0.002$)in the level of T4 hormone in patient. Compared with AHC group also significant decrease in the level of T3 hormone in patients in comparison with AHC

group while the level of TSH hormone was significantly increased (P <0.0001) in patients Compared with AHC group.

The results showed a significant decrease (P <0.011) in selenium levels in patients compared with the AHC group, whereas results showed a significant increase (P <0.0001) in malondialdehyde (MDA) level in hypothyroidism patients compared with the AHC group. The total antioxidants levels where significantly decreased (P <0.026) in patients in contrast to the AHC group also there were a significant decrease (P<0.0001) in ferritin level of patients wher compared with the AHC group. The levels of anti-thyroperoxidase and anti-thyroglobulin are significantly increased (P <0.028 and P <0.002 respectively) patient compared with the AHC group .

The current study indicated that the presence a for ferritin and selenium levels, also there was appositive correlation between thyroxin and triiodothyronine hormones with total antioxidants and thyroid stimulating hormone.

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List of Abbreviations

Symbol	Definition
AITD	autoimmune thyroid disease
Anti-TG	Anti-Thyroglobulin
Anti-TPO	Anti-Thyroid Peroxidase
ATP	Adenosine Triphosphate,
BMI	Body Mass Index
DUOX	Dual Oxidases
FT4	Free thyroxin
GPX	Glutathione peroxidase
H_2 O_2	peroxide hydrogen
HT	Hypothyroidism
I	Iodide
MDA	Malondialdehyde
Na+	Sodium
ROS	Reactive oxygen species
SE	Selenium
SIN	sodium iodide transporter
T3	3,5,3’-tri-iodothyronine
T4	Thyroxine

TAS	Total Antioxidant Status
TBARS	Thiobarbituric Acid Reactive Compounds
TBPA	albumin-binding hormone
TD	Thyroid disease
TG	Thyroglobulin
TPO	Thyroid Peroxidase
TRH	Thyroid-releasing hormone
TSH	Thyrotropin (formerly thyroid-stimulating hormone)
TSHR	Thyrotropin receptor
HDL	High-density lipoprotein

CHAPTER ONE

INTRODUCTION

1.1 Introduction:

The thyroid gland and its hormones are critical to organ and body development and the homeostatic control of basic physiological mechanisms and energy expenditure in all vertebrates (Maenhaut *et al.*, 2015). Hypothyroidism is the most common disorder of the thyroid gland, among adults, it is otherwise known as myxedema and in children as cretinism. Hypothyroidism can be primary or secondary based on its source of deficiency. More than 99.5% of instances of hypothyroidism (also known as primary hypothyroidism) are caused by hypofunction of the thyroid gland, with the remaining 0.5% being caused by pituitary and hypothalamus malfunction (Luo *et al*., 2021). Thyroid disorder can be brought on by either an increase or decrease in the thyroid hormones' output (Alyahya *et al*.,2021). Over 1.5 billion individuals are thought to be at risk for thyroid dysfunction, although there are only about 200 million confirmed cases of thyroid illness globally (Brouwer *et al* ., 2022).

Thyroid gland malfunction can be the primary cause of many disorders, including hypothyroidism, hyperthyroidism, (Grave's disease) (Hashimoto's disease). (Al-Bazi *et al*., 2021; Hu *et al*., 2022). Metals make up most trace elements. while many of them have negative consequences, some are necessary to the body functioning properly. (Mehri,2020). Hypothyroidism is the most common thyroid disorder and is often over looked. It is 5-10 times more common in females as compared to males (Vanderpump and Tunbridge., 2002). It is estimated that nearly 13 million Americans have undiagnosed hypothyroidism (Helfand, 2004). Database estimated that an annual incidence of hypothyroidism was 1.08 per million population in Japanese patients (Ono *et al*., 2017).Hypothyroidism can cause memory impairment,

slurred speech, and drowsiness. Menstruation, ovulation, and fertility are all impacted by thyroid hormone. (Shahid *et al* ., 2018).

The first phases of thyroid hormone synthesis, during iodide oxidation, already need reactive oxygen species (ROS), which are crucial. The modulation of mitochondrial activity by thyroid hormones also serves to regulate metabolism. The thyroid is especially vulnerable to oxidative injury since ROS are essential to its function (Benvenga *et al.*, 2021). Insufficient ferritin (Fe) supply, which is common in less developed countries, impairs the efficiency of the thyroid hormones biosynthetic process. (Winther *et al* ., 2020).

Selenocysteine-containing proteins provide cellular protection in addition to (H_2O_2) dependent biosynthesis and the activation of thyroid hormones, which is essential for their receptor mediated cellular activity. Common diseases associated with disturbed thyroid hormone status, such as autoimmune thyroid diseases and metabolic disorders, are caused by imbalances between the thyroid's iron and selenium contents (Köhrle, 2021). Epidemiological research have shown that Se insufficiency affects a significant portion of people worldwide. (Schomburg ,2020),and patients with abnormal thyroid profiles are more likely to have anti-thyroid antibodies than healthy people (Tipu *et al* ., 2018).

1.2 : Aim of Study

To investigate the effect of selenium and ferritin levels on the hypothyroidism patients as well as their function declines under oxidative stress. The aim of current study was achieved by using the following abjectives:

1. Determination the levels of Tri-iodothyronine (T3), tetraThyroxin (T4) and Thyroid Stimulating Hormones (TSH).

2.Determination thelevels of antibody concentrations for anti -thyroid peroxidase (anti -TPO) and anti – Thyroglobulin (anti -TG).

3. Determination the levels of malondialdehyde (MDA) and total antioxidant (TAS).

CHAPTER TWO

LITERATURES REVIEW

2. Literature Reviews

2. 1. Thyroid Gland

The thyroid gland is considered one of the most important endocrine glands in the human body, as it is located in the front part of the middle of the neck directly below the Adam's apple and specifically next to each of the trachea, esophagus and pharynx, as it surrounds the cricoid cartilage and the rings of the upper trachea and consists of two lobes, where the sternum is connected right in the left lobe through a thin band of tissue called the isthmus isthmus is what gives it the butterfly shape of the thyroid gland (Esen *et al*., 2018).

The weight of the thyroid gland varies from person to person, but it is in contrast with weight, gender and physiological conditions, as well as iodine levels in the diet, but its weight ranges from 15 to 20 grams (Kaplan *et al.,* 2015).Thyroid cells are quite epithelial: Each follicular cell is characterized by a basal side containing a nucleus, a rough endoplasmic reticulum, and an apical side. There is also another type of semicircular cell, and the latter are characterized by a small rough endoplasmic reticulum, mitochondria and a large Golgi apparatus. It is also characterized by the presence of secretory granules called C cells that secrete the hormone calcitonin (Giulea *et al*., 2019).

One of the basic functions of the thyroid gland is that the thyroid gland synthesizes, stores and releases thyroid hormones that are received by the entire body tissues through specific receptors for their importance in regulating energy and metabolism. Thyroid hormones regulate many physiological functions such as thermogenesis, reproduction, the female ovarian cycle, and lactation. They are also needed to regulate proper brain development in infants and metabolic functions in adults, thyroid gland in females is smaller than that of males and its growth is slower in females.

In both sexes, the thyroid gland increases in size with age (Al-Suhaimi and Khan ,2022). The polarity of thyroid cells is essential for the biosynthesis of thyroid hormones as this synthesis takes place in four steps, the first step in which iodide is captured for the first time at the level of sodium iodide transporter (SIN).side membrane and is transported to the intracellular milieu against the electrochemical gradient . It is then transported across the apical membrane by a passive transport process that is provided at least in part by (Na+/I-) The iodide is then instantly oxidized (Colloid), once in a colloid. Pendrin then it is linked to Peroxide hydrogen peroxide (H_2O_2)in the presence ofthyroperoxidase (TPO) by Thus, thyroglobulin (TG) residues of tyrosine are obtained, hydrolysis of thyroglobulin (TG) by lysosomes, T4 and T3 hormones are cleaved.(Benvenga *et al* ., 2018). in addition to these hormones, between the thyroid follicles or within the wall of the thyroid follicles, we find small C cells, also known as parafollicular cells. These are derived from neural crest cells and secrete a polypeptide hormone known as calcitonin. It is a peptide hormone made up of 32 amino acids and another hormone. This hormone controls the metabolism of calcium and phosphorus, which is important for the stability of endogenous calcium and phosphate in skeletal and other tissues leading to hypocalcemia. (khan and farhana .,2019).

2. 2. Regulation of Thyroid Hormones

The thyroid is part of the hypothalamic–pituitary–thyroid axis. The axis includes thyroid-releasing hormone(TRH), which is secreted by the hypothalamus. The thyroid-releasing hormone stimulates the release of thyroid-stimulating hormone (TSH) from the anterior pituitary gland. TSH, in turn, stimulates the thyroid to secrete thyroxine (T4) and triiodothyronine (T3), which are present in a free, active form and a

bound, inactive form. A negative feedback mechanism exists between TSH and thyroid hormones (Hadlow *et al*.,2013) . T3 and T4 hormones are released into the bloodstream T3 (20%) and T4 (80%) , It also binds to TBG-specific proteins and albumin-binding hormone (TBPA) for translocation into target cells (Braverman and Cooper, 2012).

T3 is four times more effective than T4, but it is present in the blood in much smaller quantities, as well as the half-life of T3 is less compared to (T4) (snyder,2012).

Thyroid hormones increases the basal metabolic rate. also control the basal metabolic rate of cells by increasing the basal oxygen consumption depending on the metabolic state, it can trigger lipolysis or fat synthesis. Stimulating the metabolism of carbohydrates and building proteins. (Pal ,2010 ; Shahid *et al*., 2018).

2.3.Types of thyroid disorders

2.3 .1 .Hyperthyroidism

It is an excessive concentration of thyroid hormones in tissues caused by increased synthesis of thyroid hormones, excessive release of preformed thyroid hormones, the most common causes of an excessive production of thyroid hormones are graves disease, toxic multinodular goiter, and toxic adenoma (Kravets, 2016). The increase in thyroid hormone synthesis is associated with an increase in iodine uptake that is stimulated by antibodies that activate the TSH receptor (Delitala *et al* ., 2015).There are two types of primary hyperthyroidism caused by Basedow disease, and Secondary hyperthyroidism resulting from an increase in the production and secretion of thyrotropin-releasing hormone from the hypothalamus stimulating the gland thyroid from the anterior lobe of the pituitary gland.Patients with long-standing untreated hyperthyroidism may develop atrial fibrillation or heart failure (Selmer *et*

al.,2012). Hypermetabolism induces weight loss despite an increased appetite.Neuromuscular symptoms include weakness of proximal muscles (Duyff *et al*.,2000).

2.3.2.Hypothyroidism

Decreased production of thyroid hormones by the thyroid gland is called as hypothyroidism; it could be either primary or secondary or twrtiary . Primary hypothyroidism refers to the abnormality in the thyroid gland itself and secondary is due to hypothalamic or pituitary disease,while (Gaitonde *et al*., 2012). Thyroid disorder is the second most common endocrine disorder, next to diabetes mellitus(Kochupillai, 2000; Unnikrishnan and Menon, 2011; Friedrich *et al*.,2020). Thyroid hormones control the basal metabolic rate of cells by increasing the basal oxygen consumption. Thyroid hormones also promote the synthesis of mitochondrial cytochromes and the activity of cytochrome oxidases. Therefore, in hypothyroidism, deficiency of thyroid hormones decreases oxygen utilization and hence, leads to hypometabolism. also affects physical and mental growth, development of central nervous system, intermediary metabolism (Pal,2010). When there is an increase in serum TSH above10mIU/L along with a decreased concentration of serum T4 and T3 is called as an overt hypothyroidism, and in Sub-clinical hypothyroidism there is an increase in serum TSH usually between 4-10mIU/L associated with a normal concentration of serum T4 and T3 (Pillai and Bennett ,2018).

Data derived from the National Health and Nutrition Examination Survey (NHANES III) suggest that about one in 1/ 300 persons in the United States has hypothyroidism(Hollowell *et al* .,2002).Clinical symptoms include fatigue, cold intolerance and weight gain the most severe manifestations are cardiac disease myxedema and myxedema

coma, which may be fatal if left unchecked (Wiersinga , 2012; Siegmann *et al*., 2018).

However, subclinical hypothyroidism progresses to overt hypothyroidism in proportion to the initial TSH level and progression is faster with the presence of anti thyroperoxidase antibodies. Hashimoto thyroiditis is one of the most common causes of primary hypothyroidism. It is a chronic autoimmune thyroiditis in which antibodies against thyroglobulin and thyroid peroxide are formed. These antibodies destroy the thyroid cells, finally leading to hypothyroidis (Friedrich *et al*., 2020).

At higher levels of TSH, and with T4 and T3 levels below the reference ranges, clinical symptoms of 'clinical' or 'overt' hypothyroidism will appear, such as weight gain, cold intolerance, constipation, edema, dry skin, bradycardia , fatigue. According to a meta analysis, autoimmune thyroiditis as a frequent cause of subclinical or overt hypothyroidism is associated with depression and anxiety disorders (Siegmann *et al*., 2018).

2.4. Etiology of Hypothyroidism

2.4.1. Age

There is a relationship between hypothyroidism with age, as the prevalence of hypothyroidism increases with age . However, the reference range for TSH also rises with age, as the population distribution of TSH concentration progressively rises with age (Leng *et al* ., 2019). Recent data from observational studies suggest that serum TSH levels increase in older people (Aggarwal salman razvi., 2013; Carlé *et al* ., 2016) .

2.4.2 **. External Radiation**

The thyroid gland is among the organs at the greatest risk of cancer from ionizing radiation. radiation exposure in childhood can cause thyroid cancer and benign thyroid nodules(Sinnott *et al* ., 2012). radiation

exposure also may induce hypothyroidism and autoimmune reactions against the thyroid The radiation dose is related to increased TPO antibody prevalence (Reiners *et al* ., 2020).

2.4.3. Chemicals

The regulation of thyroid hormones (THs) production and physiological action is complex and can be adversely disrupted by a broad spectrum of chemicals at environmentally relevant concentrations through different and intricate mechanisms, in particular pesticides(Hernández *et al*., 2020).

Polychlorinated biphenyls (PCBs) have thyroid-disrupting effects, and it is suggested that also bisphenol A, phthalates, brominated flame retardants, and perfluorinated chemicals show thyroid-disrupting characteristics . PCBs, or other persistent organochlorine compounds, disrupt thyroid hormone homeostasis, while dietary exposure to PCBs affects serum thyroid hormones and TSH in human subjects (Pearce and Braverman,2009;Boas *et al* ., 2012). Ahigh prevalence of hypothyroidism was observed in individuals exposed to polybrominated biphenyls with an associated elevation in thyroperoxidase antibodies (TPOAb)and Tg antibodies (TgAb). Bisphenol A, commonly used to manufacture plastic products, may bind to the TSH receptor (TSHR) and act as an antagonist to thriiodothyroinine (T3) thus, inhibiting its transcriptional activity (Burek *et al* ., 2009 ;Brent., 2010 ; Martínez *et al* ., 2012).

Many environmental pollutants have been shown to be toxic to thyroid cells and promote the onset of Autoimmune Thyroid Disease (AITD) (Brent ,2010).

2.4.4. Medications

Several medications may play a role in the development of autoimmune thyroid disease (AITD). lithium, amiodarone, and highly

active antiretroviral therapy are the agents most commonly associated with thyroid dysfunction (Burek and Talor., 2009). For most of these medications, patients at greatest risk of developing AITD are those with previous thyroid autoantibody positivity (Brent, 2010). Some medications, such as lithium, may not trigger autoimmunity but accelerate the autoimmune process by interfering with thyroid hormone synthesis. Thyroid function testing and measurement of TPO Ab should be considered before beginning these medications on patients (Eschler *et al* ., 2011).

2.5. Selenium

Selenium exists in two different forms, namely organic and inorganic, in which organic forms of selenium are present as selenocysteine and selenomethionine in human body. Inorganic forms such as selenite and selenate get accumulated in plants through soil. Selenium has the ability to combine with other minerals and elements of Sulphide, Copper, Silver, Nickel and Lead. Selenium holds the 67th rank for the most abundant element on the Earth's crust (USEPA , 2014).

Selenium occupies a special place among seventeen trace elements that are currently recognized as vital for human body animals and poultry minerals are inorganic nutrients, usually required in small. amounts from less than 1 to 2500 mg per day (Prashanth *et. al.,* 2015). Both selenium deficiency and toxicity are problems around the world (Santos *et al* .,2015).

2.5.1. Source of Selenium

The main source of Se is food, although its content varies in different human populations according to many factors such as ,geographical characteristics, such as soil concentration and climate, and different content in food, such as nuts, cereals, eggs, meat and fish.

Nonetheless, epidemiological data have demonstrated that Se deficiency characterizes a large percentage of individuals all over the world (Schomburg, 2020) . The amount of selenium in drinking water is not nutritionally significant in most geographic regions (Sunde, 2006).

Selenium is a trace element found in various body tissues such as the spleen, thyroid gland, kidneys, pancreas, brain, and testes. It also plays a role in liver metabolism and contributes to the maintenance of skeletal muscle, heart and sperm. Selenium is absorbed in the intestine and into the blood. and regarded as a component of glutathione peroxidase, an enzyme that plays an antioxidant role within cells. This antioxidant effect is essential in the detoxification of free radicals produced by cellular metabolism (Roman *et al*., 2014). Daily intake of selenium is highly recommended for maintaining the natural metabolism and homeostasis in the human body. The intake dosage of selenium is determined as 55 μg and 70 μg per day for adult males and females, respectively (Navarro-Alarcon and Cabrera-Vique, 2008).

2.5.2. Relationship between selenium and the thyroid gland hormones.

As an essential trace element, selenium (Se) plays an enormous role in the functioning of the human organism used in the biosynthesis of selenoproteins (proteins containing one or more selenocysteine residues). The functions of human selenoproteins in vivo are very diverse,and my Many selenoproteins have antioxidant activity in the thyroid by removing oxygen free radicals generated during the production of thyroid hormones. Being incorporated into iodothyronine deiodinases, selenium plays also an essential role in the metabolism of thyroid hormones (Rayman, 2012 ; Duntas and Benvenga , 2015;Minich, 2022).

Selenoproteins act as antioxidant warriors for thyroid regulation, male-fertility enhancement, and anti-inflammatory actions. also They participate indirectly in the mechanism of wound healing as oxidative stress reducers. Glutathione peroxidase (GPX) is the major selenoprotein present in the human body, which assists in the control of excessive production of free radicals at the site of inflammation (Hariharan and Dharmaraj,2020). The role of selenium in human body is important and crucial in stabilizing and neutralizing the body metabolism. During wound healing process, certain selenoproteins like GPX-1, GPX-4, selenoprotein S and selenoprotein P combine to perform various reactions such as antioxidant activities, inhibition of inflammatory cytokines and elimination of Peroxynitrate (a super radical ion) in inflammatory phase (Lei *et al*., 2009; Cox *et al* ., 2013 ;Talbi *et al*., 2019).

2.6.Ferritin (Fe)

Serum ferritin is an iron storage protein present in almost (Paterek *et al*., 2019). The ferritin molecule is an intracellular hollow protein shell, composed of 24 subunits surrounding an iron core that may contain as many as 4000–4500 iron atoms. In the body, small amounts of ferritin are secreted into the blood circulation,and In the absence of inflammation, the concentration of plasma or serum ferritin is positively correlated with the size of the total body iron stores (nutritional anaemias , 2017) . Iron is a micro-mineral that has a number of key functions. It's a major part of hemoglobin in red blood cells; as it carries oxygen from the lungs to all parts of the body and facilitates oxygen use and storage in muscles. In addition, every cell in the body needs iron to produce energy (Arora and Kapoor ,2012 ; Dasa and Abera, 2018). Serum ferritin, an index of iron store is present in almost all cells; however, it has been reported that an

alteration in ferritin levels occurs in patients with thyroid disease. (Malempati *et al* .,2009).

2.6.1. Relationship between ferritin and The thyroid hormones

Thyroid Peroxidase (TPO) is a thyroid-specific glycosylated hemoprotein of 110 k -Da that faces the luminal colloid of the thyroid cell. It is a key enzyme in the biosynthesis of thyroid hormone and is found on the apical membrane surface of thyroid follicular cells. It primarily catalyzes the iodide organification of tyrosine residues and the pairing of iodotyroisines residues in the molecule of thyroglobulin to produce The mature peptide, which has 919 amino acids, is followed by a preprotein that is made up of a 14 amino acid signal peptide. The human TPO mRNA is 3048 nucleotides long. DNA damage and mutation consequences might result from the high concentration of H2O2 that is created in response to a spike in TSH levels (Granner *et al*.,2003; Kollati *et al*., 2017) . Ferritin is storage form of iron and its serum level reflects iron stores of body, furthere more iron inadequacy can affect the proper functioning of the TPO enzyme that further affect the thyroid hormone production, Altered level of serum ferritin have been reported in patients with thyroid disease (Sahana and Kruthi, 2020). During iron deficiency, tissue iron start diminishing at the first. In turn synthesis of thyroid hormone is impaired by altering the activity of enzyme called hemedependent TPO (Zimmermann and Kohrle , 2002). Hypothyroidism, itself, may lead to low iron levels due to poor gut absorption as a result of decreased levels of digestive acids/ enzymes or due to associated autoimmune conditions like celiac disease (Harper *et al.*, 2007),also It may be as a result of heavy mensuration seen in some female patients (Das *et al*.,2012)

2.7. Anti-Thyroid Peroxidase and Anti-Thyroglobulin

Thyroid Peroxidase (TPO) is a key enzyme in the synthesis of the thyroid hormones, including thyroxine (T4) and triiodothyronine (T3). The thyroid hormones are made on Thyroglobulin (TG) an intra-follicle large glycoprotein, which also serves as a source for thyroid hormones. TG is secreted into the circulation where its estimated half-life is approximately 3 days (Tipu *et al.*, 2018). Serum anti-TPO Ab and anti-TG Ab titers correlate positively with an increased inflammatory reaction in the thyroid and development of hypothyroidism and there is a strong correlation between the echo graphic pattern and the anti-TPO Ab level in Hashimoto patients (Peretianu , 2005) . These antibodies can induce generation of oxidative radicals and induce apoptosis. The balance between stimulating TSHR and neutral antibodies can provide a balance between thyrocyte proliferation and apoptosis, then DNA released from apoptotic cells stimulates the immune response (Rapoport and McLachlan ,2016).

Patients with hypothyroidism at various levels is accompanied by autoantibodies (antithyroid peroxidase antibody antithyroglobulin antibody) and lymphocytic infiltration in thyroid tissue. It is more common in the 30- to 50-year-old age group and is 4–10 times more common in women than in men (Hutfess *et al*., 2011 ; McLeod *et al.*, 2014). The use of medications that may affect thyrocyte function, such as these antibodies, has also been related to an increased risk of thyroid dysfunction, The risk of hypothyroidism has also been linked to the use of drugs that may influence thyroid function, including these autoantibodies. TPO Ab positivity before treatment is linked to a 3.9-fold higher risk of thyroid disorders (TD), according to an autoimmune index association between TD and the efficacy of interferon therapy for chronic

hepatitis B (CHB).TD is a frequent side effect of pegylated interferon alpha (Peg-IFN) therapy used to treat oncology and immunology conditions, as well as when interferon is used to treat conditions other than CHB. Women had a 4.4 times higher risk of developing TD than men, so peg-IFN administration should be closely monitored in patients with thyroid antibody positivity, especially in women (Liu *et al* ., 2023).

The aging process is, likewise, associated with a greater presence of these antibodies. Moreover, these antibodies have been detected in up to 50% of women with a first degree relative with AITD and in 30% of men in the same situation and thus, suggests a dominant inheritance pattern(Vaidya *et al*., 2002).

Anti-TPO and anti-TG antibodies are related to the levels of thyroid stimulating hormone (TSH) and both alone or in combination have been used to predict development of hypo-/hyperthyroidism. It has been determined in different studies that altered levels of anti-thyroid antibodies and TSH in euthyroid subjects have been associated with development of hypothyroidism (Walsh *et al*., 2010 ;Roos *et al*., 2010).

In addition autoantibodies have shown valuable results as early diagnostic markers in many diseases such as cancer, rheumatoid arthritis, and celiac disease (Lesli *et al*., 2001 ; Allawi *et al*., 2022).

The measurement of anti-TPO in combination with TSH and T4 is useful in identifying hypothyroid subjects with potential risk of developing thyroid disease and, therefore, is useful for close monitoring, frequent follow-up, and early treatment decisions to prevent morbidity and associated diseases such as cardiovascular disease (Azizi ,2015; Kim , 2017).

2.8. Malondialdehyde (MDA)

Malondialdehyde (MDA) is one of the final products of polyunsaturated fatty acids peroxidation in the cells, an increase in free radicals causes overproduction of MDA. Malondialdehyde level is commonly known as a marker of oxidative stress and the antioxidant status in cancerous patients (Gawe *et al* .,2004).

MDA is the prototype of the so called thiobarbituric acid reactive substances (TBARS). MDA, are the most frequently measured biomarkers of oxidative stress, namely of lipid peroxidation (Tsikas, 2017). this MDA is believed to originate under stress conditions and has high capability of reaction with multiple biomolecules such as proteins or DNA that leads to the formation of adducts (Luczaj and Skrzydlewska, 2003; Zarkovic *et al*., 2013; Kudryavtseva *et al*., 2016).fur there more ROS can be produced by the cytoplasm, mitochondria, and peroxisomes (Forrester *et al* ., 2018).

2.8.1 .Relationship between Malondialdehyde (MDA) and thyroid hormones.

Reactive oxygen species (ROS) have an important role in normal thyroid function. Thyroid cells release oxidases, which catalyze ROS production (Dupuy *et al*.,1991; Ameziane,2016). Because of the reliance thyroid gland on ROS in its function, it is particularly exposed to oxidative damage Therefore, antioxidant defence system of the thyroid must effectively regulate ROS production and scavenging (Pace *et al*.,2020).

Thyroid hormones are the most important factors influencing the basal metabolic rate during normal physiological states by altering the mitochondrial oxygen consumption, the main production site of free radicals. So any changes in thyroid hormone levels might affect the

mitochondrial free-radical generation (Oziol *et al.*, 2003). Dual oxidases (DUOX), enzymes crucial for hydrogen peroxide generation, are essential for thyroid peroxidase(TPO) catalyzed hormone (Ohye and Sugawara ., 2010). Mitochondria are the main production site of free oxygen radicals, which can cause organ dysfunction by oxidation of cellular macromolecules such as carbohydrates, lipids and proteins. Oxidative stress may result from either overproduction of these species or from failure of the antioxidant defence systems, Oxidative stress is a syndrome resulting from an imbalance between antioxidant defense systems and the production of free radicals (Lushchak,2014)

Total antioxidant status (TAS) gives information about all of the antioxidants in the organism, while malondialdehyde (MDA) is a lipid peroxidation marker used to assess lipid peroxidation due to increased oxidative stress (Torun *et al* ., 2009).

MDA is not only a biomarker of oxidative stress but also a bioactive compound with several biological roles , because of its multiple biological functions (Jové *et al*., 2020) . MDA can act as a signaling messenger in insulin secretion. also as a stimulator of collagen gene expression in hepatocytes (García *et al.*, 2002). MDA has been indicated as presumably the most mutagenic molecule among ROS end products ,as well as the interaction of MDA resulting from a non-enzymatic process with other biomolecules, such as proteins, amino groups, and DNA (Onyango and Baba , 2010). the majority of MDA produced is found in the conjugated form. MDA adjuvants are highly immunogenic, that is, capable of inducing an immune response. It has been found to be associated with autoimmune diseases, such as lupus erythematosus and nephritis (Hardt *et al*., 2018). In many serious diseases, especially those related to aging, oxidative stress is the main factor,and is the case with

cancers, eye diseases (cataracts and macular degeneration) and neurodegenerative diseases (lateral sclerosis and Alzheimer's disease) (Sharifi-Rad *et al*., 2020).

2.8.2. Relationship between total antioxidant (TAS) and the thyroid hormones.

ROS and antioxidants significantly interfere with oxidation-reduction processes in cells and organisms, changing the redox (or oxidative) state of the cell; such states can stimulate or inhibit activities of various signal proteins, leading to the alteration of signal pathways. An oxidative milieu can lead to cell destruction by apoptosis or necrosis, and reducing milieu can lead to cell survival (Muchová ., *et al* 2014). The synthesis of thyroxine (T4) and triiodothyronine (T3) catalyzed by thyroid peroxidase (TPO) in thyroid follicles is a very complex process involving ROS, notably, H_2O_2 (Ohye and Sugawara ,2010). ROS are already essential in the initial stages of thyroid hormone production, during iodide oxidation. Additionally, thyroid hormones perform a metabolic regulatory function by affecting mitochondrial activity (Thanas *et al*.,2020).Therefore, the antioxidant defence system of the thyroid must effectively regulate ROS production and scavenging (Piras *et al*.,2021). In turn, at low physiological levels, ROS play a signaling role,essential for normal cellular processes,also serve as intracellular mediators produced in phagocytic cells, controlling the inflammatory response and antimicrobial defense (Marzo *et al* ., 2018).

The imbalance may be due to nutritional deficiencies in antioxidants or endogenous overproduction or exposure to environmental pro-oxidant agents. Atherosclerosis, cancer, cardiovascular disease, inflammatory diseases and aging .Oxidative stress is an abnormal condition that cells or sometimes tissues experience when they are

exposed to the internal or external production of oxidative free radicals that exceed their antioxidant capabilities. An excess of free radicals that are not neutralized by the defense system is very harmful to the essential macromolecules of cells, causing abnormal expression of genes and membrane receptors, cell proliferation or death, immune disorders, mutations, and deposition of proteins or lipids in tissues(Karajibani *et al*., 2018). Inflammation causes oxidative stress because the production of free radicals is material for immune cell activation. The interaction between oxidative stress and inflammation can play an essential role in the pathogenesis of several diseases in humans (Karajibani *et al*., 2018).

Reactive oxygen species (ROS) are molecules capable of independent existence, which contain an oxygen atom and unpaired electrons (Jakubczyk *et al*., 2020). ROS arise mainly as by-products in a series of bioenergetic processes of ATP synthesis in mitochondrial respiratory chains (Tan *et al*., 2018 ; Yang and Tian , 2020).

2.9. Thyroid hormones and obesity.

Thyroid dysfunction is associated with changes in body weight and composition, body temperature, and total resting and energy expenditure regardless of physical activity in population-based cross-sectional study of 27,097 individuals over the age of 40 with a BMI (body mass index) of 30.0 kg/m2 at least. subclinical and overt hypothyroidism is associated with a higher body mass index and a higher prevalence of obesity in both smokers and non-smokers. Both subclinical and overt hypothyroidism are often associated with weight gain, decreased thermogenesis, and decreased metabolic rate (Asvold *et al*., 2009).

Fox *et al*., (2008) study support clinical evidence that mild hypothyroidism is associated with significant changes in body weight and etween TSH and is a possible risk factor for overweight and obesity.

Body Weight Among individuals with hypothyroidism, evidence suggests that slight differences in thyroid function, contribute to the development of obesity or a tendency to be overweight, although this has not been shown. good BMI is negatively associated with serum free T4 (FT4), and fat accumulation is associated with decreased FT4, resulting in a positive correlation between TSH and progressive weight gain over time.

It is well known that thyroid hormones control the amount of heat is produced; in fact, around 30% of the heat produced to maintain body temperature depends on thyroid hormone function (Kim, 2008). Energy intake is lowered when a person has hypothyroidism, which is characterized by low thyroxine levels (Xu *et al*., 2019). Adding to the clinical evidence that mild hypothyroidism is associated with significant weight changes and may be a risk factor for obesity and overweight A healthy body mass index (BMI) and serum free T4levels are negatively connected, but FT4 levels were favorably correlated with the accumulation of fat over time. (Fox *et al.,* 2008).

Hypothyroidism induces a decreased basal metabolism and thermogenesis, an accumulation of hyaluronic acid and a decreased renal flow, all factors leading to water retention. Severe hypothyroidism states lead to a clinical picture known as myxoedema in which hyperkeratosis of the skin and facial edema could give the patient a false appearance of overweight (Malley *et al*.,200; Karmisholt *et al*., 2011).

Chronic constipation brought on by sluggish peristalsis in hypothyroid patients may contribute to weight gain. Water retention is the major cause of this weight gain, which is unrelated to an increase in fat mass (Biondi ,2010).

CHAPTER THREE
Materials and Methods

3 . Materials and Methods:

3.1. Materials :

3.1.1:laboratary equipment and apparatus

The laboratory equipment that used in the present study are mentioned in table (3.1).

Table (3.1): laboratory equipment and apparatus used in the present Study.

No	Devices and Tools	origin
1	Centrifuge	Germany
2	Cotton	NFLB/CHINA
3	ELISA	China
4	Eppendorf tube	China
5	Gavage	German
7	Gle tube	China
8	Gloves	Malaysia
9	Hitachi cup	China
10	Homogenizers	UK
11	Incubator Glass	China
12	Micro pipette	United States
13	MicrocentrifugeTubes 2ml	China
14	Micropipettesand Tips (small, large)	Gilson/ France
15	Mindray BS-240	China
16	Mindray CL-900i	China
17	Mini vidas	French Biomeriax
18	Oven	China

19	**plane tube**	**AFCO-DISPO/ Jordan**
20	**Refrigerator plastic containers**	**China**
19	**Refrigerator with freezer 20C**	**LG/Korea**
20	**Refrigeratore**	**Turkey**
21	**Roller Mixer**	**India**
22	**STEL3**	**Spain**
23	**Sterile Syringes 5ml**	**Germany**
24	**tips**	**Japan**
25	**Water Bath**	**Germany**
26	**Water bath**	**China**

3.1.2:chemical and biological materials.

The chemical and biological materials used in the present study are list of in the table (3.2).

Table (3.2): The chemical and biological materials used of in the present study.

NO	Chemicals	Manufacturing company
1	**Ascorbic acid**	**Luckmedical/China**
2	**HCl**	**Arcelik/Turkey**
3	**Na2SeO4**	**Cleanhand/Malaysia**
4	**nitric acid**	**Luckmedical/China**
5	**perchloric acid**	**Easymed/Germany**
6	**Phosphomolybdenum reagent**	**Mindray/China**
7	**selenium solutions**	**Arcelik/Turkey**

8	**streptavidin solution**	**Mindray/China**
9	**TMB**	**linear/ Spain**
10	**trichloroacetic acid**	**Mindray/China**
11	**Xylenol Orange**	**Memmert/Germany**

3.1. 3: commercial Kits:

Table (3.3): the kits that used in the currert stady are listed in the (3.3).

No	Kits	The Company (Origin)
1	**Anti TG kit**	**United States**
2	**Anti-TPO kit**	**United States**
3	**Ferritin- Kit**	**Mindray /China**

3.2. Methods

3.2.1:Study population.

seventy blood samples were collected from women patients with hypthyroidism in the endocrinology laboratory from the period from june to noveember 2022 at imam AL-sadiq hospital and Al-Marjan General Hospital also 30 blood sample were collected from healthy control define as aparently healthy control group with no evidence of hypthyroidism and chronic disease . the patients were divided in to three groups according to the age ,which are 17-20 years, 21-40 years and over 40 years Body mass index (BMI) (Kg / M ^ 2) is also taken from measurement methods Height and weight of each woman. They were divided into three groups normal weight (18.5-24.9 - 25 - 29.9)over weight 25-29.9 and obes >30 . T4, T3 and TSH levels were estimated using mini vidas. for levels. Both ferritin, anti-TG, and anti-TPO. Their concentrations are determined by the enzyme-linked immunosorbent method. and measurement of

selenium, malondialdehyde (MDA), and total antioxidants (TAS)by manual means.

3.2.2 Exclusion Criteria

People with severe disease, recent history of blood transfusion, heart Liver failure, history of hypothyroidism, radioactive iodine therapy and men They were excluded from the study.

3.2.3 Ethical approval :

The study has been conducted in accordance with the guidelines. Ethical contained in the Helsinki Declaration. Before sampling, verbal and other analytical consent is obtained for the patient. In order to obtain this permission, the study protocol, subject information, approval form and delegation were assessed by the local ethics committee in accordance with document No. 100 (which contains the number Z22120 and approval date: 6-12-2022).

3.2.4. Collection of Blood Samples

Five ml of Blood samples were taken from patients hospitalized for the purpose of conducting hormonal tests . The blood sample was taken from the vein in the left arm in front of the elbow, as the area was sterilized with a piece of medical cotton soaked in 70% ethyl alcohol, then a bandage was used over the elbow area (6-8 cm), and blood was drawn using a medical syringe with a capacity of 5 ml, The volume of the sample taken is (5) ml, and put the blood in gel tubes devoid of anticoagulant, as they contain a gelatinous substance that helps to increase the separation of the serum formed after the centrifugation process, as the samples were left for15 minutes at room temperature (20-25 °C), then placed Then inside the Centrifuge at a speed of 3000 rpm for 10 minutes for the purpose of separating the serum, the serum to measur

the were stored at -20°C .

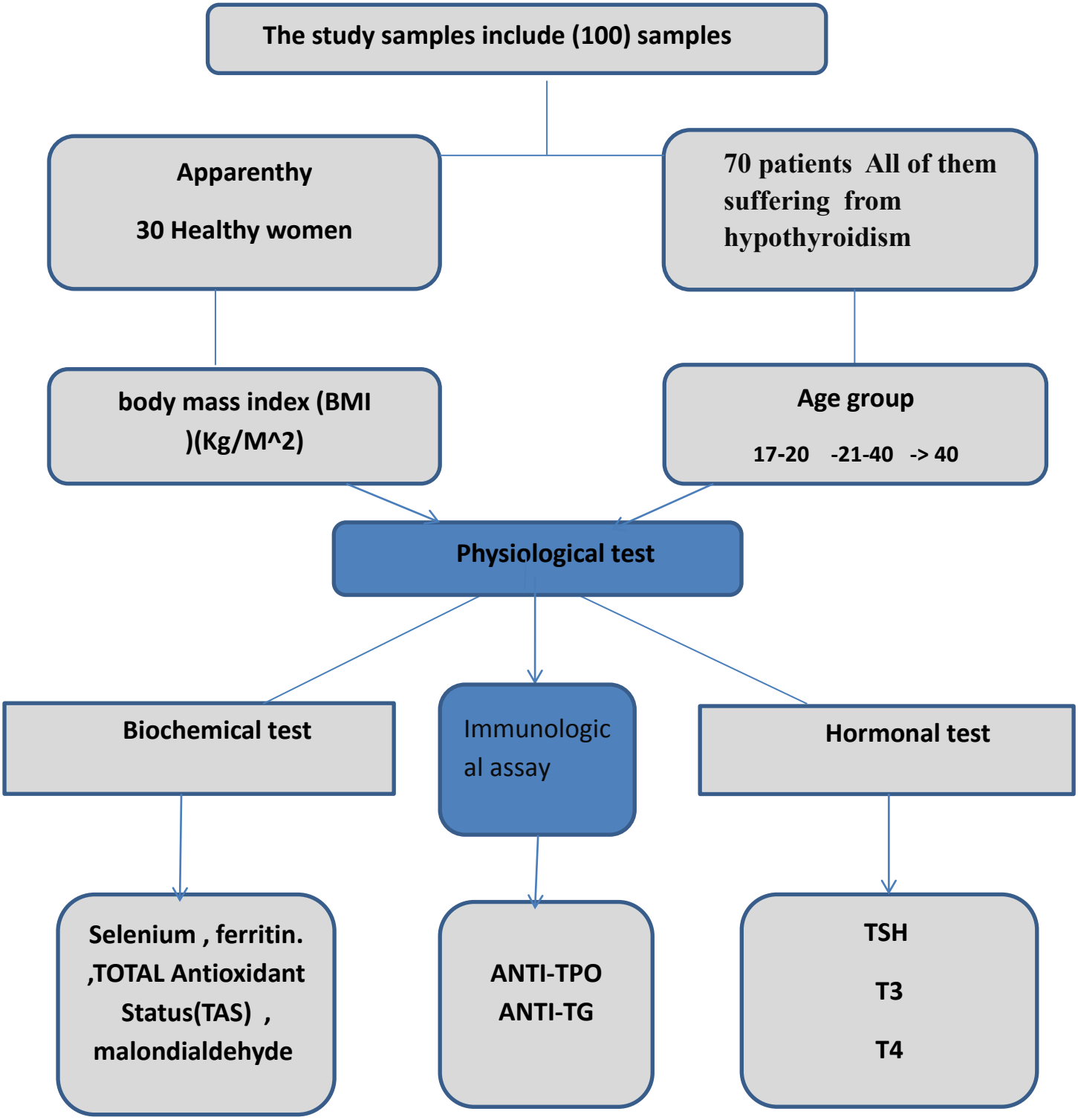


Figure (3.1): scheme of study Design

3.3.5. Survey Questionnaires.

A questionnaire was taken from the patient's age, gender and body mass index (BMI).

3.4. Anthropometric assessments.

anthropometric techniques used to measure weight and recommended by Sturm, (2007). All anthropometric measure taken with stress on body height and weight that were measured in using a portable stadiometer.

Height: is determined using a wall mounted, non-extendable measuring tape with subjects standing in an erect barefoot position, arms by side, and feet together.

Weight: measurements are taken with each subject standing at the center of the weighing scale in light clothing without shoes and socks.

Body mass index (BMI) is calculated using the formula

BMI = weight (kg)/ height² (m^2) and classifying under weight (BMI<18), normal BMI 18-24.9), overweight (BMI 25 - 29.9), obesity (BMI 30-39.9) and morbid Obesity (BMI > 40) (Sturm, 2007).

3.5. Biochemical Tests.

3.5.1. Thyroid activity Tests.

3.5.1.1. Measurement the levels of the thyroxine T4 hormone in the blood serum

The levels of the hormone thyroxine (T4) in the blood serum were measured using the Mini VIDAS device manufactured by Biomerieax franch with a ready-made kit, following the instructions attached to the test kit for the hormone T4.

3.5.1.2. Principle of assay.

The principle of interaction in measuring the concentration of the T4 hormone works on the basis of immune competition to bind to the enzme, with detection of the final radiation formed An enzyme

immunoassay.The steps for measuring the T4 hormone are carried out automatically by means of the Mini VIDAS device, and the reaction medium is moved periodically to and from the SPRs and the solutions contained in the strip several times, and the sample is included into the hole containing Anti-T4 antibodies labeled with conjugated alkaline phosphates, so the mixture (conjugate/ Sample) moves periodically to and from the SPRs, so the antigen is bound to the antibodies on the SPRs, as well as to the linker, then forming a sandwich. During the final steps of crossing, the base substance Umbelliferl phosphate 4 moves -Methyl cyclically into and out of the SPRs, and the enzyme then catabolizes the substrate into a radioactive product, which is 4-methyl umbelliferone, whose radiation is measured at a wavelength of 450 nanometers. The radiation indicates the relative concentration of the antigen present in the sample. At the end of the titration process, the results are calculated automatically by the Mini VIDAS device using the standard curve stored in the device's memory, after which the results are printed by the device.

3.5.1.3. procedure.

1. Insert the MLE card of the test kit into its designated slot on the device The Mini VIDAS, through which the device recognizes the test automatically, without which the device cannot show the result and then print it.

2. Use one (T4) SPR, STR strip for each sample of blood serum and control standard solution (Standard S1) and placed in the designated place in the device.

3. (two hundred µL) of blood serum was drawn and placed in its hole on the (T4) STR strip as well as the control and the standard solution.

4. followed the steps for the device, which are found in the practical guide of the device, so that the device starts calibration automatically, and the process takes (40) minutes.

5. After the results are crossed and the results are printed, the STR and SPRs are extracted and placed in a special container, because it is used only once.

3.5.2. Measuring the levels of triiodothyronine hormone T3 in blood serum.

Serum T3 levels are measured using the Mini VIDAS device, following the steps provided in the instruction manual attached to the T3 test kit imported from Biomerienx, as the same steps used in measuring T4 concentration were followed.

3.5.3. Measurement of TSH levels in blood serum.

Serum TSH levels are measured using the French-made Analysis Kit using Mini VIDAS, following the instructions attached to the TSH test kit.

3.5.3.1.Principle of assay .

The principle of measuring TSH levels is based on immune competition for enzyme binding with screening for endogenous radioactivity.

An enzyme immunoassay competition method with final detection (ELFA).The steps for measuring TSH hormone levels are done automatically by the(Mini VIDAS) device, and a reaction medium moves periodically to and from the SPRs and the solutions in the strip several times, and the sample is transferred into the hole containing Anti-TSH antibodies tagged with the linker Alkaline phosphatase, as the mixture (conjugate/ Sample) moves periodically to and from the SPRs, thus the antigen binds to the antibodies on the SPRs, as well as to the

linker, then forming a sandwich, and during the final steps of the titration the substrate moves.

Umbelliferl phosphate 4-Methyl is cyclic to and from SPRs, and the enzyme then works by degrading the base substance into a radioactive product, which is 4-methyl umbelliferone, for which the amount of radiation is measured at a wavelength of (450) nanometers, and the radiation indicates the relative concentration of the antigen present in the sample At the end of the crossing process, the results are automatically calculated by the Mini VIDAS device using the standard curve stored in the device's memory, and then the results are printed by the device.

3.5.3.2 .The procedure .

1. The MLE card of the test kit is inserted into the device

The (Mini VIDAS), through which the device recognizes the test automatically, without which the device cannot show the result and then print it.

2. One strip of (TSH) SPR, STR is used for each sample of blood serum, control and standard solution S1 and placed in the designated place in the device.

3. (two hundred µL) is drawn from the blood serum sample and placed in its hole on the (TSH) STR strip as well as for the Standard, Control.

4. The steps for the device in the Manual have been followed, so that the device will start calibration automatically, which takes a period of (40) minutes.

5. After the calibration is done and the results were printed, the STRs and SPRs were extracted from the device and placed in a special container, because they are used for one time only.

3.6. Measurement of Selenium levels in blood serum.

3.6.1 . The principle of assay.

A new sensitive reagent for the colorimetric detection of selenium is proposed: xylenol orange. When combined with selenite at room temperature in a slightly acidic media, the reagent creates a pink radical cation. The highest absorption for the pink species is seen at 568 nm.

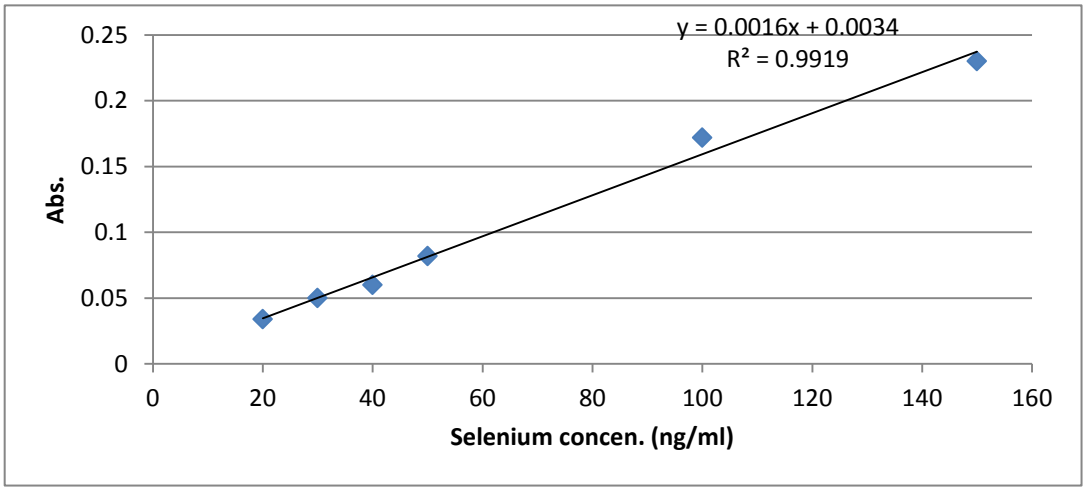
3.6.2. Procedure.

The sample is mixed to 400 μL of nitric acid and 100 μL of perchloric acid. , vortexed for 30 s and subsequently heated at 190°C (185–200°C) for 90 min . The tubes are cooled for 10 min, exposed to air, and then 500 μL of hydrochloric acid (HCl) is added. The tubes are then vortexed for 20 s and heated again for 60 min at 150°C under the same conditions as for perchloric acid digestion.

To each 100 μL of selenium solutions, add 500 μL of a universal buffer solution with a pH of 6.59 and 200 μL of a 10-3 M Xylenol Orange solution. Dilute to the proper concentration with twice-distilled water. Measure the absorbance at 568 nm in comparison to the matching reagent blank made in the same way, after thoroughly mixing (Amin and zarehb, 1996).

3.6.3 . Preparation of Standard Curve.

By dissolving sodium selenate, Na2SeO4, a stock solution of selenium (150 ng/ml) was created. From this solution, successive dilutions were produced to obtain selenium (IV) concentrations of 20, 30, 40, 50, and 100 ng ml1.



Figure(3.2) standard curve for determanation the level of selenium.

3.7 . Estimation the level of ferritin.

All reagents and samples are brought to room temperature (18 - 25ºC) before use. The removable 6-well strips are labelled as appropriate for the experiment. 50 samples containing ferritin were put to the appropriate wells along with 100 microliters of the standard for testing. After that, wells were covered and gently shaken while being incubated for 2.5 hours at room temperature. The solution was removed after incubation, and wash buffer solution was added four times. Using a multi-channel pipette or an automatic washer, 300 µL of wash buffer is poured into each well to do the washing. A successful performance depends on the complete elimination of fluids at each phaseAny wash Buffer that was still present after the final wash was aspirated or decanted. The plate was then turned over and blotted with fresh paper towels. Following that, 100 µL of produced biotin coated antibody was added to each well, and the wells were gently shaken while incubating for 1 hour at room temperature. The solution was discarded, and step four's washing procedure was followed. The next step was to pour 100 µL of the prepared streptavidin solution into each well. The plate is then gently shaken while being incubated for

45 minutes at room temperature. After that, the solution was thrown away, and step 4's washing procedure was repeated. TMB One-Step Substr 100 microliters.

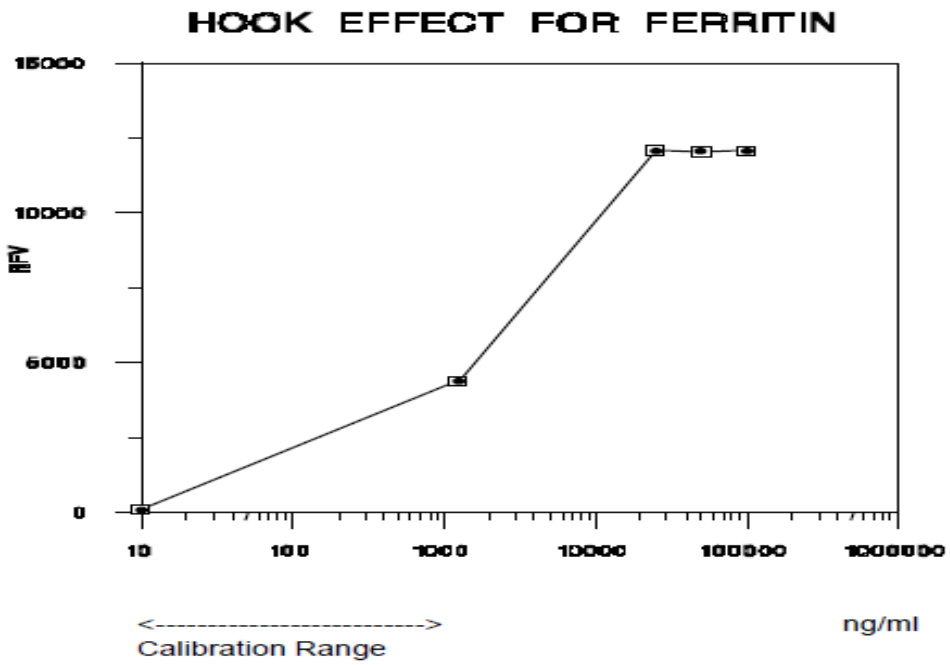


Figure (3.3): Standard Curve for determination the livel of ferritin .

3.8. Estimationthe level of Anti -TPO by ELISA .

3.8.1. Anti -TPO analysis.

3.8.1.1. Principle.

Principle of the assay The human antibody IgG enzyme-linked immunosorbent adsorption test is used for the qualitative and semi-quantitative examination of TPO in human serum. As the test system is used to aid in the diagnosis of thyroid disease, the pits of the plastic plate strips are more sensitive by thyroid mediation.

Low absorption of TPO antigen The test procedure includes three stages of incubation, namely:

1. Test sera (diluted) are incubated after being added to the antigen-coated plate. Any antigen specific to the antibody in the sample will bind

to the antigen. Wash the plate to remove non-antibody associated with other serum components.

2. Peroxidase-linked human IgG antibody is added to the pits and the plate is incubated. The bound portion will immunologically bind to the IgG in the pits, and as a result, the IgG molecules become a "sandwich" between the solid phase and the enzyme-bound antibody. Pits are washed to remove unbound antibodies.

3. The peroxidase-conjugated plate is incubated with the baseline solution. The hydrolysis of the substrate by peroxidase produces color changes. After 20 minutes, the reaction is stopped by adding a solution (H2SO4,0.7MHCL). The color intensity of the solution is measured by spectrophotometer. Light Intensity for the solution dependent on the antibody concentration in the original test sample.

3.8.1.2. Assay Procedure.

All components of the laboratory kit are brought and placed at room temperature (20-250 C) before use. The test sera of the negative control, the gradient and the positive control are diluted 21:21 (10-200L) in the dilution solution, as the dilution solution was added in the test tubes first, then the patients' sera are added with The use of micropipette tips is different for each model. 100 µl of diluted patient, gradient and control samples are distributed into appropriate wells. Add 100 microliters of the dilution solution to hole A1, labeled with Planck's reagent. I mixed well for 10 seconds and this is important to complete the mixing in this step. The plate is incubated at room temperature for (-/+25 minutes) Remove the incubated mixture by throwing out the contents of the plate .The plate is washed and cleaned five times with the rinsing buffer at a concentration of (10).Blow the plate vigorously on blotting paper to remove all remaining water droplets.Distribute 100 microliters of the

enzyme-bound reagent to each hole, including the Planck reagent hole .And blended gently for 10 seconds. Remove the incubated mixture by throwing out the contents of the plate. The plate is washed and cleaned five times with the rinsing buffer at a concentration of (10) . Blow the plate vigorously on blotting paper to remove remaining water droplets. 100 microliters of tetraTMB is added to each hole including the Planck reagent hole which leads to . The appearance of a blue color, and blended gently for 10 seconds. It was incubated at room temperature in the dark for (10-15 minutes). Stop the reaction by adding 50 microliters of the stopping solution to each well, including the holes of the Planck detector. I mixed carefully for 30 seconds and this is important to make sure that the blue color turns yellow in a uniform way Complete. I read the plate at 450 nm in the reading device.

3.8.1.3. Calculations.

The average optical density values (OD450) are calculated for each group of gradient solution, positive control, negative control and test sera. Graph as the optical density values on the x-axis and the focus on the y-axis. The average optical density values for each sample are used to determine the concentration of TPO IgG.

3.8.2. Estimation the level of Anti -TG.

Determination of the concentration of autoantibody to thyroglobulin (ANTI- TG) by adsorption method, Evaluation the concentration of Anti-Thyroglobulin by ELISA.

3.8.2.1. Principle of Examination .

The enzyme-linked immunosorbent assay for thyroglobulin Tg is used for the adsorption ability of biological materials on a plastic plate surface for the semi-quantitative determination of IgA, IgM and IgG antibodies to thyroglobulin antigens in human serum. The test plate is

covered with pure thyroglobulin antigens. When patients' sera are added, they bind to the antigens on the surface of the plate. If the antibody is specific to the antigen, an immune complex will form. After incubation, the plate is washed to remove unbound antibodies, followed by the addition of HRP peroxidase conjugated to human antibodies IgA and IgG. To etch and incubate the plate, the conjugated enzyme will bind to the immunocomplexes in the etch, then after incubation the plate is washed to remove excess quantities of the conjugated enzyme, then TMB baseline solution is added. If the antibody present in the patients' sera is specific for the antigen it will give a blue colour. After 20 minutes, the reaction is stopped by adding a solution IN (H2SO4), which leads to the color turning yellow, which represents the concentration of antibodies in the serum. The plate is read by a spectrophotometer, as the light intensity of the solution depends on the concentration of the antibody in the sample, and the proportion is direct.

3.8.2.2. Assay procedure.

All components of the laboratory kit are brought and placed at room temperature 20-25 degrees before use. The test sera, the negative control, the gradient, the high positive control and the low positive control (1:21 -200-100) are diluted in the dilution solution, as the dilution solution was added in the test tubes first, then the patients sera were added with the use of different micropipette heads for each model. Distribute 100 microliters of diluted gradient and control patient samples into appropriate wells. Add 100 microliters of the dilution solution to hole A1, known as Planck's reagent.- I mixed well for 10 seconds and this is important to complete the mixing in this step. The plate is incubated at room temperature for (5-30 minutes). Remove the incubated mixture by throwing the contents of the plate.

The plate is washed and cleaned five times with the rinsing buffer at a concentration of 20. The plate was hit vigorously on blotting paper to remove all remaining water droplets. 10 dispense 100 microliters of HRP-linked enzyme reagent to each well, including the reagent well. Blanc and mix gently for 10 seconds .

The plate is incubated at room temperature for 5-30 minutes The incubated mixture is removed by throwing out the contents of the plate. The plate was washed and cleaned five times with the rinsing buffer at a concentration of 20. Blow the plate vigorously on blotting paper to remove any remaining water droplets. 100 microliters of TMB is added to each hole including the Planck reagent hole, which leads to The appearance of a blue color, and blended gently for 10 seconds. It was incubated at room temperature in the dark for 10-15 minutes. The reaction was stopped by adding 50 microliters of the buffer solution to each hole, including the hole of the Planck detector. - I mixed carefully for about 30 seconds. This is important to make sure that the blue color turns yellow completely Complete I read the plate at 450 nm in the reading device**.**

3.10. Estimation of Malondialdehyde (MDA).

Estimation of serum malondialdehyde (MDA), malondialdehyhe was done by Thiobarbituric acid (TBA) assay according to the method of Buege and Aust, (1978) on spectrophotometer .

3.10.1. Principle.

This method quantifies lipid peroxides by measuring aldehyde breakdown products of lipid peroxidation. Basic principle of the method is the reaction of one molecule of malondialdehyde and two molecules of thiobarbituric acid to form a red MDA-TBA complex which can be measure at 535 nm. It is prepared by dissolving 15% W/V trichloroacetic

acid and 0.375% W/V thiobarbituric acid and 0.25N HCl to make 100 ml (2.1 ml of concentrated HCl in 100 ml). This solution is mildly heated to assist in the dissolution of TBA. Dissolved 15 gm TCA and 0.375 mg thiobarbituric acid in 0.25 N HCl and volume is made up to 100 ml with 0.25 N HCl.

3.10.2. Procedure.

To 0.4 ml of serum, 0.6 ml TCA-TBA-HCl reagents are added. It is mixed well and kept in boiling water bath for 10 minutes. After cooling 1.0 ml freshly prepared 1N NaOH solution is added. This absorbance of pink colour is measured at 535 nm against blank which contained distilled water in place of serum. In blank 0.4 ml distilled water and 0.6 ml TCA-TBA-HCl reagent is mixed and boiled. Blank is always taken.

(Buege and Aust ., 1978)

Calculation:

$$\text{Malondialdehyde}(\mu\text{mol/l}) = \frac{\text{Absorbance of sample}}{E_o \times L} \times D$$

Where:

E_o = Extinction coefficient 1.56×10^5 M^{-1} cm^{-1}

L= light path cm.

D = dilution factor = 6.7×10^6

3.11.Total antioxidant activity (TAS).

Principle The total antioxidant activity was determined by the phosphomolybdenum method.The absorbence is measured at 695nm using an UV/Vis spectrophotometrically. The antioxidant capacity was expressed as Ascorbic acid equivalent(AAE) by using the standard Ascorbic acid. reagents required

Standard solution:-50mg of Ascorbic acid is dissolved in 50ml standard flask using distilled water.(conc., 1mg/ml).Extract solution:- 50mg of

methanolic dried extract is dissolved in 50ml standard flask using distilled water.(conc., 1mg/ml).phosphomolybdenum Reagent:-
0.6M H2S04,28mM sodium phosphate,4mM ammonium molybdate.

procedure .

Prepare (50-250µg) concentration of standard & extract solution, from that take 0.3ml of each sample respectively. To all the tubes add 3.0ml of Phosphomolybdenum reagent. 0.3ml of water and 3.0 ml of reagent alone serves as blank. All the tubes incubate at 97°C for 90 minutes. Cooled and the absorbance was measured at 695nm using an UV/Vis spectrophotometrically against the blank . The antioxidant capacity was expressed as Ascorbic acid equivalent(AAE) by using the standard Ascorbic acid.

3.12.Statistical analysis.

Data is analyzed using SPSS(version 26, SPSS Inc. Chicago, Illinois, USA). Descriptive statistics (mean, standard Error), Statistical analysis was carried out by using Chi-square, as well as t-test student test for comparing between two groups, and by two-way ANOVA at $p \leq 0.05$ to compare by Duncan's Multiple Range test. The relationship between studied parameters is determined by Pearson's correlation coefficient (r). The value of $p < 0.05$ is considered to be statistically significant. (John,2009).

Chapter Four

Results

4.1 . Distribution of study population according to age and body mass index (BMI).

A total of 70 patients with hypothyroid sm and 30 apparently healthy women are recruited for this study. They are classified into three age groups, and it was found that, consisting of 15(21.43%), 20(28.57%), 35(50%) respectively for hypothyroidism women, and in healthy women include 10 (33.33%) women for each age group as appeared in table (4.1).

Table (4 .1) : Distribution of study samples according to age group.

Groups / Age (year)	patients women N=70	Healthy women N=30
	No. (%)	
17-20	15 (21.43)	10 (33.33)
21-40	20 (28.57)	10 (33.33)
>40	35 (50)	10 (33.33)
Total	70(100)	30 (100)

Tables (4.2) included 100 samples with different weight blocks. They were classified into three different weight groups,the first group represented by BMI(18.5-24.9) and the second(25-29.9). while third comprised BMI of more than 30 consisting of 9 (12.86), 40 (57.14), 21 (30) respectively for hypothyroidism women, white in healthy women it was 13 (43.33), 11 (36.67), 6 (20) respectively.

Table (4.2) Sample distribution according to body mass index (BMI).

Group / BMI (Kg/m^2)	patients n=70	Healthy women N=30
	No. (%)	
18.5-24.9	9 (12.86)	13 (43.33)
25-29.9	40 (57.14)	11 (36.67)
≥30	21 (30)	6 (20)
Total	70 (100)	30 (100)

4.2 . The levels of Tri-iodothyronine (T3) , Thyroxine (T4), and Thyroid Stimulating Hormone(TSH).

there was a significant decrease P<0.002 in levels of thyroxine hormone (T4) in patients compared with apparently healthy control group which reached to 55.22±11.5 and 81.81±4.2 nmol/L respectively ,also there was a significant decrease P<0.004 in the level of T3 in patients 1.41±0.1 there was a significant decrease P<0.002 in levels of thyroxine hormone (T4) in patients compared with apparently healthy control group which reached to 55.22±11.5 and 81.81±4.2 n mol/L respectively ,also there was a significant decrease P<0.004 in the level of T3 in patients 1.41±0.1 n mol/L compared with apparently healthy control group2.08±0.06 n mol/L while there was a significant increaseP<0.0001 in levels of Thyroid Stimulating hormone in patients 22.73±2.7 n mol/L compared with apparently healthy control group 1.77±0.4 nm/L as shown in table (4.3).

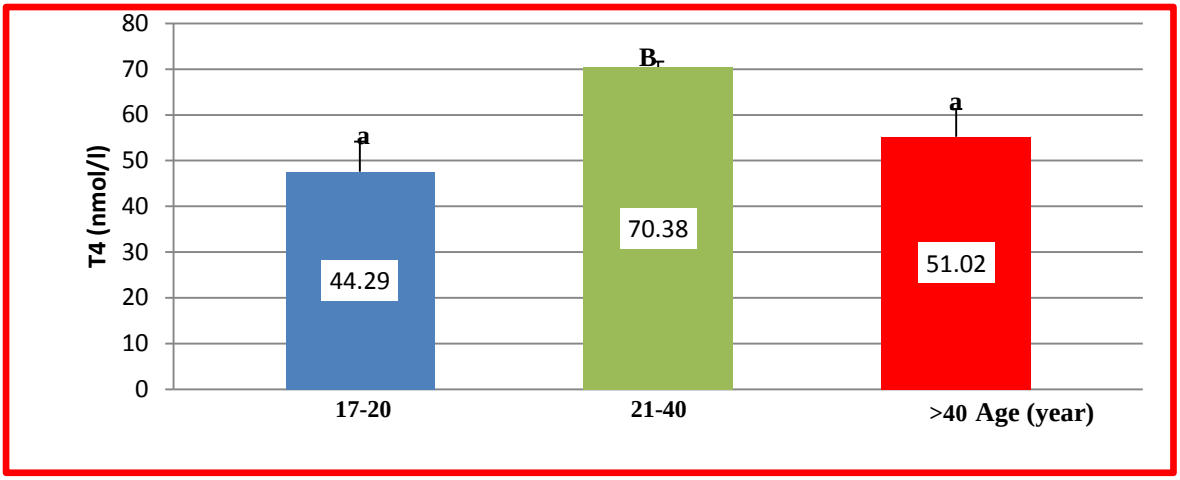
Table (4.3) Levels of thyroxin (T4) , Tri-Iodothyronine (T 3), and thyroid stimulating (TSH) hormones in patients women and healthy women groups.

Groups / Parameters	patients women	Healthy women	p-value
	Mean±S.E		
T4 nmol/L	55.22±11.5	81.81±4.2	**0.002****
T3 nmol/L	1.41±0.1	2.08±0.06	**0.040***
TSH nmol/L	22.73±2.7	1.77±0.4	**≤0.0001****

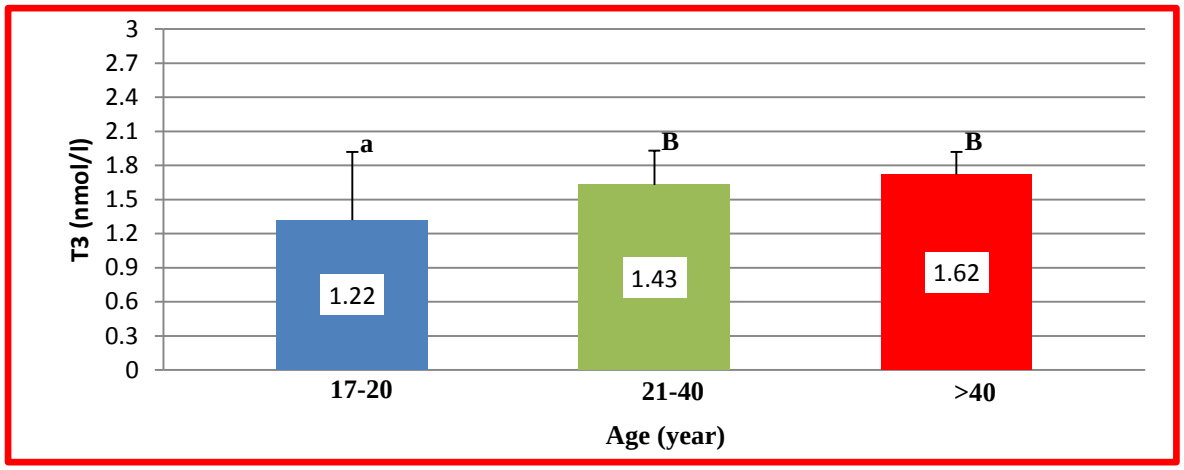
Table (4.4). There were a significant decrease between in patients compared with apparently healthy control group according to the different age groups for T4 hormone 44.29±6.6 nmol/L, 51.02±6.1 nmol/Lat first 17-20 and third <40 age groups in contrast to apparently healthy control group 82.30±4.4 nmol/L, 79.49 nmol/L in order. While there were a significant decrease for T3 hormone1.22 nmol/L, 1.43 nmol/L for first 17-20 and second 21-40 age groups in relation to apparently healthy control group 1.22±0.6 nmol/L , 2.01±0.1 nmol/L respectively. Also there was a significant increase for TSH hormone 24.27±3.6 nmol/L , 19.70±2.8 nmol/L , 24.89±3.8 nmol/L for all age groups in hypothyroidism women in compare with healthy women 2.08±1.1 nmol/L, 1.43 nmol/L, 1.77±2.7 nmol/L accordingly. The figures (4.1), (4.2) and (4.3) also show the levels of these hormones in patients group only.

table (4.4) comparing the levels of tri- iodothyronine (T 3), thyroxine (T4), and thyroid -stimulating hormone (Tsh) in patients and in healthy women groups according to age groups.

age group / Parameters	Age(year) Groups	17-20	21-40	<40
		Mean±S.E		
T4 nmol/L	patients women	44.29±6.6	70.48±1.2	51.02±6.1
	Healthy women	82.30±4.4	83.39±7.1	79.49±5.5
p-value		≤0.0001**	0.962	0.001**
T3 nmol/L	patients women	1.22±0.6	1.43±0.3	1.62±0.2
	Healthy women	2.11±0.9	2.01±0.1	2.15±0.1
p-value		≤0.0001**	0.023*	0. 502
TSH nmol/L	Patients women	24.27±3.6	19.70±2.8	24.89±3.8
	Healthy women	2.08±1.1	1.43±0.2	1.77±2.7
p-value		≤0.0001**	≤0.0001**	≤0.0001**



figure(4.1) levels of thyroxin (T4) hormones in patients according to



figure(4.2) levels of tri-iodothyronine (T3) hormones in patients according to age groups.

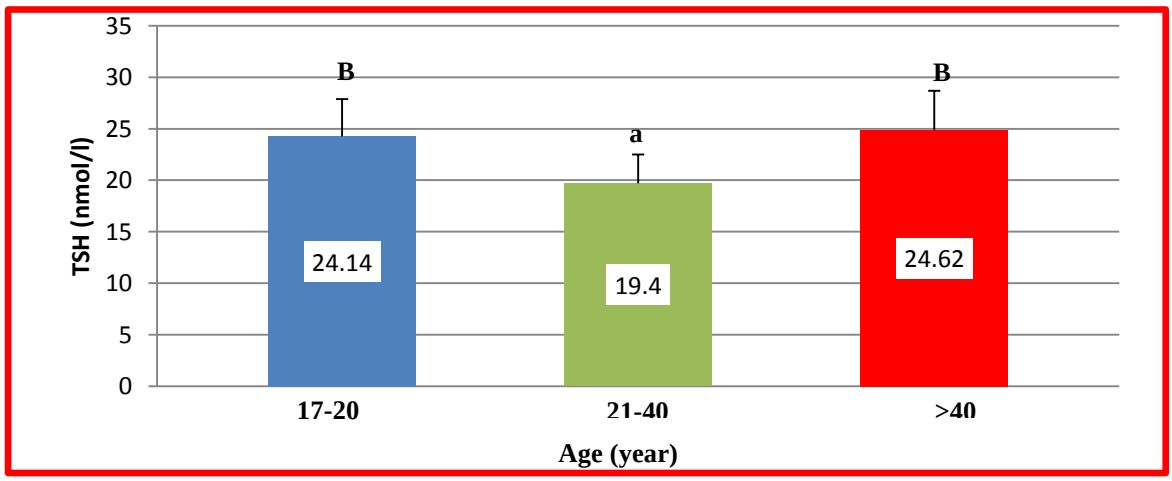


Figure (4.3). Levels of thyroid stimulating (TSH) hormones in patients according to age Groups.

Table (4.5)This table sheds light on the comparison between healthy women and women with hypothyroidism according to BMI. It showed no significant difference between the group of women with hypothyroidism and the healthy group for T4 hormone for the total of the three weights. While there was a significant decrease of T3 hormone by 1.63±0.2 nmol/L for the first weight group, 1.41±0.1nmol/L for the second weight group and 1.18±0.2 for the third group compared to healthy women 2.00±0.2 nmol/L 2.23±0.1 nmol/L and 1.18±0.2 nmol/L respectively. There was also a significant increase in TSH hormone 22.05±2.2 nmol/L , 23.09±1.8 nmol/L , 23.06±2.2 nmol/L for total weight in women with hypothyroidism compared to healthy women 1.68±0.2 nmol/L , 1.79±0.2 nmol/L , 1.85±0.2 nmol/L accordingly the figure (4.4),(4.5),and (4.6) showed that the levels of these hormones in patients groups according to the BMI .

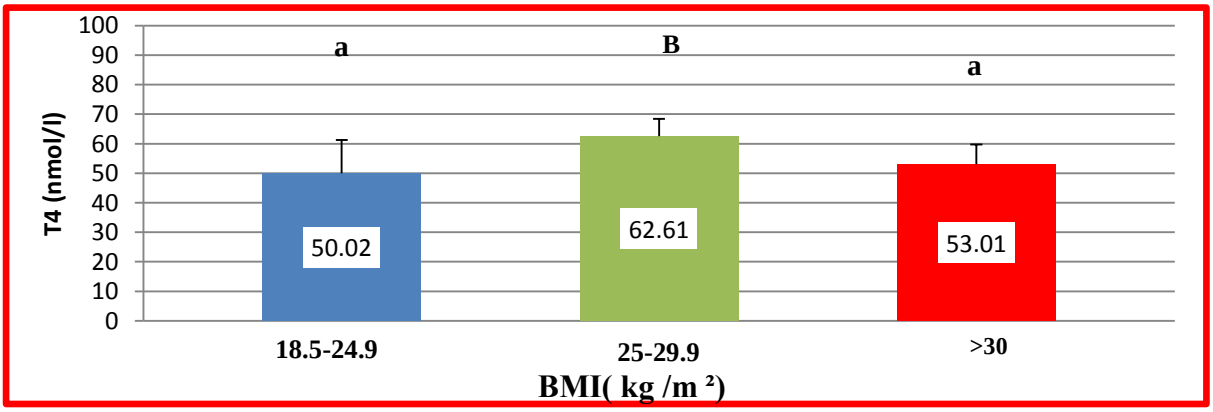
Table (4.5) Distribution the levels of thyroxin (T4)Triiodothyronine(T3)and thyroid stimulating hormones (TSH) in patients women and healthy women group according to the body mass index(BMI).

BMI Groups / para meter	BMI Groups	Normal weight 18.5-24.9	Over weight 25-29.9	Obes ≥30
		Mean±S.E		
T4 (nmol/l)	Patients women	50.02±11.2	62.61±5.8	53.01±6.7
	Healthy women	75.28±2.6	86.70±3.9	83.47±1.4
p-value		0.628	0.712	0.944
T3(nmol/l)	Patients women	1.63±0.2	1.41±0.1	1.18±0.2
	Healthy women	2.00±0.2	2.23±0.1	2.01±0.3

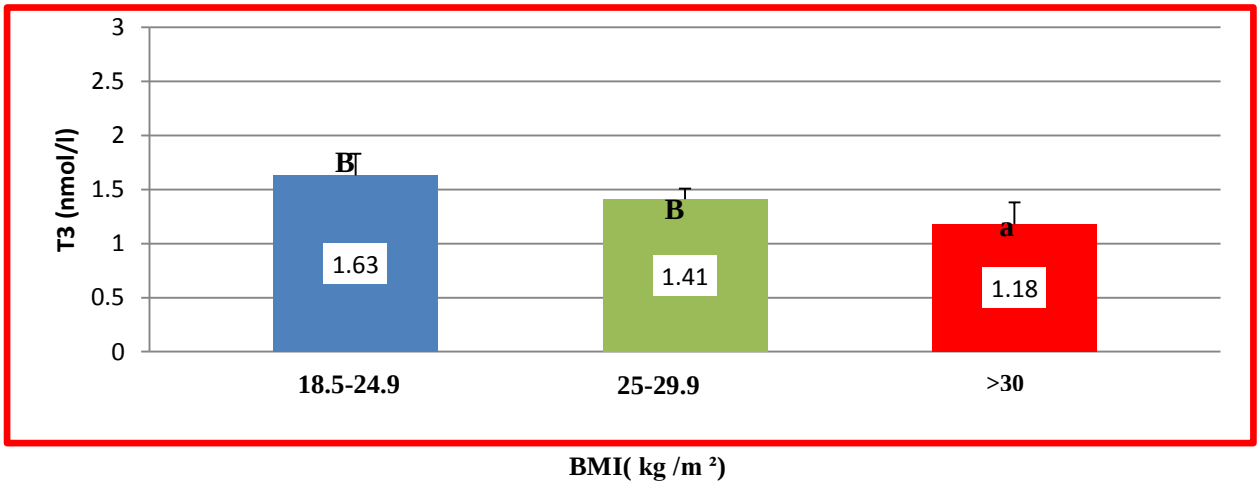
p-value		0.021*	0.006**	≤0.0001**
TSH(nmol/l)	Patients women	22.05±2.2	23.09±1.8	23.06±2.2
	Health Women	1.68±0.2	1.79±0.2	1.85±0.2
p-value		≤0.0001**	≤0.0001**	≤0.0001**

not *: 0.05

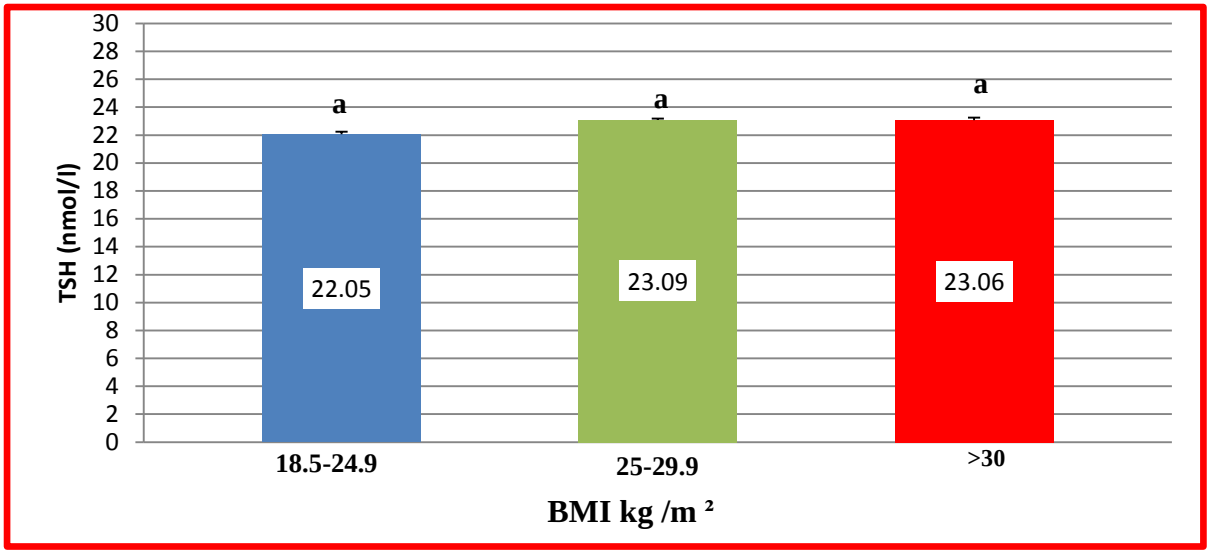
**: 0.01



Figure(4.4) Levels of Thyroxin (T4) hormone in patients women according to the body mass index (BMI).



Figure(4.5) Levels of Tri-iodothyronine (T3) hormone in patients women a according to the body mass index (BMI).



TSH

Figure (4.6). Levels of thyroid-stimulating hormone (TSH) patients women according to the body mass index (BMI).

4.3. Level of selenium, malondialdehyde (MDA) and total antioxidant status (TAS) in patients women and healthy women group .

there was asignficant decrease P<0.011 in selenium concentrations 12.14±0.9 ng / ml in patients compared with apparently healthy control group which reached to 19.75±1.7 ng / ml ,,also there was asignficant increase in Malondialdehyde (MDA) as it was 35.16±2.2 mmol/L in patients women and 26.92±2.1 mmol/L for apparently healthy control group , respectively P < 0.0001. As for the total antioxidant status (TAS), it appeared a significant decrease P<0.026 in the group of patients 175.07±3.8 mg AAE / ml compared to 196.25±5.6 mg AAE / ml in apparently healthy control group as shown in table (4.6).

Table (4.6). Level of selenium, malondialdehyde (MDA) and total antioxidant Status (TAS) in patients and Healthy Women.

Groups / Parameters	Patients women	Healthy women	p-value
	Mean±S.E		
Selenium (ng/ml)	12.14±0.9	19.75±1.7	**0.011***
MDA(m mol/l)	35.16±2.2	26.92±2.1	**≤0.0001****
TAS(mgAAE/ml)	175.07±3.8	196.25±5.6	**0.026***

table(4.7). showed that the levels of selenium ,malondialdehyde and total antioxidant in patients and apparently healthy control group according to the age group . between a group of patients and apparently healthy control group by different age groups. Selenium results showed a significant decrease ($p < 0.0001$, $p < 0.014$) for the first and third age groups (10.10±1.7 g/ml, 12.06 ± 0.4 ng/ml) compared to the apparently healthy control group (12.06 ± 0.4 ng/ml, 19.21 ± 0.2 ng/mL) for the respectively group, while (MDA) significantly increased ($p<0.022$ 0.005, $p<0.0001$) in the three age groups of female patients by age group (35.09±5.1 36.34±4.2 34.06±2.2 m mol/L)compared to the control group Health (27.87±3.2 28.46±1.8 24.37±1.5 m mol/L). As for antibodies, there were no statistically significant differences for the other age groups.

Table (4.7) Levels of Selenium ,Malondialdehyde (MDA) and Total antioxidant Status (TAS), in patients Women and Healthy Women groups according to age groups.

age groups / Parameters	Age(year) Groups	17-20	21-40	>40
		Mean±S.E		
Selenium(ng/ml)	Patients women	10.10±1.7	14.25±2.7	12.06±0.4
	Healthy women	20.85±2.4	19.20±1.9	19.21±0.2
p-value		**≤0.0001****	**0.056**	**0.014***
MDA(m mol/l)	Patients women	35.09±5.1	36.34±4.2	34.06±2.2
	Healthy women	27.87±3.2	28.46±1.8	24.37±1.5
p-value		**0.022***	**0.005****	**≤0.0001****
TAS(mgAAE/ml)	Patients women	178.57±11.2	171.45±5.5	175.18±9.4
	Healthy women	198.14±9.8	195.87±3.9	194.71±4.7
p-value		**0.122**	**0.149**	**0.744**

Showed that the levels of selenium, malondialdehyde (MDA), and total antioxidant status (TAS) in patients according to the age group in the following figures (4.7),(4.8),(4.9).

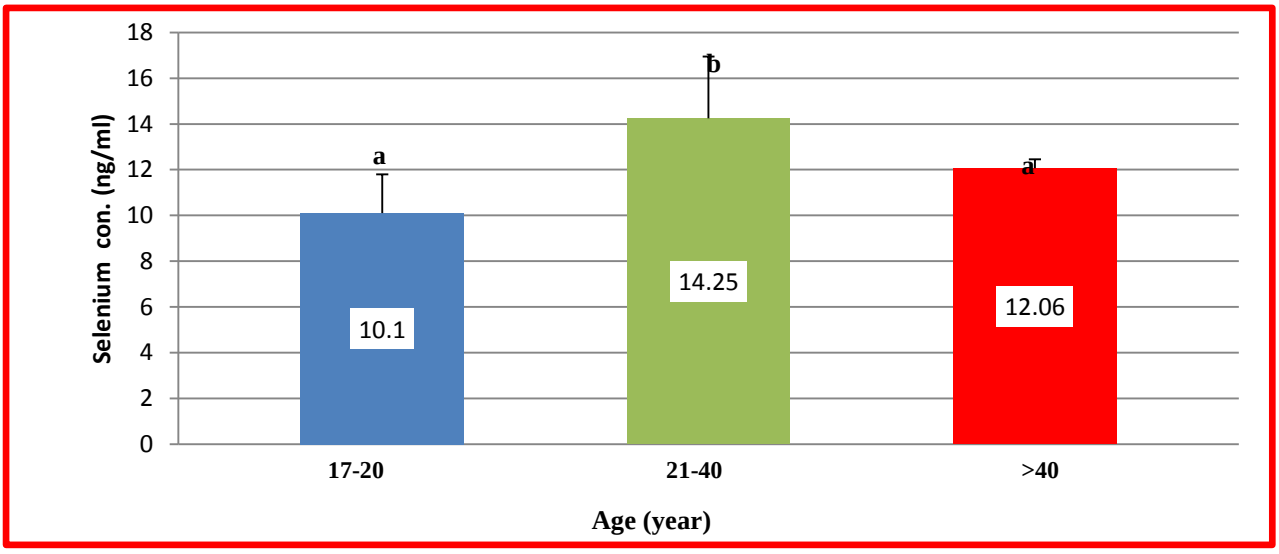


Figure (4.7) Levels of selenium in patients women according to age groups.

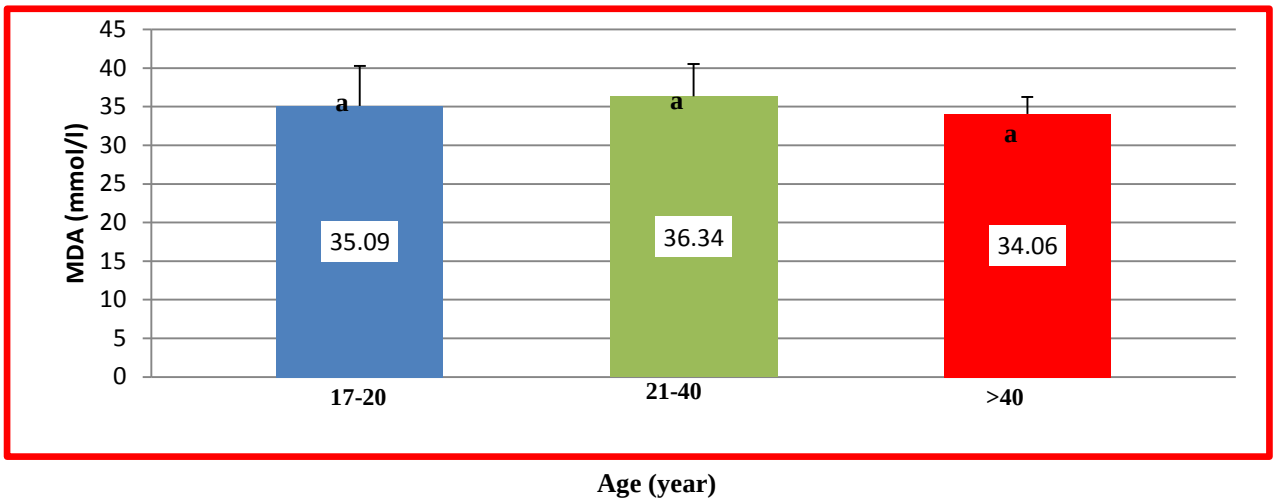


Figure (4.8)Level of malondialdehyde (MDA) in patients women according to age groups.

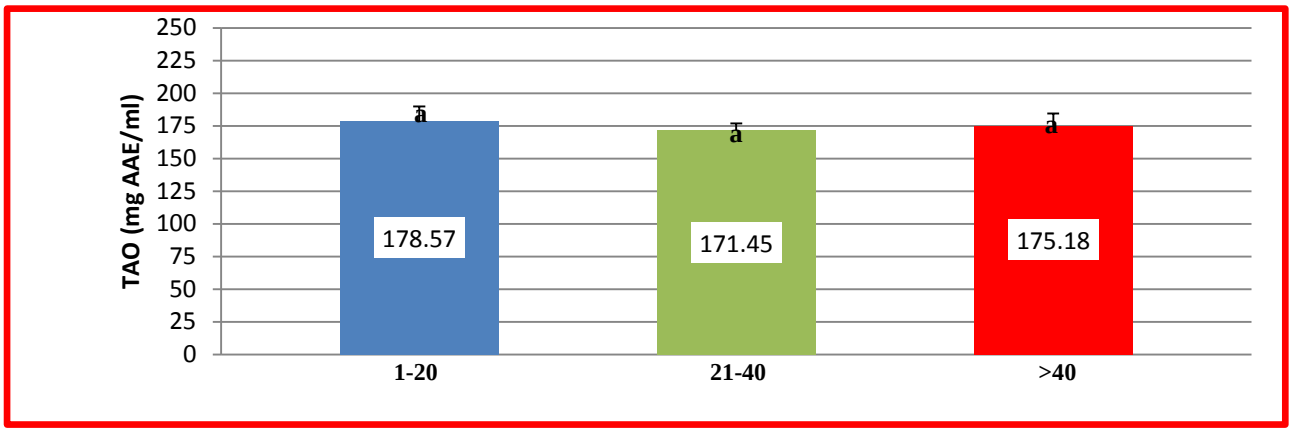


Figure (4.9) Level of total antioxidant status (TAS) in patients women according to the age groups.

table (4.8). revealed that the levles of Selenium,Malondialdehyde andTotal antioxidant in patients and apparently healthy control group and distribution according to the body mass index (BMI)group .there was significant decrease p <0.031 in levles of Selenium 12.11±1.2 ng / ml, in the first group and significant decrease p< 0.002 in the second group 10.57±0.6 ng / ml among women with in patients compared to healthy women 21.46±3.6 ng / ml22.87±1.7 ng / ml.,also there was asignfificant increase in malondialdehyde (MDA) P<0.011 ,P<0.009 in patients 33.56±5.4 mmol / L, 41.73±4.2 mmol / L for the first and second groups in patients compared to healthy women 22.87±3.2 mmol / L24.80±5.5 mmol / L, there was asignfificant decrease P<0.009, P<0.042 in levels of total antioxidant status in the the second and third groups in the patients group 172.53±4.6 mg / ml, 179.90±5.9 mg / ml compared to the second and third groups 204.86±6.1 mg / ml, 209.61±4.7 1mg / ml in the healthy women as shon in table (4.8).

Table (4.8) the levels of selenium levels, malondialdehyde (MDA) and total antioxidant status (TAS) in patients and healthy women according to the body mass index (BMI). Parameters

mass index BMI / Parameters	BMI(kg/m2) Groups	Normal weight 18.5-24.9	Over weight 25-29.9	Obes ≥30
		Mean±S.E		
Selenium (ng/ml)	Patients women	12.11±1.2	10.57±0.6	13.75±2.2
	Healthy women	21.46±3.6	22.87±1.7	14.81±1.5
p-value		**0.031***	**0.002****	**0.877**
MDA(mmol/l)	Patients women	33.56±5.4	41.73±4.2	30.18±0.7

	Healthy women	22.87±3.2	24.80±5.5	33.12±3.2
p-value		**0.011***	**0.009****	**0.412**
TAS (mgAAE/ml)	Patients women	172.85±4.7	172.53±4.6	179.90±5.9
	Healthy women	174.24±3.9	204.86±6.1	209.61±4.7
p-value		**0.511**	**0.009****	**0.042***

Showed that the levels of selenium, malondialdehyde (MDA) and total antioxidant status (TAS) in patients according to body mass index (BMI) in the following figures (4.10),(4.11),(4.12).

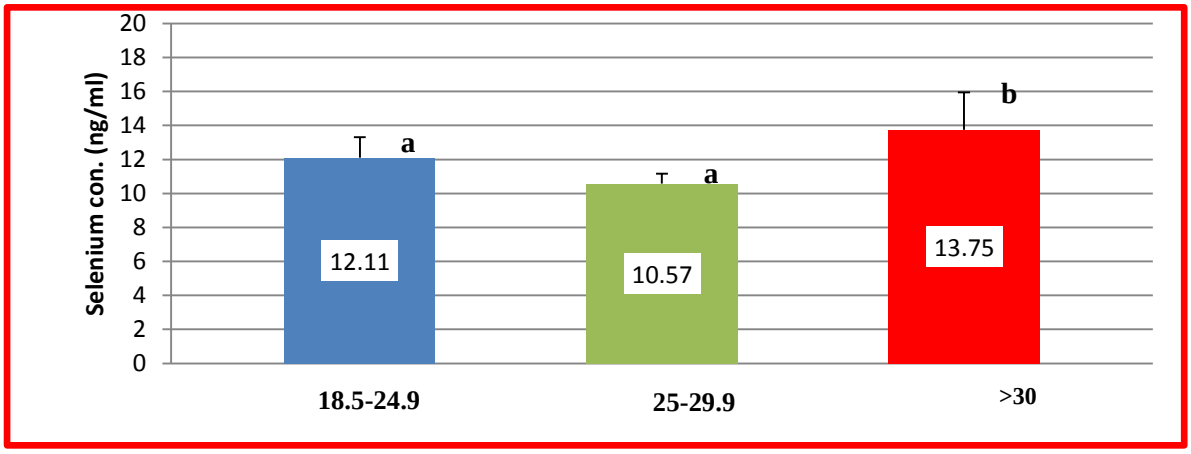


Figure (4.10) Level of selenium in patients women according to the body mass index(BMI).

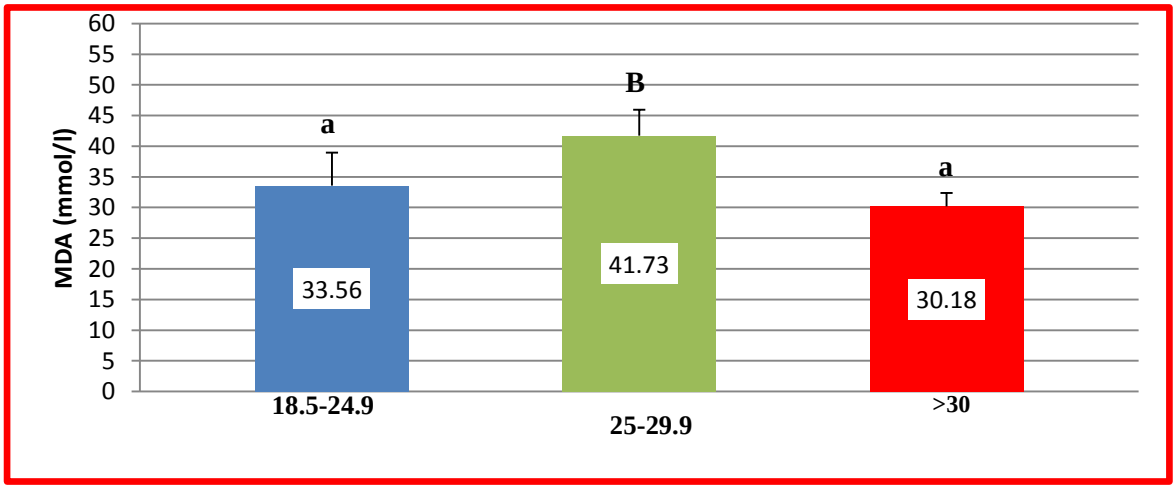


Figure (4.11) Level of malondialdehyde (MDA) in patients women according to body mass index(BMI).

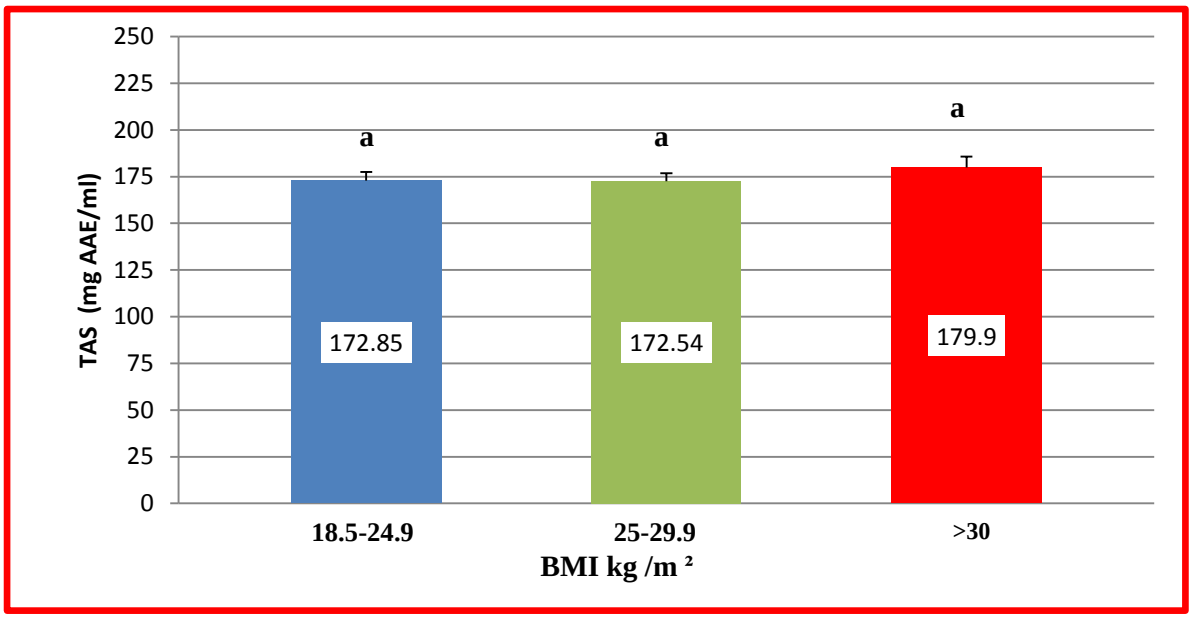


Figure (4.12) Levels of total antioxidant status (TAS) in patients women according to body mass index (BMI).

4.4. The levels of ferritin , anti-TPO and anti-TG at patients and healthy women group age and their distribution according to the body mass index (BMI) group.

the resalts revealed that a significant decrease P<0.001 in level of ferritin in patients compared with the healthy group which reached to 12.44±0.7 ng/ml and 17.97±0.8 ng/ml respectively ,whily there was significant increase P<0.028 in level of Anti-Tpo in patient 16.44±0.5 ng/ml compared with the healthy women 10.70±0.2 ng/ml, also the results stomed a significant increase P<0.002 in the level of Anti-TG in patient compared with the healthy as shown in table (4.9).

Table (4.9) the level of ferritin, Anti-TPO, and Anti-TG concentration in patients women and healthy women group.

Groups / Parameters	Patients women	Healthy women	p-value
	Mean±S.E		
Ferritin ng/ml	12.44±0.7	17.97±0.8	**≤0.0001****
Anti-TPO ng/ml	16.44±0.5	10.70±0.2	**0.028***
Anti-TG ng/ml	17.39±1.4	11.07±0.3	**0.002****

table (4.10) revealed that the levels of ferritin ,Anti-TPO and Anti-TG in patients and apparently healthy control group and distribution according to the age groups ,the results of ferritin a significant decrease P<0.044 and P<0.011 for second and third age groups11.48±2.2 ng/ml. 13.25±1.9 ng/ml in contrast to healthy group 19.24±1.6 ng/ml, 21.42±0.8 ng/ml group in order. While anti-TPO increased significantly P<0.001 in first age group 20.11±3.4 ng/ml in patients compared to healthy women 10.01±1.6 ,also there was a significant increase for anti- TG P≤0.0001, P<0.035 and P<0.022 for all age group 25.02±3. ng/ml, 13.03±1.9 ng/ml, 14.12±2.2 ng/ml in patients compared to healthy women11.02±2.1 ng/ml, 11.11±2.1 ng/ml, 11.10±1.3 ng/ml for all age group in order.

Table (4.10) the levels of the Ferritin, anti-TPO, and anti-TG levels in patients women with healthy women groups according to the age groups.

age groups / Parameter	Age(year) Groups	>20	21-40	<40
		Mean±S.E		
Ferritin (ng/ml)	Patients women	12.58±1.4	11.48±2.2	13.25±1.9
	Healthy women	13.26±2.7	19.24±1.6	21.42±0.8
p-value		0.644	0.044*	0.011**
Anti-TPO (ng/ml)	Patients women	20.11±3.4	14.20±1.3	15.01±2.3
	Healthy women	10.01±1.6	11.50±1.4	10.60±1.7
p-value		0.001**	0.744	0.365
Anti-TG (ng/ml)	Patients women	25.02±3.3	13.03±1.9	14.12±2.2
	Healthy women	11.02±2.1	11.11±2.1	11.10±1.3
p-value		≤0.0001**	0.035*	0.022*

Showed that the levels of ferritin ,Anti-Tpo , and Anti-TG in patients according to the Age Groups in the following figures (4.13), (4.14),(4.15).

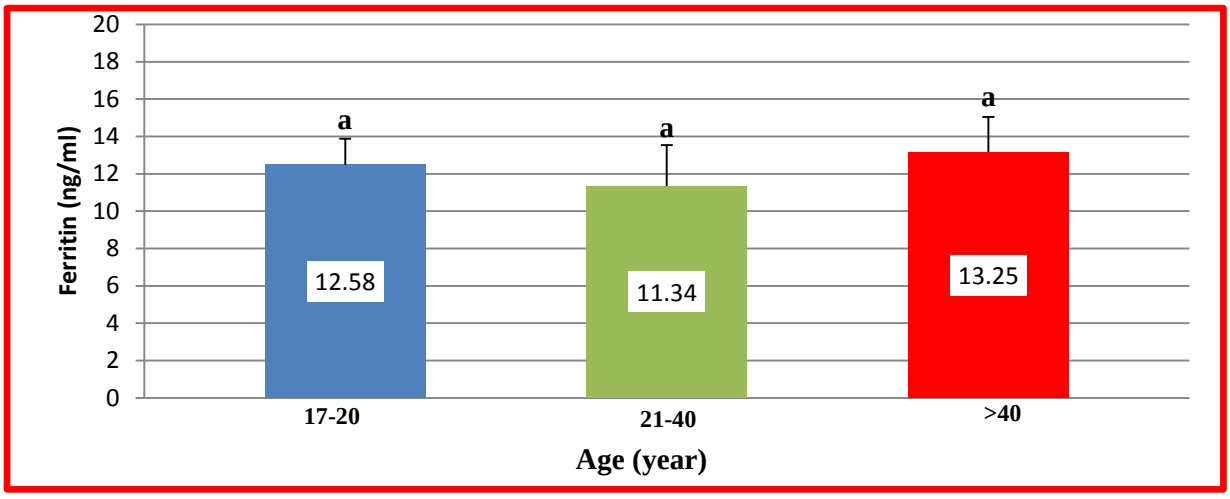


Figure (4.13) Level of Ferritin in patients Women according to the age group.

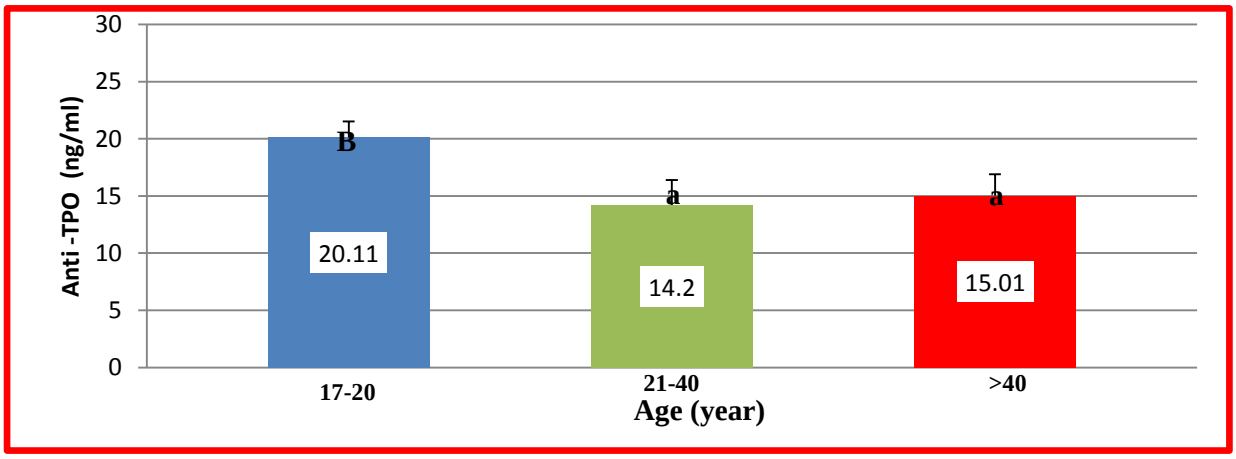


Figure (4.14) Level of Anti-TPO in patients Women according to the age group.

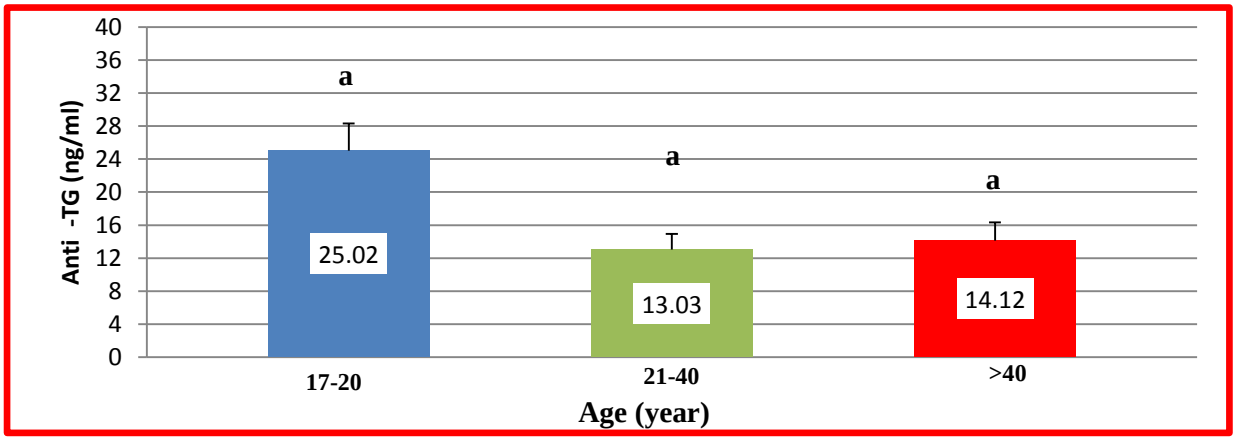


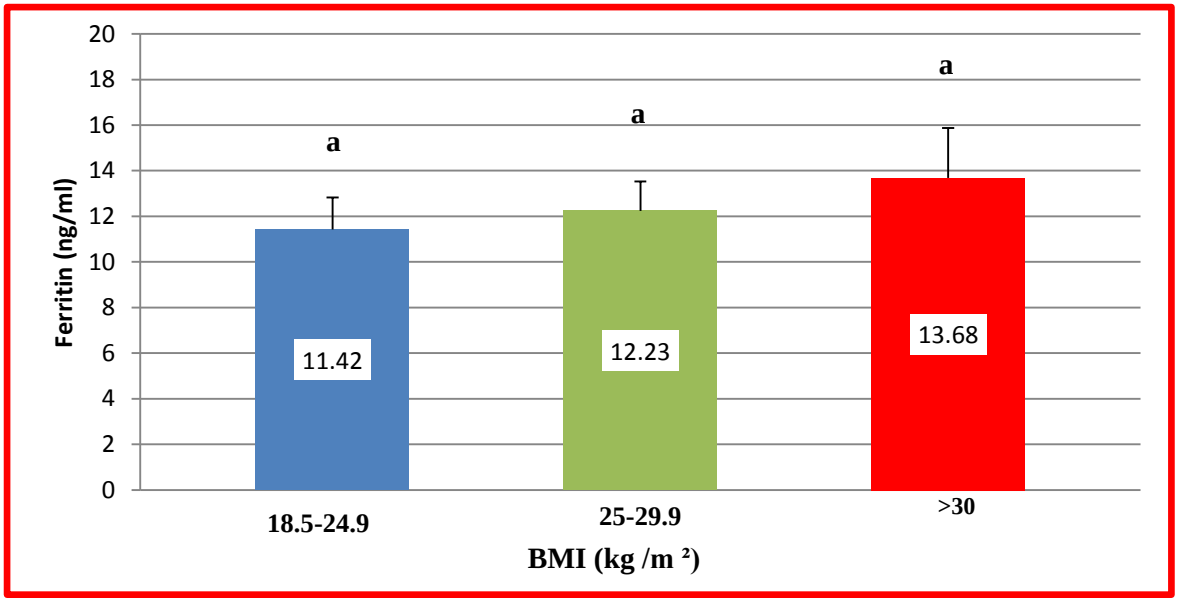
Figure (4.15) Level of Anti-TG in patients women accorging to the age group.

table (4.11). revealed that the levles of ferritin, anti-TPO, and anti-TG in patients and apparently healthy control group and distribution according to the body mass index (BMI) group . There was a significant decrease in ferritin levels (11.42±1.4, 12.23±1.3, 13.68±2.2 ng/mL) in serum levels in the three weight groups among patients women compared to healthy women (16.79±1.9, 17.56±2.2, 19.56±1.9 ng/mL) ng/ml. As for anti-TPO, there were no statistically significant differences in the three groups between patients women and healthy women, there was a significant increase in BMI in the patients gruop three weight groups for anti-TG (20.11±1.0, 15.05±1.8, 17.02±4.1 ng/mL), compared with a decrease in healthy women (11.08±0.6,11.07±1.1, 11.05±1.2ng/mL) was observed. / ml) in the three weight groups for anti-TG

Table (4.11) The levels of ferritin , Anti-TPO, and Anti-TG levels in patients women group and healthy women according to the body mass index(BMI).

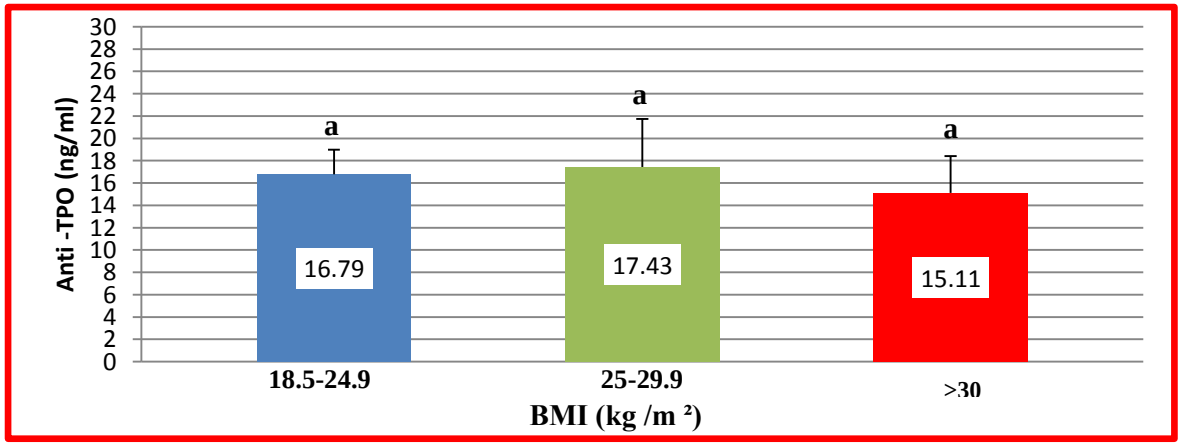
body mass index(BMI). / Parameters	BMI Groups	Normal weight 18.5-24.9	Over weight 25-29.9	Obes ≥30
		Mean±S.E		
Ferritin (ng/ml)	**Patients Women**	11.42±1.4	12.23±1.3	13.68±2.2
	Healthy Women	16.79±1.9	17.56±2.2	19.56±1.9
p-value		**0.044***	**0.037***	**0.012***
Anti-TPO (ng/ml)	**Patients Women**	16.79±1.9	17.43±4.3	15.11±3.3
	HealthyWomen	10.65±1.7	10.86±2.6	10.59±1.2
p-value		**0.069**	**0.074**	**0.822**
Anti-TG (ng/ml)	**Patie Women**	20.11±1.0	15.05±1.8	17.02±4.1
	Healthy Women	11.08±0.6	11.07±1.1	11.05±1.2
p-value		**≤0.0001****	**0.022***	**0.035***

Showed that the levels of ferritin,Anti-Tpo, and Anti-TG in patients according to the body mass index (BMI) in the following (4.16), (4.17),(4.18).



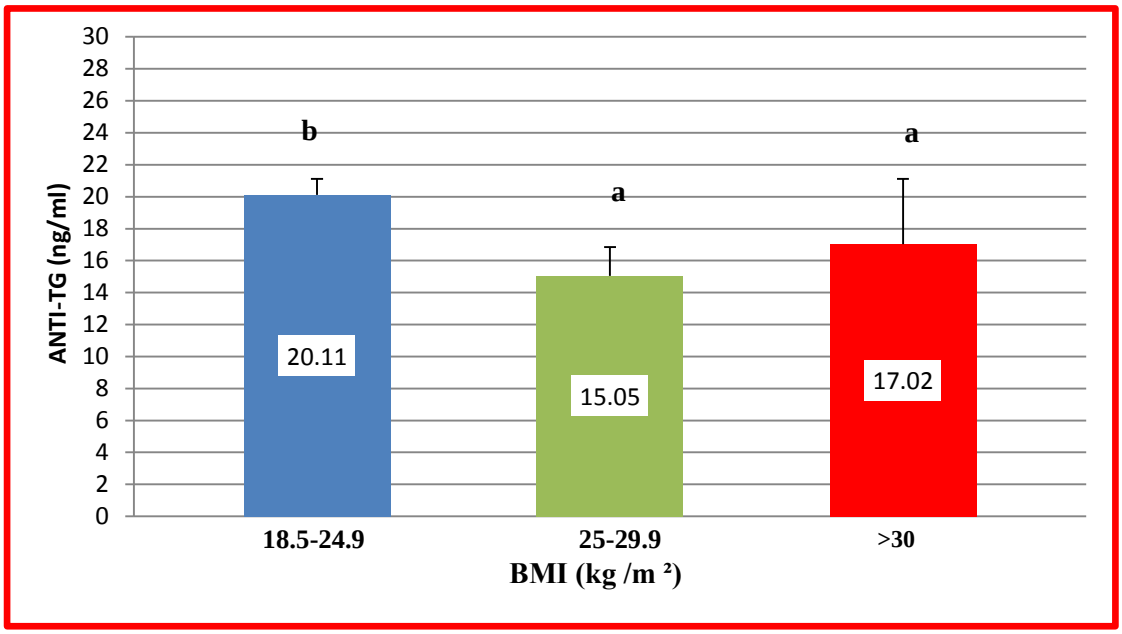
Ferritin

Figure (4.16) levels of ferritin in patients women according to the body mass index (BMI).



Anti-TPO

Figure (4.17) levels of Anti-TPO in patients women according to body mass index (BMI).



Anti-TG

Figure (4.18) Levels of Anti-TG in patients women according to the body mass index (BMI).

4.5. Correlation Coefficient among all Studied Parameters

Table (4.13) indicated that there was a significant negative relationship for both selenium level and Anti-TPO, also between Ferritin and Anti-TPO, as appeared in the following figures (4.19) and (4.20). There were a positive relationship between Triiodothyronine (T3) and Thyroxine (T4), as well as between Total Antioxidant Status (TAS) and Thyroid Stimulating Hormone (TSH) in hypothyroidism, as shown also in figures(4.21) and (4.22).

Table (4.12) Pearson's Correlation Coefficient among all Studied Parameters in patients women patients .

		Malondialdehyde (MDA) mmol/l	Total Antioxidant mgAAE/ml	T4 nmol/l	T3 nmol/l	Ferritin ng/ml	AntiTPO ng/ml	AntiTG U/ml	TSH nmol/l
Selenium (ng/ml)	R	0.090-	0.090-	0.061	.038-	0.088-	0.326-*	0.142	0.069
	Sig.	0.577	0.574	0.703	0.815	0.584	0.038	0.376	0.67
MDA (mmol/l)	R	1	0.067	0.115	0.249	.024-	0.24	0.06	0.245
	Sig.		0.677	0.473	0.116	0.881	0.131	0.71	0.123
Total Antioxidant (mgAAE/ml)	R		1	0.174-	0.074	0.051	0.023-	0.297	0.469*s*
	Sig.			0.276	0.645	0.753	0.886	0.06	0.002
T4 (nmol/l)	R			1	.504**	0.185-	0.017	0.075-	0.245-
	Sig.				0.001	0.248	0.914	0.641	0.123
T3 (nmol/l)	R				1	0.171-	0.1	0.075	0.087-
	Sig.					0.286	0.533	0.639	0.587
Ferritin (ng/ml)	R					1	0.345*	0.084-	0.032
	Sig.						0.027	0.6	0.841
Anti-TPO (ng/ml)	R						1	0.041-	0.294-
	Sig.							0.799	0.062
Anti-TG (U/ml)	R							1	0.157
	Sig.								0.327

***. Correlation is significant at the 0.05 level (2-tailed).**

****. Correlation is significant at the 0.01 level (2-tailed).**

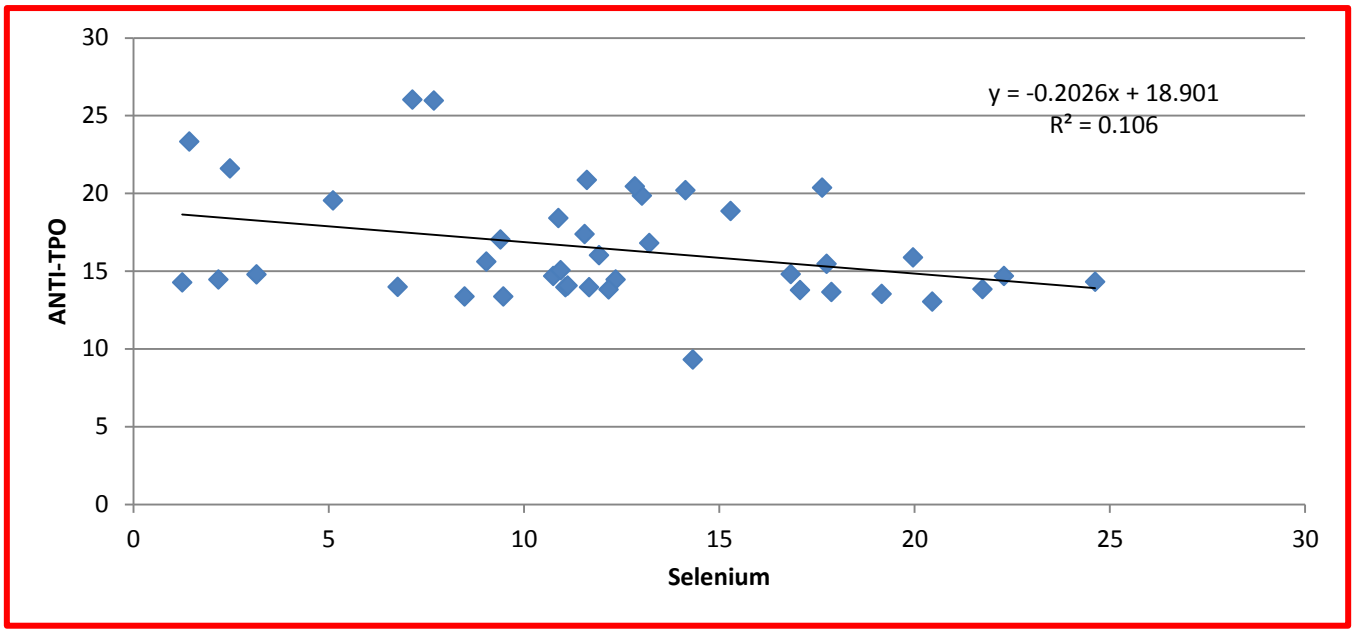


Figure (4.19) Negative Significant correlation between selenium and Anti –TPO in patients women.

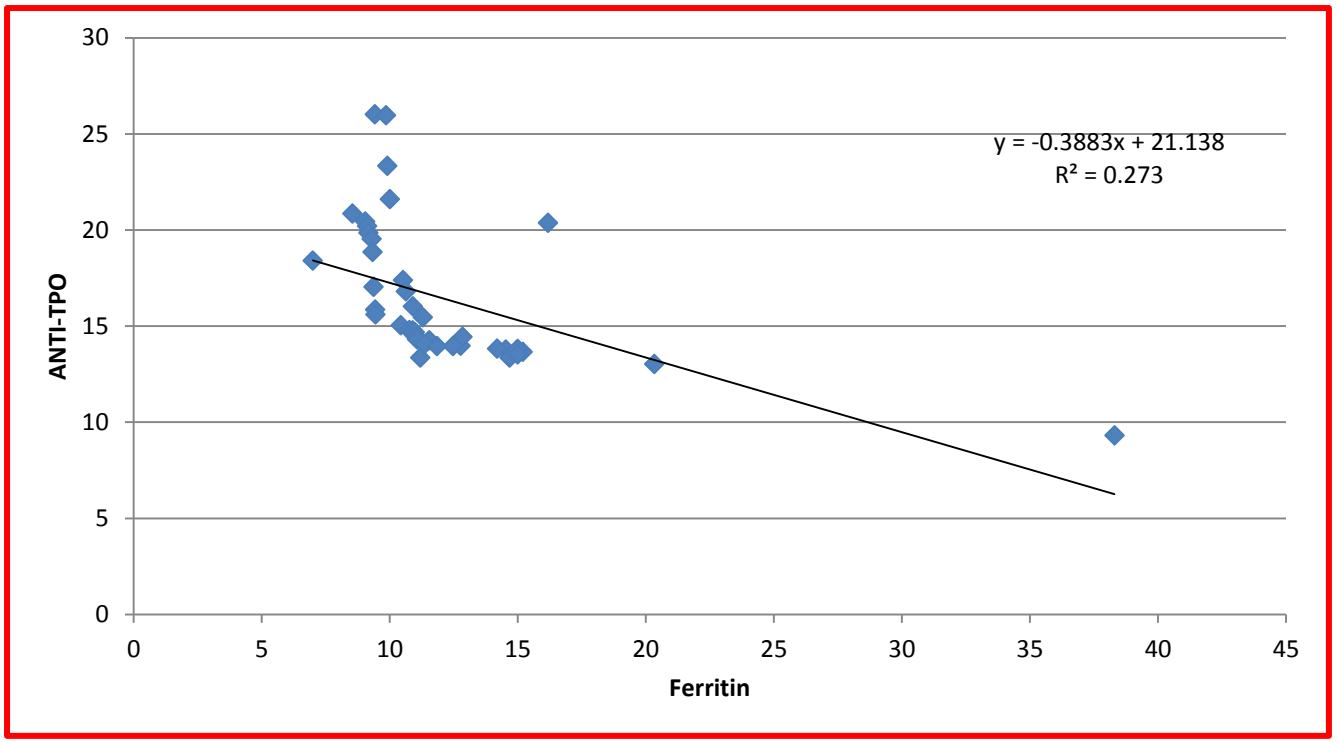


Figure (4.20) negative Significant correlation between ferritin and Anti-TPO in patients women.

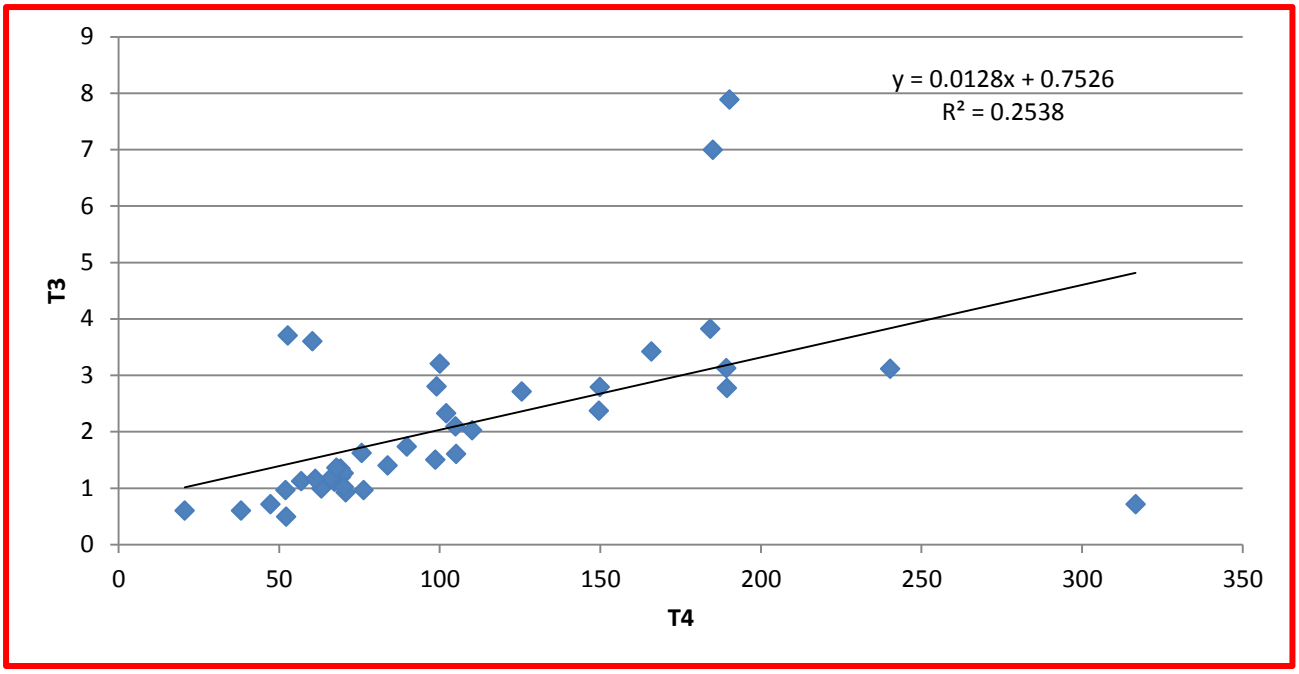


Figure (4.21) positive Significant correlation between T4 and T3 in patients women .

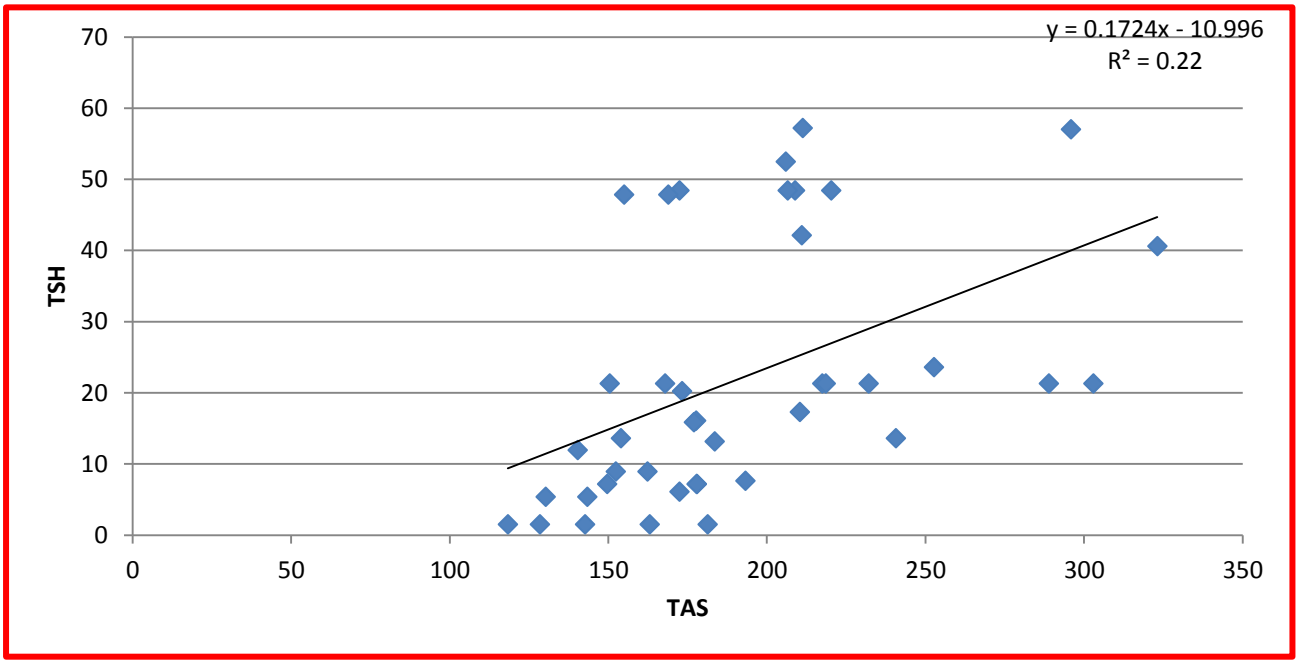


Figure (4.22) positive significant correlation between total antioxidant and thyroid stimulating hormone (TSH) in patients women .

Chapter Five

Discussion

5. Discussion

5.1 Relation Between Age, Body Mass Index, thyroid hormones in Hypothyroidism women.

The results of the current study shown in tables (4.1), (4.2), (4.3) and (4.3) showed that TSH levels in patients are much higher than in healthy women, while total T3 and T4 levels are at a lower level in healthy women. Comparing patients with healthy women. The most common cases of endocrinopathy are adult women of childbearing age, as they showed significant differences of ≤0.0001 in high TSH and low thyroid hormones. This is because thyroid hormones can affect many hormonal systems in the body, and menopause occurs due to. Changes in hormone levels such as estrogen and progesterone. Estrogen is the hormone that enhances thyroid function. If estrogen levels are low, thyroid functioning also decreases and female gender appears to be an independent risk factor for thyroid dysfunction (Strikic Dula *et al*., 2022). Women are 3-5 times more likely to be treated for thyroid disorders than men. Age-affected thyroid may change the FT4 or FT3 response point, leading to altered thyrotropin (TSH) levels in older people (Stoll, 2019; Abdalla *et al*., 2020). This study also showed that the prevalence of hypothyroidism was high.Screening should be done to prevent complications of hypothyroidism.

This is agree with the various research on the prevalence of hypothyroidism being 1-2% among premenopausal or postme nopausal women, and it is more common in older females (Vanderpump and Tunbridge, 2002; Joshi *et al*., 2011).also The results showed that The prevalence of hypothyroidism increases with age (Canaris *et al*., 2002) National Health and Nutrition Examination Survey showed the prevalence of hypothyroidism (whether overt or subclinical) by 4.6%. 8. A screening study in the United States showed a prevalence of 0.4% and 9% for overt and subclinical hypothyroidism, respectively, with the latter increasing to more than 20% among women 75 years or older. In a meta-analysis from Europe (Garmendia et al., 2000) the prevalence of overt and subclinical hypothyroidism was 0.37% and 3.8%, respectively, including diagnosed and undiagnosed cases, and the estimated incidence of hypothyroidism was 226 cases. Per 100,000. individuals annually. The results also indicated that there is a relationship between hypothyroidism in female patients and obesity, as the study indicated that a simple

disorder in the thyroid gland may contribute to changes in body weight, which could represent a significant risk factor for weight gain and obesity The TSH increases with a significant difference of $P<0.0001$ With both decreasing of T3 and T4 , This is may be due to the fact that thyroid hormones participate in multiple physiological processes regulating basal metabolic rate, enhancing the adrenergic nervous system to generate heat in response to exposure to cold, and stimulating gluconeogenesis and both lipolysis and lipogenesis.

This was similar to what Valdes.,*et al* (2017) came up with Patients with thyroid dysfunction may experience changes in body weight and body composition also slow peristalsis causing chronic constipation that may result in weight gain. This weight gain is mainly due to water retention and is not related to an increase in fat mass.

In addition (Biondi, 2010) is found a higher prevalence of subclinical hypothyroidism among obese patients. All together, these factors have probably contributed to a general belief of a direct association of hypothyroidism with obesity.

2.2. Level of Selenium, Malondialdehyde (MDA) and Total antioxidant Status (TAS) in Hypothyroidism and healthy women.

The results shown in each table (4.6),(4.7) and (4.8) found a significant decrease in the concentration of selenium in women with hypothyroidism and that selenium deficiency will increase the prevalence of several types of thyroid diseases because selenium is considered a micronutrient necessary for the biosynthesis of selenoproteins that contain selenocysteine in adults.Most selenoproteins are expressed known as glutathione peroxidase in the thyroid gland, it is involved in thyroid hormone metabolism, regulating redox status, and maintaining cellular homeostasis (Brouwer *et al*., 2022). It plays an important role in combating oxidative stress, as selenoproteins such as GPXs and TRs can scavenge H_2O_2, protect cell membrane structure and function, repair the site of molecular damage, and achieve stress. Antioxidant. and local

protective effects against oxidative stress or inflammation. In Se deficiency, GPx activity decreases, H_2O_2 degradation decreases, thyroid cells are less resistant to oxidative stress, apoptosis and cell death occur. On the other hand, Se may reduce thyroid antibodies by upregulating activated Treg cells. Se deficiency also upregulates Th1/Th2 effectors and enhances immune responses. The potential therapeutic effect of Se in HT to improve immune function was validated in a 2022 study, which showed that Se supplementation at a dose of 100 μg daily improved thyroid function and patients' quality of life by reducing interferon-gamma concentrations and increasing interleukin- 1b concentrations (Kozioł *et al*.,2022).

Ray *et al* ., (2012) indicated in his research Se and selenoproteins play a significant role in the development of thyroid cancer. It is generally agreed that oxidative stress plays an important role in cancer genesis and tumour progression. Most studies indicate an association between Se deficiency and the development of thyroid cancer, as well as significant changes in the expression and activity of various selenoproteins in different types of thyroid cancer.

The results also showed a clear decrease in selenium concentration in terms of an increase in age and weight for the group women of patients compared to the group of healthy women, which may affect the iodinase enzyme responsible for converting T4 to T3 through the need for selenoprotein.

Evidence in Zhou *et al*., (2020) research also indicated that there is a harmful effect of selenium exposure on glucose and fat metabolism This is supported by two factorsThe first is the association between selenium concentrations and the percentage of fats such as harmful cholesterol and triglycerides.

On the other hand, evidence has emerged from another trial conducted by Christensen *et al*.,(2015), that the association between adverse lipid endpoints and selenium and the response between high blood selenium concentrations and metabolic syndrome, high triglycerides and low-density cholesterol, as well as low good HDL cholesterol.

This is consistent with the researcher's opinion Minich (2022) Several selenoproteins exhibit an antioxidant effect in the thyroid gland. Selenium is an essential element in the metabolism of thyroid hormones.all indicate a relationship between selenium and metabolism and obesity.

The results also indicated the effect of selenium on antioxidants This is due toTPO enzyme responsible for Thyroid peroxidase involved in the biosynthesis of thyroid hormone. TPO has two active sites, which facilitate iodinating tyrosine residues in thyroglobulin (TG) in close conjunction with dual oxidase (DUOX) and H_2O_2 .followed by intrachain coupling of two iodotyrosines residues to form thyroid hormone (Stathatos *et al*., 2012) . Selenium is also an antioxidant , GPX needs it to get rid of free radicals resulting from the binding of active iodine to thyroid hormone by the action of TG to form a hormone, and therefore its deficiency leads to a decrease in thyroid hormones, which causes hypothyroidism(Hariharan *et al*., 2020).

Patients with selenium deficiency can benefit from selenium supplementation, while selenium supplementation in people with adequate selenium levels can exacerbate the risk of certain diseases (Rayman *et al*., 2012).

Another research (Hariharan *et al*., 2020) showed that glutathione peroxidase (GPX) is a major selenoprotein present in the human body,

which helps in controlling the excessive production of free radicals at the site of inflammation.

Regarding thyroid disease Maintaining a physiological concentration of selenium is a prerequisite for preventing thyroid disease and maintaining general health. Selenium intake in particular is associated with autoimmune disorders. This is consistent with researcher Ventura *et al*.,(2017) who showed that maintaining a physiological concentration of selenium is a prerequisite for preventing thyroid disease and maintaining health, also suggests that selenium supplementation for patients with autoimmune thyroiditis is associated with improved quality of life and improved thyroid ultrasound features and is associated with decreased levels of peroxidase antibodies.

also a selenium deficiency is a risk factor for enlarged thyroid gland size, hypothyroidism, and thyroid nodules, and Se supplementation could reasonably be suggested in Se-deficient in thyroiditis patients in Hashimoto (HT)For example, Se (100 μg/day for 6 months) ignificantly reduced the level of thyroid peroxidase antibodies when HT patients with hypothyroidism(Kozioł *et al* ., 2021).Furthermore, administration of 200 μg/day of Se yeast tablets for at least 6 months in HT patients improves thyroid antibodies and thyroid function by increasing antioxidant activity (Hu, Y., *et al* .,2021) .

A study by Wichman *et al*.,(2016) appeared low levels of Se are associated with an increased risk of developing thyroid antibodies, and Se supplementation may be can reduce TPOAb titers, that Selenium supplementation reduced thyroid antibody levels after 3, 6, and 12 months in the LT4-treated autoimmunothyroiditis(AIT) group and after 3 months in the untreated AIT group.

While a research by Ray and others (2012) illustrate that many selenoproteins exhibit antioxidant properties and may be can scavenge

reactive oxygen species (ROS) formed as by-products of molecular oxygen reactions in the process of oxidative phosphorylation in cells. ROS can also be generated by external agents, such as drugs, xenobiotics, metals, radiation, smoking, and infection.

The results of the study also showed that there was increase significantly ($P \leq 0.0001$), this increase in the level of malondialdehyde (MDA) in sick women compared to healthy women. These results are also related to obesity with age, as it increased in the three age groups with a significant difference of $P < 0.022$, $p<0.005$, $p<0.0001$ compared to healthy women, as is the case with obesity in the second and third groups, with a significant difference $p < 0.011$, $p<0.009$. . This is due to the synthesis of thyroxine (T4) and triiodothyronine (T3) catalyzed by thyroid peroxidase (TPO) in thyroid follicles, which is a very complex process. They include ROS, especially H_2O_2. ROS are already necessary in the initial stages of thyroid hormone production, during iodide oxidation. In addition, thyroid hormones perform a metabolic regulatory function by influencing mitochondrial activity(Thanas and Ziros, 2020). Because of its dependence on ROS for its function, the thyroid gland is particularly vulnerable to oxidative damage. Therefore, the thyroid antioxidant defense system must effectively regulate the production of reactive oxygen species(Szanto *et al*.,2019).

In Higher level of MDA in hypothyroid patients. Due to the increase ROS production, this may be the resulting aldehyde has cytotoxic, mutagenic and carcinogenic effects. It is expected that this is due to the increase in free radicals that will affect the cell envelope and break it due to the ability of the free electron present on the wall of the free radicals to enable them to attach and react quickly, causing damage to the cells. wall. The cell is destroyed, and thus an increase in lipid peroxidation occurs,

which rises, as malondialdehyde is its final product(Chakrabarti *et al*.,2016).

This is consistent with a study of Wang *et al*., (2023) who found that reactive oxygen species (ROS) mediate lipid peroxidation and produce 4-hydroxynonenal and other related products, which may be play an important role in the process of cell death, including apoptosis, autophagy, can promote lipid peroxidation. Phospholipids mediate mitochondrial apoptosis, endoplasmic reticulum stress, and other complex molecular signaling pathways to regulate apoptosis, Lipid peroxidation and its products also act in different stages of autophagy.

Chakrabarti et al., (2016).found that MDA concentration as a marker of oxidative stress were higher in patients with hypothyroidism prior to levothyroxine treatment and/or selenium supplementation than in the control group. MDA concentration has also been found to decrease after treatment and/or supplementation in patients with hypothyroidism. In addition, they obtained a significant positive association between MDA level and baseline TSH values.

As shown by Iglesias and Diez (2009), he concluded that oxidative stress occurs when reactive free radicals cause oxidative damage to the molecular structures of the cell. The thyroid gland plays an important role in producing general oxidative stress in disease states, as reactive oxygen levels can increase significantly and may lead to significant damage to cellular structures sometimes during disease states (such as inflammation or infection), or environmental stress (such as light Ultraviolet or heat exposure), or ionizing radiation, as oxidative stress occurs when reactive free radicals cause oxidative damage to the molecular structures of the cell The thyroid gland plays an important role in producing general oxidative stress in pathological conditions (Devasagayam *et al*.,2004).

5.3.Ferritin, Anti-TPO, and Anti-TG levels in hypothyroidism women.

In current study the result showed in table (4.9), (4.10), (4.10) a decrease in levels of ferritin, a protein that stores iron, in almost all tissues of the body. Almost all cells contain serum ferritin, a measure of the body's iron stores.

A decrease in ferritin levels was observed in women patients compared to healthy women, and this was shown in the results of a decrease in the age groups, and this is consistent with increasing age and BMI in the patient group, and this is may be due to that ferritin's may act as a protective antioxidant function, that decrease the amount of antioxidants in the body, especially ferritin, with an increase in concentration. TSH, Our results also showed that female patients suffering from hypothyroidism have significantly lower levels of ferritin in the blood compared to healthy women, which showed a negative relationship between serum ferritin and serum TSH in women patients.

Iron deficiency has been associated with decreased plasma levels of T3 and T4 ,and elevated T3 hepatic deiodination in vitro, demonstrating that iron deficiency affects thyroid hormone metabolism through an inactivation mechanism. Low iron levels correlate with hypothyroidism itself, and associated with autoimmune diseases such as celiac disease, or intestinal malabsorption caused by a deficiency of digestive acids and enzymes (Das *et al* .,2012).This explains the relationship of ferritin with BMI.

ferritin deficiency is also attributed to a deficiency of thyroid peroxidase (TPO) because ferritin is involved in the synthesis of TPO, which is one of the enzymes that carry out the first two stages in the synthesis of thyroid hormones. Iron deficiency may make it difficult for the TPO enzyme to function properly, which may stop thyroid hormone

synthesis.It has been observed that patients suffering from thyroid diseases have varying levels of ferritin in the blood. Iron levels in tissues begin to decline when iron deficiency first appears. As a result, iron levels in ferritin decrease, which changes how the heme-dependent TPO enzyme works and prevents thyroid hormone production (Sahana and Kruthi 2020). TPO acts as an important iron-dependent enzyme in the synthesis of thyroid hormone, on the one hand. Other there was an increase in oxidative stress. This is due to the fact that ferritin is included in the synthesis of GPX, which transforms free radicals from the harmful form into the beneficial form. Hypothyroidism occurs due to immune suppressants that may cause oxidative stress (Muhammad *et al*.,2014).

In this study, women are more likely to suffer from hypothyroidism. Fertile women are particularly at risk from this conste llation due to cyclic iron loss during menstruation. Blood ferritin levels have been shown to be lower in women with hypothyroidism compared to healthy women. Low blood ferritin levels indicate that hypothyroidism and these conditions are closely related, so research is still ongoing to determine whether this physiological component leads to an increased prevalence of benign and malignant thyroid hormone-related disorders in women(Maenhaut *et al*.,2015) .

This results are also consistent with Ashuma *et al*.,(2015). the relation nship between thyroid hormone characteristics and blood ferritin in hypothyroidism, where it was found that blood ferritin levels appear to be statistically lower and significantly lower in hypothyroid patients than in healthy individuals.

According to a study by Arvind and others (2018), people with subclinical hypothyroidism have significantly lower hemoglobin, blood ferritin, and red blood cells than people with a healthy thyroid.

Similar study by Kiran *et al*.,(2016) ,found that Thyroid and iron metabolism association in hypothyroidism. They concluded that iron and ferritin levels decreased significantly, especially at advanced ages, while total iron levels decreased, and the ability to bind was observed to increase significantly in patients with hypothyroidism compared to healthy woman .

The results showed that the levels of both anti-TPO and anti-TG were higher in women with hypothyroidism with a significant difference of $P<0.028$ in anti-TPO and $P<0.002$ compared to the control group. More individuals with hypothyroidism used TPO antibiotics before any thyroid dysfunction developed than did the entire control group. However, as in patients with a high level of TG antibodies, certain antibodies have been linked to an increased risk of hypothyroidism. TG serum and TPO are positively associated with the response of larger thyroid inflammation and the start of hypothyroidism. In addition, the production of reactive roots and programmed death by these antibodies can be increased (DeGroot *et al* ., 2015) .

(Balucan *et al*., 2013) His study stated that TPO antibodies are more common than thyroid antibodies (TG antibodies) and that they suggest more thyroid disease.Thyroid damage in Hashimoto thyroid inflammation is influenced by TPO antibodies. However, when the anti-protein positivity of TG alone was assessed and then compared to the joint evaluation of antibiotics, TPO or, TG, the combination of antibiotics, TPO or, anti-TG showed much higher results for comparison of anti-TG alone (Kawther *et al* ., 2022).

Results of the association of anti-TG antibiotics with increasing age and gender are as shown in Table (4.10), (4.11), These results also showed that levels of anti-TG were higher in women with hypothyroidism compared to the control group for BMI groups, due to the

association of anti-TG and anti -TPO with thyroid hormones. This is because when anti-TG and anti-TPO rise, antibiotics, TPO, or anti-TPO decrease. TG, which reduces the thyroid's production of its hormones (Hutfless *et al*., 2011). As for the thyroid is important in the metabolism process, the fat burning process decreases causing obesity. While our results did not show clear significant differences with TPO.

In addition the combination of antibiotics, TPO or anti-TG, has an important role in the oxygen free radicals accumulated in thyroid cells. They may inhibit the function of TPO and interfere with the synthesis and secretion of thyroid hormones, causing hypothyroidism. TPO deficiency prevents the synthesis of GPX, which in turn. Reducing free radicals (ROS) formed in the thyroid prevents the form of H_2O_2 from being converted into beneficial H2O, which is what happens when GPX levels decrease. Free radicals (ROS) rise, and malondialdehyde (MDA) increases. Decrease in total antioxidants (TAS), which leads to the destruction of the cell wall and then the nuclear membrane, thus destroying the cell and leading to programmed death (Sies, H and Jones,2020).

These findings were supported by the findings of researcher Balucan *et al*.,(2013) and other, Both subclinical/overt hypothyroidism showed a higher proportion of subjects with anti-TPO before the onset of thyroid dysfunction compared to the combined control group as well as in subjects with elevated TPO levels. Anti-TG, this would also be expected. Measurement of anti-TPO with TSH serves as a warning sign in the diagnosis of hypothyroid people who may be at risk for possible thyroid dysfunction.

These results are also consistent with those of Huftless *et al*.,(2011)who confirmed that anti-TPO was present in 66% of Hashimoto's patients 7 years before clinical diagnosis.

However, the opinion of Jo and Lim (2018) considered that anti-TG is less important than anti-TPO because it is a well-established marker in the diagnosis of differentiated thyroid cancer (DTC) and is considered a less specific marker in thyroid disease compared to anti-TPO.

Thus, it may be useful to consider anti-TPO and anti-TG testing in conjunction with the primary thyroid markers, TSH and FT4, to prevent long-term morbidity.

However and Frohlich and Wahl (2017). believed that anti-TPO was more important than anti-TG because it is a more accurate marker for identifying differentiated thyroid diseases. The presence of anti-TPO has been shown to indicate a potential risk for hypothyroidism even in the absence of any change in FT4 and TSH levels, and anti-TG and anti-TPO tests may be useful to prevent disease over time.

5.4. Correlation Coefficient among all Studied Parameters .

The correlation results appeared the thyroid-stimulating hormone (TSH), which is connected to aging because it rises with age and with gender since it rises higher in women than in men, This results was supported by the findings of researcher Roshangar *et al*., (2014). who found that a negative feedback mechanism maintains hormonal balance in the body. This study also showed a significant positive relationship (P< 469) between thyroid-stimulating hormone (TSH) and total antioxidant activity, According to researcher (Bjoro *et al*., 2000). In addition, accumulated oxygen free radicals in thyroid cells may inhibit TPO function and interfere with the synthesis and secretion of thyroid hormones, causing hypothyroidism, TPO deficiency prevents the synthesis of GPX, which in turn prevents the reduction of free radicals (ROS) formed in the form of H_2O_2 so that they are converted into useful H2O, which is what happens when GPX levels decrease. Free radicals

(ROS) rise, and malondialdehyde increases (MDA). Total antioxidants (TAS) decrease (Sies,H and Jones,2020).

Selenium and Anti-TPO exhibited a significant (P<326) negative relationship because of the part selenium plays in the synthesis of anti-TPO Selenium deficiency leads to TPO deficiency, which inhibits the production of thyroid hormones via idoidonase, an enzyme that promotes the bonding of glutathionine (TG) with iodine.

The results of the current research suggest that anti-TPO antibodies are frequently linked to hypothyroid dysfunction. It was more frequently observed in females who were of childbearing age. Thus, such individuals should undergo early screening testing for thyroid conditions .

Ferritin and anti-TPO also have a negative association (P<345), with ferritin deficit leading to iron insufficiency, which in turn results in TPO deficiency, which in turn results in a reduction in the synthesis of thyroid hormones T3, T4. In the same way, a lack of it raises GPX insufficiency, which decreases the quantity of thyroid hormones (Rasool *et al* ,.2021). Total antioxidants are imitated by free radicals, which might be increased the risk of breast cancer in women and diabetes (Dos.,*et al* 2020).

CHAPTER Six
Conclusions and Recommendations

Conclusions.

1. Low levels of selenium are linked to an a greater risk of producing thyroid antibodies anti-TPO anti-TG, which results in the development of thyroid illness, as well as increased ROS formation and oxidative damage.

2. Determination of serum ferritin levels may be helpful in assessing the state of thyroid hormones.

Recommendations.

1. Additional research on the biochemical effects of zinc and copper in thyroiditis patients.

2. Cellular study for selenium transporters in thyroid gland

3. Genetic stady of selenium in hypothyroidism wamen.

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الخلاصة

يعتبر قصور الغدة الدرقية من الحالات المرضية الشائعة والتي قد تؤدي الى الاصابة بامراض أكثر خطورة في حال تم اهمالها أو عدم علاجها كامراض القلب وسرطان الثدي ، اذ تسيطر هرمونات الغدة الدرقية على عملية التمثيل الغذائي وتعتبر ضرورية للنمو السليم، وتعد ثاني مرض اكثر شيوعا بعد مرض السكري.

اجريت الدراسة الحالية في قسم علوم الحياة، كلية العلوم ، جامعة بابل خلال الفترة من حزيران الى تشرين الثاني (2022). تم جمع عينات الدراسة من اناث تعاني التي من قصور الغدة الدرقية من مستشفى مدينة مرجان التعليمية ومستشفى الإمام الصادق التعليمي.

تضمت الدراسة 100 الحالية جمع عينة دم وقسمت على النحو التالي: مجموعة السيطرة وتضم 30 عينة دم (النساء التي لاتعاني من قصور الدرقية ولامراض الأخرى)، في حين ضمت مجموعة النساء المصابات بقصور الدرقية 70 عينة دم و تم تقسيم المرضى إلى ثلاث مجموعات حسب الفئة العمرية للمجموعة الأولى 20-17 سنة (15عينة) ، المجموعة الثانية أكثر من 21-40 سنة (20 عينة) و المجموعة الثالثة أكثر من 40 سنة(35 عينة). كما صنفت العينات الى ثلاث مجاميع حسب مؤشر كتلة الجسم (BMI)body mass index) (كغم) / م^{2}).

أوضحت نتائج معايير الدراسة الى وجود فروق معنوية ($P < 0.05$) بين تراكيز هرمونات الغدة الدرقية رباعي يودوثيرونين (T4) ثلاثي ايودوثيرونين (T3)، والهرمون المحفز للغدة الدرقية (TSH) لمجموعة المريضات مقارنة بمجموعة السيطرة، كما أظهرت نتائج قياس تركيز هرمون الغدة الدرقية (T4) انخفاضاً معنوياً ($P < 0.002$) لمجموعة النساء المريضات مقارنة بمجموعة السيطرة , كما اظهر تركيز هرمون يودوثيرونين انخفاضاً معنوياً ($P < 0.040$) المجموعة المريضات مقارنة بمجموعة السيطرة , في حين ازداد تركيز الهرمون المحفز للغدة الدرقية بشكل ملحوظ ($P < 0.0001$) لمجموعة النساء المريضات مقارنة بمجموعة السيطرة بينت نتائج الدراسة الحالية انخفاضا معنويا ($P < 0.011$) في تركيز عنصر السيلينيوم لدى النساء المصابات مقارنة بمجموعة السيطرة. كما ازداد العامل المضاد للاكسدة المالون الديهايد (MDA) معنويا لدى مجموعة المريضات مقارنة مع مجموعة السيطرة($P < 0.0001$) .أمابالنسبة لتركيز مضادات الأكسدة الكلية فقد انخفضت بشكل معنو($P<0.026$) في مجموعة المريضات مقارنة بمجموعة السيطرة , كذلك انخفض تركيز الفريتين معنويا ($P<0.0001$) في النساء المريضات مقارنة مع مجموعة السيطرة, في حين ازدادت معنويا ($P<0.028$) تراكيز الاجسام المضادة الى انزيم البيروكسيداز الدرقي (Anti-

TPO) والثايروغلوبولين (Anti-TG) (P<0.002) في النساء المريضات مقارنة مع مجموعة السيطرة.

لقد اشارت العلاقات بين المعايير المدروسة وجود علاقة ارتباط سلبية بين كل من تركيز الاجسام المضادة البيروكسيديز الدرقي مع كل من تركيزي عنصر السيلينيوم في حين تبين وجود علاقة إيجابية بين هرموني ثلاثي يودوثيرونين ورباعي يودوثيرونين وبين عامل مضاد الاكسدة الكلي والهرمون المحفز للدرقية.

بينت نتائج الدراسة وجود انخفاضا معنويا (P<0.011) في تركيز عنصر السيلينيوم لدى مجموعة من النساء المريضات مقارنة بمجموعة السيطرة. كما ازداد تركيز العامل المضاد للاكسدة المالون الديهايد (MDA) معنويا لدى مجموعة النساء المريضات مقارنة مع مجموعة السيطرة (P<0.0001). أما بالنسبة لتركيز مضادات الأكسدة الكلية فقد انخفضت بشكل معنوي (P<0.0026)في مجموعة المريضات مقارنة بمجموعة السيطرة . كذلك انخفض في تركيز الفريتين معنويا في النساء المريضات مقارنة مع مجموعة السيطرة(P<0.026). في حين ازدادت تراكيز معنويا (P<0.028) تراكيز الاجسام المضادة الى انزيم البيروكسيداز الدرقي (Anti-TPO) و الثايروكلوبيولين (Anti-TG) (P<0.002) في النساء المريضات مقارنة مع مجموعة السيطرة.

لقد اشارت العلاقات بين معايير الدراسة الى وجود علاقة ارتباط سلبية بين كل من تركيز الاجسام المضادة الى انزيم البيروكسيديز الدرقي مع كل من تركيزي عنصر السيلينيوم والفرتين في حين تبين وجود علاقة إيجابية بين هرموني الثايروكسين وترايايودوثايرونين وبين عامل مضاد الاكسدة الكلي والهرمون المحفز للدرقية .

جمهورية العراق
وزارة التعليم العالي والبحث العلمي
جـــامـــعـــة بـــابـــــل
كـلـيـة الـــعـــلـــوم
قـسـم عـلـــوم الحـيـاة

دراسة فسيولوجية وبايوكيميائية لدى النساء المصابات بقصور الغدة الدرقية

رسالة

مقدمة الى مجلس كلية العلوم- جامعة بابل
وهي جزء من متطلبات نيل درجة الماجستير في العلوم/علوم الحياة
من قبل

رسل مشتاق طالب سبتي عبود

بكالوريوس علوم حياة / جامعة بابل ٢٠١٧

بإشراف

أ. م. د. ولاء صالح حسن اللبان
أ. د. حسين جاسم عبيد الحربي

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