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The Effect of the Thyroid Gland on High Blood Pressure

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Abstract: ***The paper discusses the relationship between the thyroid gland and high blood pressure, specifically in the context of hyperthyroidism. It explains that hyperthyroidism, characterized by an increase in thyroid hormones, can lead to an acceleration of the heartbeat and an increase in its force, resulting in high blood pressure . The paper also mentions that high blood pressure resulting from other diseases, such as thyroid problems, is known as secondary hypertension. Treating this type of high blood pressure requires addressing the underlying medical condition causing it Additionally, the paper highlights that thyroid hormones play a role in regulating hemostasis, the process of blood clotting. Thyroxine, a thyroid hormone, is associated with the release of von Will brand factor and influences the synthesis of proteins involved in hemostasis. Thyroxine deficiency in hypothyroidism can lead to a deficiency in certain clotting factors Low serum levels of T3 can increase peripheral vascular resistance by up to 30%, leading to arterial stiffness and a decrease in cardiac output. This results in high blood pressure, particularly in systolic blood pressure, which returns to normal with treatment of the underlying thyroid dysfunction. Hypothyroidism is associated with high blood pressure, and the most common cardiovascular findings in hypothyroidism are bradycardia and high blood pressure. Thyroid hormone deficiency affects the contractility of cardiomyocytes, leading to an increase in peripheral vascular resistance and diastolic dysfunction. The presence of an antigen in patients with subclinical hypothyroidism may contribute to a potential hypercoagulable state, which can further impact blood pressure regulation.***

Keywords: Blood Pressure, Treatment, Impact, Thyroid Gland.

1. INTRODUCTION

It is a gland located in the front of the neck that is responsible for regulating the work of the

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metabolic process: adaptation to heat or cold, weight loss or gain, appetite and bowel rhythms, response to activity and inactivity[1,2,3].
In turn, this gland is regulated by thyroid-stimulating hormone, TSH, which is produced by the pituitary gland through the hypothalamic factor, TRH. [4]

High blood pressure in hyperthyroidism This is a state of hyperactivity and hypermetabolism. The affected person is agitated and agitated and may experience tachycardia, high blood pressure, weight loss, insomnia, fatigue, diarrhea, and heat intolerance[5].
The origin of these problems is diverse: nodules in the gland that loses external control and functions independently, hyperfunctioning adenomas, excess hormonal production due to an external stimulus or autoimmune forms.

The change in blood pressure is more pronounced in systolic blood pressure than in diastolic blood pressure and returns to normal when the stimulation process is treated.

Hyperthyroidism causes an increase in thyroid hormones represented by triiodothyronine (T3) and thyroxine (T4) [6], and this increase leads to an acceleration of the heartbeat and an increase in its force more than usual. Because blood pressure depends on the heart rate in part and is directly proportional to it, blood pressure The blood will rise significantly and will remain high unless the problem is treated. [7,8]

An underactive thyroid gland, which causes a lack of production of thyroid hormones, leads to weak contractile force of the heart muscle. It also reduces the speed of the heartbeat, making it lower than usual. As a result of a weak heart, the walls of the blood vessels become less elastic and harden, leading to high blood pressure. Which will continue unless the problem is treated.

High blood pressure resulting from other diseases is known as secondary hypertension, and the reason for calling it this name is that it is caused by a specific medical condition, and treating it in this case requires treating the disease causing the high blood pressure, such as thyroid problems, and not treating high blood pressure.

Hypothyroidism and Blooding Disorders
Thyroid hormones regulate the activity of coagulation factors, thereby regulating hemostasis. Thyroxine is associated with the release of von Willebrand factor from endothelial cells and is thought to influence the synthesis of proteins that regulate hemostasis at the hepatic level. For this reason, thyroxine deficiency in hypothyroidism is associated with a deficiency in certain clotting factors (factor VII, factor IX, Studies examining coagulation in overt hypothyroidism have yielded conflicting results. A study comparing moderate and severe hypothyroidism in euthyroid control patients found that patients with moderate hypothyroidism had decreased fibrinolytic activity and were more prone to clot formation, whereas patients with severe found increased fibrinolysis and decreased tissue plasminogen activating antigen in patients with hypothyroidism. Another study showed decreased antithrombin III activity and increased fibrinogen levels, which may explain a possible hypercoagulable state by plasminogen activator inhibitory antigen in patients with subclinical hypothyroidism. [9] Studies

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investigating clotting in overt hypothyroidism have given contradictory results, one study comparing moderate and severe hypothyroidism in patients with euthyroid controls found that patients with moderate hypothyroidism decreased fibrinolytic activity and were more susceptible to clot formation, while patients with severe hypothyroidism had increased fibrinolysis and lower tissue plasminogen activator antigen; another study showed decreased antithrombin III activity and increased fibrinogen levels, and plasminogen activator inhibitor antigen in patients with subclinical hypothyroidism could explain a potential hypercoagulable state.

Arterial Hypertension Due to Hypothyroidism

The most common cardiovascular physical findings found in hypothyroidism are bradycardia and high blood pressure. Thyroid hormone deficiency causes non-genomic and genomic alterations in the cardiomyocyte, deteriorating its contractility and producing an increase in peripheral vascular resistance, which is accompanied by a decrease in ventricular relaxation, resulting in diastolic dysfunction common in hypothyroidism. . Non-genomic alterations consist of the modification of sodium, potassium and calcium channels, while genomic alterations are characterized by producing a decrease or elevation in the production of regulatory and structural proteins, the important role of thyroid hormones in the Vasoconstriction of vascular myocytes explains the specific changes that we find in a given thyroid condition[10] Low serum levels of T3 can increase peripheral vascular resistance by up to 30%, caused by a decrease in the release of endothelium-derived relaxing factors (EDRF) and nitric oxide, causing arterial stiffness. The increase in total vascular resistance (TVR) results in a decrease in cardiac output, a decrease in the pulse wave, prolonging circulation time and decreasing perfusion in certain tissues. Studies demonstrate higher nitric oxide-dependent vasodilation values in patients who present subclinical hypothyroidism under treatment with L-thyroxine

2. DISCUSSION

Hypothyroidism could be a condition caused by thyroid hormone lack and happens more as often as possible in populaces with either tall immaterial or extreme iodine lacks. It is more common in ladies, individuals over 65 a long time of age, white people, patients with immune system illnesses (as portion of different immune system endocrinopathies), Down disorder and Turner syndrome. Its primary classification of the pathology is concurring to the organ that's causing hypothyroidism, partitioning into: 1) essential hypothyroidism: change of the thyroid gland, 2) auxiliary hypothyroidism: diminished incitement of the thyroid organ at the level of the pituitary gland, 3) tertiary hypothyroidism: diminished incitement of the thyroid organ at the hypothalamic level1. Similarly, it can be classified as clinical hypothyroidism, when there's an increment in thyroid-stimulating hormone (TSH) with moo free thyroxine (FT4) and indications occur1, and subclinical hypothyroidism, when there are tall TSH levels with ordinary levels. of T3 and T4 and is for the most part asymptomatic Regarding the relationship between thyroid hormones, the heart and the SVP, it is known that these hormones fulfill a cardioprotective work due to the distinctive cellular and biochemical components that they trigger, as well as creating hemodynamic changes and intervened impacts on the

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cardiomyocyte. Among the most cardiovascular conditions that hypothyroidism, both clinical and subclinical, can cause, are: cardiovascular brokenness, increment in add up to and LDL cholesterol, diastolic hypertension, endothelial weakening, systolic and diastolic brokenness of the cleared out ventricle congestive heart disappointment, torsades des pointes6, pericardial radiation with or without cardiac tamponade For a legitimate determination, a physical examination, blood tests and thyroid work tests (TSH, T3, T4) are utilized. Treatment will depend on the sort of hypothyroidism, hereditary and sociodemographic variables, and its seriousness.

3. CONCLUSION

In conclusion, the thyroid gland plays a crucial role in regulating blood pressure levels. When the thyroid gland produces an excess of thyroid hormones (hyperthyroidism), it can cause increased heart rate and cardiac output, ultimately leading to high blood pressure. On the other hand, hypothyroidism (hypothyroidism) can cause a decrease in heart rate and cardiac output, leading to low blood pressure levels. Therefore, it is important to properly monitor and manage thyroid function to effectively prevent and treat high blood pressure.

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