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A Physiological Study of Hypothyroid Patients in the City of Samawa

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Annotation: Thyroid disease can be specified as the second most common endocrine condition affecting several body systems. The purpose of the presented study has been investigating hypothyroidism effects on the fasting blood sugar level (FBS), hemoglobin level (Hb), and the prevalence of hypertension, anemia, and diabetes in such two groups. Ten healthy people, both female and male, between the ages of 30 and 65, served as controls in the study, along with 40 patients who had previously with been diagnosed hypothyroidism (40 cases). Significant differences in Hb levels have been found between the two groups, with the control group having the highest levels and the hypothyroidism group having the lowest. In comparison to the control group, the hypothyroidism group saw a significantly of increased frequency anemia. Additionally, the hypothyroidism group had a much higher diastolic blood pressure. Furthermore, in comparison with the control hypothyroidism group, the group significantly experienced increased frequency of hypertension. The FBS levels

between the two groups have shown significant differences as well. In the hypothyroidism group in comparison with the control group, the frequency regarding diabetes has been significantly higher. We advise all hypothyroid patients to undergo routine assessments for potential changes in their cardiovascular, hematological, biochemical, and dental health in light of the information acquired.

Keywords: Thyroid gland, hypothyroid, hypothyroidism, thyroxine T4, triiodothyronine T3.

Introduction:

Mild hypothyroidism, subclinical hypothyroidism, poor thyroid reserve, or preclinical hypothyroidism are all terms used to describe increased serum thyroid-stimulating hormone with the existence of normal total or free thyroxine (T4) content in serum (1). Most people with subclinical hypothyroidism either have nonspecific, vague symptoms or are asymptomatic and could be identified by routine blood tests. The prevalence regarding subclinical hypothyroidism varies between 4 and 10% depending on the geographic population. Yet, recent studies have found that subclinical hypothyroidism might be much more prevalent compared to what previously believed. Subclinical hypothyroidism has long been associated with high cholesterol, cardiovascular mortality, atherosclerosis, poor obstetric outcomes, infertility, spontaneous deep vein thrombosis, neuropsychiatric symptoms, and common bile duct stones (1–5). The most common sign of hypothyroidism, which mainly impacts the hematopoietic system, is anemia. Hypothyroidism could result in a variety of anemic diseases. There are several mechanisms involved in the pathophysiology regarding such anemia, such as macrocytic, normocytic, and microcytic ones. (6)

Hypothyroidism, a clinical disorder that results in a general reduction in metabolism, is indicated by a thyroid hormone deficiency.

The thyroid gland produces tetraiodothyronine (T4) and triiodothyronine (T3) hormones, which are essential for the human health, a healthy lifestyle, and sufficient energy levels (7). Thyroid illnesses are characterized by changes in the produced thyroid hormone. Inadequate production is the cause of hypothyroidism (8). One of the most common thyroid conditions worldwide is hypothyroidism (9). Reduced production of T3 and T4 results in thyroid inactivity (10). Low levels of the thyroid hormone in the blood, or hypothyroidism, is linked to less weight gain, a slower metabolism, poor cholesterol clearance, lipolysis, and higher levels of blood cholesterol. There are both non-genetic and genetic impacts of thyroid hormones (11). The hypothalamic-pituitary-thyroid axis is the system by which pituitary, thyroid, and hypothalamus control the production of thyroid hormones. The hypothalamus gland produces and stores thyrotropin-releasing hormone (TRH). The thyroid hormone impacts the brain development, body metabolism, and other physiological processes. T4 and T3 are frequently referred to as thyroxine. Thyroid hormone comes in 2 different metabolically active forms. Conversely, dry skin,

diarrhea, and edema are symptoms of a slowed metabolism caused by hypothyroidism. Hypothyroidism is more prevalent compared to hyperthyroidism in thyroid disease, and subclinical thyroid dysfunction is widespread (13, 14). There is a number of reasons for hypothyroidism or the underactive thyroid. A few of the causes include a lack of enzymes required for the generation of thyroid hormone, duct thyroid inflammation, autoimmune thyroiditis, prior thyroid surgery, and thyroid shortage.

Finding out how common hypertension and anemia were in these two groups, and how hypothyroidism affected hemoglobin, blood pressure, and FBS, were the goals of this investigation.

Material and method:

Along with 10 healthy individuals between the ages of 30 and 65 who acted as controls (female and male), the presented work included 40 patients who had previously been diagnosed with hypothyroidism and who visited the Al Hussein teaching hospital in Samawa. The blood pressure was measured using a mercury pressure-measuring device. The subjects were separated into three groups by the American Heart Association: hypertension, normal, and hypotension. Normal blood pressure values are between 100mmHg and 140mmHg for the systolic and 60mmHg and 90mmHg for the diastolic. Blood Hb: Five milliliters of venous blood were extracted from each individual's antecubital vein with the use of a disposable syringe. The hemoglobin content of blood samples has been determined using the Gemmy hematocrit. The subjects have been classified as polycythemia, normal, and anemia according to the typical adult range, which is 13.50–17.50g/dl for men, and 12–15.50g/dl for women. Thyroid function (T3, TSH, and T4) tests with fasting blood sugar tests: For five minutes, blood samples have been centrifuged at 5000 rpm. A clinical chemistry analyzer (Mindray BS-230) was used to quantify fasting blood sugar in the serum samples, and a small desktop immunofluorescence analyzer (AFIAS-6/biotech) was used for measuring hormones (T3, TSH, and T4). The individuals were classified as hypothyroid and control depending on the results of thyroid function tests (expected levels of T4 4.9-11, T3 1.17-3.4, and TSH 0.30-3.60µg/dl). The participants have after that been categorized as either normal or diabetic depending on the results of FBS test. The normal range for the FBS test levels is 70-100mg/dl.

Statistical analysis:

The data are represented in the form of mean \pm standard deviation (M \pm SD) and frequency of observations percentage (Cases %). The data was analyzed with the use of Fisher's test for multiple comparisons and one-way analysis of variance (ANOVA) in Stat View v. 5.0. Differences are regarded as significant in the case where p<0.050.

Results:

In the present study, 10 healthy controls (male and female) in the 30-65 age range were included, along with 40 patients who had previously been diagnosed with hypothyroidism. Table 1 describes the TSH, T4, and T3 levels in these groups.

Table 1: Characteristic hormone levels in hypothyroidism patients

	Mean TSH (µg/dl)	Mean T3 (μ g/dl) \pm	Mean T4 (µg/dl) ±
	± SD	SD	SD
Control (10 Case)	1.80±0.40	1.90±0.30	8.20±0.40
Hypothyroidism (40 Cases)	11.70±3.90	1.00±0.060	4.30±0.10

Despite the absence of polycythemia, hypothyroidism had a significantly higher incidence of anemia than control (90 and 40%, respectively), despite the latter having a higher normal (10 and 80%, respectively). The HB levels in the two groups differed noticeably. The score was highest

for control and lowest for hypothyroidism (Table2).

Table 2: HB level, anemia frequency, and normal status in thyroiditis and healthy people

	Mean HB (g/dl) ± SD	Anemia (%)	Normal (%)
Control (10 Case)	12.20±1	40	80
Hypothyroidism (40 Case)	10.50±1.5	90	10

Although the hypothyroidism and control groups did not differ significantly, the hypothyroidism group's systolic blood pressure has been considerably higher. On the other hand, the hypothyroidism group's diastolic blood pressure has been considerably greater than the control groups. The control group had a higher normal rate than the hypothyroidism group (60% and 40%, respectively), but the hypothyroidism group had a much greater frequency of hypertension (70% and 30%, respectively). None of the volunteers had any signs of hypotension. (Table 3)

Table 3: Diastolic and systolic blood pressure, the hypertension prevalence, and normal status in people with thyroid disease and those in good health

	Mean Systolic SD	Mean Diastolic SD	Hypertension (%)	Normal (%)
Control (10 Case)	11.50±1.80	9.50±0.70	30	60
Hypothyroidism (40 Cases)	13.50±2.50	9.70±1.50	70	40

Although there were no discernible differences between the frequencies of normal and diabetic status in hypothyroidism, the normal status frequency has been much higher in the control group when compared with the hypothyroidism group (40 and 20%, respectively), while the frequency of diabetic has been much higher in the hypothyroidism group (80% and 60%, respectively). The two groups' FBS levels also differed significantly from one another. The hypothyroidism group had the greatest, while the control group had the lowest (Table 4).

Table 4: FBS levels, prevalence of diabetes, and normal status in people with thyroid disease and those in good health

	Mean FBS±SD	Diabetic (%)	Normal (%)
Control (10 Case)	151±80.9	60	40
Hypothyroidism (40 Case)	192±94.7	80	20

Discussion:

Through the hyperactive proliferation regarding immature erythroid progenitors and the upregulation of erythropoietin secretion through erythropoietin gene expression, thyroid hormones are known to have a significant effect on erythropoiesis. All myeloid cell lineages exhibit hypoplasia in hypothyroidism, hyperthyroidism causes hyperplasia, and stem cells generally seem to create specific blood cell types, including granulocytes, erythrocytes, and platelets (15, 16). Hypothyroidism could cause hypochromic, normocytic, or macrocytic anemia through reducing oxygen metabolism. Microcytic anemia is often caused by poor absorption of the iron or iron loss through hemorrhage, as opposed to macrocytic anemia, which leads to malignant anemia, inadequate nutrition, and poor absorption of vitamin folic acid and B12 (16). People with hyperthyroidism, on the other hand, usually do not have anemia; in such cases, red blood cells are present, yet the look of anemia could be similar to that of hypothyroidism (17, 18). The results of the research showed that while the mean HB in the hypothyroidism group has been much lower—with anemia frequencies of 40% in the latter group and 90% in the former—the mean HB in the

control group has been within the normal range. Patients who have hypothyroidism and controls had statistically significant differences in HCT and HB, according to Dorgalaleh et al. (2013) (19). Patients with hypothyroidism and the control group did not significantly differ in RBC parameters such as HCT and HB, according to a 2012 research by Srikrishna R and Geetha J [20]. Kawa MP et al. (2010) discovered that patients with hyperthyroidism had significantly higher HB, RBC, and HCT compared to control groups, while patients with hypothyroidism had lower HB and RBC (20). It is known that hypothyroidism leads to adverse effects, whereas hyperthyroidism produces a hyperdynamic cardiovascular condition (a decrease in the systemic vascular system's resistance and an increase in the heart's output). A decrease or an increase in the way thyroid hormones affect specific molecular pathways in blood vessels and the heart might result in related cardiac arrhythmias. A higher heart rate and better left ventricular systolic and diastolic performance are associated with cardiovascular hyperdynamics (21), both hypothyroidism and hyperthyroidism-induced hypertension. Diastolic blood pressure is caused by noradrenaline being released in hypothyroidism to compensate for the decrease in thyroid hormones. Any increase in T3 results in systemic vascular resistance, or blood flow resistance, to decrease, which in turn causes systolic blood pressure to rise (23). This data explains our results that patients with hyperthyroidism had higher diastolic and systolic blood pressures compared to the control group, and that patients with hypothyroidism had a higher frequency of hypertension (70%) in comparison with 30% in the control group. Diabetes mellitus (DM) as well as thyroid problems are closely linked (24). Several studies showed that a complex combination regarding genetic, biochemical, and hormonal correlations reflects such pathological relation (24, 25). The main cause of DM associated with thyroid dysfunction is autoimmunity (26, 27). Studies have examined the association between DM and either Hashimoto's thyroiditis, a hypothyroid condition, or Graves' disease, a hyperthyroid condition (28). The results of the study showed that the prevalence regarding diabetes has been 80% in hypothyroidism against 60% in the control, and there was a significant rise in FBS in hypothyroidism relative to the control. Both systemic and oral symptoms of thyroid disease are prevalent. Malocclusion, thick lips, delayed tooth eruption, and a broad protruding tongue (macroglossia) are characteristics of childhood hypothyroidism, also known as cretinism (29). Increased susceptibility to dental cavities, enlargement of extra glandular thyroid tissue (majorly in lateral posterior tongue), periodontal disease, mandibular or maxillary osteoporosis, and accelerated dental eruption are all oral symptoms of thyrotoxicosis (30). In summary, dyspepsia, delayed eruption, delayed wound healing, altered tooth morphology, and characteristic macroglossia are common oral discoveries in hypothyroidism (30). An increased susceptibility to periodontal disease, cavities, and other diseases are among the oral symptoms of thyrotoxicosis (31). We suggested routine assessments for any changes in the cardiovascular, hematological, biochemical, and dental health of all hypothyroid patients depending on the information acquired.

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REFERENCES

- 1. Cinemre H, Bilir C, Gokosmanoglu F, Bahcebasi T.(2009) Hematologic effects of levothyroxine in iron-deficient subclinical hypothyroid patients: a randomized, double-blind, controlled study. J Clin Endocrinol Metab.;94:151-6.
- 2. Gharib H, Tuttle RM, Baskin HJ, Fish LH, Singer PA, McDermott MT. (2005) Subclinical thyroid dysfunction: a joint statement on management from the American Association of Clinical Endocrinologists, the American Thyroid Association, and the Endocrine Society. J Clin Endocrinol Metab.:90:581-7.
- 3. Michalakis KG, Mesen TB, Brayboy LM.(2011) Subclinical elevations of thyroid-stimulating hormone and assisted reproductive technology outcomes. Fertil Steril.;95:2634-7.

- 4. Squizzato A, Romualdi E, Piantanida E.(2007) Subclinical hypothyroidism and deep venous thrombosis. A pilot cross-sectional study. Thromb Haemost.;97:803.
- 5. Laukkarinen J, Kiudelis G, Lempinen M. (2007) Increased prevalence of subclinical hypothyroidism in common bile duct stone patients. J Clin Endocrinol Metab.;92:4260.
- 6. Dorgalaleh A, Mahmoodi M, Varmaghani B, Node FK.(2013) Effect of thyroid dysfunctions on blood cell count and red blood cell indices. Iran J Ped Haematol Oncol.;3(2):73-7.
- 7. Prasad R, Kumar V. (2005) Thyroid hormones increase Na+-Pi co-transport activity in intestinal brush border membrane: Role of membrane lipid composition and fluidity. Molecular and Cellular Biochemistry.; 278(1): 195-202.
- 8. Papadopoulos AS, Cleare AJ.(2012) Hypothalamic–pituitary–adrenal axis dysfunction in chronic fatigue syndrome. Nature Reviews Endocrinology.; 8(1): 22-32.
- 9. Kumar DV, Mathur DS, Tuteja DR. (2019) Effects of thyroid dysfunction on lipid profile. Int J Med Biomed Stud.; 3: 76-80.
- 10. Davis LM, Rho JM, Sullivan PG.(2008) UCP-mediated free fatty acid uncoupling of isolated cortical mitochondria from fasted animals: correlations to dietary modulations. Epilepsia.; 49: 117-119.
- 11. Chin KY, Ima-Nirwana S, Mohamed IN, Aminuddin A, Johari MH et al. (2014) The relationships between thyroid hormones and thyroid-stimulating hormone with lipid profile in euthyroid men. International journal of medical sciences.; 11(4): 349.
- 12. Rizos CV, Elisaf MS, Liberopoulos EN.(2011) Effects of thyroid dysfunction on lipid profile. The open cardiovascular medicine journal.; 5: 76.
- 13. Lieberman M, Marks AD.(2009) Marks' basic medical biochemistry: a clinical approach (2nd edition). Lippincott Williams & Wilkins.
- 14. Khan FA, Patil SKB, Thakur AS, Khan MF, Murugan K.(2014) Lipid Profile in Thyroid Dysfunction: A Study on Patients of Bastar Tiroid Fonksiyon Bozukluğu .TC, 322(51.19a).; 155-85.
- 15. Drews RE (2003) Critical issues in hematology: anemia, thrombocytopenia, coagulopathy, and blood product transfusions in critically ill patients. Clin Chest Med 24, 607-622.
- 16. Kawa MP, Grymula K, Paczkowska E, Baskiewicz-Masiuk M, Dabkowska E, Koziolek M, Tarnowski M, Klos P, Dziedziejko V, Kucia M, et al. (2010) Clinical relevance of thyroid dysfunction in human haematopoiesis: biochemical and molecular studies. Eur J Endocrinol 162, 295-305, doi: 10.1530/EJE-09-0875.
- 17. Das KC, Mukherjee M, Sarkar TK, Dash RJ & Rastogi GK (1975) Erythropoiesis and erythropoietin in hypo- and hyperthyroidism. J Clin Endocrinol Metab 40, 211-220, doi: 10.1210/jcem-40-2-211.
- 18. Fein HG & Rivlin RS (1975) Anemia in thyroid diseases. Med Clin North Am 59, 1133-1145.
- 19. Dorgalaleh A, Mahmoodi M, Varmaghani B, Kiani Node F, Saeeidi Kia O, Alizadeh S, Tabibian S, Bamedi T, Momeni M, Abbasian S, et al. (2013) Effect of thyroid dysfunctions on blood cell count and red blood cell indice. Iran J Ped Hematol Oncol 3, 73-77.
- 20. Geetha J & Srikrishna R (2012) Role of red blood cell distribution width (rdw) in thyroid dysfunction. Int J Biol Med Res 3, 1476-1478.
- 21. Fazio S, Palmieri EA, Lombardi G & Biondi B (2004) Effects of thyroid hormone on the cardiovascular system. Recent Prog Horm Res 59, 31-50.

- 22. Streeten DH, Anderson GH, Jr., Howland T, Chiang R & Smulyan H (1988) Effects of thyroid function on blood pressure. Recognition of hypothyroid hypertension. Hypertension 11, 78-83.
- 23. Prisant LM, Gujral JS & Mulloy AL (2006) Hyperthyroidism: a secondary cause of isolated systolic hypertension. J Clin Hypertens (Greenwich) 8, 596-599.
- 24. Brenta G, Danzi S & Klein I (2007) Potential therapeutic applications of thyroid hormone analogs. Nat Clin Pract Endocrinol Metab 3, 632-640, doi: 10.1038/ncpendmet0590.
- 25. Goglia F, Moreno M & Lanni A (1999) Action of thyroid hormones at the cellular level: the mitochondrial target. FEBS Lett 452, 115-120.
- 26. Kordonouri O, Maguire AM, Knip M, Schober E, Lorini R, Holl RW & Donaghue KC (2009) Other complications and associated conditions with diabetes in children and adolescents. Pediatr Diabetes 10 Suppl 12, 204-210, doi: 10.1111/j.1399-5448.2009.00573.x.
- 27. Barker JM, Yu J, Yu L, Wang J, Miao D, Bao F, Hoffenberg E, Nelson JC, Gottlieb PA, Rewers M, et al. (2005) Autoantibody "subspecificity" in type 1 diabetes: risk for organ-specific autoimmunity clusters in distinct groups. Diabetes Care 28, 850-855.
- 28. Kadiyala R, Peter R & Okosieme OE (2010) Thyroid dysfunction in patients with diabetes: clinical implications and screening strategies. Int J Clin Pract 64, 1130-1139, doi: 10.1111/j.1742-1241.2010.02376.x.
- 29. Loevy HT, Aduss H & Rosenthal IM (1987) Tooth eruption and craniofacial development in congenital hypothyroidism: report of case. J Am Dent Assoc 115, 429-431.
- 30. Poumpros E, Loberg E & Engstrom C (1994) Thyroid function and root resorption. Angle Orthod 64, 389-393; discussion 394, doi: 10.1043/0003-3219(1994)064<0389:TFARR>2.0.CO;2.
- 31. Young ER (1989) The thyroid gland and the dental practitioner. J Can Dent Assoc 55, 903-907.