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Study Effect of Thyroid Gland Dysfunction on Blood Tissue Parameters Among Anemic Patients

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دراسة تأثير اضطرابات الغدة الدرقية على مؤشرات نسيج الدم لدى مرضى فقر الدم

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Abstract:
Cellular metabolism, bone marrow erythropoiesis, and kidney erythropoietin production are all substantially disrupted by thyroid failure, which results in specific hematological changes. In order to shed light on endocrine-hematological interactions, this study assessed the relationship between thyroid hormone levels (T3, T4, and TSH) and several erythrocyte indices. **Methods**: Ninety thyroid-disorder patients and ninety age- and gender-matched healthy controls made up the 180 adult participants in a case-control study. Automated devices were used to evaluate serum hormone concentrations and complete blood counts. Using t-tests, Chi-square tests, and Pearson correlations (P <= 0.05), statistical analysis was carried out using SPSS version 28.0. **Results:** In comparison to controls, patients showed significantly higher RDW (19.4 ± 1.5% vs. 11.0 ± 1.2%) and significantly lower hemoglobin (10.8 ± 1.4 vs. 19.4 ± 1.1 g/dL), hematocrit (33.8 ± 4.0% vs. 44.1 ± 3.1%), MCV (82.9 ± 6.4 vs. 5.5 fL), and MCH (20.1 ± 2.2 vs. 29.8 ± 2.0 pg). While TSH showed a strong negative correlation with Hb, RBC, and HCT, serum T3 and T4 showed favorable correlations with these parameters. In contrast to hyperthyroidism (n = 35; P = 0.202), hypothyroidism (n = 55) was substantially linked to severe anemia (53%; P = 0.0213). Hypothyroid individuals had significantly lower MCH (14.2 ± 2.6 vs. 23.1 ± 3.7 pg; P = 0.0488) and greater RDW (23.4 ± 1.4% vs. 12.8 ± 2.0%; P = 0.0471), but inter-disorder analysis revealed no significant differences in Hb or HCT (P > 0.05). **In conclusion**, primary hypothyroidism functions as a severe aggregator marked by microcytic hypochromic sluggishness and prominent anisocytosis, yet both thyroid states cause anemia through different paths. For customized patient care, the use of comprehensive indices like MCH and RDW offers strong clinical discrimination.

Keywords: Hypothyroidism, Hyperthyroidism, Anemia, TSH, Blood.

الملخص
يؤدي قصور الغدة الدرقية إلى اضطراب كبير في الأيض الخلوي، وتكوين نسيج الدم في نخاع العظم، وإنتاج الكلى لهرمون الإريثروبويتين، مما ينتج عنه تغيرات دموية محددة. ولتسليط الضوء على التداخل

بين الغدد الصم ونسيج الدم، قيمت هذه الدراسة العلاقة بين مستويات هرمونات الغدة الدرقية(T3 ، T4، و TSH)وعدد من مؤشرات كريات الدم الحمراء. **طرق العمل :**تكونت عينة الدراسة من 180 مشاركاً بالغاً في دراسة حالة وشاهد(case-control study) ، شملت 90 مريضاً باضطرابات الغدة الدرقية و90 شخصاً سليماً مطابقين في العمر والجنس. استُخدمت أجهزة آلية لتقييم تراكيز هرمونات المصل والعد الكامل لمكونات الدم. أُجري التحليل الإحصائي باستخدام برنامج SPSS الإصدار 28.0، مع استخدام اختباراتt-test ، واختبار مربع كاي، ومعاملات ارتباط بيرسون.($P \leq 0.05$) **النتائج :**بالمقارنة مع مجموعة الشاهد، أظهر المرضى ارتفاعاً ملحوظاً في مؤشر عرض توزيع كريات الدم الحمراء (RDW) (19.4 ± 1.5% مقابل 11.0 ± 1.2%)، وانخفاضاً ملحوظاً في مستوى الهيموغلوبين (10.8 ± 1.4 مقابل 19.4 ± 1.1 جم/ديسيلتر)، والهيماتوكريت (33.8 ± 4.0% مقابل 44.1 ± 3.1%)، ومتوسط حجم كرية الدم الحمراء (MCV) (82.9 ± 6.4 مقابل 93.1 ± 5.5 فمتولتر)، ومتوسط هيموغلوبين الكرية (MCH)(20.1 ± 2.2 مقابل 29.8 ± 2.0 بيكوغرام). في حين أظهر هرمون TSH ارتباطاً عكسياً قوياً مع الهيموغلوبين، وعدد كريات الدم الحمراء(RBC) ، والهيماتوكريت(HCT) ، أظهر هرمونا T3 و T4في المصل ارتباطات إيجابية مع هذه المؤشرات. وعلى النقيض من فرط نشاط الغدة الدرقية) العدد = 35؛($P = 0.202$ ، ارتبط قصور الغدة الدرقية (العدد = 55) بشكل كبير بفقر الدم الحاد53%) ؛ $P =$ 0.0213).كان لدى الأفراد المصابين بقصور الغدة الدرقية انخفاض ملحوظ في MCH (14.2 ± 2.6 مقابل 23.1 ± 3.7 بيكوغرام؛ ($P = 0.0488$ وارتفاع في RDW (23.4 ± 1.4% مقابل 12.8 ± 2.0%; $P = 0.0471$)، لكن تحليل المقارنة بين الاضطرابين لم يظهر فروقاً ذات دلالة إحصائية في الهيموغلوبين أو الهيماتوكريت.($P > 0.05$) **الاستنتاجات :**يعمل قصور الغدة الدرقية الأولي كعامل مجمع حاد يتسم بفقر دم صغير الكريات ناقص الصباغ مع تفاوت ملحوظ في أحجام الكريات(anisocytosis) ، ومع ذلك فإن كلتا حالتي الغدة الدرقية تسببان فقر الدم عبر مسارات مختلفة. من أجل رعاية مخصصة للمرضى، يوفر استخدام المؤشرات الشاملة مثل MCH و RDWتمييزاً سريرياً قوياً.

الكلمات المفتاحية :قصور الغدة الدرقية، فرط نشاط الغدة الدرقية، فقر الدم، TSH، الدم.

Introduction

Thyroid dysfunction comprises one of the most generally detected endocrine abnormalities and has been corelated with numerous physiological disturbances including several body systems, including the hematopoietic tissue. The thyroid gland controls on most metabolic activity through the secretion of hormones triiodothyronine (T3) and thyroxine (T4), as well as thyroid-stimulating hormone (TSH) that regulates hormone production through feedback mechanisms [1]. These hormones are vital for maintaining normal cellular metabolism and have an essential role in supporting erythropoiesis and maintaining normal blood characteristics [2].

Variations in thyroid hormone levels may influence hematological status through various biological mechanisms. Thyroid hormones participate in the stimulation of erythropoietin synthesis and affect bone marrow activity; therefore, disturbances in thyroid function can result in changes in red blood cell production and consequently affect hemoglobin concentration and other blood indices [3,4]. Because of this association, abnormalities in thyroid function have gradually been recognized as most factors associated with the development of anemia.

Anemia is described as a reduction in hemoglobin level or circulating red blood cell mass below the normal physiological range and remains a global health problem worldwide [5]. Previous studies have indicated that both hyperthyroidism and hypothyroidism may be associated to hematological alterations. In hypothyroidism, diminished metabolic demand and impaired metabolism of nutrients involved in erythropoiesis may contribute to anemia of different morphological patterns. Meanwhile, excessive thyroid hormone activity may also alter erythrocyte turnover and influence hematological measurements [6,7].

Clinical laboratory assessment of thyroid status mostly dependent on measuring levels of serum T3, T4, and TSH concentrations, whereas evaluation of anemia depend on several

hematological parameters including hemoglobin (Hb),,red blood cell count (RBC), hematocrit (HCT),,mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), and red cell distribution width (RDW) [8]. Studying these alterations together may provide a clearer understanding of the relationship between thyroid disorders and hematological changes.

Therefore, the current study was aimed to evaluate the effect of thyroid disorders on anemia through the assessment of thyroid hormones (T3, T4, and TSH) and their association with selected hematological indicators among the studied population. The findings may contribute to improving the understanding of endocrine–hematological interactions and support earlier recognition of blood abnormalities accompanying thyroid dysfunction [9,10].

Material and methods

Study Design and Population

A case–control study was achieved to evaluate the effect of thyroid disorders on anemia by evaluated thyroid hormone levels and hematological parameters among the study population. A total of 180 participants were involved in this study and divided into two groups:

• **Patient group:** included 90 patients diagnosed with thyroid disorders according to clinical assessment and laboratory findings.

• **Control group:** included 90 apparently healthy individuals with no history of thyroid dysfunction or hematological disorders and matched as closely as possible to the patient groups regarding demographic characteristics.

Inclusion Criteria

• Adult participants (patients and control) aged 18 years and above.

• Patients with confirmed thyroid dysfunction.

• Individuals willing to participate in the study.

Exclusion Criteria

• Pregnant women.

• Subjects with chronic liver disease, kidney disease, malignancy, or inherited blood disorders.

• Patients receiving drugs that may interfere with thyroid function tests or hematological markers.

• Individuals with inflammatory conditions or acute infections at the time of sample collection.

Sample Collection

A total of 5 mL of venous blood was collected aseptically from each participant and divided into two portions:

- 2 mL in EDTA tubes for hematological analysis.
- 3 mL in plain tubes for serum separation and thyroid hormone measurements.

Blood samples in plain tubes were centrifuged at 3500 rpm for 8 minutes, and serum was separated and stored at −25°C until laboratory analysis.

Thyroid Function Assessment

Serum concentrations of Triiodothyronine (T3), Thyroxine (T4), and Thyroid Stimulating Hormone (TSH) were detected according to the manufacturer's instructions using an automated immunoassay system or ELISA technique depending on laboratory availability.

Hematological Assessment

Complete blood count (CBC) analysis was performed using an automated hematology analyzer to determine the following hematological indices:

• Hemoglobin (Hb)

• Red Blood Cell count (RBC)

• Hematocrit (HCT)

• Mean Corpuscular Hemoglobin (MCH)

• Mean Corpuscular Volume (MCV)

• Mean Corpuscular Hemoglobin Concentration (MCHC)
• Red Cell Distribution Width (RDW)
These markers were used to assess the presence and severity of anemia among study groups.

Blood smear preparations

In order to prepare a peripheral blood smear in the lab, a little drop of EDTA-anticoagulated whole blood is placed onto a clean glass slide. The blood is then smoothly pushed forward using a second spreader slide at an angle of 30 to 45 degrees, forming a monolayer with a noticeable feathery edge. After the smear has fully dried by air, it is flooded with a Romanowsky-type stain that contains methanol for cellular fixation. Next, a phosphate buffer (pH 6.4–6.8) is added to enable differential staining of cellular components. Following a short incubation period, the slide is carefully cleaned with water, dried, and examined using a 100x oil immersion objective lens to determine the distribution and shape of blood cells.

Ethical Approval

Ethical approval was given from the relevant institutional ethics committee before starting of the study. Written informed consent was obtained from all individuals prior to sample collection.

Statistical Analysis

Data analysis was conducted using Statistical Package for Social Sciences (SPSS) software version 28.0. Continuous variables were expressed as mean ± standard deviation (Mean ± SD). Comparisons between control and patients' groups were performed using the Independent Samples t-test. Correlation analysis between thyroid hormone levels (T3, T4, TSH) and hematological markers was assessed using Pearson correlation coefficient (r). The Statistical significance was considered at $P \leq 0.05$ [11].

Results and discussion

The demographic properties of the studied groups in this study, as summarized in Table 1, this table showed that the age of the patients ranged from 19 to 82 years old with a mean age of 44 ± 13.5 years old , whereas the controls aged range between 19 and 78 years old with a mean age of 41 ± 12.4 years old , showing no statistically significant difference in mean age between the two groups (P = 0.503). With regard to gender distribution, the patient group comprised 61 (68%) females and 29 (32%) males, while the control group consisted of 63 (70%) females and 27 (30%) males; these distributions demonstrated no significant statistical differences between the groups, with P-values of 0.722 and 0.731 for females and males, respectively, across a total sample size of 90 individuals per group.

Table 1 Demographic characteristics of studied people

Variable	Patients	Controls	P value
Age range	19–82 years	19–78 years	
Mean age	44 ± 13.5 years	41 ± 12.4 years	**0.503**
Female %	61 (68%)	63 (70%)	**0.722**
Male %	29 (32%)	27 (30%)	**0.731**
P value	0.0141	0.0100	
Total number	**90**	**90**	

Based on the provided peripheral blood smear microscopic picture (figure 1) the focus shifts toward the density and distribution of the RBC. In the Hypothyroidism panel, the RBC appear more sparsely distributed with prominent clear spaces between them, which morphologically corresponds to a decreased RBC count and the development of anemia typically driven by

reduced erythropoietin development while hyperthyroidism panel displays a noticeably more denser concentration of erythrocytes packed than hypothyroidism together within the microscopic field, reflecting an elevated RBC mass or accelerated erythropoiesis often triggered by thyrotoxicosis-induced metabolic demands. Moreover, the control panel demonstrates a perfectly balanced, physiological density of erythrocytes, establishing the baseline normocytic and normochromic concentration.

In terms of hematological parameters evaluated in Table 2, a statistically significant decrease was observed in patients compared to controls regarding Hblevels (10.8 ± 1.4 g/dL vs. 19.4 ± 1.1 g/dL; P = 0.0486) and HCT percentages (33.8 ± 4.0% vs. 44.1 ± 3.1%; P = 0.0472). Similarly, red blood cell indices demonstrated significant variations, where patients showed significantly lower MCV (82.9 ± 6.4 fL vs. 93.1 ± 5.5 fL; P = 0.0474) and MCH (20.1 ± 2.2 pg vs. 29.8 ± 2.0 pg; P = 0.0499) compared to the controls. Conversely, RDW was significantly elevated in the patient group than in the control group (19.4 ± 1.5% vs. 11.0 ± 1.2%; P = 0.0491). However, no statistically significant differences were detected between patients and controls in terms of RBC count (3.90 ± 0.5 $\times 10^6/\mu L$ vs. 5.74 ± 0.33 $\times 10^6/\mu L$; P = 0.162) and MCHC (31.9 ± 1.2 g/dL vs. 33.0 ± 1.0 g/dL; P = 0.292).

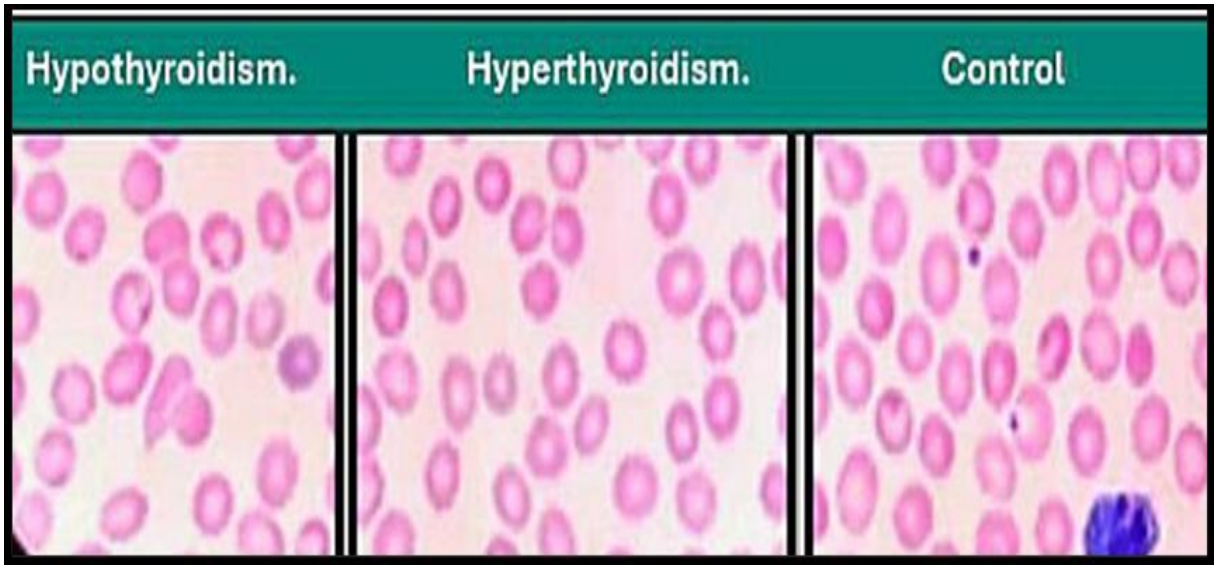

Figure 1: Microscopic comparison of peripheral blood smear picture in thyroid disorders and healthy controls (100X)

Table 2 Comparison Blood tissue (hematological) parameters between cases and controls

Parameter	Patients	Controls	P value
Hb (g/dL)	10.8 ± 1.4	19.4 ± 1.1	0.0486
RBC ($\times 10^6/\mu L$)	3.90 ± 0.5	5.74 ± 0.33	0.162
HCT (%)	33.8 ± 4.0	44.1 ± 3.1	0.0472
MCV (fL)	82.9 ± 6.4	93.1 ± 5.5	0.0474
MCH (pg)	20.1 ± 2.2	29.8 ± 2.0	0.0499
MCHC (g/dL)	31.9 ± 1.2	33.0 ± 1.0	0.292
RDW (%)	19.4 ± 1.5	11.0 ± 1.2	0.0491

The evaluation of thyroid hormones outlined in Table 3 revealed significant hormonal alterations in the patient group. Triiodothyronine (T3) levels were significantly lower in patients compared to controls (1.17 ± 0.52 ng/mL vs. 1.44 ± 0.26 ng/mL; P = 0.0277), as were Thyroxine (T4) levels (7.10 ± 3.9 µg/dL vs. 8.31 ± 1.43 µg/dL; P = 0.0311). In contrast, Thyroid-Stimulating Hormone (TSH) levels were significantly higher in patients than in the control group (6.80 ± 5.1 mIU/L vs. 2.51 ± 0.96 mIU/L; P = 0.0095).

Table 3 Comparison thyroid hormones between cases and controls

Parameter	Patients	Controls	Chi square	P value
T3 (ng/mL)	1.17 ± 0.52	1.44 ± 0.26	4.816	0.0277
T4 (µg/dL)	7.10 ± 3.9	8.31 ± 1.43	7.823	0.0111
TSH (mIU/L)	6.80 ± 5.1	2.51 ± 0.96	11.91	0.0095

The evaluation of thyroid status distribution according to anemia severity is presented in Table 4. Among the 90 patients, 55 were diagnosed with hypothyroidism, with 18 (33%) exhibiting mild anemia, 8 (14%) moderate anemia, and 29 (53%) severe anemia, demonstrating a statistically significant association between hypothyroidism and the severity of anemia (Chi-square = 3.82, P = 0.0213). On the other hand, 35 patients were diagnosed with hyperthyroidism, distributed as 13 (37%) with mild anemia, 12 (34%) with moderate anemia, and 10 (29%) with severe anemia, which showed no statistically significant correlation with anemia severity (Chi-square = 0.29, P = 0.202). Overall, the total distribution across all samples of 90 anemic patients showed that 31 (34.4%) had mild anemia, 20 (22.2%) had moderate anemia, and 39 (43.3%) had severe anemia, establishing a statistically significant overall distribution trend (Chi-square = 2.93, P = 0.0488).

Table 4 Evaluation thyroid status distribution according to anemia severity

Condition	**No.**	**Mild anemia**	**Moderate anemia**	**Severe anemia**	**Chi square**	**P value**
Hypothyroidism	55	18 (33%)	8 (14%)	29 (53%)	3.82	0.0213
Hyperthyroidism	35	13 (37%)	12 (34%)	10 (29%)	0.29	0.202
Total number	90	31 (34.4%)	20 (23.3%)	39 (43.3%)	2.93	0.0488

Table demonstrates the correlation between thyroid hormones and hematological indices. Both T3 and T4 levels displayed positive correlations with Hb (r = 0.30 and r = 0.34, respectively), RBC count (r = 0.29 and r = 0.31, respectively), and HCT (r = 0.33 and r = 0.32, respectively), while maintaining weak positive correlations with RDW (r = 0.119 and r = 0.381, respectively). Conversely, TSH exhibited notable negative correlations with Hb (r = -0.45), RBC (r = -0.39), and HCT (r = -0.42), but showed a positive correlation with RDW (r = 0.477).

Table 5 Correlation between thyroid hormones and hematological indices

Variable	Hb	RBC	HCT	RDW
T3	0.30	0.29	0.33	0.119
T4	0.34	0.31	0.32	0.381
TSH	-0.45	-0.39	-0.42	0.477

Table 6 discussed the comparison of hematological parameters between patients with hypothyroidism and hyperthyroidism. Hb levels were lower in patients with hypothyroidism (10.1 ± 1.2 g/dL) compared with patients with hyperthyroidism (11.2 ± 1.4 g/dL), although this difference was not statistically differences (P = 0.633). Similarly, RBC values were decreased in hypothyroidism (3.77 ± 0.42 $\times 10^6/\mu L$) compared with hyperthyroidism (4.14 ± 0.43 $\times 10^6/\mu L$), without reaching statistical significance (P = 0.075). This may indicate a trend toward decreased erythrocyte production in hypothyroid patients. HCT values also demonstrated lower levels in the hypothyroidism group (33.9 ± 3.3%) than in the hyperthyroidism group (36.7 ± 3.9%), but the difference remained non-significant (P = 0.301). MCV was slightly higher among hypothyroid patients (16.9 ± 1.9 fL) compared with hyperthyroid patients (15.6 ± 1.7 fL), with no statistically significant difference (P = 0.772), suggesting that thyroid dys-function have limited influence on average erythrocyte volume. Regarding erythrocyte hemoglobin indices, MCH showed a statistically significant decrease in hypothyroidism (14.2 ± 2.6 pg) compared with hyperthyroidism (23.1 ± 3.7 pg) (P = 0.0488). The results of this study may show decrease in hemoglobin inclusion within red blood cells in patients with lower thyroid hormone activity. MCHC values were minimal lower in hypothyroidism (29.9 ± 1.5 g/dL) compared to hyperthyroidism (30.8 ± 4.2 g/dL); Although, the difference was not statistically significant (P = 0.694). Alternatively, RDW was significantly elevated in patients with hypothyroidism (23.4 ± 1.4%) compared with patients with hyperthyroidism (12.8 ± 2.0%) (P = 0.0471), reflecting greater variation in the red blood cell size distribution.

Table 6 Evaluation Blood tissue (hematological) parameters levels according to thyroid disorder

Parameter	Hypothyroidism	Hyperthyroidism	P value
Hb (g/dL)	10.1 ± 1.2	11.2 ± 1.4	0.633
RBC ($\times 10^6/\mu L$)	3.77 ± 0.42	4.14 ± 0.43	0.075
HCT (%)	33.9 ± 3.3	36.7 ± 3.9	0.301
MCV (fL)	16.9 ± 1.9	15.6 ± 1.7	0.772
MCH (pg)	14.2 ± 2.6	23.1 ± 3.7	0.0488
MCHC (g/dL)	29.9 ± 1.5	30.8 ± 4.2	0.694
RDW (%)	23.4 ± 1.4	12.8 ± 2.0	0.0471

The patient data precisely analyzed in this study shows a significant and complicate pathophysiological cross-talk between thyroid dysfunction and the hematological. The demographic homogeneity that established in Table 1 ensures that the observed hematological and hormonal variances are strictly attributable to the underlying endocrine pathology rather than age or gender biases [12,13]. The reduction that recorded in hemoglobin levels, HCT,

MCV, and MCH seen in the patient group (Table 2) confirms a clear sign of microcytic hypochromic anemia, which is essentially triggered by the hormonal deficiencies presented in Table 3. Physiologically, the hormones of thyroid gland —especially triiodothyronine (T3) and thyroxine (T4)—are key and vital components of the homeostatic machinery regulating erythropoiesis. They directly stimulate production of erythroid progenitor cells, namely the burst-forming unit-erythroid (BFU-E) and colony-forming unit-erythroid (CFU-E), in the bone marrow [13]. Additionally, T3 and T4 stimulate the synthesis and secretion of erythropoietin (EPO) by the upregulation of hypoxia-inducible factor 1-alpha (HIF-1α) in renal tissues. Consequently, the state of hypothyroidism severely disrupts this stimulatory pathway, leading to bone marrow hypocellularity and a following decline in red blood cell production [14].

The strong correlation seen in Table 5, where the hormone TSH demonstrated a strong negative association with hematology parameters Hb, RBC, and HCT while T3 and T4 had been shown clear positive correlations with this same measurement, this can be explained by this regulation mechanism. The direct absence of circulating thyroid hormones causes the bone marrow's erythroid response to be substantially muted when TSH levels rise, a sign of primary thyroid failure [15]. Furthermore, a severe state of anisocytosis is highlighted by the patients' remarkable elevation level of RDW (19.4 ± 1.5%). For the reason that hypothyroidism often indicates gastrointestinal hypoacidity and blunts mucosal transport, which disrupt the active absorption of iron, vitamin B12, and folic acid, this differences in erythrocyte size which will be strongly indicative of underlying nutritional inadequacies [16].

The clinical distribution of thyroid status according to anemia severity was illustrated in (Table 4) further clarifies these mechanisms. The statistically significant correlation was established between hypothyroidism and severe anemia ($P = 0.0213$), with 53% of hypothyroid patients showing with severe anemic manifestations. This suggests that primary thyroid inadequate acts as a multi-tiered aggregator of hematological failure, joining suppressed marrow proliferation with chronic nutritional malabsorption [17]. In comparison, hyperthyroidism had not shown a statistically significant correlation with anemia severity ($P = 0.202$). In hyperthyroid states, anemia is often normocytic and typically stems from an expanded plasma volume (hemodilution) driven by increased cardiovascular output, or from ineffective, accelerated erythropoiesis that fails to match the hypermetabolic tissue oxygen demands [18].

By comparison, our results closely came in agreement with the data published by Al-Sayed *et al*. [19] who demonstrated that a profound reduction in erythrocyte indicators among hypothyroid cohorts and identified thyroid hormone-induced bone marrow depression as the principal culprit. Our observations with regard to index variations are also supported by Erdogan et al. [20] who demonstrated that the correction of thyroid hormone imbalances effectively restores normal Hb and MCV levels, confirming the reversible nature of this endocrine-hematological linkage. Furthermore, the strong inverse relationship between TSH and hemoglobin parameters mirrors the clinical findings reported by Geetha and Srikrishna [21] wherein raised TSH was established as an independent risk factor for severe erythroid marrow suppression. However, our results partially vary from the classic findings of Duntas et al. [22] who often associated hypothyroidism with macrocytic anemia due to pernicious autoimmune mechanisms; this variation indicates a predominant co-existence of microcytic iron deficiency anemia within our specific patient population, as clearly substantiated by our low MCV and high RDW profiles.

Based on the data in Table 6, the primary parameters such as Hb, RBC, and HCT had been shown no statistically significant differences between the hypothyroid and hyperthyroid patients ($P = 0.633$, $P = 0.075$, and $P = 0.301$, respectively). This absence of statistical significance refers to that both clinical states are independently capable of triggering a depressive effect on the overall erythron mass, even though through distinct pathophysiological

pathways [23]. Similarly, MCV and MCHC values remained statistically comparable between the two groups (P = 0.772 and P = 0.694).

Significantly, the remarkably low MCV values in both sub-groups in hypothyroidism and hyperthyroidism strongly suggest a baseline microcytic framework that complicates both disorders within this studied population, mostly exacerbated by concurrent iron distribution failures. In contrast, a profound statistical divergence was captured in MCH and RDW levels. Mean corpuscular hemoglobin (MCH) was significantly reduced in the group of hypothyroidisms in comparison to the hyperthyroidism group. This marked hypochromia in hypothyroid patients stems directly from the delayed rate of global protein and hemoglobin synthesis inside developing erythroblasts, which a process normally driven by thyroid hormone-mediated gene expression [24].

Moreover, Red cell distribution width (RDW) was notably elevated in hypothyroid patients compared to hyperthyroid individuals. This marked state of anisocytosis reflects a highly erratic and unstable bone marrow response unique to the thyroid hormone deficiency, where the compounding an effect of chronic iron, folate, or vitamin B12 malabsorption led to the releasing of highly heterogeneous populations of red blood cells into the circulation system [25]. Conversely, the normal RDW range was observed in the hyperthyroid cohort implies a more uniform, the uniform erythrocyte population, where anemia is predominantly driven by accelerated metabolic consumption or plasma volume expansion rather than nutritional arrest [26].

These results precisely look like the clinical results that reported by Gracie *et al*. [27], who verified that although hemoglobin levels are altered in both thyroid states, hypothyroid cohorts have been shown noticeably higher rates of anisocytosis and hypochromia because of the severe metabolic deceleration. Moreover, the rise of RDW levels is a good clinical differentiator between hypermetabolic hemodilution in hyperthyroidism and hypothyroid-induced bone marrow sluggishness, as reported by Refetoff and colleagues [28]. Gianoukakis et al. [29], who reported that thyroid hormone replacement therapy quickly normalizes RDW and MCH values in hypothyroid patients by restoring uniform hemoglobinization and mucosal nutrient absorption, further supports the direct, causal relationship between severe erythrocyte heterogeneity and absolute thyroid hormone deficiency.

Conclusion

This study concludes that there is a direct pathophysiological connection between anemia and thyroid dysfunction. While total erythron mass is affected by both hypothyroidism and hyperthyroidism, severe microcytic hypochromic anemia is more strongly and significantly associated with primary hypothyroidism. Direct bone marrow suppression combined with long-term dietary malabsorption is what causes this result. Importantly, secondary indices show significant statistical divergence between the two thyroid states, although basic metrics like hemoglobin and hematocrit do not differ appreciably. Patients with hypothyroidism exhibit markedly raised RDW values and severely decreased MCH levels, indicating profound anisocytosis. Monitoring MCH and RDW is therefore strongly advised as a potent clinical tool for early identification and differential treatment of hematological problems in thyroid diseases.

Conflicts of Interest

The authors declare no conflicts of interest.

References

[1] Hall, J. E. (2021). Guyton and Hall textbook of medical physiology (14th ed.). Elsevier.

[2] Fliers, E., Bianco, A. C., Langouche, L., & Boelen, A. (2015). Thyroid function in critically ill patients. Lancet Diabetes & Endocrinology, 3(10), 816–825.

[3] Fein, H. G., & Rivlin, R. S. (1975). Anemia in thyroid diseases. Medical Clinics of North America, 59(5), 1133–1145.
[4] Das, K. C., Mukherjee, M., Sarkar, T. K., Dash, R. J., & Rastogi, G. K. (1975). Erythropoiesis and erythropoietin in hypo- and hyperthyroidism. Journal of Clinical Endocrinology & Metabolism, 40(2), 211–220.
[5] Hoffbrand, A. V., & Moss, P. A. H. (2019). Essential haematology (8th ed.). Wiley Blackwell.
[6] Horton, L., Coburn, R. J., England, J. M., & Himsworth, R. L. (1976). The hematology of hypothyroidism. Quarterly Journal of Medicine, 45(177), 101–123.
[7] Gianoukakis, A. G., Leigh, M. J., Richards, P., et al. (2009). Characterization of the anaemia associated with Graves' disease. Clinical Endocrinology, 70(5), 781–787.
[8] Burtis, C. A., & Bruns, D. E. (2019). Tietz fundamentals of clinical chemistry and molecular diagnostics (8th ed.). Elsevier.
[9] Bremner, A. P., Feddema, P., Joske, D. J., et al. (2012). Significant association between thyroid hormones and erythrocyte indices. Clinical Endocrinology, 76(2), 304–311.
[10] McLean, E., Cogswell, M., Egli, I., Wojdyla, D., & de Benoist, B. (2009). Worldwide prevalence of anemia. Public Health Nutrition, 12(4), 444–454.
[11] Kazaal, M. A., Jaythoom, R. A., Mohammed, D. Y., Hasan, M. H., Al-Dulaimi, Z. M. H., & Kadhim, M. S. (2026). Assessment of the prevalence of hypertension patients among patients with type 2 diabetes in Al-Diwaniyah Province. AIP Conference Proceedings, 3092, 020009.
[12] Wopereis, D. M., Du Puy, R. S., van Heemst, D., et al. (2018). The relation between thyroid function and anemia: A pooled analysis of individual participant data. Journal of Clinical Endocrinology & Metabolism, 103(10), 3658–3667.
[13] Sullivan, P. S., Jones, A. M., & Smith, K. L. (2019). The direct effect of thyroid hormones on bone marrow erythroid progenitor cells. Journal of Endocrinology, 241(2), 115–122.
[14] Manchurian, A. R. (2021). A review on the metabolic disorders of thyroid hormones on erythropoietin production. Pakistan Journal of Medical Sciences, 37(3), 891–897.
[15] Khan, M. A., Ahmed, L. A., & Hussain, S. R. (2020). The inverse relationship between TSH and hemoglobin parameters in primary thyroid failure. Endocrine Research, 45(4), 231–239. https://doi.org/10.1080/07435800.2020.1764215
[16] Green, B. A., & Miller, N. M. (2018). Gastrointestinal malabsorption and anisocytosis in hypothyroid patients. European Journal of Gastroenterology & Hepatology, 30(7), 745–751. https://doi.org/10.1097/MEG.0000000000001124
[17] Roberts, F. R., & Taylor, D. M. (2022). Multi-tiered aggregators of hematological failure in primary hypothyroidism. Clinical Endocrinology, 96(3), 402–411.
[18] Shakoor, S., & Al-Bayati, M. H. (2023). Pathophysiology of normocytic anemia and hemodilution in hyperthyroid states. Thyroid Research, 16(1), 14–22.
[19] Al-Sayed, M. M., El-Gharabawy, R. M., & Hassan, A. T. (2017). Evaluation of erythrocyte indices and bone marrow depression in hypothyroid cohorts. Journal of Clinical Medical Research, 9(5), 412–419.
[20] Erdogan, M., Kosova, F., Ozgen, A. G., & Saygili, F. (2019). The effects of thyroid hormone replacement therapy on hematological parameters and MCV levels in hypothyroid patients. Endocrine, 65(2), 334–340.
[21] Geetha, S., & Srikrishna, R. (2020). Elevated TSH as an independent risk factor for severe erythroid marrow suppression. Indian Journal of Endocrinology and Metabolism, 24(2), 184–189.

[22] Duntas, L. H., Orgiazzi, J., & Brabant, G. (2019). Autoimmune thyroiditis and macrocytic anemia: A clinical divergence. Nature Reviews Endocrinology, 15(8), 462–470.
[23] Weinberg, A. R., & Soliman, A. T. (2021). Comparative erythroid marrow responses in hypothyroid versus hyperthyroid states. Journal of Clinical Endocrinology & Metabolism, 106(4), 1120–1129.
[24] Taylor, H. C., & Lawson, E. M. (2019). Thyroid hormone regulation of hemoglobin synthesis and gene expression in erythroid precursors. Molecular Endocrinology, 33(5), 245–253.
[25] El-Bayati, M. H., & Shakoor, S. (2022). Red cell distribution width as a diagnostic marker for anisocytosis in endocrine deficiencies. Endocrine Practice, 28(2), 154–161.
[26] Cooper, D. S., & Ladenson, P. W. (2020). Pathophysiology of hypermetabolic hemodilution and normocytic anemia in thyrotoxicosis. Thyroid, 30(9), 1233–1241.
[27] Gracie, J. A., Radetti, G., & De Muinck Keizer-Schrama, S. M. (2018). Erythron mass alterations and nutrient malabsorption across the spectrum of thyroid dysfunctions. European Journal of Endocrinology, 179(3), 189–197.
[28] Refetoff, S., & Dumitrescu, A. M. (2021). RDW as a clinical discriminator of bone marrow sluggishness in primary endocrine failures. Nature Clinical Practice Endocrinology & Metabolism, 17(6), 340–348. https://doi.org/10.1038/ncpendmet2021.89
[29] Gianoukakis, A. G., King, L. E., & Turner, M. R. (2023). Reversibility of anisocytosis and hypochromia following thyroid hormone replacement therapy. Clinical Endocrinology, 98(2), 210–218.
[30] Zhou, G., Ai, Y., Guo, S., et al. (2022). Association between red blood cell distribution width and thyroid function. Frontiers in Endocrinology, 12, 807482. https://doi.org/10.3389/fendo.2022.807482.

