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Evaluation of the interrelationship between selected blood components and thyroid physiology in Atherosclerosis

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ABSTRACT

Atherosclerosis is a chronic inflammatory disease characterized by the accumulation of fatty plaques in arterial walls, leading to luminal narrowing and reduced blood flow. Because thyroid function regulates lipid metabolism and inflammatory pathways, thyroid status and circulating inflammatory markers are important indicators of atherosclerotic risk. This study examined the relationship between thyroid function and blood parameters in atherosclerosis, evaluated the effect of thyroid dysfunction on disease development, and considered its implications for prevention and treatment. The study enrolled 140 participants divided into three groups: a control group (n = 40 healthy individuals), a conservative-treatment group (n = 50 patients receiving conservative therapy for atherosclerosis), and an atherosclerosis group (n = 50 patients taking anticoagulant drugs). Thyroid hormones, including total thyroxine (TT4), total triiodothyronine (TT3), free thyroxine (fT4), free triiodothyronine (fT3), and thyroid-stimulating hormone (TSH), and hematologic/biochemical parameters (red blood cells, white blood cells, platelets, hemoglobin, hematocrit, urea, creatinine) were measured in all participants. Compared with controls, the conservative-treatment and atherosclerosis groups showed significantly lower TT4, TT3, fT4, and fT3, along with higher TSH. In some patients, red blood cell count, hemoglobin, and hematocrit were decreased, while urea and creatinine were increased. These findings indicate an association between atherosclerosis and thyroid dysfunction, characterized by reduced thyroid hormone levels with elevated TSH, and suggest that atherosclerosis also affects blood components and renal function. These results may inform strategies for the prevention and treatment of atherosclerosis.

Keywords: *Arteriosclerosis, Blood components, Mutual relationship, Thyroid gland*

1 INTRODUCTION

Atherosclerosis is a widespread chronic inflammatory disease characterized by the gradual accumulation of fatty plaques in arterial walls, leading to luminal narrowing and reduced blood flow [1]. It is the leading cause of cardiovascular disease and is responsible for approximately 18 million deaths annually worldwide due to myocardial infarction, stroke, and related conditions [2, 3]. The disease disproportionately affects older adults and individuals with metabolic syndrome. Despite major advances in lowering low-density lipoprotein cholesterol (LDL-C), residual inflammation persists in 30-40% of patients, underscoring the need to explore immune-modulating therapies [4]. Early modification of traditional risk factors, such as hypertension, hypercholesterolemia, and smoking, is essential to slow disease progression and prevent major vascular events [5].

Attention has increasingly focused on the role of thyroid function in atherosclerosis [6, 7]. Thyroid hormones triiodothyronine (T3) and thyroxine (T4) are central to metabolic and cardiovascular regulation [8, 9]. They act via genomic and non-genomic mechanisms, including binding to nuclear receptors and modulating intracellular signaling pathways, thereby influencing energy expenditure, heart rate, and vascular tone [10]. Thyroid hormones increase mitochondrial activity and oxygen

consumption [11]. They promote catabolic processes (e.g., lipid oxidation), anabolic processes (e.g., gluconeogenesis) [12, 13], enhance insulin-dependent glucose uptake, stimulate gluconeogenesis and glycogenolysis, and interact with adrenergic signaling to amplify adaptive thermogenesis in brown adipose tissue [14]. They also regulate cholesterol metabolism through liver X receptor (LXR) and peroxisome proliferator-activated receptor (PPAR) pathways and support cardiovascular function by improving contractile performance through calcium handling and myosin ATPase activation, augmenting nodal activity and heart rate [15, 16], and reducing systemic vascular resistance and diastolic blood pressure [17]. These systemic effects highlight how disturbances in thyroid regulation can contribute to disorders ranging from hematologic abnormalities to heart failure [18].

Blood parameters reflect overall health status and are influenced by thyroid function [19, 20]. Lipid measures (cholesterol, triglycerides) and inflammatory markers are key indicators for assessing atherosclerotic risk. Risk stratification often relies on blood-based biomarkers that index lipid metabolism and inflammation. Elevated LDL-C is associated with the extent of subclinical atherosclerosis [21–23], and triglyceride-rich lipoproteins (TGRLs) may promote atherogenesis via lipolysis products and residual lipoprotein cholesterol (LP-C). Persistent residual inflammation in statin-treated patients has motivated interest in IL-1β/NLRP3-targeted therapies [24, 25]. Accordingly, this study investigates the relationship between thyroid function and blood parameters in atherosclerosis, explores the impact of thyroid status on disease development, and considers its potential use in prevention and treatment strategies.

2 MATERIALS AND METHODS

A total of 10 mL of venous blood was collected from the control group, patients with atherosclerosis, and the treatment group at Al-Kadhimiya Educational Hospital during the period from May 2022. Participants were aged 50–75 years.

2.1 Study setting and period

The research was conducted at Al Ramadi Teaching Hospital for Maternity and Children, Fallujah Teaching Hospital, and two private medical laboratories from December 2023 to August 2024. The study included 55 women who underwent legal abortion following intrauterine fetal death.

Approximately 2.5 mL of each sample was placed into ethylenediaminetetraacetic acid (EDTA) tubes (anticoagulant) and used immediately, without storage, for blood smear analysis. The remaining blood was transferred into clean, dry (plain) tubes and left at 37 °C for 10 minutes, then centrifuged at 3000 rpm for 10 minutes to separate the serum.

Urea and creatinine were measured directly from the obtained serum. The remaining serum was stored at -20 °C until thyroid hormone and thyroid-stimulating hormone (TSH) testing was conducted [26].

2.2 Patients groups

The study included 50 patients receiving conservative management and 50 patients with atherosclerosis who were taking anticoagulant drugs (e.g., aspirin), all aged 50-75 years. The control group comprised healthy individuals without cardiovascular or hematologic disease, diabetes, or thyroid disorders; this group included 40 men aged 50-75 years.

2.3 Creatinine concentration

Serum creatinine was measured by a colorimetric method with protein deproteinization using a diagnostic device from RANDOX (UK). The absorbance (A) of the sample solution and the standard solution was read at 520 nm against a blank solution [27].

2.4 Urea concentration

Serum urea was measured by an enzymatic method using a diagnostic kit from bioMérieux (France).

2.5 Complete blood count (cbc)

An automated hematology analyzer (Sysmex, Japan) was used to quantify blood cells in all samples. White blood cells (WBC) were counted in the WBC channel using the direct current (DC) method. Red blood cells (RBC) and platelets were counted in their respective channels using the DC method. Hemoglobin (HGB) concentration was measured in the HGB channel using the non-cyanide hemoglobin method (SYSMEX KX-2IN operating manual, 1999).

2.6 Measurement of serum

Serum TT4, TT3, TSH, T4, and T3 were measured using a miniVIDAS analyzer (bioMérieux, France) according to the manufacturer's kit instructions.

2.7 Statistical analysis

Data were collected and analyzed using SPSS version 14 for Windows (SPSS Inc., Chicago, IL, USA). One-way analysis of variance (ANOVA) was performed, and least significant difference (LSD) post hoc testing was used to identify pairwise differences. Age groups were not analyzed because the research did not require it. Ethical approval was granted according to order no. 285 on 26/12/2024.

3 RESULTS AND DISCUSSION

Table 1 shows significant decreases in the mean values of hematologic indicators, red blood cell (RBC) count, white blood cell (WBC) count, and hematocrit, in both the conservative-treatment group and the anticoagulant group compared with the control group ($p < 0.05$). The table also indicates a significant difference in hematocrit in the anticoagulant group compared with the conservative-treatment group ($p < 0.01$). In addition, RBC count was significantly lower in the anticoagulant group than in the conservative-treatment group ($p < 0.05$). No significant differences in platelet count were observed among the three groups.

Although total white blood cell (WBC) counts were within reference ranges in all three groups, the anticoagulant group showed a significant reduction in WBC count compared with the other groups ($p < 0.05$). On differential counting, the anticoagulant group demonstrated a significant increase in the percentage of WBCs compared with the control group ($p < 0.001$) and a significant decrease compared with the conservative-treatment group ($p < 0.05$). The table also shows a significant reduction in the percentage of lymphoid WBCs (lymphocytes) in patients with chronic atherosclerosis compared with controls.

Across the three groups, complete blood count parameters (RBC, HGB, HCT, PLT, WBC, LYM, NEUT) showed significantly lower red blood cells and hemoglobin in the conservative-treatment and atherosclerosis groups compared with controls, consistent with anemia due to reduced erythropoietin production, iron loss, and chronic inflammation.

Platelet (PLT) levels in atherosclerosis were slightly lower than in controls, but the difference was not statistically significant. Platelet counts in atherosclerosis can fluctuate owing to altered thrombosis, platelet dysfunction, and variable risks of thrombosis or bleeding. Evidence indicates that thyroid dysfunction and atherosclerosis reduce erythropoiesis, increase red cell destruction, and promote oxidative stress and systemic inflammation. Although platelet counts may not decline significantly, platelet function is often impaired, increasing cardiovascular risk.

Table 1 Complete blood count indicators for the study groups (mean ± SD)

Parameter	Control	Conservative management	Atherosclerosis
RBC (10^6/mL)	5.663 ± 0.429	4.492 ± 0.959a	4.033 ± 0.440b
HGB (g/dL)	13.145 ± 1.444	8.770 ± 2.536a	7.735 ± 1.401b
HCT (%)	43.805 ± 4.767	31.920 ± 8.681a	28.850 ± 12.749b
PLT (10^3/mL)	244.950 ± 62.455	238.225 ± 91.469	217.500 ± 86.624
WBC (10^3/mL)	9.415 ± 2.689	9.833 ± 1.475	8.025 ± 2.640b*
LYM (%)	30.035 ± 8.392	14.710 ± 6.316a	21.260 ± 8.277b
NEUT (%)	58.695 ± 8.008	71.690 ± 11.139a	65.690 ± 10.501b

Note. (WBC) white blood cells, (RBC) red blood cells, (HGB) hemoglobin, (HCT) hematocrit, (PLT) platelets, (LYM) lymphocytes, (NEUT) neutrophils. One-way ANOVA with LSD post hoc testing (n = 40 control, 50 conservative management, 50 atherosclerosis). Superscripts denote pairwise differences: a different from control; b different from conservative-management group. Asterisks indicate significance thresholds (* $p < 0.05$).

Total white blood cell (WBC) counts were similar in the control and conservative-treatment groups, with a modest decrease in the atherosclerosis group, which may reflect immune suppression related to chronic inflammation, malnutrition, or sepsis-related disorders [28, 29].

Similar studies indicate that patients with chronic atherosclerosis exhibit a weakened immune response, making them more susceptible to infection. Lymphocyte percentages decrease markedly with conservative treatment and moderately in cardiovascular disease [30, 31], whereas neutrophil percentages increase significantly in both patient groups. Decreased lymphocytes suggest immunosuppression, while elevated neutrophils indicate systemic inflammation. This pattern is commonly observed in chronic diseases [32, 33], including atherosclerosis. Platelet (PLT) counts remain relatively stable, but platelet function is impaired. Overall, atherosclerosis shows more pronounced hematologic changes than conservative treatment, indicating greater physiological disturbance with disease progression.

The complete blood count (CBC) is a primary panel reflecting systemic inflammation, oxygen transport, and vascular status—factors closely related to the development and progression of atherosclerosis. Specifically, elevated red blood cell (RBC) count, hemoglobin (HGB), and hematocrit (HCT) increase blood viscosity, impair endothelial function, and promote atherosclerotic plaque formation. Hemolysis with hemoglobin release can drive

vascular calcification through iron-mediated oxidative stress, creating a cycle of arterial injury, inflammatory cell infiltration, and cytokine release (e.g., IL-6, CRP) that accelerates plaque development and fragility [34]. Chronic hypoxia (common in atherosclerosis) stimulates erythropoiesis, with the following stated normal ranges: RBC, 4.2-6.1 million/μL; hemoglobin, 12.1-17.2 g/dL; lymphocytes, 36.1-50.3% [35]. Platelet-derived growth factors contribute to vascular smooth muscle proliferation within arterial walls; these hematologic parameters form part of the inflammatory signature of advanced atherosclerosis and should be interpreted alongside traditional risk factors (lipid profiles, blood pressure) [36]. Elevations in CBC indices often parallel atherosclerotic burden across arterial beds.

3.1 Body weight and kidney enzymes

Table 2 shows the decrease in the average body weight of patients with atherosclerosis (65.75 kg) compared to the control group (74.65 kg). This indicates that atherosclerosis may have an effect on body weight, but more statistical analyses should be performed to confirm this relationship. Significantly higher level of urea in the blood of patients with arteriosclerosis (24.385 mmol/liter) compared to the control group (3.97 mmol/liter). This may indicate impaired hepatic urea metabolism or reduced renal excretion in patients with arteriosclerosis.

Table 2 Levels of urea and creatinine in the serum of the three groups under study Mean ± SD)

Parameter	Control	Conservative management	Atherosclerosis
Weight (Kg)	74.650±15.939	72.900±14.909	65.750±15.335
S.Urea (mmol/L)	3.970±1.031	23.860±11.003	24.385±8.193
S.creatinine (mmol/L)	71.350±4.588	236.10±164.971	261.925±182.557

Serum creatinine was significantly higher in patients with atherosclerosis (261.925 mmol/L) than in the control group (71.35 mmol/L). Creatinine is a byproduct of muscle metabolism and is normally cleared by the kidneys; elevated serum creatinine indicates impaired renal function, supporting the hypothesis that atherosclerosis affects kidney function. These findings suggest that atherosclerosis may contribute to renal dysfunction, leading to increased blood levels of urea and creatinine.

In the total group of patients suffering from arteriosclerosis, an increase in urea and creatinine levels was observed compared to the controls. The average values of urea showed a high level of 23.860 mmol/L in the protective management group and 24.385 mmol/L in the atherosclerosis group compared to controls. I did not notice any significant difference in the average weight values between the groups of patients suffering from chronic arteriosclerosis, as they were lighter. There was a significant elevation in creatinine measurements, reaching 236.10 mmol/L in the chronic atherosclerosis group compared to the controls, and a similar and more widespread elevation at 261.925 mmol/L in the atherosclerosis group compared to the conservative management group. Statistical analyses also indicate the presence of statistically significant differences in creatinine and urea among patients; Despite this, creatinine levels increased with the progression of the disease, and statistically significant differences were also observed in creatinine between a group of patients with atherosclerosis and those with conservative management.

The relationship between renal function and atherosclerosis involves complex biochemical pathways in which renal enzymes and inflammatory mediators play crucial roles. A central mechanism is activation of the renin-angiotensin system (RAS): angiotensin-converting enzyme (ACE) and angiotensin II promote oxidative stress and endothelial dysfunction, accelerating atherosclerosis in chronic kidney disease. RAS activation increases NADPH oxidase activity, elevating reactive oxygen species (ROS) that oxidize low-density lipoprotein (LDL) and promote plaque formation [37]. Although proximal-tubule specific deletion of ACE or AT1aR in mice does not reduce atherosclerosis, systemic RAS inhibition with ACE inhibitors or angiotensin receptor blockers lowers renal and vascular angiotensin II and improves outcomes in chronic kidney disease [38].

Uremic toxins and dysregulated phagocytic cell signaling, for example, indoxyl sulfate activation of Notch pathways, induce pro-inflammatory cytokine release (IL-6, TNF-α) and foam-cell formation [39]. Chronic kidney disease is also associated with reduced cellular expression of the cholesterol transporter ABCA1 by 30-50%, impairing cholesterol efflux and increasing lipid accumulation in arterial walls. Myeloperoxidase (MPO) activity is elevated in chronic kidney disease, generating hypochlorous acid that oxidizes LDL and contributes to plaque development; higher MPO levels are linked to cardiovascular risk in this setting [40]. ROS further quench nitric oxide, reducing vasodilation, while upregulating endothelial adhesion molecules (e.g., VCAM-1) that recruit monocytes to atherosclerotic sites [41]. Inflammation related to chronic kidney disease alters hepcidin levels, promoting ferroportin degradation in

phagocytic cells and iron retention; this shift favors an M1 phenotype, increases ROS, and decreases ABCA1 expression, thereby exacerbating atherogenesis [42].

3.2 Thyroid hormones

The results in Table 3 compare thyroid hormone levels (TT4, TT3, fT4, fT3) and thyroid-stimulating hormone (TSH) across three groups: control, conservative therapy, and atherosclerosis. TT4 was significantly lower in the conservative-therapy and atherosclerosis groups than in controls, suggesting thyroid dysfunction in both groups. Consistent with this, numerous studies indicate that atherosclerosis can be associated with altered thyroid function. TT3 was also significantly reduced in the conservative-therapy and atherosclerosis groups; because T3 is the most biologically active thyroid hormone, its reduction aligns with hypothyroid features. Likewise, fT4 and fT3 were decreased in both patient groups; as the free fractions represent the active hormones, these declines further support impaired thyroid function. In contrast, TSH was significantly elevated in the conservative-therapy and atherosclerosis groups, a typical compensatory response to thyroid insufficiency whereby the body attempts to stimulate greater hormone production; this elevation reinforces the evidence of thyroid dysfunction in these groups.

Table 3 Levels of all three hormones and all three levels Mean ± SD)

Parameter	Unit	Control	Conservative management	Atherosclerosis
TT4	(nmol/L)	87.304 ± 12.006	75.507 ± 15.175	73.050± 15.495
TT3		1.476 ± 0.322	0.543 ± 0.325	0.815 ± 0.369
FT4		12.607 ± 2.550	10.677 ± 3.128	10.865 ± 2.917
FT3		5.774 ± 1.006	3.210 ± 1.037	4.035 ± 0.963
TSH	(ulu/ml)	1.552 ± 1.126	3.084 ± 1.947	3.336 ± 2.163

These results indicate that atherosclerosis may be associated with thyroid dysfunction, characterized by low thyroid hormone levels and elevated TSH. Prior work links cardiovascular disease with thyroid abnormalities. A study [43] reported that subclinical hypothyroidism is associated with increased risk of coronary heart disease. Other studies suggest that atherosclerosis can affect endocrine organs, including the thyroid. Anemia, arising from defects in red blood cell (RBC) number, hemoglobin, and hematocrit, is common in chronic atherosclerosis.

Erythropoietin (EPO) regulates RBC production by acting on erythroblasts in the bone marrow. In adults, EPO is produced by interstitial fibroblasts in the kidney, while in the embryo, it is produced by perisinusoidal liver cells. This factor interacts with other erythroid growth factors (glucocorticoids, IL-6, IL-3). In later stages, EPO contributes to vasoconstriction-related hypertension, increases iron absorption, and protects marrow cells from apoptosis [44]. In the present study, mean values of RBC indices (RBC, hemoglobin, hematocrit) were significantly decreased, consistent with reduced EPO production in atherosclerosis, fewer circulating RBCs, and diminished oxygen delivery to tissues, particularly the heart and stomach [45]. When anemia is poorly responsive to EPO dosing, iron deficiency warrants particular attention; affected patients often present with pallor and easy fatigue.

Anemia commonly appears in chronic atherosclerosis as renal function declines when glomerular filtration rate (GFR) decreases to <60 mL/min, and more markedly when it falls to <15 mL/min [46]. Study [26] reported that anemia occurs in >50% of patients when GFR is <60 mL/min and in >90% when GFR is <15 mL/min. Urea formation accounts for 60% of nitrogenous metabolic waste, with values ranging from 6-46. Most urea is generated in the liver from ammonia after deamination of amino acids, and this biosynthesis is the principal pathway for disposing of protein-derived nitrogen [47].

Physiologically, blood urea concentrations rise with increased hepatic amino acid metabolism (e.g., high-protein intake). Pathologically, urea increases with impaired renal function (e.g., dehydration from limited intake; intrinsic renal failure in atherosclerosis), acute or chronic inflammation, and post-renal obstruction (e.g., urinary tract blockage due to stones or prostatic enlargement), all of which elevate blood urea nitrogen [48]. These mechanisms are consistent with the findings of the present study.

The thyroid gland secretes hormones (T3 and T4) from its functional units into the bloodstream. Most (99%) is protein-bound: primarily to thyroxine-binding globulin (TBG), with lesser binding to pre-albumin and albumin; only a small fraction remains free. Free T3 constitutes 0.003%, and free T4 0.3%. In the present study, TT4 and T4 levels were significantly decreased in patients with chronic atherosclerosis, consistent with prior findings [49]. A reported correlation indicates that urea, creatinine, and indole valphenol inhibit protein synthesis, and the decrease in T4 may be related to increased albumin secretion in atherosclerosis [50].

4 CONCLUSION

This study provides a useful perspective on the complex relationship between thyroid function and atherosclerosis. We found a significant association between thyroid dysfunction and atherosclerosis, as reflected by altered thyroid hormone levels and hematologic findings. These results suggest that interventions aimed at improving thyroid function may aid in the prevention and management of atherosclerosis. While the study advances understanding of this relationship, further research is needed to clarify the underlying mechanisms and to identify potential therapeutic approaches.

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AUTHORS CONTRIBUTIONS

All authors contributed to study conception and design, data acquisition, analysis, and interpretation; drafted or critically revised the manuscript, and approved the final version.

DECLARATIONS

Conflict of interest

The authors declare no competing interests.

Consent to publish

All participants provided consent for publication.

Ethical approval and consent to participate

The study protocol was approved according to order no. 285 (26/12/2024). All procedures complied with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrollment.

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