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January 2022

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Editor in Chief

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Dr. Abdelwahab Arrazaghi, was born in Gharian, a mountain city in Libya, he started school in the capital city Tripoli, Libya. He completed his elementary school in Saudi Arabia and Spain where he travelled with his diplomatic family. He completed high school at Hay Al Andalus and joined the University of Tripoli Faculty of Medicine in 1984. He completed medical school in 1993 and travelled to Toronto to pursue his higher medical education at the University of Toronto, where he joined in the postgraduate program for internal medicine followed by adult cardiology program and Echocardiography fellowship.

He showed outstanding performance throughout his training years and was actively involved in teaching junior and senior medical residents at the University of Toronto internal medicine and cardiology program. He lectured on a regular basis on many topics in the internal medicine and cardiology grand rounds. Over all his presentations are known to be up to date, exciting and well organized. He was the only trainee ever to publish two medical books during his training years and was awarded for that by the Department for Cardiology at Toronto general hospital. He made the first 200 copies of his books free and delivered and distributed to the major hospitals in his homeland, Libya. Several copies were distributed to the major hospitals in Toronto, including Toronto general hospital, St. Michael's hospital and Sunnybrook Hospital.

Amongst his many other successors Dr. Arrazaghi has earned the title of one of the best leading physicians in the world alongside his recent nomination for the year 2020-2021 as one of three top rated cardiologists in Toronto. He is the creator and chief editor of *The Canadian Medical* magazine, a public magazine curated to empower the general popular with medical knowledge and realistic views on hot medical topics.

He initiated free teaching courses for the Canadian international medical physicians, where he provided review medical lectures, introductory courses to the North American health system and practical hands-on supervised patient interactions. He is a registered University of Toronto medical examiner for graduate and postgraduate medical programs. Furthermore, he is a certified director for the Advanced Life Cardiac Support program with the Heart & Stroke Foundation of Canada. Dr. Arrazaghi is known as an excellent medical lecturer and is involved in CME lectures to the medical community in Ontario, Canada.

He is currently the executive director of Toronto Heart and Women's Health Center located in Scarborough, Ontario. He is also the director and CEO of Richmond Hill Cardiac center, carrying more than 200,000 patients seen and managed by him.

He is patient advocate both nationally and internationally by helping patients to achieve the best of health. He facilitated the transfer of patients from different third world countries to receive treatments in both Canada and Europe. Dr. Arrazaghi is known to help the poor communities in Sudan, Palestine and Libya by sending medical equipment and medical supplies on a regular basis. He is known to be civil right advocate and an active member in Immensity International and Animal Rights group, and a member of the Canadian Race Relation Foundation, and active member of Islamic Relief Canada.

At a personal level, Dr. Arrazaghi is a loving father, son and husband. He has a never ending love of sports including; boxing, swimming, soccer and basketball. He has travelled to over 21 countries to explore nature and nations around the world and has a loving passion for connecting with his community.

Dr. Arrazaghi lives by the notion *"heart to heart"* offering care where it matters and devoting his life to helping others. He has offered not only medical care but an emotional connection with all his patients. Dr. Arrazaghi is a idol to his community, being that he came to Canada as an immigrant and has since created a successful foundation, his story inspires many like him to follow the path of passion.

HIS STRATEGY IN LIFE IS WHAT WAS NARRATED FROM THE PROPHET MOHAMMED (PPUH):

"PREPARE FOR YOUR LIFE AS IF YOU ARE TO LIVE FOREVER, AND PREPARE FOR THE DAY AFTER AS IF YOU ARE DYING TOMORROW."

JAN 2022

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The Canadian Medical is published monthly online and in print.

ISSN: 2563-9404 (Print)
ISSN: 2563-9412 (Online)

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The Canadian Medical offers real facts and stories that uncover the truth behind many mainstream medically biased procedures, medications and trends that may harm the public health. We equip our readers with the information they need to to stay empowered in our constantly changing society. *Empowering the public with Medical Knowledge.*

The Canadian Medical

Toronto Heart & Women's Health Corp.

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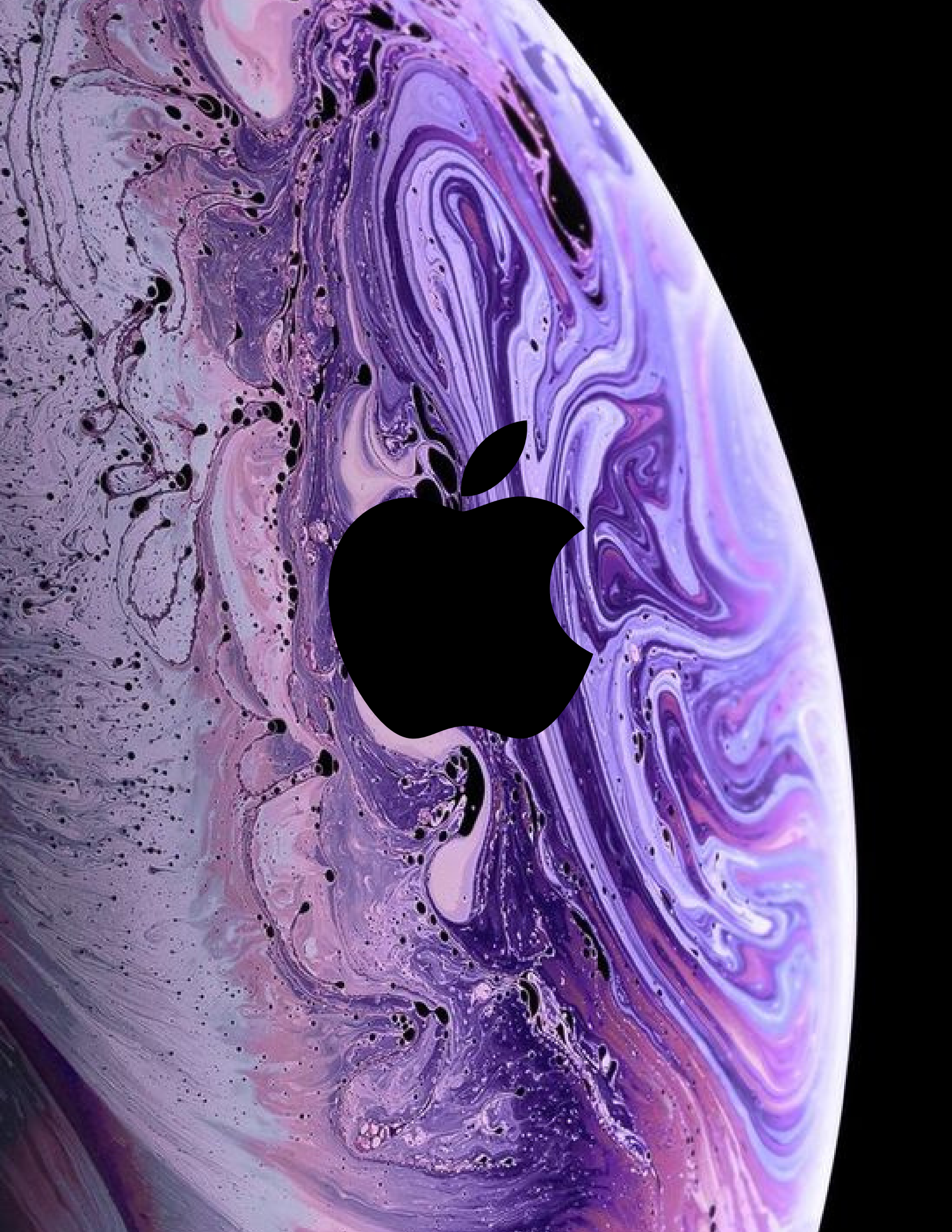
Primary Hyperaldosteronism, also known as Conn's syndrome, is a rare disease caused by an excess in the synthesis of a naturally occurring hormone in our bodies known as aldosterone.

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A Hydration Support Formula with Ultra-Pure, Vegan Hyaluronic Acid
Acide Hyaluronique 2% + B5
Une Formule de Soutien d'Hydratation avec de l'Acide Hyaluronique Végétalien Ultra-Pur
Clinical Formulations with Integrity.
The Ordinary.
Natural Moisturizing Factors + HA
Surface Hydration Formula
Facteurs Naturels d'Hydratation + HA
Formule d'Hydratation de Surface

Sheehan's Syndrome

"An unforgettable disease"

Written by: Serife Gulmus, MD
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A lady who was in her first pregnancy was admitted to the hospital for her delivery. Everything went well until the time the placenta, an organ that develops in a woman's womb (uterus) to nourish the baby during pregnancy, came out and bleeding was developed due to the absence of uterine contractions. Luckily, she was diagnosed earlier and treated by blocking the bleeding vessel in the operating room. She also got enough blood transfusions which helped her to get back to normal life and enjoy being a new mother. Unfortunately, her story didn't end here. Around a week after she went back home, she suffered from seizures, which happen when the brain develops abnormal electrical activities. After a thorough and detailed exploration at the hospital, she was diagnosed with "Sheehan's Syndrome", a collection of hormonal changes in the body due to the lack of blood supply to the Pituitary gland.

Let's begin with a brief explanation of the anatomy and physiology of the pituitary gland to understand the pathophysiology of Sheehan's Syndrome. This pea-sized major endocrine gland lies at the base of the brain and its frontal part is responsible for the growth, development, and functioning of other endocrine glands such as adrenals, thyroid gland, ovaries and testicles. The hormonal signals such as CRH (Corticotropin-Releasing Hormone), TRH (Thyroid Releasing Hormone), GnRH (Gonadotropin-Releasing Hormone) which are created from another part of the brain, called the Hypothalamus, regulates other hormones to be secreted from the Pituitary gland, ACTH (Adrenocorticotropic Hormone), TSH (Thyroid Stimulating Hormone), and LH (Luteinizing Hormone) and FSH (Follicular Stimulating Hormone), respectively.

TRH also down-regulates the Prolactin hormone which is essential for a mother to produce milk for her baby. Those hormones then stimulate and cause changes in the peripheral secretory organs in the body. When the target organ has enough hormones to function, it sends signals to the Pituitary gland to stop sending more hormones. This same negative feedback regulation is also in place between the pituitary gland and the hypothalamus. Any disruption or malfunction in these systems causes clinical symptoms. One of the malfunctions of this system happens in the post-partum period- the time after a woman delivers her baby- when the pituitary gland does not have enough blood supply to create regulatory hormones and shrinks in the sella, the place where the gland sits in the brain. The sum of the clinical findings then is called Sheehan's Syndrome.

The syndrome was first described by Glinski and Simmond in the beginning of 20th century as a severe anterior pituitary necrosis which is found in the biopsy results of a postpartum woman. They described the disease as a result of the postpartum hemorrhage and puerperal sepsis, which happened due to bacterial emboli or thrombosis in the vessels that water the pituitary area. In 1939, however, Harold Leeming Sheehan, a British physician and pathologist, described the disease as the necrosis of the anterior pituitary gland following the intensive blood loss after the delivery.

As mentioned, clinical findings mostly result from hormonal absence and those differ from woman to woman. Some examples are;

- In the acute term headache, hypotension, visual impairment, failure of lactation due to blood loss,
- Infertility, amenorrhea, absence or failure of the secretion of milk, breast atrophy, decreased libido due to Prolactin and Gonadotropin hormone deficiency,
- Dry and sparse hair, pale skin, constipation, cold intolerance, mental slowing due to thyroid hormone deficiency,
- Loss of pigmentation of the skin, fatigue, hypotension due to cortisol deficiency, Impaired quality of life and body composition differences due to the lack of growth hormone effects,
- Psychiatric disturbances

Diagnosis requires high suspicion and knowledge of the disease. In the developed part of the world, disease prevalence has decreased significantly due to advancement of fast recognition and treatment. However, Sheehan's Syndrome still can be seen in developing and underdeveloped countries and causes morbidity and mortality among women. Some women show vague symptoms such as fatigue and postpartum blues that result in a delay for the diagnosis and treatment. Definitive diagnosis is made by seeing the 'empty sella', the necrotic finding of the pituitary gland in the radiological studies such as CT and MRI along with the support of the laboratory tests of decreased hormones.
Treatment requires adequate fluid and blood transfusion in the acute term. In the long-term, when hypopituitarism is developed, thyroid and glucocorticoid replacement should be done immediately to protect the hypothalamic–pituitary–adrenal axis.

"What happened to our case later on? Well, her laboratory test results showed a lack of thyroid and prolactin hormone levels, and adrenal insufficiency along with hypoglycemia. She was given thyroxine and hydrocortisone and was discharged from the hospital with improvement of her symptoms. She developed infertility later on and was able to conceive with induction of ovulation, a method used to stimulate ovaries to produce eggs."

THYRO TOXICOSIS

Written by: Dr. Hajra Saleem MD
Reviewed by: Dr.Abdulwahab Arrazaghi MD. FDABIM. FRCPC Specialist of internal medicine and cardiovascular diseases, University of Toronto, Canada.

Thyrotoxicosis is characterized by the overproduction of thyroid hormone and in turn its action at the tissue level due to inappropriately high circulating thyroid hormone levels. Hyperthyroidism, a subset of thyrotoxicosis, refers specifically to excess thyroid hormone synthesis and secretion by the thyroid gland. It is a clinical state that results from any condition leading to high thyroid hormone action in tissues. It affects females more than men, approximately 2% of females and 0.2% of males. Although the condition is rare, the incidence increases with age. The clinical presentation varies, ranging from asymptomatic or subclinical, to life-threatening thyroid storm.

Subclinical hyperthyroidism/thyrotoxicosis
It is defined biochemically as the low levels of thyroid-stimulating hormone (TSH) and normal free thyroxine (FT4) and free triiodothyronine (FT3) concentrations. Patients can present with symptoms of thyrotoxicosis or they could be asymptomatic.

Overt primary thyrotoxicosis
It is a condition with suppressed TSH and with high levels of FT4, FT3, or both. Most commonly patients are presented with symptoms of hyperthyroidism.

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Hyperthyroidism
It is a form of thyrotoxicosis, which is caused by high synthesis and release of thyroid hormone by the thyroid gland. It is sometimes called overactive thyroid.

Thyrotoxic crisis (thyroid storm)
It is a severe form of thyrotoxicosis, which is acute with sudden collapse of homeostasis. It develops as a result of undiagnosed or inadequately treated thyrotoxicosis and involving altered mental status causing coma, cardiac and multiorgan failure, shock, and even death.

Signs & Symptoms Of Thyrotoxicosis:

- Unexplained weight loss
- Irregular heartbeat
- Rapid heartbeat (tachycardia) — usually a heart rate higher than 100 beats per minute
- Muscle weakness
- Shakiness
- Nervousness, anxiety, irritability
- Heat intolerance
- Changes in your menses
- High grade fever
- Diarrhea and feeling sick
- Loss of consciousness or confusion, if severe
- Eyelid retraction and stare
- Optic nerve involvement in severe cases
- Thin and fragile nails
- Hair loss

Causes of Thyrotoxicosis:
There are many conditions that can cause thyrotoxicosis, namely:

- Graves' disease: It is the most common cause of hyperthyroidism -- and thyrotoxicosis. It leads the body's immune system to attack the cells of your thyroid gland with antibodies mistakenly considering them as invaders. The etiology is not clear, but it makes the gland grow and make too much thyroid hormone. The condition tends to run in families, so the genes may play a vital role in whether you get it or not.

- Nodules: These are small growths that can develop on your thyroid and affect the secretion of the hormone. There can be a single nodule and is called toxic nodular adenoma, or multiple nodules as the case in multinodular goiter or Plummer's disease.

- Struma ovarii: It is a rare type of ovarian tumor, which is made mostly of thyroid tissue. Data has shown that it can cause hyperthyroidism.

- Thyroiditis: Any infection, viral or bacterial, some medications like lithium and amiodarone, or one's own immune system can cause the inflammation of the thyroid gland and make it release excessive thyroid hormone into the bloodstream. It can also happen post delivery, after having a baby and the condition is called postpartum thyroiditis.

Thyroid Hormone Medication: Thyroid hormone is prescribed to treat a medical condition called hypothyroidism (in which thyroid doesn't make enough hormone). You can have too much in your blood if your prescription is off or if it is not taken as prescribed.

Risk Factors:

- Family history of thyroid disease, especially Graves' disease
- Female gender
- Age above 60
- Autoimmune diseases, like type 1 Diabetes, pernicious anemia and Addison's disease

Diagnosis:
It requires:

- A comprehensive history
- Physical examination
- Blood work
- Imaging

Along with asking for signs and symptoms, inquiry about family history of thyroid problems, medication history, history of autoimmune disorders is crucial to reach to the causative factors. Evaluation of serum TSH levels and, if abnormal (low), FT4 and FT3 levels and examine the thyroid gland for enlargement or presence of any nodules in the parenchyma. Also TSH receptor antibodies (TRAb), that allow for a reliable differential diagnosis of autoimmune (Graves Disease) and non-autoimmune causes of thyrotoxicosis.

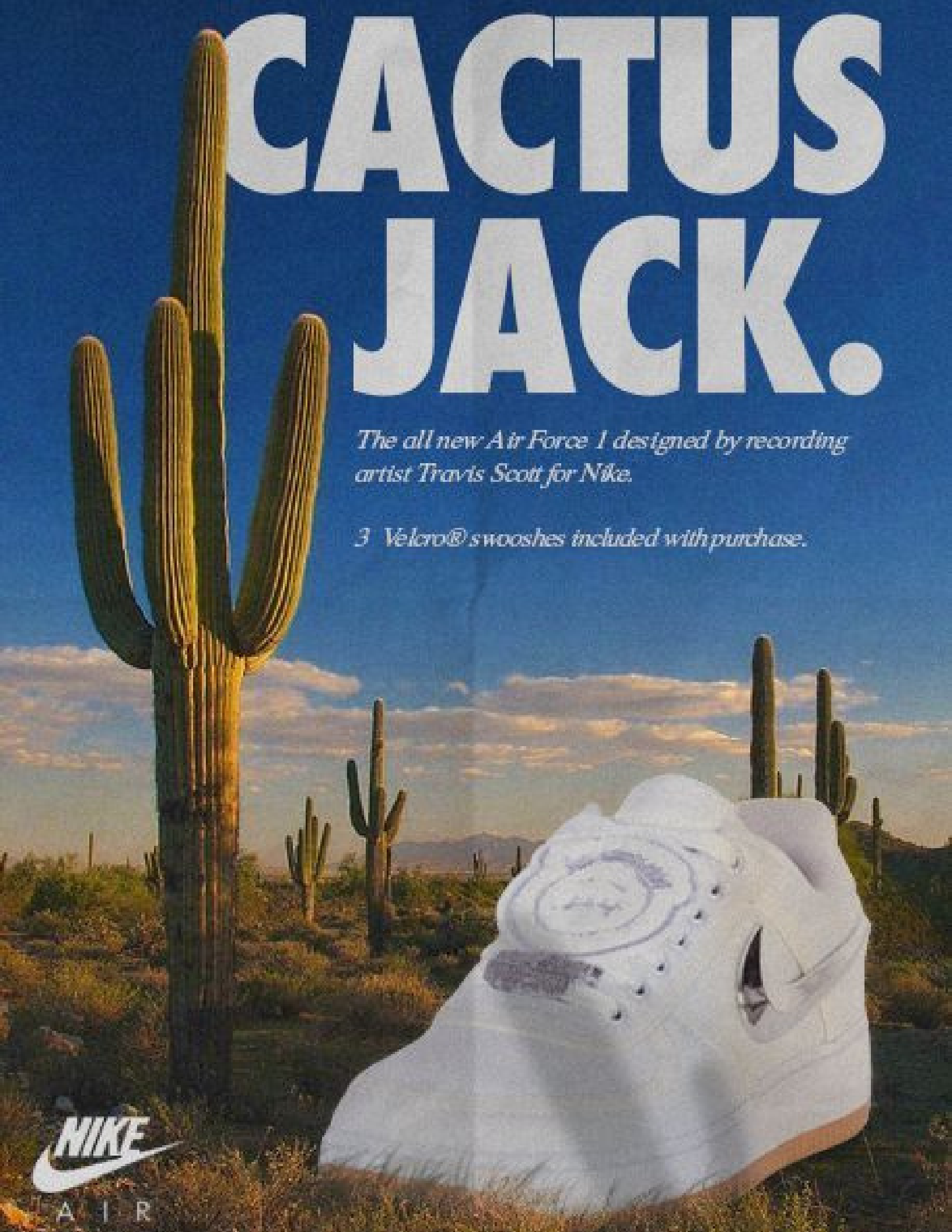
Imaging is required to have a definitive diagnosis. Various imaging modalities are available like ultrasonography of the thyroid gland, thyroid scan (scintigraphy), radioactive iodine uptake test and orbital CT scan or MRI if there is eye involvement. Moreover, cytology is also carried out in case of the nodules to classify them as malignant or non-malignant.

Treatment:
It is difficult to prevent thyrotoxicosis but it can be treated and the choice of treatment depends on the cause of thyrotoxicosis. Treatment options for thyrotoxicosis include:

- Antithyroid drugs: Methimazole and propylthiouracil (PTU) block the thyroid from making hormones.
- Radioactive iodine: It can be taken by mouth. The overactive thyroid cells absorb the radioactive iodine, which damages the cells. This causes the thyroid to shrink and reduce the thyroid hormone levels. This usually leads to permanent destruction of thyroid, which then causes hypothyroidism. Because of this, most people who receive this treatment have to take thyroid hormone drugs for the rest of their lives to maintain appropriate hormone levels.
- Surgery: Sometimes it is recommended to remove the thyroid gland through surgery, which is called thyroidectomy. This will correct hyperthyroidism, but it will usually cause hypothyroidism. That is why people who undergo thyroidectomy have to take thyroid hormone drugs for the rest of their lives to maintain appropriate hormone levels.
- Beta-blockers: These drugs block the action of thyroid hormones on the body. They help control symptoms, such as rapid heartbeat, anxiety and shakiness that are caused by thyrotoxicosis.
- Glucocorticoids: Glucocorticoids are a type of corticosteroid and they inhibit the conversion of T4 to T3. Conditions like thyroiditis that cause pain, can be treated with glucocorticoids.

Complications of Thyrotoxicosis:

- Atrial fibrillation, stroke and congestive heart failure
- Thyroid storm- a life-threatening complication
- Osteoporotic fractures - excessive thyroid hormone makes it difficult for the body to store calcium in the bones and bones need calcium in order to be strong and stable. Hence, brittle bones
- Grave's ophthalmology – blurring of vision, bulging or redness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CONN'S SYNDROME

WRITTEN BY: DR. ESSAM ASMAA ELSEFY, MD.
REVIEWED BY: DR.ABDULWAHAB ARRAZAGHI MD. FDABIM. FRCPC SPECIALIST OF INTERNAL.MEDICINE AND CARDIOVASCULAR MEDICINE UNIVERSITY OF TORONTO, CANADA

Primary Hyperaldosteronism, also known as Conn's syndrome, is a rare disease caused by an excess in the synthesis of a naturally occurring hormone in our bodies known as aldosterone. This hormone is responsible for the regulation of the electrolytes sodium and potassium in our blood, hence regulating our blood pressure and maintaining the rhythm of our hearts. As much as this hormone is crucial for survival, its excess could lead to catastrophic consequences.

To better understand the nature of this disease, it is important to understand the fundamental principles regarding this hormone production and its regulation.

Aldosterone is synthesized in the adrenal glands, two triangular shaped glands that sit on top of the kidneys. Different areas of the gland are responsible for the production of different hormones, for example the Zona glomerulosa produces aldosterone, the zona fasciculata produces corticosteroid, and the zona reticularis produces the sex hormone androgen.

Aldosterone, like any other hormone, is regulated by another hormone that is released from the kidneys known as Renin. The kidneys have a group of cells (juxta glomerular cells) that are highly sensitive to the pressure of blood flowing through the kidneys. If the blood pressure drops, the kidney releases Renin that leads to the release of aldosterone, which in turn acts on the kidney tubules to retain sodium and water to maintain the normal blood pressure level. Once the blood pressure is controlled , the kidney stops producing renin and hence exerts its negative feedback and consequently, less aldosterone is released.

THE NATURAL (PHYSIOLOGICAL) ROLE OF ALDOSTERONE

Aldosterone is primarily responsible for the regulation of the blood pressure through sodium and water retention from the kidneys. Holding back of sodium causes the excretion of potassium, therefore, excess functioning of the hormone causes an excess in sodium in the blood (which causes high blood pressure) accompanied by a low potassium level, a condition known as hypokalemia.

MORE DOES NOT ALWAYS MEAN BETTER !

So what causes this hormone to be produced in excess? As mentioned earlier, aldosterone is synthesized in the adrenal glands. The most common cause of primary hyperaldosteronism is a condition known as bilateral adrenal hyperplasia. To break it up into simpler terms, bilateral indicates both adrenal glands, and hyperplasia is the increase in the size of the glands. Another cause could be due to a tumor on the adrenal gland, known as a cortical adenoma. The resultant mass is usually non-cancerous , however, cancerous tumors may rarely arise as well.

WHEN IS HIGH BLOOD PRESSURE CONSIDERED A RED FLAG?

Not every case of blood pressure leads physicians to considering Conn's syndrome. High blood pressure is most often primary with no underlying organic disease. However, there are certain signs that, if present, would warrant further investigations to dig for the underlying condition causing the high blood pressure. Some of these are:

-High blood pressure in patients less than 30 years
-Three or more blood pressure lowering medications are required to control the high blood pressure
-An abdominal mass (indicates an adrenal mass maybe present)
-Lab tests showing low potassium level

HOW IS CONN'S SYNDROME DIAGNOSED?

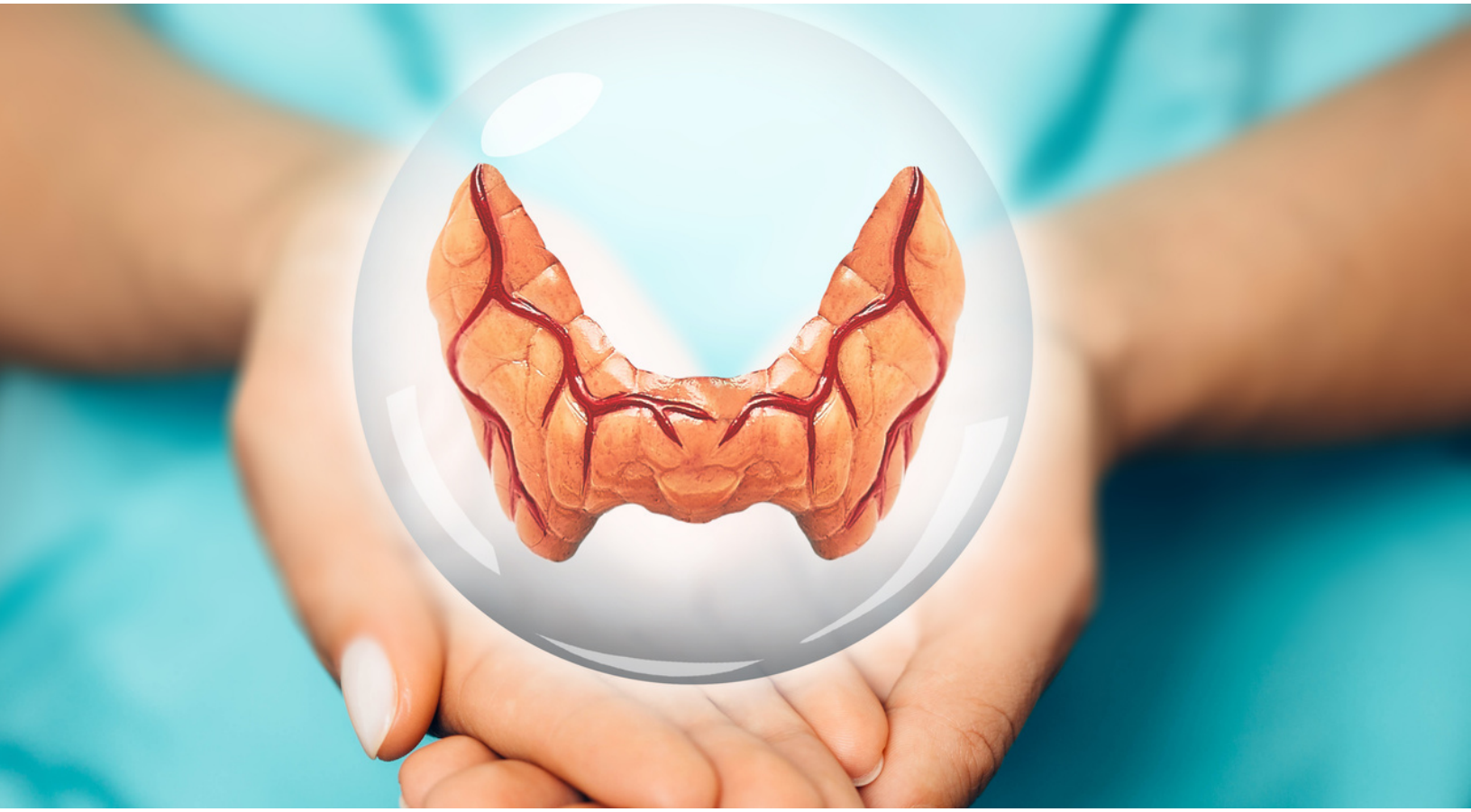
Initially, a high blood pressure is noted usually while taking the vital signs of the patient. Basic blood tests would reveal an imbalance in the electrolyte level (ie, a high sodium and a low potassium level). Hormonal analysis would reveal a shooting aldosterone level and a low renin level. Measuring renin would allow the physician to determine if the cause of high aldosterone was primary due to an adrenal cause or secondary to another underlying cause
Ps: Renin is the hormone responsible for the negative feedback on aldosterone, and if low would indicate a high aldosterone level secondary to an adrenal cause- Primary hyperaldosteronism). Once primary hyperaldosteronism is suspected, a CT scan or an MRI can be done on the abdomen to visualize the adrenal mass.
Once primary hyperaldosteronism is suspected, a CT scan or an MRI can be done on the abdomen to visualize the adrenal mass.

TREATMENT: MEDICATIONS OR SURGERY?

Conn's syndrome can only be completely cured by excising the tumor, and this is possible if the hyperplasia or tumor arises from a single adrenal gland. However, as mentioned earlier, most cases of Conn's syndrome are due to hyperplasia of both adrenal glands, making medications the preferred modality of treatment to control the disease.

Medications that are used in treatment of Conn's syndrome mainly target blocking of the action of aldosterone on the kidneys (known as anti-aldosterone). The most common drug used is Spironolactone, a common diuretic that can be used for its anti-aldosterone "side effect".
Antagonizing aldosterone would lead to an elevation in salt and water excretion and an increase in potassium retention, leading to a lower blood pressure with a "potassium sparing" effect.

The most common adverse effects of spironolactone are due to its feminizing effects as it also acquires anti-androgen properties. If the patient is disturbed by these side effects, an alternative drug can be used, called eplerenone, which has a similar anti- aldosterone action, and spares the anti-androgen effects.



HYPOTHYROIDISM

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Hypothyroidism is a disorder of the endocrine system in which the thyroid gland doesn't create and release enough thyroid hormone to meet the body's needs. Also known as underactive thyroid.
The thyroid is a small, butterfly-shaped gland in the front of the neck. Thyroid hormones control and regulate the way our body uses energy. It is responsible for providing energy to nearly every organ in the body.

Hypothyroidism is a fairly common condition. It affects approximately 2 individuals in 100. It's prevalence increases with age, and people over 60 years old experience it more frequently. Women are more likely to have an underactive thyroid. In fact, 1 in 8 women will develop hypothyroidism.
Hypothyroidism can also affect infants and young children causing congenital hypothyroidism (previously termed endemic cretinism). It is a major cause of intellectual disability. All newborn babies are routinely screened for thyroid hormone deficiency after birth.

Symptoms may vary from one person to the other, depending on the severity of the hormone deficiency. Some people have no symptoms, while others have dramatic symptoms or rarely life-threatening symptoms. It usually starts slowly, often over a number of years. Hard to notice as it may mimic other conditions like depression.
Various organ systems may be affected by the decrease in the thyroid hormone, the most common signs and symptoms include:
- Metabolic manifestations: Cold intolerance, weight gain, hypothermia - Neurologic manifestations: Forgetfulness, paresthesias of the hands and feet - Psychiatric manifestations: Personality changes, depression, dull facial expression, dementia
- Dermatologic manifestations: Facial puffiness, coarse and dry hair and skin - Gastrointestinal manifestations: Constipation
- Cardiovascular manifestations: Slow heart rate, pericardial effusion develop slowly and rarely causes hemodynamic distress
- Other manifestations: hoarse voice, slow speech, Periorbital swelling, and occasionally there may be swelling of the front part of the neck due to goiter.

In approximately 95 percent of cases, hypothyroidism is due to a problem in the thyroid gland itself called primary hypothyroidism. Worldwide, the most common cause of primary hypothyroidism is too little iodine in the diet. Hashimoto's thyroiditis is the most common cause of hypothyroidism in countries with sufficient dietary iodine. It is an autoimmune disorder, this means the immune system starts to attack itself by making antibodies against the thyroid gland. Less common causes include treatment for an overactive thyroid gland, that may include radioactive iodine therapy, medications, or surgery. A condition called secondary hypothyroidism can also happen. It is when the hypothalamus produces insufficient thyrotropin-releasing hormone (TRH) or the pituitary gland produces insufficient thyroid-stimulating hormone (TSH).

In the past, hypothyroidism was not diagnosed until symptoms had been present for a long time. However, simple blood tests can now detect hypothyroidism at an early stage
Laboratory testing of thyroid stimulating hormone (TSH) levels in the blood is considered the best initial test for hypothyroidism. TSH is the most sensitive test because it can be elevated even with a small decrease in thyroid function. Free thyroxine (T4), the main product of the thyroid gland, may also be measured to confirm and assess the degree of hypothyroidism. Lower-than-normal T4 levels usually mean you have hypothyroidism. However, some people may have increased TSH levels while having normal T4 levels. This is called subclinical (mild) hypothyroidism. It is believed to be an early stage of hypothyroidism. In some cases, where goiter is felt upon palpation, a thyroid ultrasound can be ordered to evaluate for any nodules (lumps) in the enlarged thyroid gland.

The goal of treatment for hypothyroidism is to return blood levels of thyroid-stimulating hormone (TSH) and thyroxine (T4) to the normal range and to relieve symptoms. Most people are treated with a synthetic long-acting form of thyroid hormone, known as levothyroxine (L-thyroxine). Blood free thyroxine(T4) and TSH levels are monitored to help determine whether the dose is adequate or not. This is done 4–8 weeks after the start of treatment or a change in levothyroxine dose. Once the adequate replacement dose has been established, the tests can be repeated after 6 and then 12 months, unless there is a change in symptoms.

The one word we don't have in our dictionary.

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GRAVES' DISEASE

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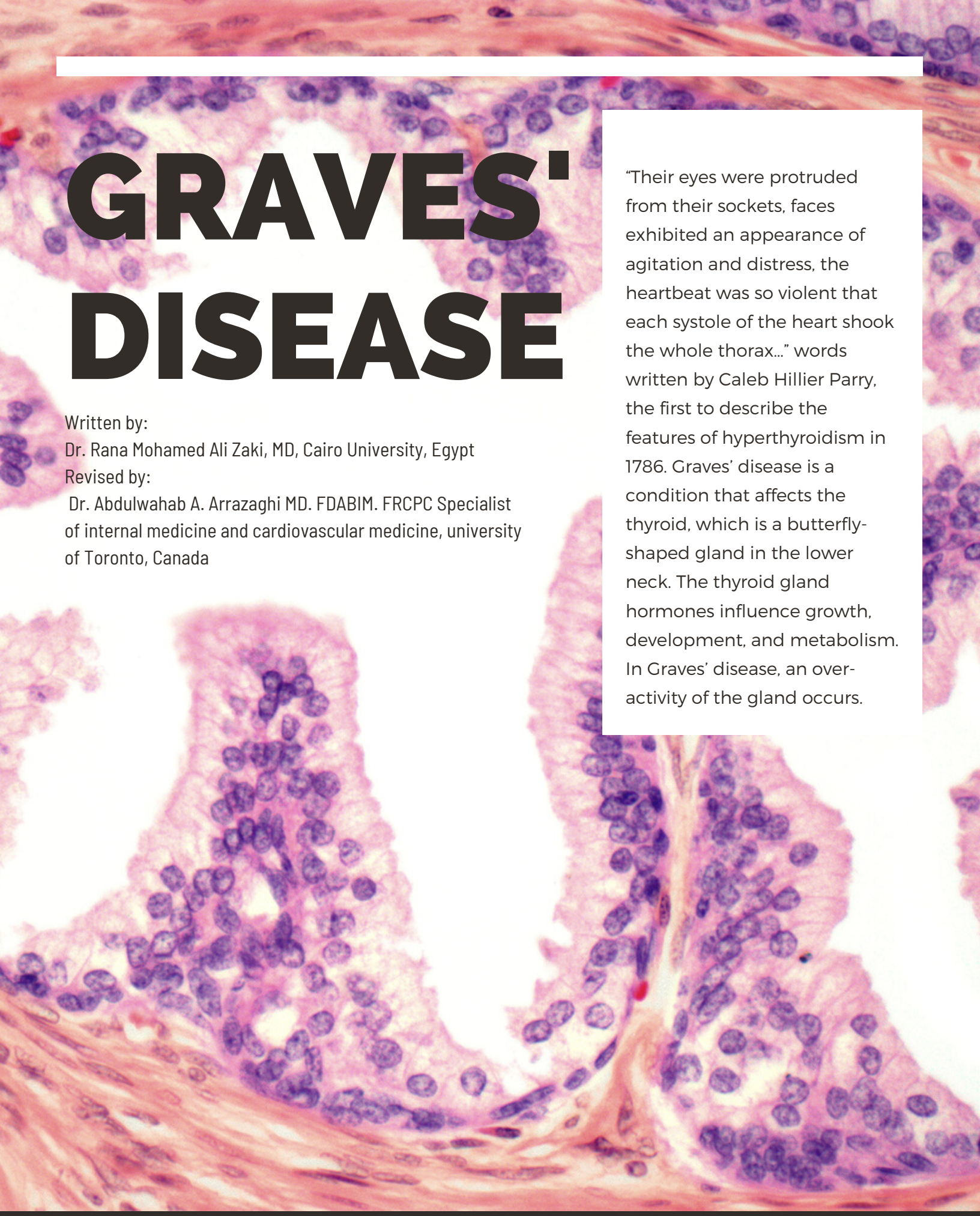
Revised by:
Dr. Abdulwahab A. Arrazaghi MD. FDABIM. FRCPC Specialist of internal medicine and cardiovascular medicine, university of Toronto, Canada

"Their eyes were protruded from their sockets, faces exhibited an appearance of agitation and distress, the heartbeat was so violent that each systole of the heart shook the whole thorax..." words written by Caleb Hillier Parry, the first to describe the features of hyperthyroidism in 1786. Graves' disease is a condition that affects the thyroid, which is a butterfly-shaped gland in the lower neck. The thyroid gland hormones influence growth, development, and metabolism. In Graves' disease, an over-activity of the gland occurs.



The symptoms of thyroid disease can be mild while others may experience significant features. These may include rapid and irregular heartbeats, strokes, heart failure and osteoporosis, trouble sleeping, weight loss in spite of an increased appetite. Just 10 to 20% may develop significant eye disease known as Graves' ophthalmopathy, which is characterized by bulging eyes, double vision, dry eyes, and swollen eyelids, light sensitivity, and in rare instances, vision loss. This complication may be due to the compression of the optic nerve connecting the eye and the brain. Others may develop nervousness or anxiety, extreme tiredness, hand tremors, frequent bowel movements or diarrhea, increased sweating, and difficulty tolerating hot conditions. A small percentage of people with Graves' disease develop a skin abnormality called pretibial myxedema. This abnormality causes the skin on the front of the lower legs and the tops of the feet to become thick, lumpy, and red. It is not usually painful.

Regarding women, they may experience menstrual irregularities, such as an unusually light menstrual flow and infrequent periods. Pregnancy problems may include miscarriage and stillbirth. The majority of people with thyroid issues do not develop goiters. However, some people may develop an enlargement of the thyroid. Depending on its size, it can cause the neck to look swollen and may interfere with breathing and swallowing.

Many children with Graves' disease suffer from a family history. The condition can arise at any point during childhood but is most likely to begin in adolescence. Teenage girls may have lighter and less frequent menstrual periods. They may also have difficulty concentrating, restlessness, fatigue, or anxiety, which can sometimes be mistaken for conditions such as attention deficit hyperactivity disorder or anxiety disorders. Over time, it can lead to growth problems.

Graves' disease is relatively common affecting one in every 100 Canadians and about one in every 200 Americans. According to the national organization for rare disorders: Graves' disease affects females more often than males by a ratio of 5-10 to 1. The disorder usually develops during middle age with a peak incidence of 40-60, but can also affect children, adolescents, and the elderly. Graves' disease occurs in almost any part of the world. Graves' disease is estimated to affect 2%-3% of the general population. Graves' disease is the most common cause of hyperthyroidism. Further, According to the British Thyroid Foundation, Cigarette smoking is identified to have a key influence on Grave's disease and on thyroid eye disease (TED).

Graves' disease is thought to result from a combination of genetic factors and other environmental factors. Graves' disease is classified as an autoimmune disorder, where the immune system creates an antibody called thyroid-stimulating immunoglobulin that signals the thyroid to increase its production in an uncontrollable manner. It is estimated that 70% of graves' disease is genetic and then something in the environment triggers the production of antibodies such as virus, pregnancy or
menopause, diet, drugs, or possibly extreme stress. Moreover, people with Graves' disease have an increased risk of developing other autoimmune disorders, including rheumatoid arthritis, pernicious anemia, systemic lupus erythematosus, Addison disease, celiac disease, type 1 diabetes, and vitiligo.

About 60 percent of people with thyroid disease are usually undiagnosed. A TSH (thyroid stimulating hormone) blood test, in combination with physical examination helps doctors make the most accurate diagnosis of thyroid disease. A lump on the neck is not necessarily an enlarged thyroid (goiter). Other causes of a neck lump could be a swollen lymph node or cyst. Often a radioactive iodine uptake and scan are ordered in hyperthyroid patients to differentiate Graves' disease from thyroiditis or hyper functioning thyroid nodule(s). Sometimes measurement of TSH receptor antibodies is used as well. Only five percent of thyroid nodules are cancerous. Aggressive thyroid cancer is uncommon but these nodules will be large, firm, fixed, and fast-growing. A thyroid ultrasound may be ordered to confirm the diagnosis as well.

Diet cannot alone regulate problems of the thyroid. Moreover, Excess iodine can trigger thyroid dysfunction. Eating a well-balanced diet with plenty of vitamins and minerals is recommended. A gluten free diet may be helpful with patients with Hashimoto's disease as they may be suffering as well from other autoimmune conditions such as Celiac disease. Some antioxidants and nutrients may help alleviate symptoms or reduce flares. These include calcium-rich foods preventing brittle bones and osteoporosis such as broccoli, almonds, kale, sardines, and okra.

The purpose of medical treatment for Graves' disease is to stop your thyroid from producing too much hormone, while still giving you enough hormone so that your body can function properly. The most common antithyroid drug is methimazole. Some women either cannot tolerate it, want to become pregnant or who are in their first trimester cannot take it. In these cases, doctors prescribe propylthiouracil. However, recent evidence of side effects of Propylthiouracil on liver function, especially in children, the FDA has issued a warning for its use. Another medication that can be given to treat the symptoms of hyperthyroidism is Propranolol or other beta-blockers, which block the effects of excess thyroid hormones on the heart, blood vessels, and nervous system. An iodine supplement of 150mcg daily is recommended for women in the UK pre-conception, during pregnancy, and during breastfeeding. If hyperthyroidism persists or recurs then consideration of radioactive iodine or surgery is recommended.

Cardiovascular disease, as heart attacks, heart failure, and strokes, is the most common cause of death in patients with hyperthyroidism. Another potentially life-threatening complication is known as a thyroid storm. This occurs in case of untreated hyperthyroidism suffering from major stress, such as trauma, heart attack, or infection. It is characterized by an attack of fever, confusion or agitation, irregular heart rhythms, or heart failure. Patients who develop thyroid storms have a 20 to 50% chance of dying.

Like humans, Hyperthyroidism in dogs is a serious condition, which can be caused by an error in medications usually including a synthetic form of thyroxine. Fresh real food is advised for dogs with hyperthyroidism, preferably lightly cooked or raw. Highly processed food is not recommended. If left untreated the condition can cause heart and kidney failure. It is advisable to avoid unnecessary chemicals or environmental hazards, which are all possibilities that contribute to the disease as over-vaccinating, Flea and tick preventatives, Unnecessary Steroids, antibiotics and NSAIDs, Food preservatives especially red dye found in processed food and treats.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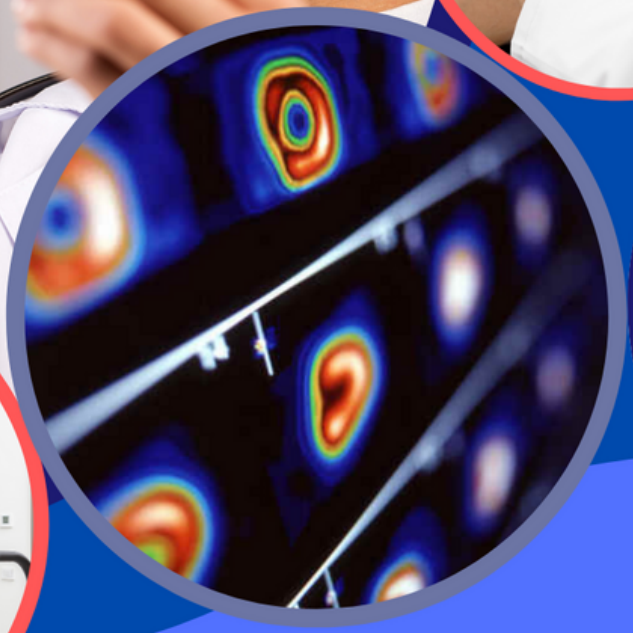

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