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Study The effect of Some Hormones in Sera of Alopecia Areata Patients in Al-Ramadi City

A Thesis Submitted to the Council of the College of Science - University of Anbar as a Partial Fulfillment of the Requirements for Master Degree in Chemistry.

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بِسمِ ٱللَّهِ ٱلرَّحمَٰنِ ٱلرَّحِيمِ

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Dedication :

To my parents (Tariq & Fatima), for their love, prayers, caring and sacrifices for educating and preparing me for my future. They are my ultimate role models.

To My husband and my life partner (Mohammad) for his love, understanding, continued support and walks with me to my dreams.

To my sisters, brothers and aunts, the gentle pure hearts and innocent souls to the winds of my life.

To my friends and everyone who helped me and you were always by my side to help me with all their effort and time.

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Roaa Tariq Hammad

Summary:

Context:

Alopecia areata is an autoimmune or organic disease that occur hair loss, and alopecia areata is characterized by patchy hair loss, which includes the scalp and other areas of the head, including eyelashes, beard and entire body hair, which may also be affected. A patient with alopecia areata notes the sudden appearance of a circular patch of hair loss, which may lead to baldness of the entire scalp and is called (Alopecia areata totalis) or loss of full body hair called (Alopecia areata universals).
The etiopathogenesis of the disease is still unclear, but the role of autoimmunity is strongly suggested. AA is commonly associated with Thyroid disorders; the most frequent among them is autoimmune thyroid disorders.

The aim of our study to determine whether Alopecia Areata (AA) is associated with some hormone like: Thyroid hormones (T3, T4 and TSH) Follicle Stimulating Hormone (FSH) and Luteinizing Hormones (LH) in patients with AA in Al- Ramadi city environment.

The study includes 72 cases with age ranging from (10-50 years) ; (Group A) 42 patients with AA (27 male and 15 female) and (Group B) 30 healthy volunteers (7 male and 23 female) are selected as a control group. All samples are obtained from Dermatology outpatient clinic, Al-Ramadi Hospital, Al-Anbar Governorate, Iraq. during the period from December 2019 to march 2020. Every case and control are subjected to history taking, dermatological examination and complete general Venous blood samples are taken from controls and patients after taking their agreement for measurement of some hormones such; Thyroid Hormones (TSH, T3, T4) and LH, FSH. We found, there were statistically significant differences between patients and controls regarding Thyroid Hormones levels of TSH, T3 and T4.

Also There were significant differences between Patients and controls regarding of Follicle Stimulating Hormone (FSH) and Luteinizing Hormones (LH)

So, Follow the current study with same parameters with more samples number to confirm the current results and It's preferable to screened of Hormones every patient with Alopecia Areata such as thyroid hormones (TSH, T3, T4), Follicle Stimulating Hormone (FSH) and Luteinizing Hormones (LH) ,even in absence of manifestations suggestive of Hormone disorders and associated diseases.

Keywords:

Alopecia**,** Thyroid hormones (TSH, T3, T4), Follicle Stimulating Hormone (FSH) and Luteinizing Hormones (LH)

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List of Abbreviation

AA	Alopecia areate
AT	Alopecia Totalis
AU	Alopecia Universalis
ATP	Adenosine Triphosphate
CIA	Chemotherapy- Induced Alopecia
DHT	Di-Hydro-Testosterone
Fig	Figure
FSH	Follice Stimulating Hormone
GnRH	Gonadotropin-releasing hormone
HCG	Human Chorionic Gonadotropin
HF	Hear Follicle
IRMA	Immunoradiometric assay
IVF	In Vitro Fertilization
LH	Luteinizing Hormone
NS	No Significant
P value	Probability Value
PCOS	Polycystic ovary syndrome
SIA	Stress Induced Alopecia
SD	Standard Deviation
SPSS	Statistical Package for Social Science
TH	Thyroid hormone

T3	Triiodothyronine
T4	Thyroxin
TSH	Thyroid - Stimulates Hormone
TBG	Thyroxin-Binding Globulin
TRH	Thyrotropin-releasing hormone
TG-AB	Thyroglobulin autoantibodies bind thyroglobulin
TPO-AB	Thyroid peroxidase autoantibody
TRH	Thyrotropin Releasing Hormone

Chapter One

Introduction and

Literature review

1.1 Introduction: -

Alopecia areata (AA) is an autoimmune or organic disease, chronic in nature, is a T-cell mediated of hair follicles characterized by sudden, recurrent, and psychologically devastating hair loss with a pattern of relapsing immune-mediated inflammation of the hair follicles. [1] This inflammatory process affects the hair follicle size resulting in it becoming smaller with associated retardation of hair production. AA is a common human disease with a lifetime risk of 1.7% in the general population.[2]

It is characterized by a hair loss most commonly involving the scalp although other regions of the head, including eyelashes and beard, may also be affected. The patient with [AA] notes the sudden appearance of circular patch of hair loss. [AA] is not life-threatening but rather psychologically and socially disturbing. [AA] may be observed at any age with no sex predominance but likely to happen in adolescents [3]. In a study of patients with alopecia areata who were 16 years of age, the median age at onset was 10 years and the male : female ratio was 1.4:1 ; the disorder was more severe in boys and in those with an onset in early childhood [4].

The disease may sometimes lead to complete scalp baldness called (Alopecia areata totalis) It is a status characterized by the complete loss of hair on the scalp. It is a progressing form of alopecia areata a status that causes round patches of hair loss [5]. Although the exact cause of Alopecia areata totalis is obscure, it is thought to be an autoimmune status in which the immune system mistakenly attacks the HF follicles [6][7].

About 20% of influenced people have a family member with alopecia, suggesting that genetic factors may take part to the development of Alopecia areata totalis [8]. There is currently no medicament for Alopecia areata totalis, but sometimes hair regrowth occurs on its own, even after many years [9], or even total body hair loss called Alopecia areata universals, it is characterized by the whole loss of hair on both the body and scalp. Most of Alopecia areata universals Patients do not have other symptoms and signs, but some may experience a itching, sensation or burning.[10]

Alopecia areata universals can be associated in some cases with other status such as thyroid disorders, atopic dermatitis, and/or nail changes (like pitting).[11] disorders, Anxiety, depression, personality and paranoid disorders are more popular in people with different forms of alopecia areata [12]. AA of the neck is called the ophiasis type [13].

Alopecia areata is a non-contagious autoimmune skin disease in which your body's immune system sees your own hair follicles as strange, and so, attacks them causing follicle cell destruction and hair growth to completely stop. there are several possibilities Causes Alopecia Areata, Totalis And Universalis, such as [4][14] :

- **Congenital ;** (they were born with it) - A few cases have been notified for babies with congenital areata, but it's not known for sure whether this disorder developed from was due to genetics or from some unknown interaction within the womb.

- **Genetics**; in 20% of cases, areata does view to have a family connection. But, whether this is congenital or genetic is still obscure. However, atopic conditions definitely can have a genetic connection, and often coincide with areata as well.
- **Atopic conditions**; These involve asthma and eczema, both of which have been associated with this type of hair loss.

  Both areata and atopic conditions can involve an autoimmune response, so therein lies an obvious connection. In fact, these two conditions can coincide in up to 50% of areata cases.
- **Langerhans cells and helper T lymphocytes;** an excess of some autoimmune cell types might be the cause.
- **Other autoimmune diseases;** Your chances of promote areata might be higher if the following autoimmune diseases run in your family: diabetes type I, Addison's disease (adrenal gland disorder), rheumatoid arthritis, vitiligo (patchy loss of skin color) [15][16].
- **Psychological trauma;** several studies have behold that emotional stress participate to the appearance of alopecia areata, given the noticing that emotional trauma precedes the process, with each other with the high prevalence of psychological disorders happen in these patients [17][18].

1.2 Literature review

1. 2. 1 Alopecia Areata Diagnosis:

The diagnosis of AA is made on clinical grounds. The course of AA is unpredictable and typically characterized by phases of acute hair loss followed by spontaneous hair regrowth and waxing and waning of the lesions and hair loss can persist for many years. Very often AA shows a mild clinical course with only few small bald patches, and hair regrowth after some weeks or months. The prognosis for AA is defined by Age at onset, duration and progression of disease, personal and family history of similar disease as there are many conditions that can be associated with Alopecia. A decrease in the number of red blood cells due to B12 deficiency (pernicious anaemia), vitiligo, lupus, menopause, as well as pregnancy, and infections of hair follicles and scalp can all cause hair loss. As a basic diagnostic step, the doctor will examine scalp for the appearance and pattern of hair loss.

Patterns of smooth patches with short broken hairs around the borders can be characteristic of alopecia. There is no blood test to confirm or to rule out the diagnosis If an autoimmune condition is suspected, addition tests may be undertaken these can include[19][20]: Immunological tests of antinuclear antibodies, Iron levels, Type 1 diabetes mellitus, Free and total testosterone, Thyroid hormones, Follicle stimulating and luteinizing hormones and measures of inflammation .

1.2.2 Symptoms:

As in all autoimmune conditions, the body's immune system enters into a confused state and begins to mistakenly attack its own systems. In the case of Alopecia, the hair follicles are targeted. Whilst the condition is not life threatening and in general does not have associated physical pain, the loss of hair can cause immense psychosocial impacts as well as symptoms related to loss of protective hair coverings. For example [20][21]:

• The loss of nasal eyelashes and hair can result in increased irritation of nasal and eyes passages with increased risk of foreign body intrusion such as dust.

• The pitting of toe and finger nails is common therefore; adherence to careful feet, hand, and nail care regimes can help reduce impacts.

• Bare patches of skin are more susceptible to sun burn so increased vigilance in adhering to common sun protection measures as wearing eyewear and protective clothing and use sunscreen are recommended.

1.2.3 Associated with Other Autoimmune Diseases:

AA may be associated with other autoimmune diseases, like Vitiligo or Type 1 diabetes mellitus, Rheumatoid arthritis, increasing risk factors for alopecia and 20% of people with alopecia also report to have a close family member with the condition as well, A decrease in the number of red blood cells due to B12 deficiency (pernicious anaemia), Down's syndrome, and candidiasis, Follicle stimulating and luteinizing hormones.

Thyroiditis tends to be more common in AA. The prevalence of thyroid disease in patients with AA ranges from 8% to 28% [22] .

The prevalence of vitiligo in AA patients is 3% to 8%. These disease associations suggest a relationship between autoimmunity and AA. .[4] AA is associated with psychiatric morbidity, especially anxiety and depression.[23]

Atopic disorders, namely allergic rhinitis, asthma, and atopic dermatitis have been related to AA. They have been found to happen in more than 40% of patients with AA, whereas their diffusion in the general population is estimated to be about 20% [24]. Ocular alterations, like asymptomatic punctate lens opacities, and fundus changes can happen in up to 50% of patients with AA [25].

The thyroid gland is one of the largest of the endocrine glands located immediately below the larynx on each side of and anterior to the trachea. Thyroid gland is secretes two biologically active thyroid hormones: Thyroxin (T4) and 3,5,3'-triiodothyronine (T3) [26]. Luteinizing Hormone (LH) and Thyriod-stimulating Hormone (TSH) are key regulators of the gonadal functions. [27][28]

Luteinizing Hormone (LH) and Follicle-stimulating Hormone (FSH) are both made from similar genes, and thus have similar properties. They are both glycoproteins made up of an alpha and beta subunit. The alpha subunit is the same between the two hormones, and the beta subunit of each is different and gives each hormone its biological specificity[29]. Psychiatric Morbidity.

- **Thyroid diseases**

Thyroid disorders involve all the organ systems of the body and the skin is no exception, The first symptoms of Some dermatological skin findings and diseases may be thyroid disease [30]. Thyroid hormone stimulates protein synthesis, epidermal oxygen consumption, determination of epidermal thickness and mitosis [31] Thyroid hormone is an important organizer of epidermal homeostasis[32]. In tissue culture studies using replacement for DNA expression, T3 has been shown to catalyze growth of both epidermal keratinocytes and dermal fibroblastes, also thyroid hormone appears to be necessary for both maintenance and the initiation of hair growth and normal secretion of sebum [33][34] .

Hypothyroidism and Hyperthyroidism both are cause skin change. Hypothyroidism may result from target cell resistance to hormonal action or either inadequate circulating levels of thyroid hormone, Primary hypothyroidism the most common cause is as a result of glandular failure and most frequently result from autoimmune disease [35] .

In hypothyroidism, the skin is pale, cold and xerotic. The dryness of hypothyroid skin results from decreased eccrine gland secretion, Hypohidrosis, possibly accompany by diminished epidermal sterol biosynthesis [36][37] .

In hyperthyroidism, the skin is soft, warm, smooth and moist. The epidermis is thin but not atrophic, While the smooth skin is an epidermal finding, the warmth is caused by increased cutaneous blood flow and the moisture is a reflection of the underlying metabolic state, the warmth is often chaperoned by a persistent flush of the face, redness of the elbows and palmar erythema Scalp hair may be fine and soft, and may be chaperoned by a diffuse non scarring alopecia. The resulting abnormal ities in Thyroid hormone serum levels effect on the function of skin and its appendages [38] [39] .

Hypothyroid patients display hair follicles that are brittle, dry and dull due to a lower sebum production, 50% of the patients experience from diffuse alopecia which seems to result from slower growth and a shorter anagen phase [40][41]. The hair follicles of hyperthyroid individuals show a thin and fine texture and appear fatty. Alopecia occurs in hyperthyroidism also but only in 20-40% of patients, this is explained by a shortening of hair cycling with a shortened telogen and anagen phase [42] .

1.2.4 Types of Alopecia:

Alopecia is a generic term that is generally used for hair fall. Alopecia can be classified into two broad categories, Cicatricle (Scarring) hair loss accompanied with scars that destroy the hair follicle, which resulted into permanent hair loss. And Non-Cicatricle (Non-Scarring) hair loss is not permanent [43] .

1.2.4.1 Non-Cicatricle alopecia

1.2.4.1.1 Alopecia areata (AA)

Alopecia areata is one of the most common autoimmune diseases. Most common reasons of AA are Hormone pills, sexually transmitted disease like syphilis, pregnancy, thyroids disorder, anemia and arthritis [44].

1. Classic forms

A - Alopecia areata in unifocal plaque or single in this shape there is oval or a single, round, smooth alopecic plaque in which with hair of a normal appearance, the skin coloration is normal.

FIG (1_1) : Multifocal AA [45]

B - Alopecia areata in multifocal or multiple plaques in this shape typical alopecic plaques happen that affect the scalp or other pilar areas.

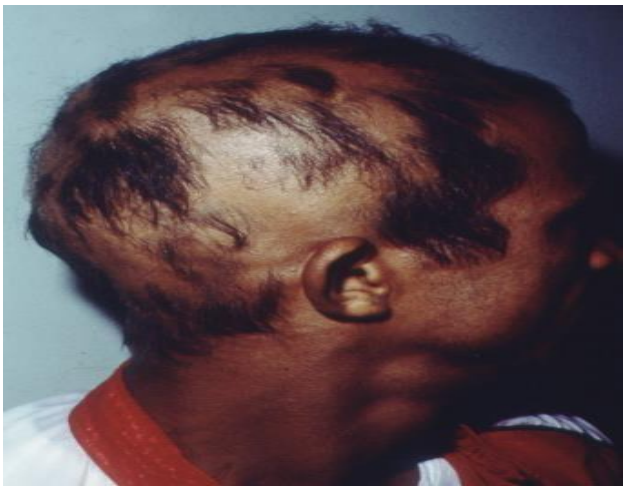


FIG (1 -2) : Multifocal AA [45]

C - Ophiasic alopecia areata in this type, the hair loss happens along the line of temporo occipital implantation, giving rise to a wide alopecic area, in a band that extent the inferior margins of the scalp.

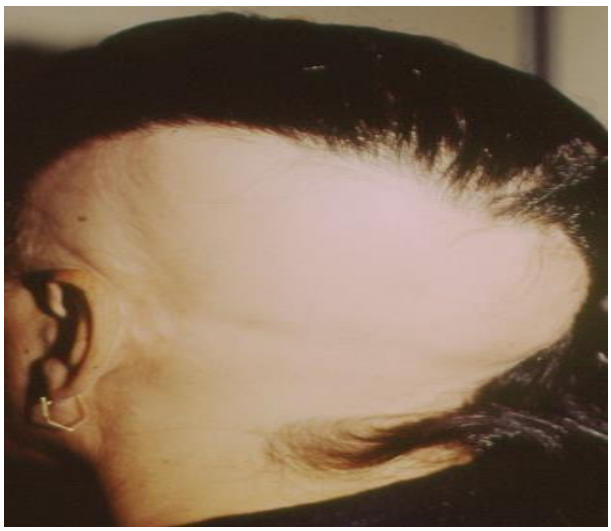


FIG (1-3):Ophiasic AA [45]

D - Alopecia totalis (AT) There is whole loss of terminal hair of the scalp without affecting other body hair.

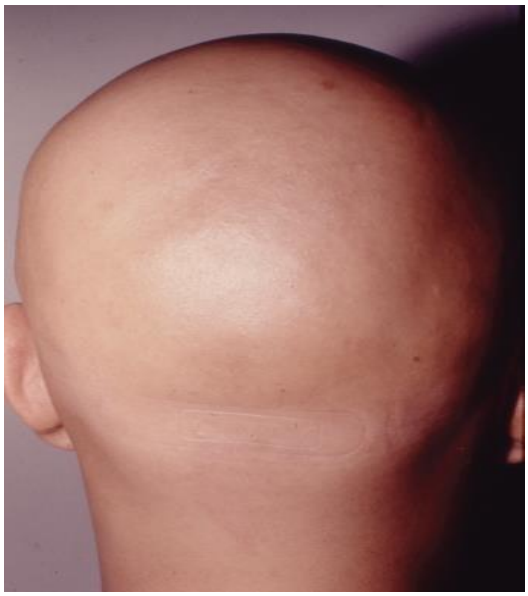


FIG (1-4) : AA totalis [45]

E - Alopecia universalis (AU) There is whole loss of body hair, include the beard, eyelashes, eyebrows, scalp, and mustache, genital areas and armpits [46].

There are atypical presentations of AA , In addition to these types that are considered classic:

2. Atypical forms

A - Sisaifo type AA (inverse ophiasis) It is the inverse clinical image of the ophiasis form, In this type , the hair loss involves the entire scalp excluding for the lower margins, along the line of temporo occipital implantation .

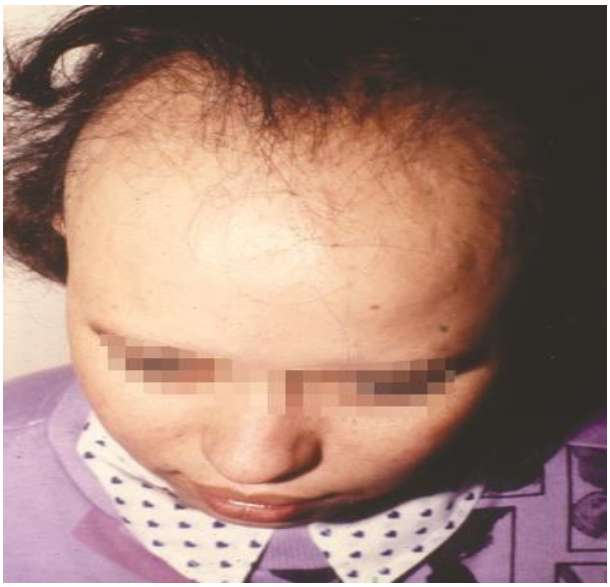


FIG (1-5) : Sisaifo type AA [45]

B - Reticular alopecia areata in this form, multiple alopecia plaques happen separated by narrow bands of preserved hair, accord a reticulated aspect to the picture.

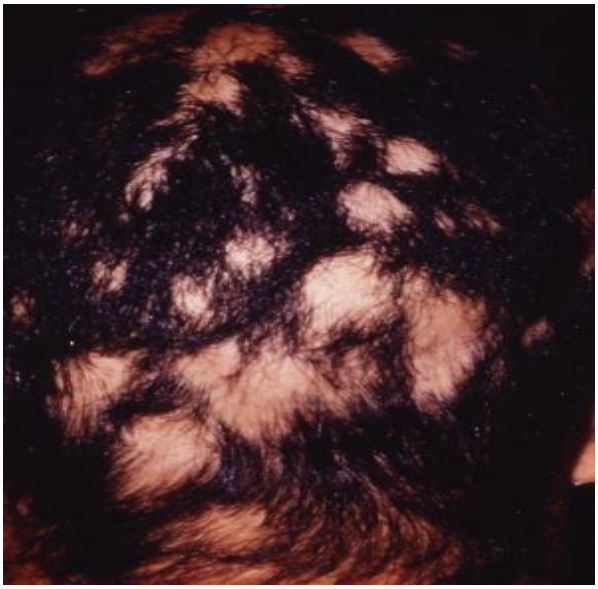


FIG (1-6): Reticular A [45]

C - Diffuse alopecia areata in this form, the hair loss is sharp and prevalent. It can be the premier form, at most among adolescents and children, or can develop from plaque forms. Most of these cases develop into the more dangerous AT or AU forms. It is the most difficult form to diagnose, demanding a differential diagnosis with sharp telogen deffluvium, alopecia syphilitica and also androgenetic alopecia. Thus, require complementary test in general and also histopathological test by biopsy.

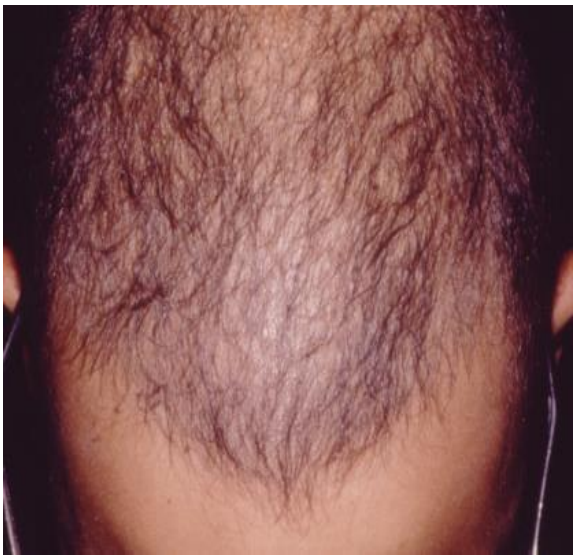


FIGURE (1-7) : Diffuse AA [45]

1.2.4.1.2 Androgenic alopecia:

Is a common hereditary thinning of hair induced by androgens and this condition is known as common female-pattern baldness in women and as baldness in men. In this type, hair follicles decrease in specialized patterns and specific over the scalp in female and male. Androgen especially testosterone is required for androgen dependent alopecia in men.

In women, there is no evidence that the hair loss are truly dependent on hormone, although both male and female pattern alopecia results in decrease in hair follicle size [47].

1.2.4.2 Cicatricle alopecia:

Cicatricle (scarring) alopecia is linked with inflammation that destroy the hair follicle, resulting in permanent hair loss. is categorized into:

1.2.4.2.1 primary cicatricle alopecia: hair follicles are affected by inflammation of cell by lymphocytic or it's predominant such as Discoid lupus erythematous

1.2.4.2.2 secondary cicatricle alopecia : inflammation due to systemic disorder an example of secondary cicatricle alopecia is granulomatous is involved and sarcoidosis [48].

1.2.4.3 Other types of alopecia:

1.2.4.3.1 Chemotherapy–induced alopecia (CIA) :

One of the most emotional and distractive side effects with chemotherapy is hair loss, hairs do eventually again regrow. after chemotherapy treatment because the cycling follicular stem cells regenerate a new hair follicle [49][50].

1.2.4.3.2 Stress induced alopecia (SIA) :

Stressed condition doesn't cause hair loss directly but emotional and behavioral changes lead to hair fall. Thus, reduction in stress level, balanced diet and exercise is the best treatment for Stress induced alopecia [51].

1.2.5 Hormones:

The word hormone was formed by deriving the Greek word (hormao) that denotes excite, move or stimulate. Hormones are chemicals messengers (peptides, proteins, and steroids) secreted by the endocrine glands; produced and secreted by specialized cells [52], acting as messengers, released into the bloodstream to influence behavioral physical, physiological, changes, They have continued action to keep body balance, enable continuous adaptation to the environment and organ function [53][54]. In general, the functions of hormones under following heads:

1- **Permissive function.** The endocrine gland affects the functioning of one another not only does affect a number of processes, so some hormones demand the presence of another hormone for the expression of their activity, this helps in preserve a perfect hormonal balance. Disruption of this balance, either experimental or clinical, result a variety of metabolic aberrations
2- **Integrative function.** the endocrine system has integrative properties steady and slow whereas those of the nervous system are fast. This Close association between the two systems has led to evolution of a new discipline of science called neuroendocrinology**.**

3- **Morphogenetic function.** The hormones administer the ontogenetic development of an individual from the embryonic to the adult state.

Chemically, the hormones are divided into 3 groups Depending on the structure:

1- Steroid (cholesterol derivatives).
2- Derivatives of an animated acid (Thyroid hormones).
3- Peptides (made from several amine acids) such as LH, FSH and TSH [55] .

Hormones regulate different functions in the body, including:

1- Metabolism.
2- Development and Growth.
3- Psychological state.
4- Reproduction and Sexual function [56] .

The hormones come in contact with all the organs of the body, growth and reproduction, exercising, sleep, their control over metabolism, mood and more. Any hormonal imbalance is followed by some disease ,AA associated with some disease or hormone [57] One of the key roles is attributed to hormones, of which we list the most important:

1.2.5.1 Thyroid hormones:

Thyroid gland is a chief endocrine gland secretes two biologically active thyroid hormones: thyroxin 3,5,3',5'-tetraiodo-L-thyronine (T4) and 3,5,3'-triiodothyronine (T3).

They are composed of a phenyl ring attached via an ether linkage to a tyrosine molecule. T4 has two iodine atoms on its phenyl (outer) ring, whereas T3 has only one. Both have two iodine atoms on their inner tyrosine ring [26] .

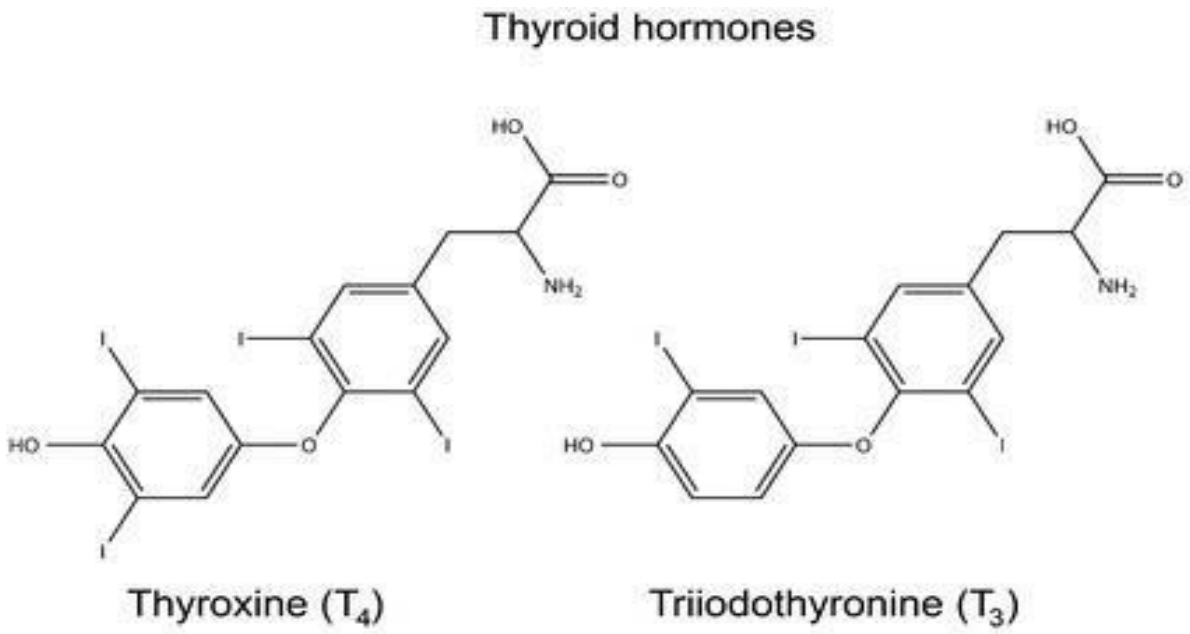


Figure (1- 8) Chemical structure of the thyroid hormones

The thyroid hormone, (T3), is produced by the thyroid gland. An important component in the synthesis isiodine. (T3) is considered the active hormone. About 20% – 30% of the circulating T3 is secreted by the thyroid gland and the remainder is produced by monodeiodination of T4 in extract thyroid tissues, kidney, notably the liver, brain, and pituitary.

T3 is biologically the most metabolically active hormone (3-4 times more powerful than T4) although Circulating levels of T4 are much greater than T3 levels, but although its effect is shorter due to its briefer half-life compared to T4.

In hypothyroidism (T3) and (T4) levels are decreased. T3 levels are often low in sick or hospitalized euthyroid patients.

In hyperthyroidism, both (T4) and (T3) levels are usually elevated, but in a small subset of hyperthyroid patients only T3 is elevated (T3 toxicosis).

The interaction of (T3) with its receptors (TRa and TRß) affects epidermal differentiation and promote its responsiveness to growth factors. These effects of T3 are particularly important for the function of eccrine, apocrine glands and sebaceous, synthesis of protein and glycosaminoglycan and growth of hair follicles by dermal fibroblasts [39][58] .

Thyroxin (T4) is the principal hormone synthesized in the thyroid gland, All the T4 in Blood circulation is derived from thyroidal secretion, T4 catalyze the proliferation of hair follicle keratinocytes and T3 prevent their apoptosis [34]

T4 is metabolized to (T3) peripherally by deiodination. T4 is considered a reservoir or prohormone for T3, the biologically most active thyroid hormone. About 0.05% of circulating T4 is in the free or unbound portion. The remainder is bound to Thyroxin-binding Globulin (TBG), prealbumin, and albumin. The hypothalamus excretes Thyrotropin-releasing hormone (TRH),

which catalyze the pituitary to release thyrotropin (previously thyroid catalyzing hormone: TSH). TSH catalyze the thyroid to secrete T4.

T4 is partially transform to (T3). High amounts of (T4) and (T3) (mostly from peripheral transform of T4) cause hyperthyroidism. (T3) and (T4) cause positive feedback to hypothalamus and the pituitary with resultant deactivation or catalyze of the thyroid gland such as:

Hypothyroidism if (T4) or (T3) is low, increase of TSH. Hyperthyroidism if (T3) or (T4) is high, decrease of TSH.

Thyroid hormones may also affect hair follicle stem cells, since T3 and T4 were found to prevent apoptosis and stimulate differentiation, and clonal growth of hair follicle epithelial stem cells [59].

Thyroid Stimulates Hormone (TSH) is glycoprotein hormone with molecular weight about 30,000, composed of an alpha unit, also present in other anterior pituitary hormones (FSH, LH) and a beta unit responsible for its specific actions, TSH has a half-life = 60 min and typical plasma level are (0.4 - 4.8 mU/L) in those with normal thyroid function. [60] TRH is major controller of TSH secretion, Thyroid hormone release is controlled by an inverse feedback system on TRH and TSH release, TSH is secreted from cells called thyrotrophs in the anterior pituitary gland (TSH) stimulates thyroid growth and production of thyroid hormones. TSH-producing cells constitute approximately 5% of the cells of the normal anterior pituitary. Antibodies to TSH are used in a panel to sub classify pituitary adenomas [61]

Diffuse loss of scalp hair follicles, the lateral eyebrows and body hair are clinical signs associated with hypothyroidism, Hyperthyroid hair follicles produce a thinner hair shaft diameter and brittle, greasy hair [62][63][64]. Also, early greying has been claimed to be related to hypothyroidism, autoimmune thyroid disease, and non-autoimmune [65][66].

1.2.5.2 Follicle stimulating (FSH) and luteinizing hormones (LH):

Follicle stimulating hormone (FSH) is a hormone produced by the anterior pituitary, is a glycoprotein dimer with alpha and beta subunits. The alpha subunit (92 amino acids) is the same as in TSH, HCG, and LH, while the beta subunit (111 amino acids) is unique to FSH. It causes follicular growth and maturation , promote production and secretion of estrogens [67][68].

Excessive FSH causes hyper ovarian follicle maturation, hyperplasia of secondary sexual organs, and hyper secretion of estrogens. While deficiency of FSH causes oocyte maturation and scanty spermatogenesis, obesity, hypogonadism, decrease of estrogen secretion, and hair growth defect [69].

In male, FSH, in conjunction with testosterone (which is under the control of LH), is required for the initiation and maintenance of quantitatively and qualitatively normal spermatogenesis [70].

In the female FSH is necessary for the chosen and growth of ovarian follicles and for the production of estrogens from androgen substrates[71].

The biological activity of FSH is the sum of a complex combination of processes: survival in the circulation, release from the pituitary, transport to the site of action [72].

Luteinizing hormone (LH) is a peptide hormone released by the pituitary gland in response to luteinizing hormone- releasing hormone. plays a key role in gonadal function. is one of the main factors prompt oocyte maturation and ovulation in vertebrates, the release of LH was organized by gonadotropin-releasing hormone (GnRH) in the hypothalamic pituitary- gonadal axis.

LH has a heterodimer structure consisted of α-subunit is a glycoprotein which is common to other pituitary hormones thyroid-stimulating hormone (TSH) and follicle-stimulating hormone (FSH) and β-subunit is specific to LH, and is also a glycoprotein, with a total molecular weight of approximately 29,000 [73][74].

1.2.5.3 Testosterone :

Testosterone is a steroid hormone involved in many bodily processes, including morphology (such as; development of secondary sexual characteristics), reproductive physiology (such as; spermatogenesis), behavior (such as; aggression), psychology (such as; sexual desire), and each of which plays an important role in reproduction and survival [75].

Function: Testosterone has often been rated to be a "youth hormone" it is sometimes used for remedy of persons who have impairment developed muscles Because of the ability of testosterone to increase the size and strength

of bones, it is often used in aging to treat osteoporosis. such as the androgens, estrogens are also in charge of for the development of secondary sex characters in males.

In Male, most of testosterone (90%) produce from the testicles, the other 10% produce from the adrenal glands is Small glands on top of the kidneys. Di-hydro-testosterone DHT, synthesized by the activity of the enzyme 5-α reeducates is the most important metabolite of testosterone concerning hair loss and hair growth [76].

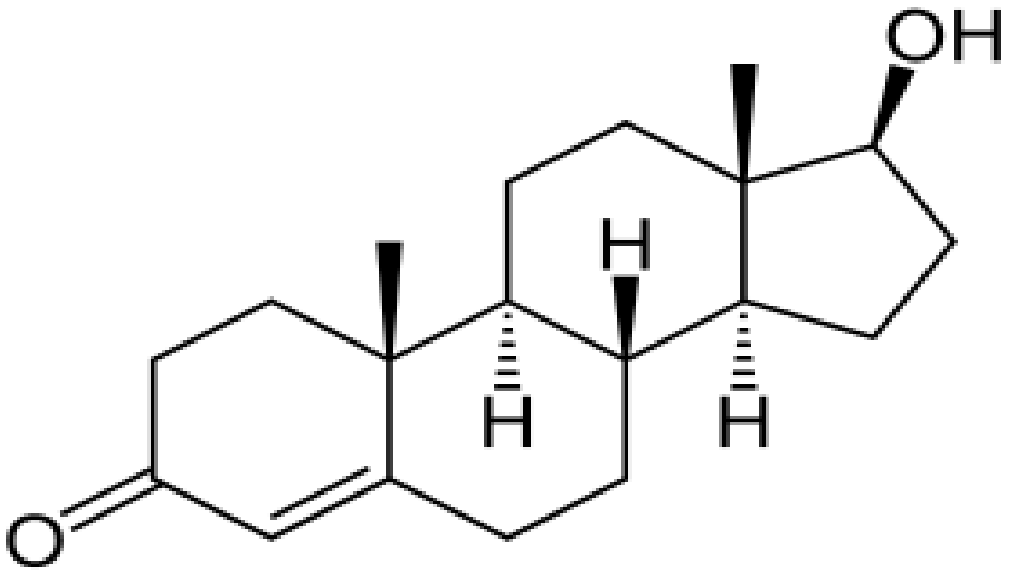


Figure (1-9): Testosterone structure

1.2.6 Previous work Survey

Alopecia areata (AA) is a common kind of hair loss; it is an autoimmune disease, the second-most frequent non-scarring alopecia is Alopecia areata (AA) , after male and female pattern alopecia, It is described by the loss of hair in patches, whole loss of body hair (alopecia universalis) or whole loss of scalp hair (alopecia totalis). The etiopathogenesis of the disease is still vague, but there is proof that endocrine dysfunction and autoimmunity may be embroiled. The aim of this study is to define whether Alopecia areata is statistically connected with hormones and the diseases which hormones cause such as; thyroid autoimmunity.

In **Emina Kasumagić-Halilović** [77] study, Thyroid hormones (triiodothyronine (T3), thyroxine (T4), thyroid stimulating hormone (TSH) and thyroid antibodies) were measured in all patients. Total triiodothyronine (T3) (normal range: 1.3-3.3 nmol / L) and total parathyroid hormone (T4) (normal range: 70-180 nmol / L) were determined using radioimmunoassay (RIA); Thyroid stimulating hormone (TSH) (normal range: 0.3-4.2 ml / L) was determined using an immunoassay (IRMA) (BRAHMS Aktiengesellschaft, Hennigsdorf, Germany). In 8 (11.4%) patients with alopecia areata they found functional abnormalities of the thyroid gland.

In (**Ola A B akry**)[78] study, Measurement of T3, T4, TSH, Tg-Ab and TPO-Ab by used blood sampling: 10 ml of A sample of venous blood was put into tube, left for 10 mints to stand, then put it for 5 mints in centrifuged at

4000 rpm. T3 and T4 were determination by IMMULITE 2000 which is a solid stage, The liquid stage consists of alkaline phosphatase coupled to T3 and to T4 respectively. TSH was determination by IMMULITE 2000 which is a solid stage , Two-site chemiluminescent immunometric assay [79]. Among studied patients, found hypothyroidism in eight cases (16%). (4%) two cases were with moderate Alopecia areata and (12%) Six cases of them were with severe Alopecia areata. hypothyroidism is diagnosed when serum TSH is above the reference range but levels of T4 and T3 are normal, in this study, there were statistically significant variations among diverse. subgroups of Alopecia areata as regards TSH, T3 and T4 levels [80].

also**, Kakourou et al.**, [81] announce that in estimate of 157 cases with Alopecia areata , there were 5% of the cases with Alopecia areata and thyroid disorder.

Thomas and Kadyan made clinical study agreement with these results[82] who declare that hypothyroidism was the most common form of thyroid function abnormalities related with Alopecia areata among the thyroid disorders. it is probable that the volume of impact of thyroid hormone (TH) on hair growth is changeful and its expression may be conditioned by other hormonal influences and local factors [62].

Also, **Hongfeng Wang** [83], Serum anti thyroid autoantibodies and T3, T4 and TSH were checked by chemiluminescence (Bayer AG, Leverkusen, Germany). The values of positivity for TPOAb and TgAb were 34 and 115 IU / ml , respectively. The normal values for T4, T3 and TSH were 12-22 pmol /L, 3.1-6.8 pmol /L, 0.27-4.2 pmol /L, respectively. physical

examination and Thyroid ultrasonography were proceeded to check thyroid magnitude which could be split into I, II- and III-degree tumefaction[83]. However, Alopecia areata (AA) had abnormalities of thyroid hormone and thyroid autoantibodies about (34.18%) 54 of 158 cases, significantly (16%) higher than normal controls.

In other study (**Kasumagic-Halilovic and Begovic**) [84] Blood samples were taken, thyroid sonography was performed and a physical examination. Thyroid autoantibodies and thyroid hormones (triiodthyronine (T3), thyroxin (T4), and thyroid stimulating hormone (TSH) were measured in all subjects. Total triiodthyronine T3(normal range:1.3-3.3nmol/L) and total thyroxineT4 (normal range:70-180nmol/L) were determines by use of radioimmunoassay (RIA); TSH (normal range: 0.3-4.2 mlU/L) was measured by use of immunoradiometric assay (IRMA), (BRAHMS Aktiengesellshaft, Hennigsdorf, Germany). Hormonal abnormalities in thyroid functional were found in (11,43%) 8 patients, only one patient in the control group had abnormalities in hormonal status.

1.3 The Aims of Study:

Association of some Hormones in patients with Alopecia areata in Al- Ramadi city environment.

The following hormonal variables are chosen to be under study and research based on the pathological conditions that induce alopecia areata:

1- **Thyroid hormones**

- Triiodothyronine (T3)
- Thyroxin (T4)
- Thyroid stimulating hormone (TSH)

2- **Luteinizing hormone (LH).**

3- **Follicle stimulating hormone (FSH).**

Chapter Two

Materials and Methods

2.1 Materials

2.1.1 Apparatuses and Equipment

Throughout our study we use some Apparatuses and Equipment which are listed in table (2-1) with their Manufacture.

Table (2-1) Apparatuses and Equipment and their manufacture company

Apparatuses and Equipment	Manufacture
Automatic pipet	Chain
Centrifuge	Germany
Cylinder	Chain
ELISA Microwells	USA
Gel Tube	Chain
Incubator	Germany
Tubes Rack	Chain
Wight Tube	Chain

2.1.2 The Kit and chemicals Materials

The kits and chemicals materials which are used in this study listed in Table (2-2) with the reagent and their company and manufacture.

Table (2-2) Chemicals and Reagents

Kit	**Reagent**	company and manufacture
Kit of measurement T3	R1 (Human serum References) R2 (T3 Enzyme Reagent) R3 (T3\T4 conjugate) R4 (T3 Antibody Coated Plate) R5 (wash solution concentrate) R6 (substrate A) R7 (substrate B) R8 (stop solution) R9 (product Instruction)	Monobind Inc \USA
Kit of measurement T4	R1 (Human serum References) R2 (T4 Enzyme Reagent) R3 (T3\T4 conjugate) R4 (T4 Antibody Coated Plate) R5 (wash solution concentrate) R6 (substrate A) R7 (substrate B) R8 (stop solution) R9 (product Instruction)	Monobind Inc \USA

Kit of measurement TSH	R1 (Thyrotropin Calibrators) R2 (TSH Enzyme Reagent) R3 (Streptavidin Coated Plate) R4 (wash solution concentrate) R5 (Substrate A) R6 (Substrate B) R7 (Stop solution) R8 (Product Instruction)	Monobind Inc \USA
Kit of measurement of LH	R1 (LH Calibrators) R2 (LH Enzyme Reagent) R3 (Streptavidin Coated Plate) R4 (wash solution concentrate) R5 (Substrate A) R6 (Substrate B) R7 (Stop solution) R8 (Product Instruction)	Monobind Inc \USA
Kit of measurement FSH	R1 (FSH Calibrators) R2 (FSH Enzyme Reagent) R3 (Streptavidin Coated Plate) R4 (wash solution concentrate) R5 (Substrate A) R6 (Substrate B) R7 (Stop solution) R8 (Product Instruction)	Monobind Inc \USA

2.2 Methods and Biochemical tests

2.2.1 Patients and healthy controls:

This study includes 72 cases with age ranging from (10-50 years); (Group A) 42 patients with alopecia areata (27 male and 15 female) and (Group B) 30 healthy volunteers (7 male and 23 female) were selected as a control group. All samples were obtained from Dermatology outpatient clinic, Al-Ramadi Hospital, Al-Anbar Governorate, Iraq. during the period from December 2019 to march 2020. after informed agreement, following Helsinki guidelines.

All cases underwent to:

whole history taking concerning:

- onset and duration of Alopecia areata
- Family and Past history of Alopecia areata

Blood sampling:

A sample of 10 mL of vein blood was put into tube, installed for 10 minutes, then centrifuged for 5 minutes at 4000 rpm, and then used the serum for measurement of T3, T4, TSH hormones and LH, FSH hormones.

2.2.2 Measurement of Triiodothyronine (T3) :-

2.2.2.1 Principle

The major reagents required for a solid stage enzyme immunoassay comprise immobilized antibody, native antigen and enzyme-anligen conjugate. when mixing immobilized antibody, a serum containing the native antigen and enzyme-antigen conjugate, an opposition reaction results between the native antigen and the enzyme-antigen conjugate for a limited number of insolubulized binding sites. The reaction is explained by the following equation: [85].

$$\mathbf{Enz\ Ag + Ag + Ab_{c.w.}} \underset{Ka^*}{\overset{Ka}{\rightleftarrows}} \mathbf{AgAb_{c.w.} + Enz\ AgAb_{c.w}}$$

$Ab_{c.w}$ = Monospecific Immobilized Antibody (constant Quantity)

Ag = native Antigen (Variable Quantity)

Enz Ag = Enzyme- Antigen Conjugate (Constant Quantity)

$AgAb_{c.w.}$ = Antigen-Antibody Complex

Enz $AgAb_{c.w.}$ = Enzyme- Antigen Conjugate- Antibody Complex

Ka = Rate constant of Association

Ka* = Rate constant of Disassociation

K = Ka \ Ka* = Equilibrium Constant

After equilibrium is obtained, separate the antibody bound fraction is from unbound antigen by decantation or aspiration. The enzyme activity in the antibody-bound fraction is backward proportional to the native antigen concentration.

Many different serum references of known antigen concentration, a dose response curve can be created from Determining the antigen concentration of an unknown can be ascertained.

2.2.2.2 Reagent preparation:

1- Working Reagent A= T3-Enzyme conjugate solution:

We diluted T3 enzyme conjugation 1:11 with T3 / T4 conjugate buffer in a suitable container, e.g., we diluted 160 conjugate with 1,6 mL of buffer for 16 wells (a slight excess of solution is made). Use this detector within 24 hours to achieve maximum assay performance.

General Formula:

Amount of Buffer required = Number of wells * 0.1

Quantity of T3_ enzyme necessary = # of wells * 0.01

i.e. = 16 * 0.1 = 1.6 mL for Total T3/T4 conjugate

Buffer 16 * 0.01 = 0.16 mL for T3 enzyme conjugate.

2- Wash Buffer:

We diluted the washing compound to 1000 mL with distilled or deionized water in a suitable storage container. The diluted buffer was stored at 2-30 ° C for up to 60 days.

3- **Working Substrate Solution:**

The contents of the vial of amber labeled with solution "A" were poured into the transparent solution labeled "B.". The yellow cap has been placed on the transparent vial for easy identification. Mixed and labeled accordingly, store at 2-8 ° C.

2.2.2.3 Test procedure

Before proceeding with the assay, all reagents, serum references, and controls were brought to room temperature (20-27 ° C) and the test was performed by a skilled individual or trained professional.

1- Format the microplates wells for each serum reference, controls and patient specimen to be assayed in duplicate. any unused microwell strips back into the aluminum bag are replaced, sealed and stored at 2-8c .
2- Pipette 0.05 mL (50) of the appropriate serum reference. control or specimen into the assigned well .
3- Added 0.1 mL (100) of working reagent A, T3 enzyme reagent to all wells,
4- Gently rotate delicate dishes for 20-30 seconds to mix and coat.

5- Incubate 60 minutes at room temperature .

6- The contents of the microplate are removed by pouring or suction. If casting, the plate is dried with absorbent paper.

7- 350 wash buffer added. Pour (faucet stain) or douche. Repeat twice (2) for a total of 3 washes. An automatic or manual washer can be used. If a squeeze bottle is used, fill each well by squeezing the container (air bubbles should be avoided) to distribute the wash. Pour the wash and repeat two (2) more times.

8- Added 0.100 mL of working substrate solution to all wells. Always added reagent in the same order to minimize reaction time differences between wells.

" Do not shake the plate after substrate additional "

9- Incubate at room temperature for 15 minutes.

10- Added 0.05 mL of stop solution to each well and gently mix for 15-20 second. always added reagent in the same order to minimize reaction time differences between wells.

11- Read the absorbance in each well at 450 nm (using a reference wavelength of 620 to 630 nm to reduce well defects) in a microplate reader, and the results read within 30 minutes of adding the stop solution.

2.2.3 Measurement of Thyroxin (T4) :-

2.2.3.1 Principle

The major reagents required for a solid stage enzyme immunoassay comprise immobilized antibody, native antigen and enzyme anligen conjugate. when mixing immobilized antibody, a serum containing the native antigen and enzyme-antigen conjugate, an opposition reaction results between the native antigen and the enzyme-antigen conjugate for a limited number of insolubilized binding sites. The reaction is explained by the following equation: [86][87].

$$\mathbf{Enz\ Ag + Ag + Ab_{c.w}} \underset{Ka^*}{\overset{Ka}{\longleftrightarrow}} \mathbf{AgAb_{c.w.} + Enz\ AgAb_{c.w.}}$$

$Ab_{c.w}$ = Monospecific Immobilized Antibody (constant Quantity)

Ag = native Antigen (Variable Quantity)

Enz Ag = Enzyme- Antigen Conjugate (Constant Quantity)

$AgAb_{c.w.}$ = Antigen-Antibody Complex

Enz $AgAb_{c.w.}$ = Enzyme- Antigen Conjugate- Antibody Complex

Ka = Rate constant of Association

Ka* = Rate constant of Disassociation

K = Ka \ Ka* = Equilibrium Constant

After equilibrium is obtained, separate the antibody bound fraction is from unbound antigen by decantation or aspiraion. The enzyme activity in the antibody-bound fraction is backward proportional to the native antigen concentration. Many different serum references of known antigen concentration, a dose response curve can be created from Determine the antigen concentration of an unknown can be ascertained.

2.2.3.2 Reagent preparation:

1- Working Reagent A= T4- Enzyme conjugate solution:

T4 enzyme conjugation was diluted 1:11 with total T3 / T4 conjugate buffer in an appropriate container, e.g.; Dilute 160 mL of conjugated with 1.6 mL of buffer for 16 wells (a slight excess of solution is administered). This detector is used within 44 hours to achieve maximum assay performance at 2-8 ° C.

General Formula:

Amount of Buffer required = Number of wells * 0.1

Quantity of T4_ enzyme necessary = # of wells * 0.01

i.e. = 16 * 0.1 = 1.6 mL for Total T3/T4 conjugate

Buffer 16 * 0.01 = 0.16 mL for T4 enzyme conjugate.

2- Wash Buffer :

Dilute the washing compound to 1000 mL with distilled or deionized water in an appropriate storage container. Diluted buffer was stored at 2-30 ° C for up to 60 days.

3- Working Substrate Solution:

The contents of the vial of amber labeled Solution A were poured into the transparent solution labeled "B." Place the yellow cap on the clear vial for easy identification. The dough was mixed accordingly and stored at 2-8 ° C.

2.2.3.3 Test procedure

Before proceeding with the assay, all reagents, serum references, and controls were brought to room temperature (20-27 ° C) and the test was performed by a skilled individual or trained professional.

1- Format the microplates wells for each serum reference, controls and patient specimen to be assayed in duplicate. any unused microwell strips back into the aluminum bag are replaced, sealed and stored at 2-8c .
2- Pipette 0.05 mL (50) of the appropriate serum reference. control or specimen into the assigned well .
3- Added 0.1 mL (100) of working reagent A, T4 enzyme reagent to all wells,
4- Gently rotate delicate dishes for 20-30 seconds to mix and coat.

5- Incubate 60 minutes at room temperature.

6- The contents of the microplate are removed by pouring or suction. If casting, the plate is dried with absorbent paper

7- 350 wash buffer added. Pour (faucet stain) or douche. Repeat twice (2) for a total of 3 washes. An automatic or manual washer can be used. If a squeeze bottle is used, fill each well by squeezing the container (air bubbles should be avoided) to distribute the wash. Pour the wash and repeat two (2) more times.

8- Added 0.100 mL of working substrate solution to all wells. Always added reagent in the same order to minimize reaction time differences between wells.

" Do not shake the plate after substrate additional "

9- Incubate at room temperature for 15 minutes.

10- Added 0.05 mL of stop solution to each well and gently mix for 15-20 second. always added reagent in the same order to minimize reaction time differences between wells.

11- Read the absorbance in each well at 450 nm (using a reference wavelength of 620 to 630 nm to reduce well defects) in a microplate reader, and the results read within 30 minutes of adding the stop solution.

2.2.4 Measurement of Thyroid Stimulating Hormone (TSH) :-

2.2.4.1 Principle

The major reagents required for an immunoenzymorietric assay involved specificity antibodies and high affinity (enzyme immobilized and conjugated) with distinct and different epitope recognition, in excess, and original antigen. In this process, [88] the immobilization takes place through the check at the surface of a micro plate well during the interaction of streptavidin plated on the well and exogenously added biotinylated monoclonal anti-TSH antibody.[89] when mixing the enzyme- labeled antibody, monoclonal biotinylated antibody and a serum containing the native antigen, reaction results between the antibodies and the native antigen , without steric hindrance or competition , to form a double sandwich complex. The interaction is illustrated by the equation in the following below :[90]

$$^{Enz}\mathbf{Ab}_{(p)} + \mathbf{Ag}_{TSH} + {}^{Btn}\mathbf{Ab}_{(m)} \underset{Ka^*}{\overset{Ka}{\longleftrightarrow}} {}^{Enz}\mathbf{Ab}_{(p)}\text{-}\mathbf{Ag}_{TSH}\text{-}{}^{Btn}\mathbf{Ab}_{(m)}$$

$^{Btn}Ab_{(m)}$ = Biotinylated Monoclonal Antibody (Excess Quantity)

Ag_{TSH} = native Antigen (Variable Quantity)

$^{Enz}Ab_{(p)}$ = Enzyme polyclonal Antibody (Excess Quantity)

$^{Enz}Ab_{(p)}\text{-}Ag_{TSH}\text{-}{}^{Btn}Ab_{(m)}$ = Antigen-Antibody Sandwich Complex

Ka = Rate constant of Association

Ka* = Rate constant of Disassociation

In the same time, the complex is deposited to the well during the high affinity reaction of biotiny'ated antibody and streptavidin. This reaction is illustrated below:

$$^{Enz}Ab_{(p)}\text{-}Ag_{TSH}\text{-}{}^{Btn}Ab_{(m)} + Streptavidin_{cw} \longrightarrow \textbf{immobilized complex}$$

Streptavidinc.w. = Streptavidin immobolized on well
Immobilized complex = sandwich complex bound to the well surface

After equilibrium is attained, the antibody-bound fraction is separated from unbound antigen by aspiraton or decantation. The enzyme activity in the antibody bound fraction is directly proportional to the native antigen concentration. By utilizing several different serum references of known antigen values, a dose response curve can be generated from which the antigen concentration of an unknown can be ascertained.

2.2.4.2 Reagent preparation:

1- Wash Buffer:

The washing concentrates were diluted to 1000 mL with distilled or deionized water in an appropriate storage container. Store the diluted buffer at 2-30 ° C for up to 60 days.

2- Working Substrate Solution:

The contents of the vial of amber labeled with solution "A" were poured into a transparent bottle with "solution B." written on it. Place the yellow cap on the clear vial for easy identification. Mixed and labeled accordingly and stored at 2-8 ° C.

2.2.4.3 Test procedure

Before proceeding with the assay, all reagents, serum references, and controls were brought to room temperature (20-27 ° C) and the test was performed by a skilled individual or trained professional.

1- Format the microplates wells for each serum reference, controls and patient specimen to be assayed in duplicate. any unused microwell strips back into the aluminum bag are replaced, sealed and stored at 2-8c.
2- Pipette 0.05 mL (50) of the appropriate serum reference. control or specimen into the assigned well.
3- Added 0.1 mL (100) of working reagent A, T4 enzyme reagent to all wells.
4- Gently rotate delicate dishes for 20-30 seconds to mix and coat
5- Incubate 60 minutes at room temperature.
6- The contents of the microplate are removed by pouring or suction. If casting, the plate is dried with absorbent paper

7- 350 wash buffer added. Pour (faucet stain) or douche. Repeat twice (2) for a total of 3 washes. An automatic or manual washer can be used. If a squeeze bottle is used, fill each well by squeezing the container (air bubbles should be avoided) to distribute the wash. Pour the wash and repeat two (2) more times.

8- Added 0.100 mL of working substrate solution to all wells. Always added reagent in the same order to minimize reaction time differences between wells .

" Do not shake the plate after substrate additional "

9- Incubate at room temperature for 15 minutes .

10- Added 0.05 mL of stop solution to each well and gently mix for 15-20 second . always added reagent in the same order to minimize reaction time differences between wells.

11- Read the absorbance in each well at 450 nm (using a reference wavelength of 620 to 630 nm to reduce well defects) in a microplate reader, and the results read within 30 minutes of adding the stop solution.

2.2.5 Measurement of Luteinizing Hormones (LH): -

2.2.5.1 Principle

The major reagents required for an immunoenzymorietric assay involve specificity antibodies and high affinity (enzyme immobilized and conjugated) with distinct and different epitope recognition, in excess, and original antigen.[91]

In this process, the immobilization takes place through the check at the surface of a micro plate well during the interaction of streptavidin plated on the well and exogenously added biotinylated monoclonal anti-LH antibody.[92]

when mixing the enzyme- labeled antibody, monoclonal biotinylated antibody and a serum containing the native antigen, reaction results between the antibodies and the native antigen, without steric hindrance or competition, to form a souble sandwich complex. The interaction is illustrated by the equation in the following below:

$$^{Enz}\mathbf{Ab}_{(p)} + \mathbf{Ag}_{LH} + {}^{Btn}\mathbf{Ab}_{(m)} \underset{Ka^*}{\overset{Ka}{\longleftrightarrow}} {}^{Enz}\mathbf{Ab}_{(p)}\text{-}\mathbf{Ag}_{LH}\text{-}{}^{Btn}\mathbf{Ab}_{(m)}$$

$^{Btn}Ab_{(m)}$ = Biotinylated Monoclonal Antibody (Excess Quantity)

Ag_{LH} = native Antigen (Variable Quantity)

$^{Enz}Ab_{(p)}$ = Enzyme polyclonal Antibody (Excess Quantity)

$^{Enz}Ab_{(p)}\text{-}Ag_{LH}\text{-}{}^{Btn}Ab_{(m)}$ = Antigen-Antibody Sandwich Complex

Ka = Rate constant of Association

Ka* = Rate constant of Disassociation

In the same time, the complex is deposited to the well during the high affinity reaction of biotiny'ated antibody and streptavidin. This reaction is illustrated below:

$$^{Enz}\mathbf{Ab}_{(p)}\text{-}\mathbf{Ag}_{TSH}\text{-}{}^{Btn}\mathbf{Ab}_{(m)} + \mathbf{Streptavidin}_{cw} \longrightarrow \mathbf{immobilized\ complex}$$

Streptavidinc.w. = Streptavidin immobolized on well

Immobilized complex = sandwich complex bound to the well surface

After equilibrium is attained, the antibody-bound fraction is separated from unbound antigen by aspiraton or decantation . The enzyme activity in the antibody bound fraction is directly proportional to the native antigen concentration. By utilizing several different serum references of known antigen values, a dose response curve can be generated from which the antigen concentration of an unknown can be ascertained.

2.2.5.2 Reagent preparation:

1- Wash Buffer:

We diluted the washing compound to 1000 mL with distilled or deionized water in a suitable storage container. Diluted buffer was stored at 2-30 ° C for up to 60 days.

2- Working Substrate Solution :

The contents of the vial of amber labeled with solution "A" were poured into a transparent bottle with "solution B." written on it. Place the yellow cap on the clear vial for easy identification. It was mixed and placed on the label accordingly kept at 2-8 ° C.

2.2.5.3 Test procedure

Before proceeding with the assay, all reagents, serum references, and controls were brought to room temperature (20-27 ° C) and the test was performed by a skilled individual or trained professional.

1- Format the microplates wells for each serum reference, controls and patient specimen to be assayed in duplicate. any unused microwell strips back into the aluminum bag are replaced, sealed and stored at 2-8c .
2- Pipette 0.05 mL (50) of the appropriate serum reference. control or specimen into the assigned well .
3- Added 0.1 mL (100) of working reagent A, LH enzyme reagent to all wells,
4- Gently rotate delicate dishes for 20-30 seconds to mix and coat
5- Incubate 60 minutes at room temperature .
6- The contents of the microplate are removed by pouring or suction. If casting, the plate is dried with absorbent paper
7- 350 wash buffer added. Pour (faucet stain) or douche. Repeat twice (2) for a total of 3 washes. An automatic or manual washer can be used. If a squeeze bottle is used, fill each well by squeezing the container (air bubbles should be avoided) to distribute the wash. Pour the wash and repeat two (2) more times.
8- Added 0.100 mL of working substrate solution to all wells. Always added reagent in the same order to minimize reaction time differences between wells .

" Do not shake the plate after substrate additional "

9- Incubate at room temperature for 15 minutes.
10- Added 0.05 mL of stop solution to each well and gently mix for 15-20 second . always added reagent in the same order to minimize reaction time differences between wells.

11- Read the absorbance in each well at 450 nm (using a reference wavelength of 620 to 630 nm to reduce well defects) in a microplate reader, and the results read within 30 minutes of adding the stop solution.

2.2.6 Measurement of Follicle Stimulating Hormone (FSH) :-

2.2.6.1 Principle

The major reagents required for an immunoenzymorietric assay involved specificity antibodies and high affinity (enzyme immobilized and conjugated) with distinct and different epitope recognition, in excess, and original antigen. In this process, the immobilization took place through the check at the surface of a micro plate well during the interaction of streptavidin plated on the well and exogenously added biotinylated monoclonal anti- FSH antibody. when mixing the enzyme- labeled antibody, monoclonal biotinylated antibody and a serum containing the native antigen, reaction results between the antibodies and the native antigen , without steric hindrance or competition , to form a souble sandwich complex. The interaction is illustrated by the equation in the following below : [93][94]

$$^{Enz}\mathbf{Ab}_{(p)} + \mathbf{Ag}_{FSH} + {}^{Btn}\mathbf{Ab}_{(m)} \underset{Ka^*}{\overset{Ka}{\longleftrightarrow}} {}^{Enz}\mathbf{Ab}_{(p)} - \mathbf{Ag}_{FSH} - {}^{Btn}\mathbf{Ab}_{(m)}$$

$^{Btn}Ab_{(m)}$ = Biotinylated Monoclonal Antibody (Excess Quantity)

Ag_{FSH} = native Antigen (Variable Quantity)

$^{Enz}Ab_{(p)}$ = Enzyme polyclonal Antibody (Excess Quantity)

$^{Enz}Ab_{(p)}$ -Ag_{FSH} - $^{Btn}Ab_{(m)}$ = Antigen-Antibody Sandwich Complex

Ka = Rate constant of Association

Ka* = Rate constant of Disassociation

In the same time, the complex is deposited to the well during the high affinity reaction of biotiny'ated antibody and streptavidin. This reaction is illustrated below:

$$^{Enz}Ab_{(p)} - Ag_{FSH} - {}^{Btn}Ab_{(m)} + Streptavidin_{cw} \longrightarrow \text{immobilized complex}$$

Streptavidinc.w. = Streptavidin immobolized on well

Immobilized complex = sandwich complex bound to the well surface

After equilibrium is attained, the antibody-bound fraction is separated from unbound antigen by aspiration or decantation. The enzyme activity in the antibody bound fraction is directly proportional to the native antigen concentration. By utilizing several different serum references of known antigen values, a dose response curve can be generated from which the antigen concentration of an unknown can be ascertained.

2.2.6.2 Reagent preparation:

1- Wash Buffer:

We diluted the washing compound to 1000 mL with distilled or deionized water in a suitable storage container. Diluted buffer was stored at 2-30 ° C for up to 60 days.

2- Working Substrate Solution:

The contents of the vial of amber labeled with solution "A" were poured into a transparent bottle with "solution B." written on it. Place the yellow cap on the clear vial for easy identification. It was mixed and placed on the label accordingly kept at 2-8 ° C.

2.2.5.3 Test procedure

Before proceeding with the assay, all reagents, serum references, and controls were brought to room temperature (20-27 ° C) and the test was performed by a skilled individual or trained professional.

1- Format the microplates wells for each serum reference, controls and patient specimen to be assayed in duplicate. any unused microwell strips back into the aluminum bag are replaced, sealed and stored at 2-8c .
2- Pipette 0.05 mL (50) of the appropriate serum reference. control or specimen into the assigned well.
3- Added 0.1 mL (100) of working reagent A, TSH enzyme reagent to all wells,
4- Gently rotate delicate dishes for 20-30 seconds to mix and coat
5- Incubate 60 minutes at room temperature.
6- The contents of the microplate are removed by pouring or suction. If casting, the plate is dried with absorbent paper

7- 350 wash buffer added. Pour (faucet stain) or douche. Repeat twice (2) for a total of 3 washes. An automatic or manual washer can be used. If a squeeze bottle is used, fill each well by squeezing the container (air bubbles should be avoided) to distribute the wash. Pour the wash and repeat two (2) more times.

8- Added 0.100 mL of working substrate solution to all wells. Always added reagent in the same order to minimize reaction time differences between wells .

" Do not shake the plate after substrate additional "

9- Incubate at room temperature for 15 minutes.

10- Added 0.05 mL of stop solution to each well and gently mix for 15-20 second. always added reagent in the same order to minimize reaction time differences between wells.

11- Read the absorbance in each well at 450 nm (using a reference wavelength of 620 to 630 nm to reduce well defects) in a microplate reader, and the results read within 30 minutes of adding the stop solution.

Chapter Three

Results and Discussion

Results and Discussion

3.1 The Results

This study explains the relationship between Alopecia Areata and Some of Hormones. It included 72 samples, 42 patients with Alopecia areata (27male–15female), 30 healthy sample as control (7male –23female). AA patients and control groups were (10 – 50) years.

3.2 Alopecia Areata with Triiodothyronine Hormone (T3) and Thyroxin Hormone (T4)

- Triiodothyronine Hormone **(T3)**

Table (3-1) Distribution of Age for Alopecia areata patient and control group. There were significant differences between the difference Age of patients with Alopecia Areate and control group. There was decrease in level of Hormone T3 in Alopecia Areata patients was **(0.85 ng/dl),SD= 0.426** in group (10_22Year) compare with control group which was **(1.77),** SD= **0.418** and significant at (P<0.01)**.**

group (23_ 37year) was **(0.78 ng/dl), SD= 0.278** while control group was (**1.29 ng/dl)** when SD= **0.298** and significant at (P< 0.01)**.**

when group (38– 50 year) was **(0.72 ng/dl), SD=0.413** compare with control group was **(1.37 ng/dl),** SD= **0.491** significant at (P< 0.05)**.**

Table (3-1) Distribution of Age for Alopecia areata patient and control group for Hormone T3

T3 ng/ml	**Healthy Group**			**patients**		
parameters	**N**	**Mean**	**SD**	**N**	**Mean**	**SD**
10-22 year	11	1.77	0.418	15	0.85	0.426 b
23-37 year	16	1.29	0.298	20	0.78	0.278 b
38-50 year	3	1.37	0.491	7	0.72	0.413 a
Total	30			42		

Table (3-2) Distribution of Gender for Alopecia areata patient and control group. There was significant decrease in concentration of T3 hormone, where the level of T3 was (**0.852 ng/dl**) in male compared to control (**1.764 ng/dl**) , while in female was (**0.738 ng/dl**) compared to control (**1.604 ng/dl**) meaning there are significant differences between AA patients and control, but, in male there was a highly significant increase with female .

Table (3-2) Distribution of Gender for Alopecia areata patient and control

T3 ng/ml	**Healthy Group**			**Patients**		
parameters	**N**	**Mean**	**SD**	**N**	**Mean**	**SD**
Male	**7**	**1.764**	**0.142**	**27**	**0.852**	**0.132**
Female	**23**	**1.604**	**0.085**	**15**	**0.738**	**0.076**
Total	30			42		

- Thyroxin Hormone **(T4)**

Table (3-3) Distribution of Age for Alopecia areata patient and control group. There were significant differences between the difference Age of patients with Alopecia Areate and control group. There was decrease in levels of Hormone T4 in Alopecia Areata patients was **(7.14 ng/dl)**, **SD= 1.186** in group (10_22 Year) compare with control group which was **(7.46 ng/dl),** SD= **0.91** and significant at (P<0.05)**.**

when group (22_ 37 year) was **(7.33 ng/dl), SD=1.21** compare to control group which was (**7.79 ng/dl)** when SD= **1.03** and significant at (P<0.01)**.**

when group (38– 50 year) was **(6.975 ng/dl), SD=1.19** compare with control group which was **(7.38 ng/dl)** SD= **1.18** and significant at (P<0.01)**.**

Table (3-3) Distribution of Age for Alopecia areata patient and control group for Hormone T4.

T4 ng/ml	**Healthy Group**			**Patients**		
parameters	**N**	**Mean**	**SD**	**N**	**Mean**	**SD**
10-22 year	11	7.46	0.91	15	7.14	1.186 a
23-37 year	16	7.79	1.03	20	7.336	1.21 b
38-50 year	3	7.38	1.18	7	6.975	1.199 b
Total	30			42		

Table (3-4) Distribution of Gender for Alopecia areata patients and control group. There was significant decrease in Average of T4 hormone, where hormone T4 average was (**5.676 ng/dl**) in male compared to control (**9.324 ng/dl**), while in female was (**4.918 ng/dl**) compared to control (**7.894 ng/dl**) meaning there are significant differences between AA patients and control. but, in male there were a highly significant increase compared to female.

Table (3-4) Distribution of Gender for Alopecia areata patient and control group of hormone T4 .

T4 ng/ml	**Healthy Group**			**Patients**		
parameters	**N**	**Mean**	**SD**	**N**	**Mean**	**SD**
Male	7	9.32	0.551	27	5.676	0.496
Female	23	7.89	0.480	15	4.91	0.504
Total	30			42		

That differences in level of Hormones T3 and T4 in Alopecia Areata patients It may be because that Alopecia Areata occurs in association with other autoimmune disorders. Thyroid diseases were observed to be the most common comorbid disorder in AA patients, including hypothyroidism, Hashimoto's thyroiditis, Graves' disease and simple goiter [95].

In our study, we find significant differences between cases and controls regarding levels of T3 and T4. T3 and T4 were decrease in patients with AA compare to control group.

our results agreement with Kakourou et al.[81] who informed that there were 5% of patients with concomitant AA and thyroid disorder. Those have had subclinical hypothyroidism of autoimmune etiology that was revealed at the time of investigation.

Also, Kasumagić-Halilović [84] founded that thyroid functional abnormalities in the form of hypothyrodism were found in 11.4% of AA patients.

As well , Seyrafi et al., [26] found thyroid function abnormalities in form of hypothyroidism in 8.9% of the studied AA cases.

Also Andrzej Lewihski, et al.[96] agreement with our study result. They were performed " The plasma concentrations of (T3) and (T4) were determined by radioimmunoassay and thyroid-stimulating hormone (TSH) concentration was measured by the imrnunoenzymatic method (Abbott Laboratories DiagnosticDiv., Abbott Park, Ill.), thyroid abnormalities was found in patients with AA (78%) than in the control group (33%).

3.4 Alopecia Areata with Thyroid Stimulate Hormone (TSH)

Table (3-5) Distribution of Age for Alopecia areata patient and control group. There were significant differences between the difference Age of patients with Alopecia Areate and control group. There was increase in levels of Hormone TSH in Alopecia Areata patients was **(6.31** µl U/ml**), SD= 2.19** in group (10_22 Year) compare with control group which was (**3.41** µl U/ml**), SD= 1.34** and significant at ($P<0.01$)**.**

in group (23_ 37 year) was **(6.29**µl U/ml**), SD=1.69** while control group was (**3.76** µl U/ml**), SD= 1.81** and significant at (P<0.01)**.**
when group (38– 50 year) was **(6.4** µl U/ml**), SD=2.77** compare with control group which was **(2.89** µl U/ml**)** SD= **1.93** and significant at (P<0.001).

Table (3-5) Distribution of Age for Alopecia areata patient and control group of hormone TSH .

TSH µl U / ml	**Healthy Group**			**Patients**		
parameters	**N**	**Mean**	**SD**	**N**	**Mean**	**SD**
10-22 year	11	3.41	1.34	15	6.31	2.19 b
23-37 year	16	3.76	1.81	20	6.29	1.69 b
38-50 year	3	2.89	1.93	7	6.4	2.77 c
Total	30			42		

Table (3-6) Distribution of Gender for Alopecia areata patients and control group. There were significant decrease in concentration of TSH hormone, where the concentration of TSH was (**7.134** µl U/ml) in male compared to control (**5.734** µl U/ml) , while in female was (**6.248** µl U/ml) compared to control (**5.174** µl U/ml) meaning there are significant differences between AA patients and control , but, in male there was a highly significant increase than female

Table (3-6) Distribution of Age for Alopecia areata patient and control group of hormone TSH .

TSH μl U / ml	**Healthy Group**			**Patients**		
parameters	**N**	**Mean**	**SD**	**N**	**Mean**	**SD**
Male	7	5.734	0.468	27	7.134	0.640
Female	23	5.174	0.692	15	6.248	0.348
Total	30			42		

That differences in concentration of Hormone TSH in Alopecia Areata patients It may be cause that the main associations is with thyroid abnormalities. The incidence of thyroid disease has varied from 8 to 28% in patients with AA [95]. Milgraum et al. also found an apparent association between thyroid disease and AA [84]. Subsequently Lewinski et al confirmed the frequent co-existence of AA and thyroid abnormalities [96].

In our study, we found significant differences between cases and controls regarding levels of TSH. our results agreement with Kakourou et al. [81] who informed that in evaluation of 157 patients with AA, there were 5% of patients with concomitant AA and thyroid disorder. Those have had subclinical hypothyroidism of autoimmune etiology that was revealed at the time of investigation.

Also, Kasumagić-Halilović [77] founded that thyroid functional abnormalities in the form of hypothyrodism were found in 11.4% of AA patients.

As well , Seyrafi et al.[26], found thyroid function abnormalities in form of hypothyroidism in 8.9% of the studied AA cases.

Also Ola A Bakry, Mohamed A Basha et al [78]. agree with our study result. They explained the effects of hypothyroidism on hair include changes in hair texture and scalp alopecia by delayed or failure of resumption of anagen hair due to decreased metabolic rate which leads to loss of hair without replacement as well as increased telogen hair counts (club hair) before shedding. Also, it is likely that the magnitude of effect of thyroid hormone on hair growth is variable and its expression may be conditioned by local factors and other hormonal influences. Bakry and colleagues reported significantly higher levels of TSH .

Also, in one of the largest sample study, Park and colleagues [97], evaluated 1408 patients and observedan increased incidence of thyroid dysfunction and thyroid autoimmunity in AA patients. particularly in those having severe AA.

3.5 Alopecia Areata with Hormone (LH)

Table (3-8) Distribution of Age for Alopecia areata patient and control group. There were significant differences between the difference Age of patients with Alopecia Areate and control group. There was increase in levels of Hormone LH in Alopecia Areata patients was **(6.81** µl U/ml**), SD= 1.60** in group (10_22 Year) compare with control group which was (**3.41** µl U/ml**), SD**= **1.91** and significant at (P<0.05).

When group (23_ 37 year) was **(6.97** µl U/ml**), SD=1.91** while control group was (**4.43** µl U/ml**),** SD= **1.83** and significant at (P<0.01)**.**
when group (38– 50 year) was **(6.82** µl U/ml**),SD= 2.43** compare with control group (38_50 Year) was **(4.61)** SD= **2.14** and significant at (P<0.001)

Table (3-7) Distribution of Age for Alopecia areata patient and control group of hormone LH .

LH µl U / ml	**Healthy Group**			**Patients**		
parameters	**N**	**Mean**	**SD**	**N**	**Mean**	**SD**
10-22 year	11	3.41	1.91	15	6.81	1.60 a
23-37 year	16	4.43	1.83	20	6.97	1.91 b
38-50 year	3	4.61	2.14	7	6.82	2.43 c
Total	30			42		

Table (3-8) Distribution of gender for Alopecia areata patient and control group of LH.

LH µl U / ml	**Healthy Group**			**Patients**		
parameters	**N**	**Mean**	**SD**	**N**	**Mean**	**SD**
Male	7	6.42	0.337	27	7.754	0.550
Female	23	5.746	0.485	15	6.868	0.518
Total	30			42		

That differences in concentration of Hormone LH in Alopecia Areata patients, informed that Hormonal disorders specific to female may be the cause, the most important of which is Polycystic Ovaries, known for short as P.C.O, which is very common, especially in the Middle East, and one of the main symptoms of hair loss. Polycystic ovary syndrome (PCOS) is the hallmark of androgen excess disorders; In the case of PCO, the pituitary gland secretes high amounts of the hormone (LH) and the ovary also secretes high amounts of the male hormone androgen and testosterone, which affect in hair and cause hair lose. The intense secretion of the hormone androgen can cause hair loss [98][99].

We found significant differences between cases and controls regarding levels of LH. our results agreement with Schmidt J.B. [102] who informed that a broad range of hormones was determined in males and females with hair loss. The androgens testosterone, androstenedione, and sex hormone binding globulin were evaluated in 65 male and 46 female patients. Besides the hypophyseal hormones LH, FSH, were investigated. Hormone levels were compared with those of 58 age-matched male and 45 female controls. In 38 of the 46 female patients, Our findings showed a significant elevation of hormones level in both male and female patients compared to controls.

Also, our result agree with Geraldine Cheyana Ranasinghe et al [100]. who reported a significant association between AA and hyperandrogenism, Based on study they provide to follow when androgen excess is of question for patients presentation. Who were diagnosed with AA at the Department of Dermatology at the Cleveland Clinic Foundation the diagnosis of AA and

patients were categorized into one of four subtypes: patchy alopecia, alopecia ophiasis, alopecia totalis, alopecia universalis. A total of 220 patients met the inclusion criteria for the study (abnormal hormones levels, and/or history of ovarian dysfunction, and/or clinical evidence of androgen excess). And they evaluated laboratory values for progesterone, FSH, LH, fasting glucose, insulin, prolactin, serum ferritin, testosterone free and total testosterone. They dysfunction identified in 220 patients with AA and all subtypes 42.5% (n=96) ($p<0.001$) Androgen excess /PCOS was the most common and (17.3%) with hormonal abnormalities levels Testosterone free and total Testosterone, FSH and LH.

3.6 Alopecia Areata with Hormone (FSH)

Table (3-9) Distribution of Age for Alopecia areata patient and control group. There were significant differences between the difference Age of patients with Alopecia Areate and control group. There was increase in levels of Hormone LH in Alopecia Areata patients was **(6.89** µl U/ml**), SD= 2.09** in group (10_22 Year) compare with control group which was (**4.5** µl U/ml**), SD**= **2.04** and significant at ($P<0.001$).

When group (23_ 37 year) was **(7.41** µl U/ml**), SD=1.97** while control group was (**4.77** µl U/ml**),** SD= **1.089** and significant at ($P<0.001$)**.**

when group (38– 50 year) was **(7.88** µl U/ml**),SD= 1.64** compare with control group (38_50 Year) was **(4.91)** SD= **1.213** and significant at ($P<0.01$)

Table (3-9) Distribution of Age for Alopecia areata patient and control group of FSH .

FSH μl U / ml	**Healthy Group**			**Patients**		
parameters	**N**	**Mean**	**SD**	**N**	**Mean**	**SD**
10-22 year	11	4.5	2.04	15	6.89	2.09 c
23-37 year	16	4.77	1.089	20	7.41	1.97 c
38-50 year	3	4.91	1.213	7	7.88	1.64 b
Total	30			42		

Table (3-10) and Distribution of Gender for Alopecia areata patients and control group. There were significant increase in concentration of FSH hormone, where the concentration of FSH was (**8.082**),**SD= 0.730** in male compared to control (**6.218**),**SD=0.404**.

while in female was (**8.844**), **SD= 0.586** compared to control (**6.854**), **SD= 0.413** meaning there are significant differences between AA patients and control , but, in female there was a highly significant than male .

Table (3-10) Distribution of Gander for Alopecia areata patient and control group of hormone FSH.

FSH μl U / ml	**Healthy Group**			**Patients**		
parameters	**N**	**Mean**	**SD**	**N**	**Mean**	**SD**
Male	7	6.218	0.404	27	8.082	0.730
Female	23	6.854	0.413	15	8.844	0.586
Total	30			42		

That differences in concentration of Hormone FSH in Alopecia Areata patients It may be cause that Gonadotropin hormones are considered one of the most important hormones responsible for hair health, as FSH or the stimulating hormone is secreted, in addition to the luteinizing hormone LH through the gonads, which play a large role directly in the secretion of estrogen and testosterone in female and male, these hormones interact with the hormone progesterone and affect the How hair grows, and gonadotropins play an indirect role in hair growth because it works on the production of all the hormones we mentioned, and directly affects hair growth.

Also, Testosterone hormone plays a major role in hair loss and hormonal baldness, and this is due to the transformation of the testosterone hormone into the dihydrotestron hormone (DHT) , which is circulating in the blood until it reaches the hair follicles and is linked to them and causes them to weaken, causing hair weakening and falling, and with time the hair follicle dries up And it stops producing hair [101]

we found significant differences between cases and controls regarding levels of LH. our results agreement with Schmidt J.B. [102] who informed that A broad range of hormones was determined in males and females with hair loss. The androgens testosterone, androstenedione, and sex hormone binding globulin were evaluated in 65 male and 46 female patients. Besides the hypophyseal hormones LH, FSH [102], were investigated. Hormone levels were compared with those of 58 age-matched male and 45 female controls. In 38 of the 46 female patients. Our findings showed a significant elevation of hormones level in both male and female patients compared to controls.

Also, our results agree with Geraldine Cheyana Ranasinghe et al. [100] who reported a significant association between AA and hyperandrogenism, Based on study they provide to follow when androgen excess is of question for patients presentation. Who were diagnosed with AA at the Department of Dermatology at the Cleveland Clinic Foundation the diagnosis of AA and patients were categorized into one of four subtypes: patchy alopecia, alopecia ophiasis, alopecia totalis, alopecia universalis. A total of 220 patients met the inclusion criteria for the study (abnormal hormones levels, and/or history of ovarian dysfunction, and/or clinical evidence of androgen excess). And they evaluated laboratory values for progesterone, FSH, LH, fasting glucose, insulin, prolactin, serum ferritin, testosterone free and total testosterone. They dysfunction identified in 220 patients with AA and all subtypes 42.5% (n=96) ($p<0.001$) Androgen excess /PCOS was the most common and (17.3%) with hormonal abnormalities levels Testosterone free and total Testosterone, FSH and LH.

Conclusions:

From this study we can support the idea of association between some Hormones and Alopecia Areata.

- ❑ We found different significant correlation between levels of Thyroid hormones in control group and Alopecia Areata patients;
- T3 and T4 decreased significantly. Decrease of T3 was more pronounced and effective than decrease of T4. On the other hand, a significant increase in serum TSH levels was observed in alopecia areata patients compared to the control group. The effect of T3 and TSH in AA patients is more than T4 compare to control group.
- ❑ Also, we found significant different correlation between levels of FSH and LH hormones in control group and Alopecia Areata patients.
- LH and FSH was increase significantly in Alopecia areata patients compare to control.

Recommendations:

- Follow the current study with same parameters with more samples number to confirm the current results.
- Study the effect and association of other hormones with alopecia areata such as hormones: Cortisol, testosterone, and androgen ...etc.
- It’s preferable to screen of Hormones every patient with Alopecia Areata even in absence of manifestations suggestive of Hormone disorders and associated diseases.

References:

1. Renert-Yuval Y, Guttman-Yassky E. A novel therapeutic paradigm for patients with extensive alopecia areata. *Expert Opin Biol Ther*. 2016;16(8):1005-1014.
2. Gilhar A, Schrum AG, Etzioni A, Waldmann H, Paus R. Alopecia areata: animal models illuminate autoimmune pathogenesis and novel immunotherapeutic strategies. *Autoimmun Rev*. 2016;15(7):726-735.
3. Gilhar A, Paus R, Kalish RS. Lymphocytes, neuropeptides, and genes involved in alopecia areata. *J Clin Invest*. 2007;117(8):2019-2027.
4. Xiao F-L, Yang S, Liu J-B, et al. The epidemiology of childhood alopecia areata in China: a study of 226 patients. *Pediatr Dermatol*. 2006;23(1):13-18.
5. Pratt CH, King LE, Messenger AG, Christiano AM, Sundberg JP. Alopecia areata. *Nat Rev Dis Prim*. 2017;3(1):1-17.
6. Miller P. *Alopecia Areata*. Cavendish Square Publishing, LLC; 2015.
7. Simakou T, Butcher JP, Reid S, Henriquez FL. Alopecia areata: A multifactorial autoimmune condition. *J Autoimmun*. 2019;98:74-85.
8. Gilhar A, Etzioni A, Paus R. Alopecia areata. *N Engl J Med*. 2012;366(16):1515-1525.
9. Dainichi T, Kabashima K. Alopecia areata: What's new in epidemiology, pathogenesis, diagnosis, and therapeutic options? *J Dermatol Sci*. 2017;86(1):3-12.

10. Migacz-Gruszka K, Branicki W, Obtulowicz A, Pirowska M, Gruszka K, Wojas-Pelc A. What's new in the pathophysiology of alopecia areata? the possible contribution of skin and gut microbiome in the pathogenesis of alopecia--Big opportunities, big challenges, and novel perspectives. *Int J Trichology*. 2019;11(5):185.
11. Lee S, Paik SH, Kim H-J, et al. Exomic sequencing of immune-related genes reveals novel candidate variants associated with alopecia universalis. *PLoS One*. 2013;8(1):e53613.
12. Cho HH, Jo SJ, Paik SH, et al. Clinical characteristics and prognostic factors in early-onset alopecia totalis and alopecia universalis. *J Korean Med Sci*. 2012;27(7):799-802.
13. Bertolini M, McElwee K, Gilhar A, Bulfone-Paus S, Paus R. Hair follicle immune privilege and its collapse in alopecia areata. *Exp Dermatol*. 2020;29(8):703-725.
14. Alsaleh QA, Nanda A, AL-HASAWI F, EL-KASHLAN M. Concurrent appearance of alopecia areata in siblings. *Pediatr Dermatol*. 1995;12(3):285-286.
15. Muller SA, Winkelmann RK. Alopecia areata: an evaluation of 736 patients. *Arch Dermatol*. 1963;88(3):290-297.
16. Shellow WVR, EDWARDS JE, KOO JYM. Profile of alopecia areata: a questionnaire analysis of patient and family. *Int J Dermatol*. 1992;31(3):186-189.
17. Baker GH. Invited review: Psychological factors and immunity. *J Psychosom Res*. Published online 1987.

18. Colón EA, Popkin MK, Callies AL, Dessert NJ, Hordinsky MK. Lifetime prevalence of psychiatric disorders in patients with alopecia areata. *Compr Psychiatry*. 1991;32(3):245-251.
19. Messenger AG, McKillop J, Farrant P, et al. British Association of Dermatologists' guidelines for the management of alopecia areata 2012. *Br J Dermatol*. 2012;166(5):916-926.
20. Alkhalifah A, Alsantali A, Wang E, McElwee KJ, Shapiro J. Alopecia areata update: part I. Clinical picture, histopathology, and pathogenesis. *J Am Acad Dermatol*. 2010;62(2):177-188.
21. Price VH. Double-blind, placebo-controlled evaluation of topical minoxidil in extensive alopecia areata. *J Am Acad Dermatol*. 1987;16(3):730-736.
22. Kyriakis KP, Paltatzidou K, Kosma E, Sofouri E, Tadros A, Rachioti E. Alopecia areata prevalence by gender and age. *J Eur Acad Dermatology Venereol*. 2009;23(5):572-573.
23. Ruiz-Doblado S, Carrizosa A, Garc\'\ia-Hernández MJ. Alopecia areata: psychiatric comorbidity and adjustment to illness. *Int J Dermatol*. 2003;42(6):434-437.
24. Eckert J, Church RE, Ebling FJ. The pathogenesis of alopecia areata. *Br J Dermatol*. 1968;80(4):203-210.
25. Messenger AG, Slater DN, Bleehen SS. Alopecia areata: alterations in the hair growth cycle and correlation with the follicular pathology. *Br J Dermatol*. 1986;114(3):337-347.

26. Seyrafi H, Akhiani M, Abbasi H, Mirpour S, Gholamrezanezhad A. Evaluation of the profile of alopecia areata and the prevalence of thyroid function test abnormalities and serum autoantibodies in Iranian patients. *BMC Dermatol*. 2005;5(1):1-5.
27. Haller-Kikkatalo K, Salumets A, Uibo R. Review on autoimmune reactions in female infertility: antibodies to follicle stimulating hormone. *Clin Dev Immunol*. 2012;2012.
28. Webster R, Fahie-Wilson M, Barker P, Chatterjee VK, Halsall DJ. Immunoglobulin interference in serum follicle-stimulating hormone assays: autoimmune and heterophilic antibody interference. *Ann Clin Biochem*. 2010;47(4):386-389.
29. Goodman HM. Discovery of the luteinizing hormone of the anterior pituitary gland. *Am J Physiol Metab*. 2004;287(5):E818--E819.
30. Mullin GE, Eastern JS. Cutaneous signs of thyroid disease. *Am Fam Physician*. 1986;34(4):93-98.
31. Mullin GE, Eastern JS. Cutaneous consequences of accelerated thyroid function. *Cutis*. 1986;37(2):109-114.
32. Safer JD, Crawford TM, Fraser LM, et al. Thyroid hormone action on skin: diverging effects of topical versus intraperitoneal administration. *Thyroid*. 2003;13(2):159-165.
33. Holt PJA. In vitro responses of the epidermis to triiodothyronine. *J Invest Dermatol*. 1978;71(3).
34. Leonhardt JM, Heymann WR. Thyroid disease and the skin. *Dermatol Clin*. 2002;20(3):473-481.

35. Pazos-Moura CC, Moura EG, Breitenbach MMD, Bouskela E. Nailfold capillaroscopy in hypothyroidism and hyperthyroidism: blood flow velocity during rest and postocclusive reactive hyperemia. *Angiology*. 1998;49(6):471-476.
36. Silva JE. The thermogenic effect of thyroid hormone and its clinical implications. *Ann Intern Med*. 2003;139(3):205-213.
37. P Delitala A, Fanciulli G, M Pes G, Maioli M, Delitala G. Thyroid hormones, metabolic syndrome and its components. *Endocrine, Metab Immune Disord Targets (Formerly Curr Drug Targets-Immune, Endocr Metab Disord*. 2017;17(1):56-62.
38. Christianson HB. Cutaneous manifestations of hypothyroidism including purpura and ecchymoses. *Cutis*. 1976;17(1):45.
39. Safer JD. Thyroid hormone action on skin. *Dermatoendocrinol*. 2011;3(3):211-215.
40. Fistarol SK, Itin PH. Disorders of pigmentation. *JDDG J der Dtsch Dermatologischen Gesellschaft*. 2010;8(3):187-202.
41. Molina PE, Education M-H. *Endocrine Physiology*. Lange Medical Books/McGraw-Hill New York; 2006.
42. Nanda S, De Bedout V, Miteva M. Alopecia as a systemic disease. *Clin Dermatol*. 2019;37(6):618-628.
43. Duncan FJ, Silva KA, Johnson CJ, et al. Endogenous retinoids in the pathogenesis of alopecia areata. *J Invest Dermatol*. 2013;133(2):334-343.

44. Olsen EA, Hordinsky MK, Price VH, et al. Alopecia areata investigational assessment guidelines--Part II. *J Am Acad Dermatol*. 2004;51(3):440-447.
45. Rivitti EA. Alopecia areata: a revision and update Alopecia areata: revisão e atualização.
46. Cole GW, Herzlinger D. Alopecia universalis in identical twins. *Int J Dermatol*. 1984;23(4):283.
47. Sonthalia S, Sahaya K, Arora R, et al. Nocebo effect in Dermatology. *Indian J Dermatology, Venereol Leprol*. 2015;81(3):242.
48. Olsen EA. *Disorders of Hair Growth: Diagnosis and Treatment*. McGraw-Hill; 2003.
49. Carelle N, Piotto E, Bellanger A, Germanaud J, Thuillier A, Khayat D. Changing patient perceptions of the side effects of cancer chemotherapy. *Cancer*. 2002;95(1):155-163.
50. Wikramanayake TC, Amini S, Simon J, et al. A novel rat model for chemotherapy-induced alopecia. *Clin Exp Dermatology Exp dermatology*. 2012;37(3):284-289.
51. Peters EMJ, Handjiski B, Kuhlmei A, et al. Neurogenic inflammation in stress-induced termination of murine hair growth is promoted by nerve growth factor. *Am J Pathol*. 2004;165(1):259-271.
52. Ghaderinia P, Shapouri R. Assessment of immunogenicity of alginate microparticle containing Brucella melitensis 16M oligo polysaccharide tetanus toxoid conjugate in mouse. *Banat J Biotechnol*. 2017;8(16):83-92.

53. Ouis N, Hariri A. Antioxidant and antibacterial activities of the essential oils of Ceratonia siliqua. *Banat J Biotechnol*. 2018;9(17).
54. Aramesh M, Ajoudanifar H. Alkaline protease producing Bacillus isolation and identification from Iran. *Banat J Biotechnol*. 2017;8(16).
55. Jahan S, Chowdhury SF, Mitu SA, Shahriar M, Bhuiyan MA. Genomic DNA extraction methods: a comparative case study with gram--negative organisms. *Banat J Biotechnol*. 2015;6(11):61-68.
56. Hariri A, Ouis N, Bouhadi D, Benatouche Z. Characterization of the quality of the steamed yoghurts enriched by dates flesh and date powder variety H'loua. *Banat J Biotechnol*. 2018;9(17).
57. Egu UN, Okonkwo JC. EFFECT OF GONADOTROPHIN (DICLAIR®) ON SEMEN CHARACTERISTICS, HORMONAL PROFILE AND BIOCHEMICAL CONSTITUENTS OF THE SEMINAL PLASMA OF MATURE BALAMI RAMS. *Banat J Biotechnol*. 2017;8(15).
58. Slominski AT, Zmijewski MA, Skobowiat C, Zbytek B, Slominski RM, Steketee JD. Sensing the environment: Regulation of local and global homeostasis by the skin neuroendocrine system. *Adv Anat Embryol Cell Biol*. 2012;212:v.
59. Tiede S, Bohm K, Meier N, Funk W, Paus R. Endocrine controls of primary adult human stem cell biology: thyroid hormones stimulate keratin 15 expression, apoptosis, and differentiation in human hair follicle epithelial stem cells in situ and in vitro. *Eur J Cell Biol*. 2010;89(10):769-777.

60. Hamilton TE, van Belle G, LoGerfo JP. Thyroid neoplasia in Marshall Islanders exposed to nuclear fallout. *Jama*. 1987;258(5):629-636.
61. Bizhanova A, Kopp P. The sodium-iodide symporter NIS and pendrin in iodide homeostasis of the thyroid. *Endocrinology*. 2009;150(3):1084-1090.
62. Freinkel RK, Freinkel N. Hair growth and alopecia in hypothyroidism. *Arch Dermatol*. 1972;106(3):349-352.
63. Schell H, Kiesewetter F, Seidel C, Hintzenstern J V. Cell cycle kinetics of human anagen scalp hair bulbs in thyroid disorders determined by DNA flow cytometry. *Dermatology*. 1991;182(1):23-26.
64. Trüeb RM. Hormones and hair growth. *Hautarzt*. 2010;61(6):487-495.
65. Kaminski C. Alopecia in systemic diseases. *Trichology Dis pilosebaceus follicle Madrid Anca Media Gr*. Published online 1997:500-505.
66. Lerner AB. On the etiology of vitiligo and gray hair. Published online 1971.
67. Barbieri RL. The endocrinology of the menstrual cycle. In: *Human Fertility*. Springer; 2014:145-169.
68. Touyz RM, Jiang L, Ram Sairam M. Follicle-stimulating hormone mediated calcium signaling by the alternatively spliced growth factor type I receptor. *Biol Reprod*. 2000;62(4):1067-1074.
69. Pouwer AW, Farquhar C, Kremer JAM. Long-acting FSH versus daily FSH for women undergoing assisted reproduction. *Cochrane Database Syst Rev*. 2015;(7).

70. McLachlan RI, Wreford NG, O'donnell L, De Kretser DM, Robertson DM. The endocrine regulation of spermatogenesis: independent roles for testosterone and FSH. *J Endocrinol*. 1996;148(1):1-9.
71. Kumar TR, Wang Y, Lu N, Matzuk MM. Follicle stimulating hormone is required for ovarian follicle maturation but not male fertility. *Nat Genet*. 1997;15(2):201-204.
72. Richards JS. Hormonal control of gene expression in the ovary. *Endocr Rev*. 1994;15(6):725-751.
73. Kalra SP, Kalra PS. Neural regulation of luteinizing hormone secretion in the rat. *Endocr Rev*. 1983;4(4):311-351.
74. Sower SA, Freamat M, Kavanaugh SI. The origins of the vertebrate hypothalamic--pituitary--gonadal (HPG) and hypothalamic--pituitary--thyroid (HPT) endocrine systems: new insights from lampreys. *Gen Comp Endocrinol*. 2009;161(1):20-29.
75. Geniole SN, Bird BM, Ruddick EL, Carré JM. Effects of competition outcome on testosterone concentrations in humans: An updated meta-analysis. *Horm Behav*. 2017;92:37-50.
76. Alsantali A, Shapiro J. Androgens and hair loss. *Curr Opin Endocrinol Diabetes Obes*. 2009;16(3):246-253.
77. Kasumagić-Halilović E. Thyroid autoimmunity in patients with alopecia areata. *Acta Dermatovenerologica Croat*. 2008;16(3):0.
78. Bakry OA, Basha MA, El Shafiee MK, Shehata WA. Thyroid disorders associated with alopecia areata in Egyptian patients. *Indian J Dermatol*. 2014;59(1):49.

79. Hay ID, Bayer MF, Kaplan MM, Klee GG, Larsen PR, Spencer CA. American Thyroid Association assessment of current free thyroid hormone and thyrotropin measurements and guidelines for future clinical assays. The Committee on Nomenclature of the American Thyroid Association. *Clin Chem*. 1991;37(11):2002-2008.
80. Sehgal VN. Vitiligo and alopecia areata associated with subclinical/clinical hypothyroidism. *Skinmed*. 2011;9(3):192-194.
81. Kakourou T, Karachristou K, Chrousos G. A case series of alopecia areata in children: impact of personal and family history of stress and autoimmunity. *J Eur Acad Dermatology Venereol*. 2007;21(3):356-359.
82. Emy Abi Thomas RS. Alopecia areata and autoimmunity: a clinical study. *Indian J Dermatol*. 2008;53(2):70.
83. Wang H, Gan H, Mei L, Yang G, Fang F. The association between alopecia areata and thyroid autoimmunity in Chinese adult patients: a controlled study. Published online 2017.
84. Milgraum SS, Mitchell AJ, Bacon GE, Rasmussen JE. Alopecia areata, endocrine function, and autoantibodies in patients 16 years of age or younger. *J Am Acad Dermatol*. 1987;17(1):57-61.
85. Braverman LE, Cooper D. *Werner & Ingbar's the Thyroid: A Fundamental and Clinical Text*. Lippincott Williams & Wilkins; 2012.
86. Charkes ND. The many causes of subclinical hyperthyroidism. *Thyroid*. 1996;6(5):391-396.
87. Gharib H, Mazzaferri EL. Thyroxine suppressive therapy in patients with nodular thyroid disease. *Ann Intern Med*. 1998;128(5):386-394.

88. Spencer CA, Takeuchi M, Kazarosyan M, MacKenzie F, Beckett GJ, Wilkinson E. Interlaboratory/intermethod differences in functional sensitivity of immunometric assays of thyrotropin (TSH) and impact on reliability of measurement of subnormal concentrations of TSH. *Clin Chem*. 1995;41(3):367-374.
89. Braverman LE. Evaluation of thyroid status in patients with thyrotoxicosis. *Clin Chem*. 1996;42(1):174-178.
90. Hopton MR, Harrop JS. Immunoradiometric assay of thyrotropin as a" first-line" thyroid-function test in the routine laboratory. *Clin Chem*. 1986;32(4):691-693.
91. Danzer H, Braunstein GD, Rasor J, Forsythe A, Wade ME. Maternal serum human chorionic gonadotropin concentrations and fetal sex prediction. *Fertil Steril*. 1980;34(4):336-340.
92. Lenton EA, Neal LM, Sulaiman R. Plasma concentrations of human chorionic gonadotropin from the time of implantation until the second week of pregnancy. *Fertil Steril*. 1982;37(6):773-778.
93. Simoni M, Gromoll J, Nieschlag E. The follicle-stimulating hormone receptor: biochemistry, molecular biology, physiology, and pathophysiology. *Endocr Rev*. 1997;18(6):739-773.
94. Vitt UA, Kloosterboer HJ, Rose UM, et al. Isoforms of human recombinant follicle-stimulating hormone: comparison of effects on murine follicle development in vitro. *Biol Reprod*. 1998;59(4):854-861.
95. Sharma VK, Dawn G, Kumar B. Profile of alopecia areata in Northern India. *Int J Dermatol*. 1996;35(1):22-27.

96. Lewiński A, Broniarczyk-Dyła G, Sewerynek E, Zerek-Mełeń G, Szkudliński M. Abnormalities in structure and function of the thyroid gland in patients with alopecia areata. *J Am Acad Dermatol*. 1990;23(4):768-769.
97. Park S-M, Oh Y-J, Lew B-L, Sim W-Y. The association among thyroid dysfunction, thyroid autoimmunity, and clinical features of alopecia areata: A retrospective study. *J Am Acad Dermatol*. 2019;81(2):602-605.
98. Carmina E, Rosato F, Janni A, Rizzo M, Longo RA. Relative prevalence of different androgen excess disorders in 950 women referred because of clinical hyperandrogenism. *J Clin Endocrinol Metab*. 2006;91(1):2-6.
99. Azziz R, Sanchez LA, Knochenhauer ES, et al. Androgen excess in women: experience with over 1000 consecutive patients. *J Clin Endocrinol Metab*. 2004;89(2):453-462.
100. Ranasinghe GC, Piliang M, Bergfeld W. Androgen Excess in Alopecia Areata, an Unexpected Finding. *Med J Obstet Gynecol*. 2017;5(3).
101. Ranasinghe C, Bergfeld W, Piliang M. Prevalence of hormonal and endocrine dysfunction in lichen planopilaris patients: a retrospective data analysis of 413 patients: 2827. *J Am Acad Dermatol*. 2016;74(5).
102. Schmidt JB. Hormonal basis of male and female androgenic alopecia: clinical relevance. *Skin Pharmacol Physiol*. 1994;7(1-2):61-66.

الخالصـــة :-

داء الثعلبة هو مرض جلدي ُظهر على شكل مناطك دائرّة خالّة تماما من الشعر فً منطمة او اكثر)مثل الراس او الذلن او الشارب او الحواجب او الجسم واالطراف(, وتكون على شكل دائرة او بُضاوّة وتحدث فً الجنسٌن من عمر)١٠_١٠(سنة غالباً. ولد تصّب الثعلبة اي منطمة فً الجسم , ولكن فروة الراس ةً االكثر عرضة.. كذلن تصّب الثعلبة االعمار كلها من االطفال الى الكبار , لكن االطفال هم اكثر اصابة بها وتصّب الذكور واالناث على نحو سواء . ٌُعد المرض بشكل عام على نوعٌن: الثعلبة الشاملة او البمعٌة فً هذا النوع ُتساطل الشعر بشكل مفاجئ من جمٌع فروة الرأس . الثعلبة الكاملة وفً هذا النوع ُتساطل الشعر من كامل جسم المصاب. ال ُزال سبب المرض غٌر واضح لكن ُمترح بشدة دور المناعة الذاتٌة وعادة ما ُرتبط داء الثعلبة مع اضطرابات الغدة الدرلٌة , واغلبها شٌوعا اضطرابات الغدة الدرلٌة المناعٌة الذاتٌة.

ُهدف بحثنا لدراسة ارتباط بعض الهرمونات المحفزة لإلصابة بالمرض لدى المصابون بداء الثعلبة فً بٌئة مدٌنة الرمادي مثل : هرمونات الغدة الدرلٌة اوالهرمون المنبه للجرٌب والهرمون اللوتٌنً

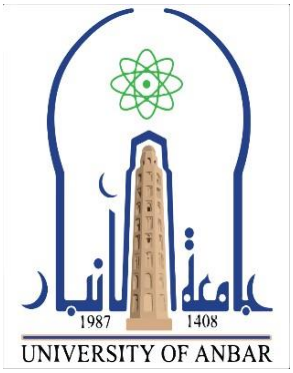
شملت الدراسة على)٧٢(حالة تتراوح اعمارهم بٌن)١٠-١٠سنة( )المجموعة االولى(شملت)٧٠(مرٌضاً بداء الثعلبة)٧٢ ذكور, ٠٠ اناث(بٌنما)المجموعة الثانٌة(شملت)١٠ (متطوعاً سلٌماً)٢ ذكور و٠٧ أناث(كمجموعة سٌطرة االصحاء وتم جمع العٌنات من العٌادات الخارجٌة لألمراض الجلدٌة, مستشفى الرمادي ومستشفى النسائٌة واالطفال فً محافظة االنبار/ العراق . للفترة من شهر كانون االول ٢٠١٧ ولغاٌة شهر اذار ١٧١٧

وتم تثبٌت المعلومات الخاصة لكل من المرضى واالصحاء والتً تضمنت العمر, تارٌخ االصابة واذا كان هنان امراض اخرى, واخذ موافمتهم على ألاس بعض الهرمونات مثل: ألاس هرمونات الغدة الدرلٌة والهرمون المنبه للجرٌب والهرمون اللوتٌنً عند اجراء المٌاسات وجدنا فروق ذات داللة احصائٌة بٌن المرضى واالصحاء فٌما ٌتعلك بمستوٌات بٌنما لوحظ الهرمونات حٌث لوحظ انخفاض ملحوظ فً مستوٌات هرمونات الغدة الدرلٌة)3T،3T(ارتفاع ملحوظ فً مستوٌات هرمون)3ST(فً المرضى ممارنة باألصحاء.

كما لوحظ وجود فروق ذات دالئل احصائُة بُن المرضى واالصحاء فُما ُتعلك بكل من الهرمون المنبه للجرٌب)FSH (والهرمون اللوتُنً) LH(حُث لوحظ ارتفاع ملحوظ فً كل من الهرمونُن فً المرضى ممارنه باألصحاء .

لذا ُفضل فحص الهرمونات لكل مرٌض مصاب بداء الثعلبة حتى فً غُاب االعراض التً توحً باضطرابات الهرمونات واألمراض المرتبطة بها. كما نوصً بّتبع الدراسة الحالُة بنفس المعلمات مع عدد أكبر من العُنات لتأكُد النتائج الحالُة. وكذلن دراسة تأثُر ارتباط هرمونات أخرى بداء الثعلبة مثل هرمون: الكورتُزول والتستوستُرون واألندروجُن ... إلخ.

جوهرَت العراق
وزارة التعلُن العالٍ والبحث العلوٍ
جاهعت االنبار
كلُت العلىم
قسن الكُوُاء



دراست تأثُر بعض الهرهناث فٍ هصل هرضً داء الثعلبت فٍ هدَنت الرهادٌ

رسالت
مقدمت الى مجلس كليت العلوم / جامعت االنبار كجزء من متطلباث
نيل شهادة الماجستير في الكيمياء

هن قبل الطالبت
رؤي طـارق حوـاد الشكـت
بكالىرَس كُوُاء

اشراف

أ.د. وجُه ًَنس هحوـد
أ.م.د. عبد هلا صالح الحسن

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