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INFLAMMATORY STATUS AND THYROID DYSFUNCTION AMONG PREGNANT WOMEN WITH AND WITHOUT ANEMIA : A CROSS-SECTIONAL OBSERVATIONAL STUDY

Biochemistry

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ABSTRACT

Background: Anemia and thyroid dysfunction are common gestational disorders that share overlapping pathophysiological pathways, and inflammation has been proposed as a biological link connecting the two. Data evaluating inflammatory and thyroid parameters concurrently in anemic and non-anemic pregnant women remain limited, particularly from Indian settings. **Objectives:** To compare thyroid profile and C-reactive protein (CRP) between anemic and non-anemic pregnant women and to assess their correlation with hemoglobin. **Methods:** This observational cross-sectional study enrolled 128 pregnant women (20–35 years) attending the antenatal clinic of a tertiary care institute, equally divided into anemic (n=64, hemoglobin <11 g/dL) and non-anemic (n=64) groups. Serum total T3, total T4, thyroid-stimulating hormone (TSH), CRP, hematological indices, and routine biochemical parameters were estimated and compared using the unpaired t-test; Pearson correlation was used to examine relationships between hemoglobin, CRP, and TSH. $p \leq 0.05$ was considered significant. **Results:** Anemic women had significantly higher TSH (4.54±1.07 vs 2.01±0.88 μIU/mL) and CRP (7.76±2.26 vs 3.09±1.54 mg/L) and lower total T3 and T4 than non-anemic women (all $p<0.001$). Thyroid dysfunction, predominantly subclinical hypothyroidism, was present in 59.4% of anemic versus 15.6% of non-anemic women ($p<0.001$). Hemoglobin correlated inversely with TSH ($r=-0.69$, $p<0.001$), while CRP correlated positively with TSH ($r=0.57$, $p<0.001$). **Conclusion:** Gestational anemia is strongly associated with elevated inflammatory activity and impaired thyroid function, predominantly subclinical hypothyroidism. Combined screening for anemia, inflammatory markers, and thyroid status may facilitate earlier identification and management of at-risk pregnancies.

KEYWORDS

Anemia in Pregnancy; Thyroid Dysfunction; C-Reactive Protein; Thyroid-Stimulating Hormone; Inflammation; Subclinical Hypothyroidism

INTRODUCTION

Pregnancy imposes substantial physiological demands on the maternal hematopoietic and endocrine systems, both of which must adapt in a coordinated manner to support fetal growth.[1,2] Anemia and thyroid dysfunction are among the most prevalent gestational disorders worldwide, with the World Health Organization estimating that nearly half of pregnant women globally are anemic, a burden that disproportionately affects low- and middle-income countries including India.[3,4] Concurrently, pregnancy-induced changes in thyroid-binding globulin, human chorionic gonadotropin-mediated thyroid stimulation, and increased metabolic demand frequently unmask latent thyroid dysfunction, particularly subclinical hypothyroidism.[5,6]

A growing body of evidence suggests that these two conditions are mechanistically interlinked rather than coincidental. Thyroid hormones, particularly triiodothyronine, stimulate erythropoietin production and enhance erythroid progenitor responsiveness, so that hypothyroid states can precipitate anemia.[7,8] Conversely, iron is an essential cofactor for thyroid peroxidase, and iron deficiency can impair thyroid hormone synthesis, producing a bidirectional relationship between the two disorders.[9,10] Systemic inflammation may represent an additional connecting pathway: inflammatory cytokines upregulate hepcidin, sequester iron, suppress erythropoiesis, and interfere with thyroid hormone metabolism and hypothalamic-pituitary-thyroid axis regulation.[11,12] Elevated C-reactive protein (CRP) and interleukin-6 have been reported to precede or accompany both iron-deficiency anemia and subclinical hypothyroidism, suggesting that inflammatory activation may be an early, shared feature of both conditions.[13,14]

Despite accumulating international literature linking iron status, inflammation, and thyroid function, comprehensive data assessing all three parameters concurrently in anemic and non-anemic pregnant women remain scarce, particularly in the Indian population where the prevalence of both anemia and thyroid dysfunction is high. Clarifying this relationship could support the rationale for combined antenatal screening strategies. We therefore undertook this study to compare thyroid function and inflammatory status (CRP) between anemic and non-anemic pregnant women and to evaluate their inter-correlations.

MATERIALS AND METHODS

Study Design and Setting

This observational, cross-sectional study was conducted jointly by the Departments of Biochemistry and Obstetrics & Gynaecology at a tertiary care teaching institute in North India over a period of one year, following approval from the Institutional Ethics Committee (Reference: IEC/2024/51). The study is reported in accordance with the STROBE statement for cross-sectional studies.

Study Participants

Pregnant women aged 20–35 years attending the antenatal outpatient department were screened consecutively for eligibility. Women with a confirmed pregnancy who provided written informed consent were enrolled and stratified into two groups based on hemoglobin concentration: an anemic group (hemoglobin <11 g/dL, n=64) and a non-anemic group (hemoglobin ≥11 g/dL, n=64). Women with a pre-existing diagnosis of thyroid disorder or severe anemia already on treatment, acute illness within the preceding three months, bleeding disorders, high-risk or multiple pregnancy, threatened miscarriage, or comorbid conditions such as diabetes mellitus, hypertension, hepatic or renal disease, hemolytic disorders, HIV infection, or malignancy were excluded. The sample size of 128 (64 per group) was determined using G*Power software based on expected effect sizes for thyroid and inflammatory parameters derived from comparable prior studies.

Sample Collection and Biochemical Analysis

Venous blood samples were collected under aseptic precautions from all participants. Complete hemogram parameters (hemoglobin, red blood cell count, packed cell volume, mean corpuscular volume, mean corpuscular hemoglobin, mean corpuscular hemoglobin concentration, and red cell distribution width) were measured using an automated hematology analyzer. Serum total triiodothyronine (T3), total thyroxine (T4), and thyroid-stimulating hormone (TSH) were estimated by chemiluminescence immunoassay, and serum CRP was measured by an immunoturbidimetric method. Fasting blood glucose, urea, creatinine, total protein, and albumin were estimated using standard automated biochemical assays. Thyroid status was categorized as euthyroid, subclinical hypothyroid (elevated TSH with normal T3/T4), or overt hypothyroid (elevated TSH with reduced T3/T4) using trimester-appropriate reference ranges.

Statistical Analysis

Data were analyzed using SPSS software (version 26.0). Continuous variables are expressed as mean ± standard deviation and compared between groups using the unpaired Student's t-test. Categorical variables were compared using the chi-square test and are presented as frequencies and percentages. Pearson's correlation coefficient was used to assess the relationship between hemoglobin, CRP, and TSH. A two-tailed p-value ≤0.05 was considered statistically significant.

RESULTS

A total of 128 pregnant women were enrolled and equally divided into anemic (n=64) and non-anemic (n=64) groups. The two groups were comparable in age (28.52±4.74 vs 28.80±3.96 years; p=0.717) and gestational age (25.67±10.48 vs 22.75±9.52 weeks; p=0.101), with most participants in the second and third trimesters in both groups, minimizing potential confounding by these variables (Table 1).

Table 1. Baseline Demographic Characteristics of Study Participants

Variable	Anemic (n=64)	Non-anemic (n=64)	p-value
Age (years)	28.52 ± 4.74	28.80 ± 3.96	0.717
Gestational age (weeks)	25.67 ± 10.48	22.75 ± 9.52	0.101

As expected by group definition, mean hemoglobin was significantly lower in the anemic group (9.61±0.85 g/dL) than the non-anemic group (12.30±0.71 g/dL; p<0.001). Anemic women also showed significantly lower red blood cell count, packed cell volume, mean corpuscular volume, and mean corpuscular hemoglobin, along with significantly higher red cell distribution width, consistent with an iron-deficiency-like hematological pattern; mean corpuscular hemoglobin concentration did not differ significantly between groups (Table 2).

Table 2. Comparison of Hematological Parameters Between Study Groups

Parameter	Anemic (Mean ± SD)	Non-anemic (Mean ± SD)	p-value
Hemoglobin (g/dL)	9.61 ± 0.85	12.30 ± 0.71	<0.001
RBC ($\times 10^6$/µL)	3.80 ± 0.23	4.48 ± 0.30	<0.001
PCV (%)	28.76 ± 2.96	36.65 ± 2.40	<0.001
MCV (fL)	80.28 ± 4.53	90.33 ± 4.30	<0.001
MCH (pg)	25.40 ± 2.88	27.55 ± 2.48	<0.001
MCHC (g/dL)	32.97 ± 0.93	33.02 ± 0.92	0.775
RDW (%)	14.55 ± 0.59	12.45 ± 0.56	<0.001

Thyroid profile analysis revealed significantly higher TSH and significantly lower total T3 and total T4 in the anemic group compared with the non-anemic group (all p<0.001; Table 3). Correspondingly, thyroid dysfunction was considerably more prevalent among anemic women: subclinical hypothyroidism was observed in 56.3% and overt hypothyroidism in 3.1% of anemic women, compared with 15.6% and 0%, respectively, among non-anemic women (Table 4). Overall, thyroid dysfunction (subclinical plus overt) was present in 59.4% of anemic versus 15.6% of non-anemic women, a highly significant association (χ^2 test, p<0.001; Table 5).

Table 3. Comparison of Thyroid Profile Between Anemic and Non-anemic Pregnant Women

Parameter	Anemic (Mean ± SD)	Non-anemic (Mean ± SD)	P-value
TSH (µIU/ML)	4.54 ± 1.07	2.01 ± 0.88	<0.001
Total T3 (NG/DL)	129.35 ± 20.49	152.22 ± 24.10	<0.001
Total T4 (µG/DL)	7.00 ± 1.19	9.65 ± 1.34	<0.001

Table 4. Distribution of Thyroid Status Among Study Groups

Thyroid Status	Anemic, N (%)	Non-anemic, N (%)
Euthyroid	26 (40.6)	54 (84.4)
Subclinical Hypothyroid	36 (56.3)	10 (15.6)
Overt Hypothyroid	2 (3.1)	0 (0.0)

Table 5. Association Between Anemia Status and Thyroid Dysfunction

Thyroid status	Anemic, n (%)	Non-anemic, n (%)	p-value
Euthyroid	26 (40.6)	54 (84.4)	<0.001
Thyroid dysfunction (subclinical + overt)	38 (59.4)	10 (15.6)	

Serum CRP, the marker of systemic inflammation, was significantly higher in anemic women (7.76±2.26 mg/L) than in non-anemic women (3.09±1.54 mg/L; p<0.001; Table 6). Raised CRP levels were more frequently observed among women with thyroid dysfunction than among those with normal thyroid status, and this association was statistically significant (p<0.01), suggesting a link between inflammatory activation and thyroid dysfunction independent of anemia grouping alone.

Table 6. Comparison of Inflammatory Marker (CRP) Between Study Groups

Parameter	Anemic (Mean ± SD)	Non-anemic (Mean ± SD)	p-value
CRP (mg/L)	7.76 ± 2.26	3.09 ± 1.54	<0.001

Correlation analysis showed a strong negative correlation between hemoglobin and TSH (Pearson r=-0.69, p<0.001), indicating that lower hemoglobin levels were associated with higher TSH. A significant positive correlation was also observed between CRP and TSH (r=0.57, p<0.001), supporting an association between inflammatory status and thyroid dysfunction. Both TSH and CRP showed a progressive rise from the first to the third trimester across the study population, indicating that inflammatory and thyroid stress accentuate as gestation advances. Other routine biochemical parameters, including fasting glucose, urea, and creatinine, were comparable between groups (p>0.05), whereas total protein and albumin were significantly lower in anemic women (p<0.01 and p<0.001, respectively), suggesting a degree of concomitant nutritional compromise.

DISCUSSION

This study demonstrates that gestational anemia is accompanied by a substantially higher burden of thyroid dysfunction and systemic inflammation compared with non-anemic pregnancy, and that these three parameters are significantly inter-correlated. The demographic comparability of the two groups with respect to age and gestational age strengthens the inference that the observed biochemical differences reflect true pathophysiological associations rather than confounding by these variables, consistent with similar matched comparisons reported by Singh et al. and Wang et al.[15,16]

The hematological pattern in the anemic group—reduced red cell count, packed cell volume, mean corpuscular volume, and mean corpuscular hemoglobin with increased red cell distribution width—is characteristic of iron-deficiency-type anemia and is concordant with earlier reports describing similar red cell indices in anemic pregnant women with coexisting thyroid abnormality.[17,18] The markedly higher TSH and lower total T3 and T4 levels among anemic women in the present study parallel findings from Li et al., who reported significantly elevated TSH and reduced free T4 in iron-deficient pregnant women, with the greatest derangement in those with iron-deficiency anemia, and from a meta-analysis by Luo et al. demonstrating a consistent inverse relationship between iron status and thyroid hormone concentrations in pregnancy.[9,19] The predominance of subclinical hypothyroidism (56.3%) over overt hypothyroidism (3.1%) among anemic women in our cohort is also consistent with previous Indian and international studies, which have reported subclinical hypothyroidism as the dominant pattern of thyroid dysfunction associated with anemia in pregnancy.[20,21]

The strong inverse correlation between hemoglobin and TSH observed here (r=-0.69) reinforces the concept that hemoglobin, rather than iron-storage markers such as ferritin alone, may better reflect functional iron availability for thyroid hormone synthesis, particularly given that ferritin behaves as an acute-phase reactant and can be confounded by concurrent inflammation.[22,23] This is biologically plausible: thyroid peroxidase, the enzyme catalyzing thyroid hormone synthesis, requires iron as an essential cofactor, and reduced iron availability—whether from true deficiency or inflammation-mediated iron restriction—can impair thyroid hormonogenesis and elevate TSH through negative feedback.[10,24]

The significantly elevated CRP levels in the anemic group, together with the positive correlation between CRP and TSH, support a role for systemic inflammation as a mechanistic bridge linking anemia and thyroid dysfunction. Inflammatory cytokines are known to upregulate hepcidin, which restricts iron availability for erythropoiesis and thyroid hormone synthesis alike, while also potentially disrupting hypothalamic-pituitary-thyroid axis regulation.[11,25] Similar inflammatory signatures have been reported in women with isolated hypothyroxinemia, who demonstrate persistently elevated CRP and interleukin-6 alongside low ferritin across gestation, and in women

with preeclampsia, in whom combined micronutrient deficiency and inflammatory-oxidative stress pathways have been implicated.[13,26] The progressive rise in TSH and CRP observed from the first to the third trimester in our study further suggests that the physiological demands of advancing pregnancy may amplify both inflammatory activation and thyroid stress, highlighting early pregnancy as a potentially important window for screening and intervention.[27]

The lower total protein and albumin levels observed among anemic women may reflect coexisting nutritional compromise or an inflammation-driven redistribution of hepatic protein synthesis, both of which could further influence thyroid hormone transport and free hormone bioavailability.[28] Taken together, these findings support a unifying model in which iron deficiency, systemic inflammation, and thyroid dysfunction interact bidirectionally during pregnancy, each potentially exacerbating the others, rather than representing isolated and unrelated conditions.[12,29] Such a model has direct clinical relevance: isolated screening for anemia or thyroid dysfunction alone may under-detect at-risk women, and combined evaluation—potentially including CRP as an adjunct marker—may enable more effective risk stratification and earlier targeted management to mitigate adverse maternal and fetal outcomes associated with both conditions.[30]

This study has certain limitations. Its cross-sectional design precludes causal inference regarding the temporal sequence of anemia, inflammation, and thyroid dysfunction. Ferritin, hepcidin, and thyroid autoantibody levels were not measured, limiting further mechanistic interpretation of autoimmune or iron-storage contributions. The single-center design and modest sample size may also limit generalizability of the findings to broader populations. Longitudinal studies incorporating a more comprehensive biomarker panel are warranted to clarify causal pathways and evaluate whether correction of anemia or modulation of inflammation improves thyroid function during pregnancy.

CONCLUSION

Anemic pregnant women demonstrate a significantly higher burden of thyroid dysfunction, predominantly subclinical hypothyroidism, along with elevated CRP levels compared with non-anemic pregnant women. The strong correlations observed between hemoglobin, CRP, and TSH support a biologically plausible interrelationship linking iron deficiency, systemic inflammation, and thyroid dysregulation during pregnancy. These findings underscore the potential value of integrated antenatal screening for anemia, inflammatory status, and thyroid function to enable earlier diagnosis and management, thereby improving maternal and fetal outcomes.

Ethical Approval

The study was approved by the Institutional Ethics Committee (Reference: IEC/2024/51), and written informed consent was obtained from all participants prior to enrollment.

Conflict of Interest

The authors declare no conflict of interest.

Source of Funding

None declared.

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