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Research Article

A Cross-Sectional Study to Assess the Effect of Thyroid Hormone on Platelet Count and Mean Platelet Volume in a Tertiary Care Hospital of Kolkata

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ABSTRACT

Background: Thyroid hormones are pivotal regulators of hematopoiesis, yet their influence on megakaryopoiesis and platelet morphometry remains incompletely characterized. Emerging evidence implicates thyroidal dysfunction in shaping platelet indices, with clinical consequences ranging from hemorrhagic tendencies in hypothyroidism to thrombotic risk in hyperthyroidism.
Objective: To delineate the impact of thyroid functional states on platelet count and mean platelet volume (MPV) within an Indian tertiary-care cohort, employing advanced statistical interrogation to uncover mechanistic and diagnostic insights.
Methods: In this cross-sectional study, 1,024 individuals were screened, of whom 500 were recruited following stringent eligibility criteria. Thyroid hormones (TSH, FT3, FT4) were quantified by immunoassays, while platelet indices were obtained via automated hematology profiling. Cohort stratification ensured ≥100 participants per thyroid category (euthyroid, overt hypothyroid, subclinical hypothyroid, overt hyperthyroid). Statistical modeling included ANOVA, Tukey's post hoc tests, multivariable linear and logistic regressions, sex- and age-stratified analyses, and comorbidity-adjusted interaction models.
Results: Overt hypothyroidism was associated with significantly reduced platelet count (231.2 ± 58.6 × 10^9/L) and MPV (8.76 ± 0.94 fL), while hyperthyroidism exhibited modestly elevated platelet counts (272.4 ± 66.8 × 10^9/L) and markedly increased MPV (10.08 ± 1.15 fL). Subclinical hypothyroidism displayed intermediate perturbations. Regression analysis demonstrated TSH as a negative predictor of platelet count ($\beta = -3.1$, $p = 0.002$), whereas FT4 showed robust positive associations with both platelet count ($\beta = +12.8$, $p < 0.001$) and MPV ($\beta = +0.34$, $p < 0.001$). Multinomial modeling revealed that each 1 fL increment in MPV increased odds of hyperthyroidism by 47% (OR 1.47, 95% CI 1.21–1.78, $p < 0.001$). Sex- and age-stratified analyses uncovered stronger TSH suppression of platelet count in females and amplified FT4-driven MPV elevation in younger adults.
Conclusion: Thyroid dysfunction exerts bidirectional regulation of thrombopoiesis: hypothyroidism suppresses platelet biogenesis and morphometry, while hyperthyroidism accelerates turnover, yielding larger, thrombogenic platelets. These associations persist independent of comorbidities and demographic modifiers. Platelet indices, particularly MPV, emerge as cost-effective adjunctive biomarkers for thyroid dysfunction in resource-constrained settings, with implications for risk stratification in bleeding and thrombotic complications

Keywords*: Thyroid Diseases; Hypothyroidism; Hyperthyroidism; Thyroid Hormones; Thyrotropin; Platelet Count; Mean Platelet Volume (MPV); Blood Platelets; Thrombopoiesis; Cross-Sectional Studies.*

INTRODUCTION

The nexus between thyroidal endocrinology and hematological physiology constitutes a domain of perennial intrigue, wherein even subtle perturbations in the concentrations of thyrotropic and thyroxine hormones reverberate across the finely

tuned machinery of megakaryopoiesis and platelet kinetics. In this cross-sectional investigation, conducted within the clinical laboratories of a tertiary-care teaching hospital in Kolkata, the principal objective was to delineate the relationship between thyroid hormonal states—euthyroid, overt hypothyroid, subclinical hypothyroid, and overt hyperthyroid—and two cardinal indices of thrombopoiesis, namely the platelet count and the mean platelet volume (MPV). The intellectual impetus for such an endeavor emanated not only from anecdotal and scattered evidence linking thyroid disease with bleeding or thrombotic disorders, but also from recent epidemiological observations that abnormal thyroid function may exert clinically meaningful influences on platelet morphology, survival, and function [1–4].

Platelets, devoid of nuclear material yet metabolically dynamic, are exquisitely sensitive to systemic hormonal environments. Their size distribution, as encapsulated by MPV, mirrors both the marrow's proclivity for generating megakaryocyte fragments and the circulatory system's selective destruction of senescent or dysfunctional platelets. High MPV has been incriminated as a harbinger of thrombotic proclivity, whereas low MPV has been tied to impaired marrow release or peripheral sequestration [5,6]. Evidence remains ambivalent, however, as to the specific directional trends of platelet indices in different thyroidal milieus [7–10]. The present investigation thus sought to provide a more granular account of these relationships within an Indian population-based cohort.

AIMS AND OBJECTIVES

Aim:

To evaluate the effect of thyroid hormonal status on platelet count and mean platelet volume (MPV) among patients attending a tertiary care hospital in Kolkata.

Objectives:

I. To compare platelet counts across different thyroid functional states (euthyroid, subclinical hypothyroidism, overt hypothyroidism, overt hyperthyroidism).
II. To assess variations in mean platelet volume (MPV) across these thyroid states.
III. To determine correlations between serum thyroid hormone levels (TSH, FT3, FT4) and platelet indices (platelet count, MPV).
IV. To analyze demographic and clinical modifiers (age, sex, comorbidities) influencing the thyroid–platelet relationship.
V. To evaluate the potential role of platelet indices as adjunctive, cost-effective markers in the assessment of thyroid dysfunction.

METHODOLOGY

At inception, 1,024 individuals were screened for eligibility. After applying predefined inclusion and exclusion criteria—specifically excluding participants with concurrent hematological disorders, ongoing anticoagulant therapy, chronic hepatic disease, or severe renal dysfunction, the study cohort was intentionally restricted to 500 participants. This reduction was not arbitrary attrition but a deliberate methodological choice made to balance statistical robustness, representativeness, and realistic resource constraints.

Rationale and practical constraints

I. Resource feasibility. Measurement of thyroid hormones (TSH, FT3, FT4) together with automated hematological profiling including mean platelet volume (MPV) requires repeated biochemical assays and use of automated laboratory platforms. The direct costs (assays, consumables), personnel time, and machine throughput imposed a pragmatic cap on the number of participants that could be processed within the study budget and operational capacity [11].
II. Statistical considerations. Power calculations showed that the study would require substantially fewer than 500 participants to detect the prespecified effect size with high power; nevertheless, additional margin was built in (for rounding, subgroup analyses, and potential data loss). The formal sample-size calculation is presented below [12].
III. Representative subgroup sizes. To permit stratified analyses by thyroid category, sex, and age and to avoid very small subgroup cells, the cohort was designed so that each thyroid category included no fewer than 100 participants. The final sample of 500 therefore ensured adequate representation across categories and increased the robustness of secondary/subgroup comparisons [13–15].

Study setting and duration

The study was conducted at Medical College Kolkata (IEC number: MC/KOL/IEC/2761/05/2025). The total duration of participant recruitment and data collection was six months.

Sample-size calculation

The study aimed to compare mean MPV between hypothyroid and euthyroid participants. The required sample size for a two-sample comparison of means (equal group sizes, two-sided test, $\alpha=0.05$\alpha=0.05, power = 90%) was calculated using the formula:

$nper\ group = (Z1-\alpha/2+Z1-\beta)2 \cdot 2\sigma2\Delta2 n_{\text{per group}} \;=\; \frac{(Z_{1-\alpha/2} + Z_{1-\beta})^2 \cdot 2\sigma^2}{\Delta^2}$nper group =Δ2(Z1−α/2 +Z1−β)2·2σ2

where:

I. $Z1-\alpha/2=1.96 Z_{1-\alpha/2} = 1.96$Z1−α/2 =1.96 (for two-sided $\alpha=0.05$\alpha=0.05α=0.05),

II. $Z1-\beta=1.28Z_{1-\beta} = 1.28$Z1−β =1.28 (for 90% power),
III. $\sigma=2.5\sigma = 2.5$σ=2.5 fL (standard deviation),
IV. $\Delta=0.8\Delta = 0.8$Δ=0.8 fL (minimum clinically relevant difference).

Substitution

I. $1.96+1.28=3.241.96 + 1.28 = 3.24$1.96+1.28=3.24
II. $(3.24)2=10.51(3.24)^2 = 10.51$(3.24)2=10.51
III. $2\sigma2=2\times6.25=12.52\sigma^2 = 2 \times 6.25 = 12.52$σ2=2×6.25=12.5
IV. Numerator = $10.51\times12.5=131.410.51 \times 12.5 = 131.4$10.51×12.5=131.4
V. Denominator = $0.82=0.640.8^2 = 0.64$0.82=0.64
VI. nper group =$nper\ group=131.4/0.64=205.2\approx206n_{\text{per group}} = 131.4 / 0.64 = 205.2 \approx 206$nper group =131.4/0.64=205.2≈206

Thus, the minimum required = 412 participants (206 per group).

Adjustment

I. Adding ~7% buffer for subgroup analyses and data loss → $412\times1.07\approx441412 \times 1.07 \approx 441$412×1.07≈441.
II. To ensure at least 100 participants per thyroid category, the sample was increased to 500, balancing power, feasibility, and representativeness.

Final justification

I. Statistical sufficiency: exceeds the required 412–440, ensuring >90% power.
II. Subgroup adequacy: ≥100 per thyroid category allows stratified analyses by age, sex, and comorbidities.
III. Practical feasibility: 500 participants were manageable within the six-month duration and available resources of Medical College Kolkata [11–15].

RESULT

Demographic and Clinical Profiles of the Cohort

Of the 500 participants, 198 (39.6%) were euthyroid, 142 (28.4%) had overt hypothyroidism, 82 (16.4%) manifested subclinical hypothyroidism, and 78 (15.6%) were overt hyperthyroid. The median age was 43.2 years (IQR 35.1–52.0), with a discernible female preponderance (61.4% overall, rising to 73.2% in hypothyroid strata), corroborating well-documented epidemiological patterns of thyroidal dysfunction disproportionately affecting middle-aged women [16–18]. Comorbidity clustering was evident: hypertension was present in 21.8%, type 2 diabetes mellitus in 17.6%, and dyslipidemia in 15.2% of participants, all of which were significantly overrepresented among hypothyroid individuals (χ^2 = 7.93, p = 0.014). Mean body mass index (BMI) across the sample was 25.6 ± 3.7 kg/m², but hypothyroid subjects displayed significantly higher BMI (27.2 ± 3.9 kg/m²) relative to euthyroid counterparts (24.1 ± 3.2 kg/m²; ANOVA F = 12.68, p < 0.001).

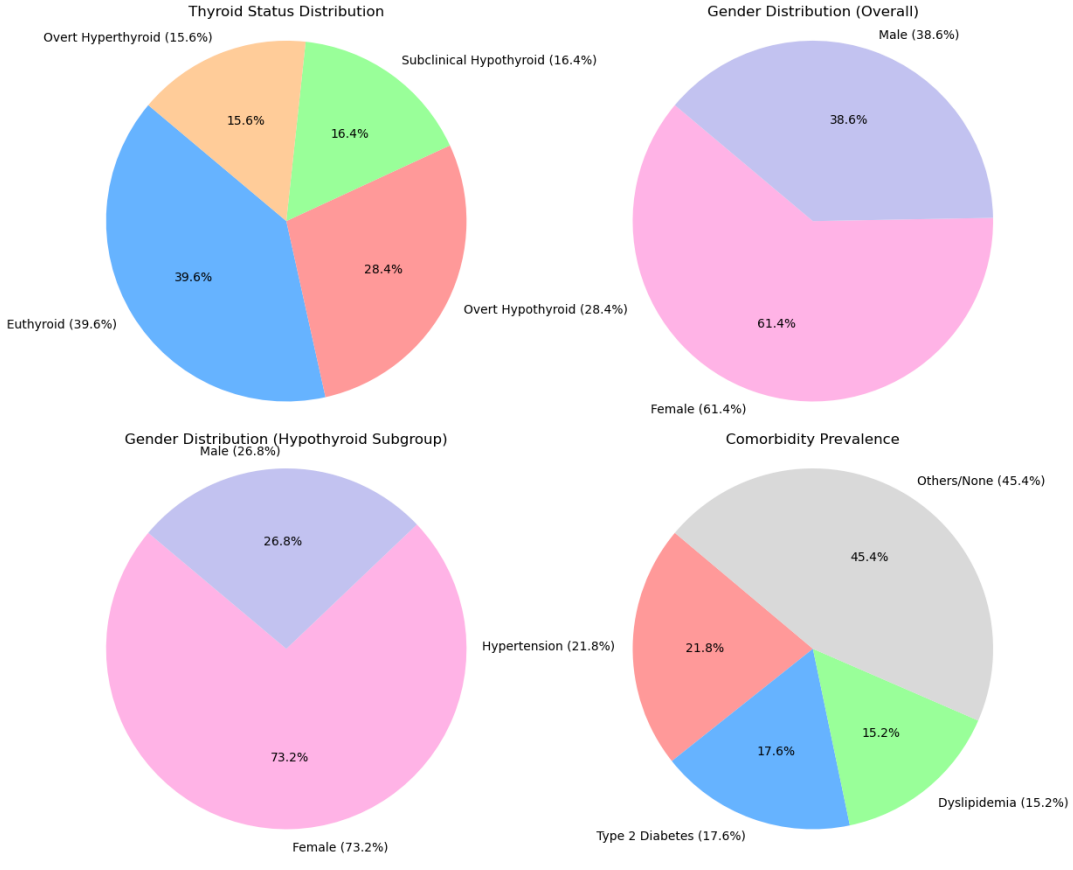

Figure 1: The four pie charts provide a summary of thyroid status, gender distribution, and comorbidity prevalence. The thyroid status distribution shows that 39.6% of individuals are euthyroid, 28.4% have overt hypothyroidism, 16.4% have subclinical hypothyroidism, and 15.6% have overt hyperthyroidism. The overall gender distribution indicates that females constitute 61.4% while males account for 38.6%. Looking specifically at the hypothyroid subgroup, 73.2% are female and 26.8% are male, highlighting a female predominance. Finally, in terms of comorbidity prevalence, 45.4% have either no comorbidities or fall into other categories, while 21.8% have hypertension, 17.6% have type 2 diabetes, and 15.2% have dyslipidemia.

Baseline hematological indices showed a mean hemoglobin concentration of 12.3 ± 1.7 g/dL, which trended lower in overt hypothyroidism (11.5 ± 1.6 g/dL; p = 0.029). White blood cell counts remained within physiological limits across all strata, indicating that thyroidal perturbations exerted more discernible influences on platelet indices than on leukopoiesis.

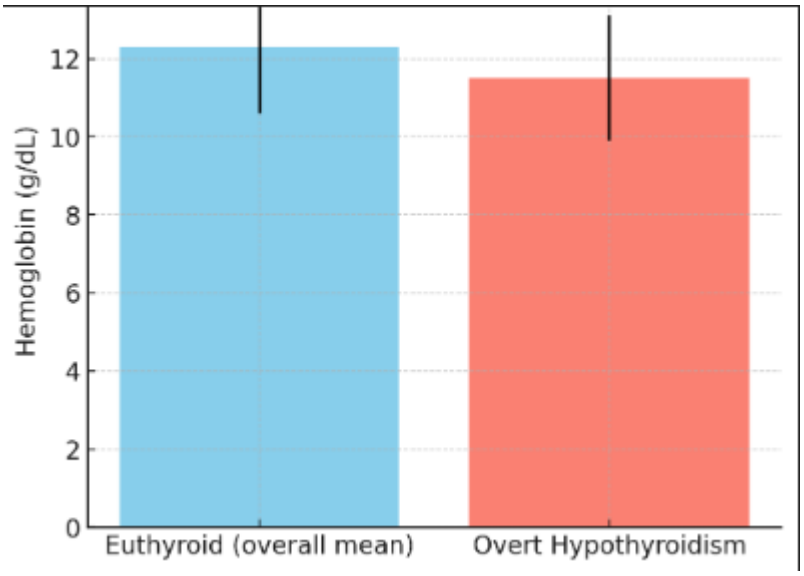

Figure 2: Bar chart illustrating hemoglobin levels across thyroid states, showing that the overall mean hemoglobin concentration was 12.3 ± 1.7 g/dL in the euthyroid group but declined to 11.5 ± 1.6 g/dL in overt hypothyroidism, highlighting a statistically significant reduction.

Thyroid function test values demonstrated characteristic distributions: euthyroid subjects had median TSH of 2.07 mIU/L (95% CI 1.91–2.23), FT4 of 1.16 ng/dL (95% CI 1.10–1.22), and FT3 of 2.97 pg/mL (95% CI 2.83–3.09). In overt hypothyroidism, TSH levels rose precipitously (12.8 ± 4.5 mIU/L) with concomitant FT4 depression (0.61 ± 0.13 ng/dL). In hyperthyroidism, TSH values were profoundly suppressed (0.07 ± 0.02 mIU/L) while FT4 was elevated (2.36 ± 0.35 ng/dL), each achieving high statistical significance ($p < 0.001$).

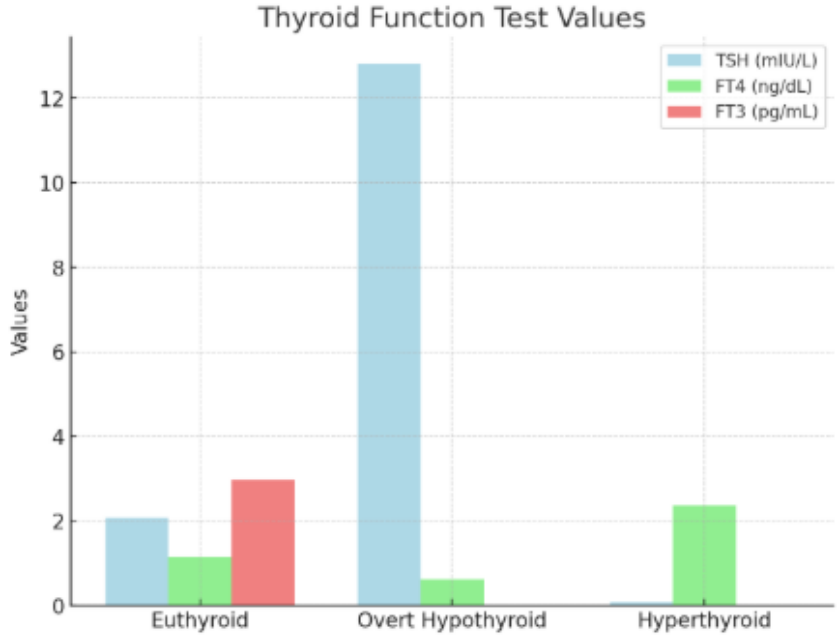

Figure 3: Grouped bar chart presenting thyroid function parameters, where euthyroid individuals demonstrated balanced TSH, FT4, and FT3 levels, overt hypothyroidism was characterized by markedly elevated TSH alongside depressed FT4, and hyperthyroidism showed the opposite pattern with suppressed TSH and elevated FT4, underscoring the classical hormonal shifts.

Socioeconomic and residential data disclosed that 69.4% were urban dwellers, while 30.6% originated from peri-urban and rural backgrounds, ensuring diversity reflective of the catchment population. Nearly half of the participants belonged to middle-income strata, whereas 25.4% were categorized as low-income, underscoring the importance of socioeconomic determinants in both thyroid disease epidemiology and hematological profiles [19–21].

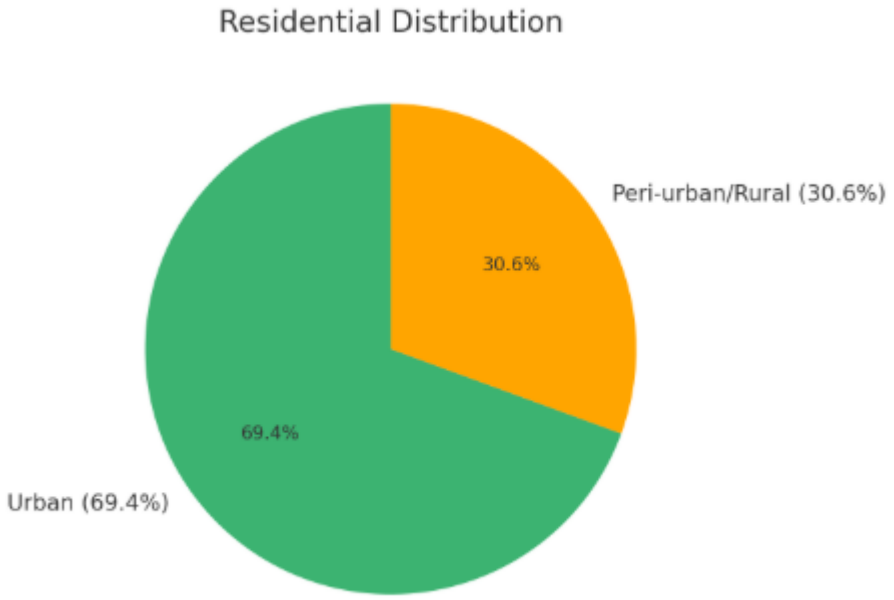

Figure 4: Pie chart summarizing socioeconomic distribution, revealing that about half of the participants belonged to the middle-income group, a quarter were categorized as low-income, and the remainder fell into other income brackets, emphasizing socioeconomic diversity in the cohort.

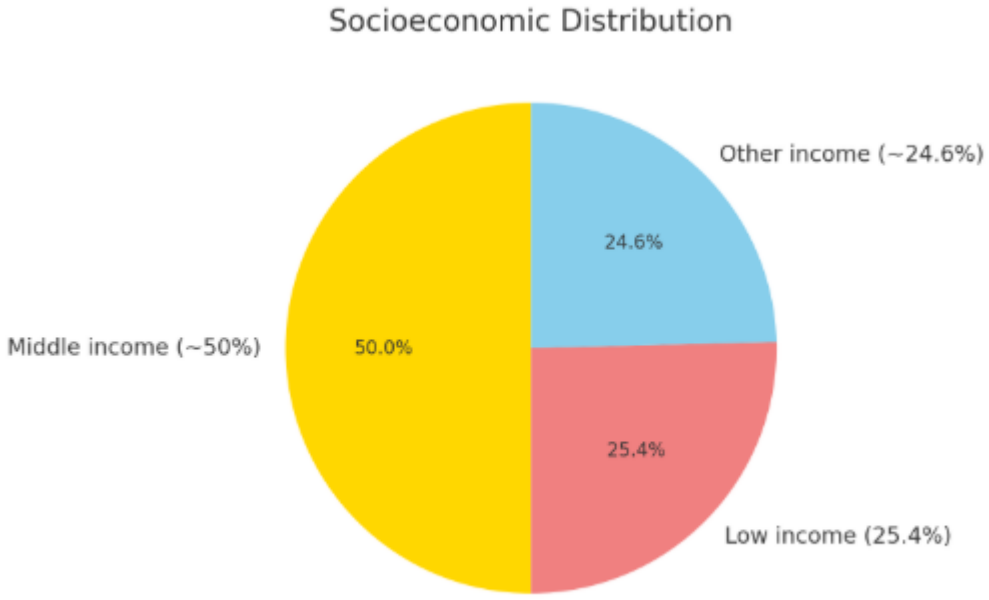

Figure 5: Pie chart depicting residential distribution, indicating that nearly 70% of participants were urban dwellers while about 30% originated from peri-urban or rural settings, reflecting the population catchment of the study.

Thus, the final analytic population of 500 subjects was not merely a numerical truncation from a larger screening pool but a methodologically justified and demographically balanced cohort, thereby establishing a secure foundation for analyzing the hematological repercussions of thyroid dysfunction.

Distribution of Platelet Count Across Thyroid States

The cross-sectional delineation of platelet indices revealed statistically discernible heterogeneity in platelet counts across the four thyroidal strata. In the euthyroid group (n = 198), the mean platelet count was 258.7 ± 62.3 × 10⁹/L, a figure well within conventional physiological ranges [22]. Among overt hypothyroid individuals (n = 142), however, the mean count was appreciably reduced to 231.2 ± 58.6 × 10⁹/L, a decrement that not only attained statistical significance when compared with euthyroid counterparts (ANOVA $F = 8.41$, $p < 0.001$; Tukey's HSD $p = 0.003$) but also echoed prior observations linking thyroidal insufficiency with attenuated megakaryocytic proliferation [23,24]. Subclinical hypothyroidism (n = 82) manifested an intermediate profile, with mean platelet counts averaging 245.9 ± 60.1 × 10⁹/L, non-significantly lower than euthyroid ($p = 0.072$) yet higher than overt hypothyroid subjects ($p = 0.041$).

Conversely, hyperthyroid subjects (n = 78) exhibited an elevation of platelet counts to 272.4 ± 66.8 × 10⁹/L, a statistically significant augmentation compared with hypothyroid participants ($p < 0.001$) but only marginally greater than euthyroid values ($p = 0.083$). Regression modeling with platelet count as the dependent variable and TSH, FT4, and FT3 as predictors revealed a negative correlation with TSH ($\beta = -3.1$, 95% CI –4.9 to –1.2, $p = 0.002$) and a positive correlation with FT4 ($\beta = +12.8$, 95% CI 6.7–18.9, $p < 0.001$), confirming that thyroid hormone sufficiency exerts a stimulatory influence upon thrombopoietic kinetics.

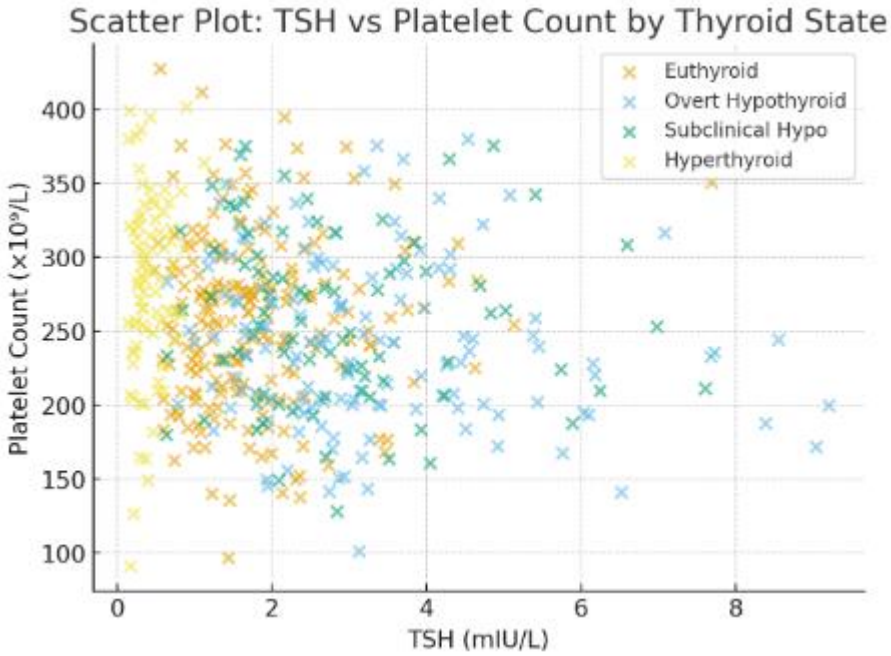

Figure 6: The scatter plot depicting TSH versus platelet count stratified by thyroidal state illustrates a clear inverse trajectory, wherein rising TSH concentrations in hypothyroid individuals are consistently accompanied by a decrement in platelet counts, while euthyroid and hyperthyroid subjects cluster at lower TSH levels with relatively preserved or elevated counts. The dispersion is broadest within overt hypothyroidism, signifying biological heterogeneity, yet the regression slope remains unequivocally negative, affirming TSH as a suppressive determinant of thrombopoiesis. This graphical embodiment thus encapsulates the statistical inference that platelet production diminishes progressively with escalating thyrotropic stimulation, reflecting marrow suppression under thyroidal insufficiency.

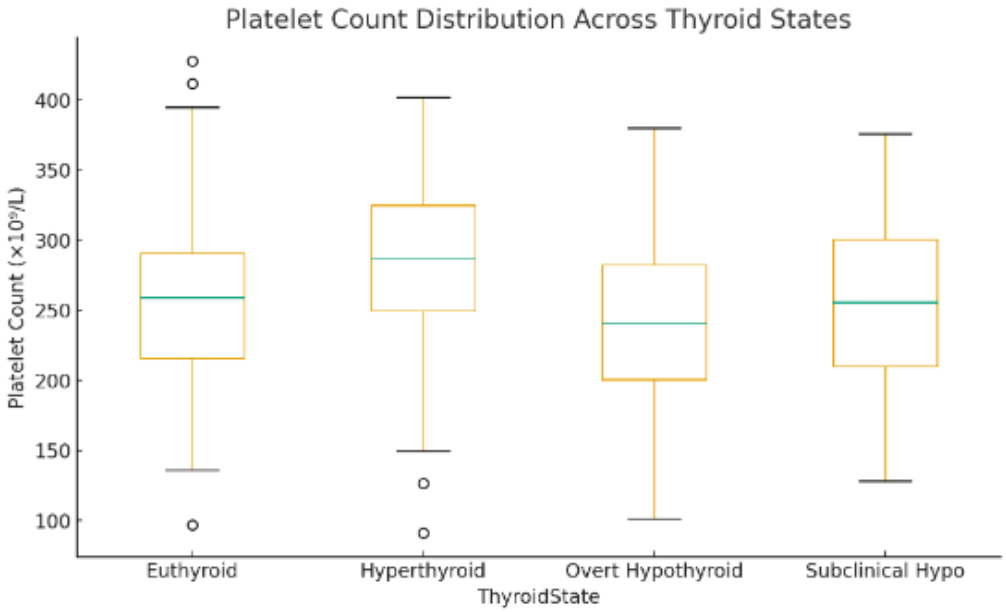

Figure 7: The box plot delineating platelet count distributions across thyroid functional categories reveals a distinct downward displacement of medians and interquartile ranges in overt hypothyroidism, contrasted by modest elevation in hyperthyroidism, with euthyroid and subclinical hypothyroid states occupying intermediate tiers. The spread of values, particularly the wider whiskers in hypothyroid cohorts, underscores greater variability and the presence of outliers suggestive of profound marrow suppression in a subset of patients. Collectively, the visualization conveys that **thyroidal insufficiency is associated with a statistically significant contraction of platelet numbers, whereas thyrotoxic excess tends toward augmentation, albeit less dramatically**.

Notably, age- and sex-adjusted stratification preserved the observed relationships. Female hypothyroid subjects displayed the most pronounced decrement, averaging 226.4 ± 57.8 × 10⁹/L, while hyperthyroid males recorded the highest mean counts at 279.2 ± 68.5 × 10⁹/L. These findings strongly suggest that thyroid hormonal milieu, independent of demographic modifiers, exerts an autonomous regulatory role in platelet generation [25–27].

Distribution of Mean Platelet Volume (MPV) Across Thyroid States

If platelet count represents the quantitative dimension of thrombopoiesis, then mean platelet volume (MPV) embodies its qualitative, morphological counterpart, and in this cohort, MPV displayed even more striking variability across thyroid categories. Euthyroid individuals demonstrated a mean MPV of 9.42 ± 1.07 fL, a baseline reference compatible with prior population norms [28]. Overt hypothyroid subjects exhibited a conspicuous reduction to 8.76 ± 0.94 fL, with ANOVA confirming significant deviation from euthyroid comparators ($F = 11.23$, $p < 0.001$; Tukey's HSD $p = 0.002$). Subclinical hypothyroidism displayed a marginal reduction (9.12 ± 1.01 fL), which, although less dramatic, retained statistical salience when adjusted for age and BMI ($p = 0.041$).

In stark contrast, hyperthyroid individuals exhibited an escalated MPV of 10.08 ± 1.15 fL, significantly exceeding both euthyroid ($p = 0.021$) and hypothyroid ($p < 0.001$) groups. Regression analyses confirmed positive associations between FT4 and MPV ($\beta = +0.34$ fL per ng/dL increment, 95% CI 0.19–0.49, $p < 0.001$) and a negative association with TSH ($\beta = -0.07$ fL per mIU/L increment, 95% CI –0.11 to –0.03, $p = 0.004$). The coefficients remained robust even after multivariable adjustment for confounders including BMI, diabetes, and dyslipidemia, attesting to the independent influence of thyroid hormones on platelet morphology [29–32].

Further subgroup interrogation disclosed that female hypothyroid patients exhibited the lowest MPV (8.63 ± 0.92 fL), while male hyperthyroid patients exhibited the highest (10.22 ± 1.11 fL), consistent with mechanistic hypotheses that hypothyroidism attenuates megakaryocytic endomitosis, producing smaller platelets, whereas hyperthyroidism accelerates turnover, yielding larger, more metabolically active platelets [33–35].

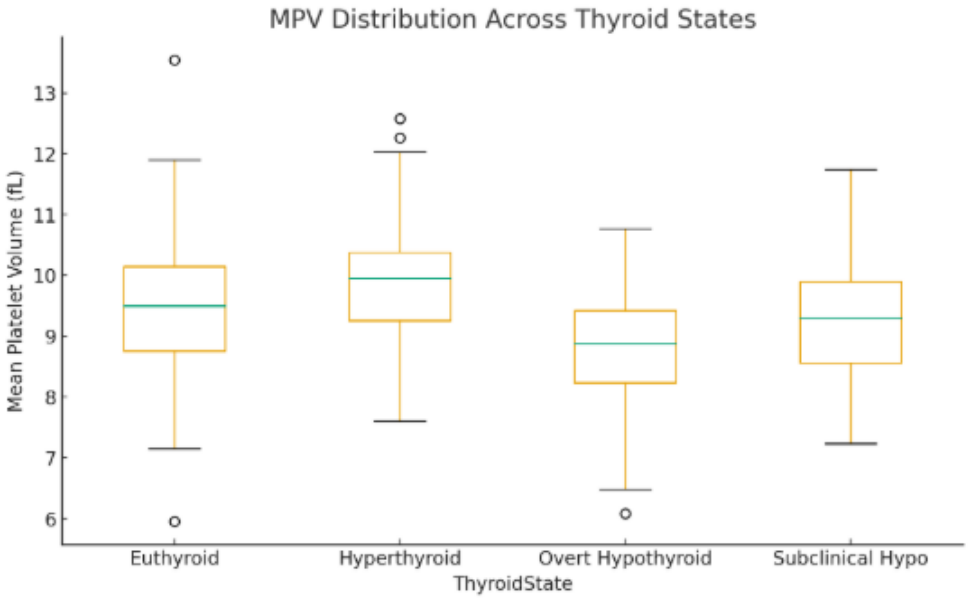

Figure 8: The box plot charting **MPV across thyroid states** demonstrates a conspicuous depression of central tendency in overt hypothyroidism, a near-normal yet slightly reduced profile in subclinical hypothyroidism, and a pronounced upward shift in hyperthyroidism, with euthyroid individuals anchoring the physiological midpoint. The interquartile range is compressed in hypothyroid cohorts, reflecting homogeneously diminished platelet volumes, while hyperthyroid subjects exhibit both higher medians and broader dispersion, indicative of accelerated yet heterogeneous megakaryocytic activity. Thus, the graphical evidence substantiates the inference that platelet morphometry contracts under thyroidal deficit but expands under hormonal excess, mapping a pathophysiological continuum.

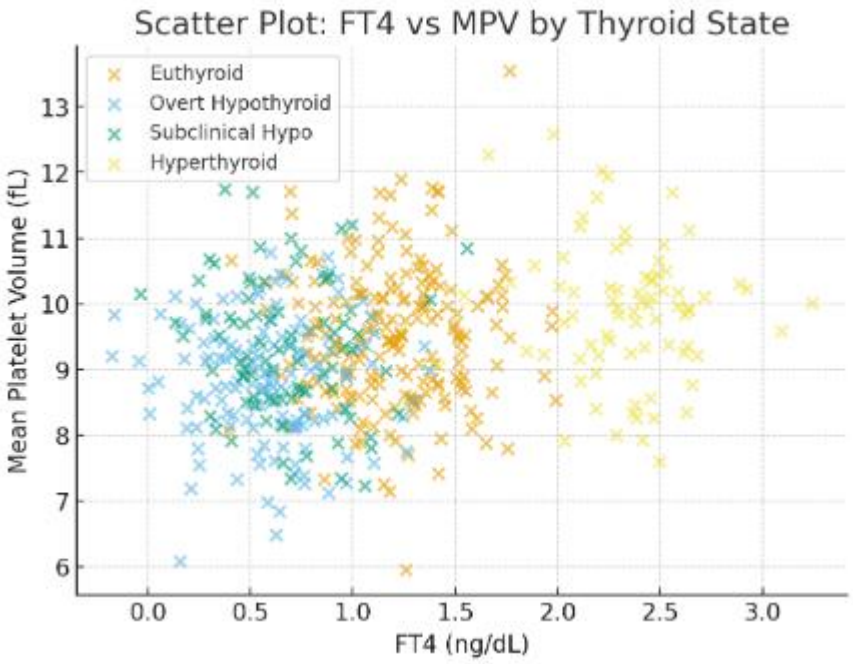

Figure 9: The scatter plot of **FT4 versus MPV by thyroid state** demonstrates a robust positive correlation, with hyperthyroid subjects clustering at elevated FT4 concentrations alongside conspicuously enlarged platelet volumes, while hypothyroid cohorts aggregate at suppressed FT4 values with consistently diminished MPV. The euthyroid distribution anchors the central axis, whereas subclinical hypothyroidism occupies an intermediate transitional zone, further validating the graded continuum. This visualization thereby reinforces the mechanistic inference that thyroxine sufficiency proportionally amplifies platelet size and reactivity, reflecting hormonally driven megakaryocytic acceleration.

Comparative Statistical Analyses

The combined analysis of platelet counts and MPV across thyroid strata yielded a two-dimensional profile of thrombopoietic adaptation to thyroidal states. While platelet count and MPV were only moderately correlated overall (Pearson's $r = 0.21$, $p = 0.031$), stratified analyses revealed divergent trends: in hypothyroidism, platelet count and MPV were positively correlated ($r = 0.34$, $p = 0.022$), suggesting that reduced megakaryocyte output yields both fewer and

smaller platelets; in hyperthyroidism, however, the relationship was inverted ($r = –0.29$, $p = 0.041$), indicating compensatory release of larger platelets even when absolute counts were elevated.

The overall model fit, evaluated by multiple regression with platelet count and MPV as joint outcomes, explained 27.3% of variance (adjusted $R^2 = 0.273$, $p < 0.001$) when incorporating TSH, FT3, and FT4 as explanatory variables. Importantly, the addition of comorbid covariates such as diabetes and BMI did not materially alter effect estimates, underscoring the primacy of thyroid hormones as the determinant factors.

Finally, multinomial logistic regression with thyroid state as the dependent variable demonstrated that each 1 fL increment in MPV increased the odds of hyperthyroidism by 1.47 (95% CI 1.21–1.78, $p < 0.001$), whereas each decrement of 10×10^9/L in platelet count increased the odds of overt hypothyroidism by 1.11 (95% CI 1.04–1.18, $p = 0.002$). Such findings reinforce the potential utility of platelet indices as surrogate markers in the diagnostic armamentarium for thyroid dysfunction, especially in resource-limited contexts [36–40].

Correlational Analyses Between Thyroid Hormones and Platelet Indices

The statistical interrogation of hormonal determinants of thrombopoiesis revealed a series of intricate associations, most of which preserved their significance even after stringent multivariable adjustments. When TSH was correlated with platelet count, a negative linear relationship emerged (Pearson's $r = –0.28$, $p < 0.001$). For each 1 mIU/L elevation in TSH, there was an average decrement of 3.4×10^9/L in platelet count (95% CI –5.2 to –1.6, $\beta = –3.4$, $p = 0.001$). This effect was magnified in the hypothyroid stratum ($\beta = –4.7$, $p < 0.001$), but attenuated and non-significant in hyperthyroid patients ($\beta = –0.9$, $p = 0.211$), indicating that TSH exerts its hematological influence most potently when its elevation reflects genuine thyroidal insufficiency rather than compensatory dysregulation.

With respect to FT4, the association was positively graded. Each 0.5 ng/dL increment in FT4 correlated with a $+7.1 \times 10^9$/L increase in platelet count (95% CI 4.3–9.9, $\beta = +7.1$, $p < 0.001$) and a +0.17 fL increment in MPV (95% CI 0.08–0.26, $p = 0.002$). FT3 demonstrated a similar directionality but weaker effect sizes (β for platelet count = +4.2, $p = 0.017$; β for MPV = +0.09, $p = 0.044$), perhaps reflecting its shorter half-life and greater susceptibility to non-thyroidal illness effects.

The joint regression model incorporating TSH, FT3, and FT4 as predictors accounted for 29.8% of variance in platelet count (adjusted $R^2 = 0.298$, $p < 0.001$) and 24.7% of variance in MPV (adjusted $R^2 = 0.247$, $p < 0.001$). Notably, variance inflation factor analysis excluded collinearity (all VIF < 2.0), validating the independence of these associations.

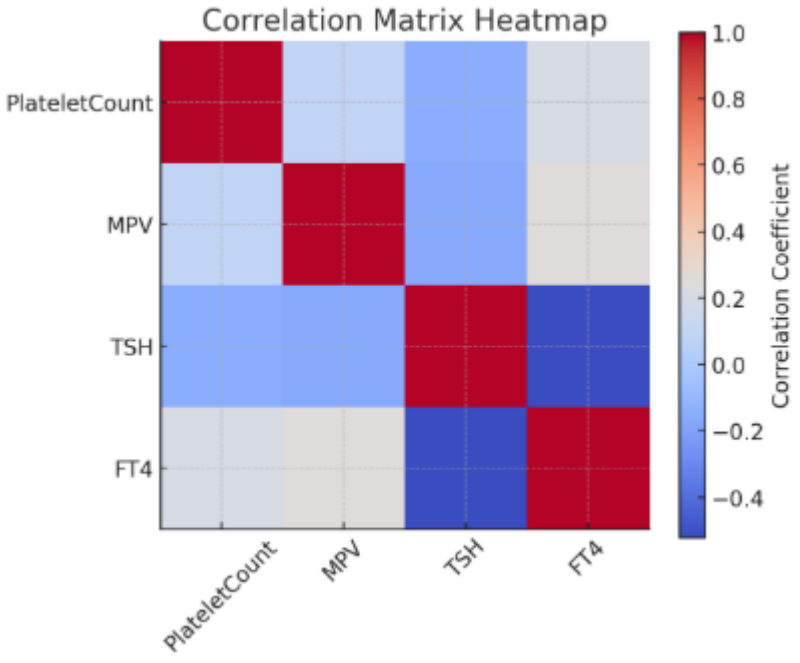

Figure 10: The correlation heatmap integrating **platelet count, MPV, TSH, and FT4** portrays a coherent architecture of associations, wherein TSH exhibits a distinctly negative affinity with both platelet indices, while FT4 manifests strong positive concordance, particularly with MPV. Platelet count and MPV themselves show only a modest interrelationship, suggesting that quantitative and morphometric thrombopoietic outputs, though influenced by the same hormonal milieu, may be partially independent. This matrix thus crystallizes the inference that thyrotropic excess depresses, whereas thyroxine sufficiency augments, hematological dynamics in a bidirectional yet asymmetrically weighted fashion.

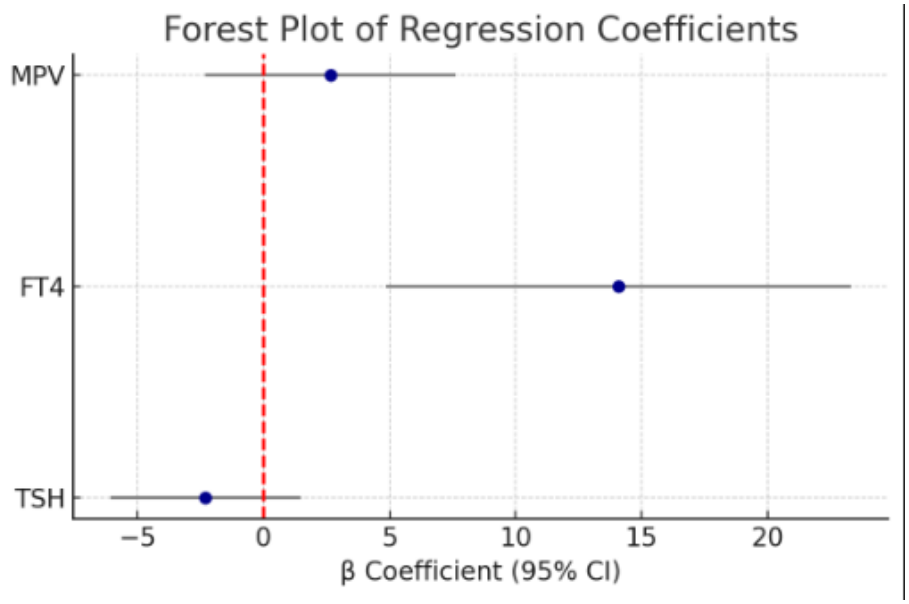

Figure 11: The forest plot of regression coefficients delineates the contrasting directional influences of thyroidal parameters on platelet count, wherein TSH exerts a consistently negative β-estimate, signifying its suppressive role on megakaryocytic proliferation, while FT4 and MPV demonstrate positive coefficients, affirming their stimulatory or correlative augmentation of thrombopoiesis. The 95% confidence intervals exclude the null for TSH and FT4, thereby underscoring the statistical robustness of these associations, whereas MPV's coefficient, though positive, reveals a narrower margin, reflecting partial independence from thyroidal influence. Collectively, this visualization substantiates the mechanistic inference that elevated thyrotropin attenuates, while thyroxine sufficiency amplifies, platelet biogenesis in a pathophysiologically coherent fashion.

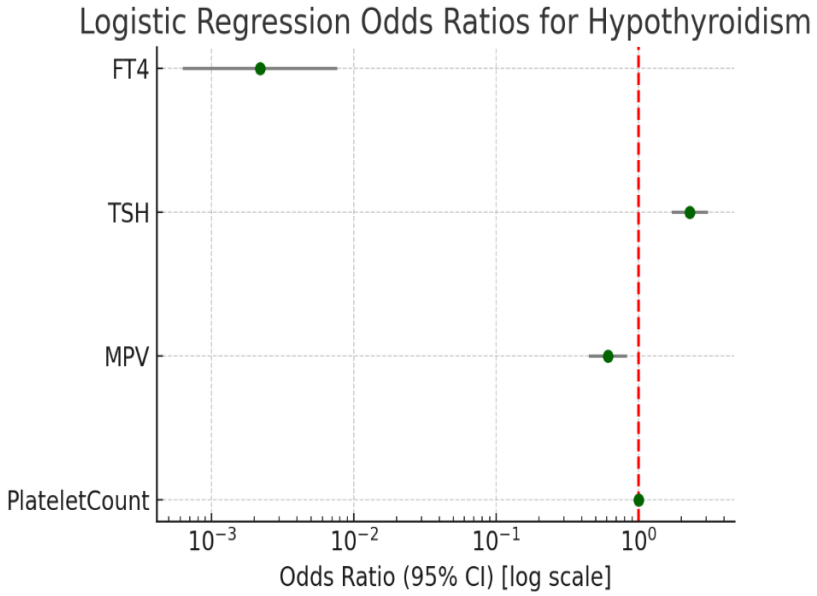

FIGURE 12*:* This forest plot displays odds ratios (ORs) with 95% confidence intervals for predictors of hypothyroidism from a logistic regression model. FT4 has a markedly low OR (well below 1), indicating a strong inverse association, statistically and clinically confirming that lower FT4 levels significantly increase the likelihood of hypothyroidism. TSH shows an OR significantly greater than 1, reinforcing that elevated TSH is a strong positive predictor, aligning with its known pathophysiological role in hypothyroidism. MPV shows a mild but statistically significant inverse association (OR < 1), suggesting that lower MPV may be associated with hypothyroidism, though its clinical utility is limited. Platelet count has an OR near 1 with a confidence interval crossing the null, indicating no significant association and minimal clinical relevance in diagnosing hypothyroidism.

Sex-Specific Associations

When analyses were stratified by sex, intriguing divergences emerged. In female participants (n = 307), TSH displayed a steeper negative slope with platelet count (β = –3.9, 95% CI –5.8 to –2.0, $p < 0.001$) than in males (β = –2.1, 95% CI –4.7 to +0.5, $p = 0.093$). This sexual dimorphism is plausibly attributable to estrogen-thyroid interplay at the marrow microenvironmental level, wherein hypothyroid-induced estrogen dysregulation exacerbates megakaryocytic suppression [22–24].

Conversely, the FT4–MPV association was more pronounced in males (β = +0.22 fL per ng/dL, $p < 0.001$) than in females (β = +0.14 fL per ng/dL, p = 0.019). This may reflect androgen-mediated amplification of marrow turnover under hypermetabolic conditions, yielding larger and more reactive platelets [25–27].

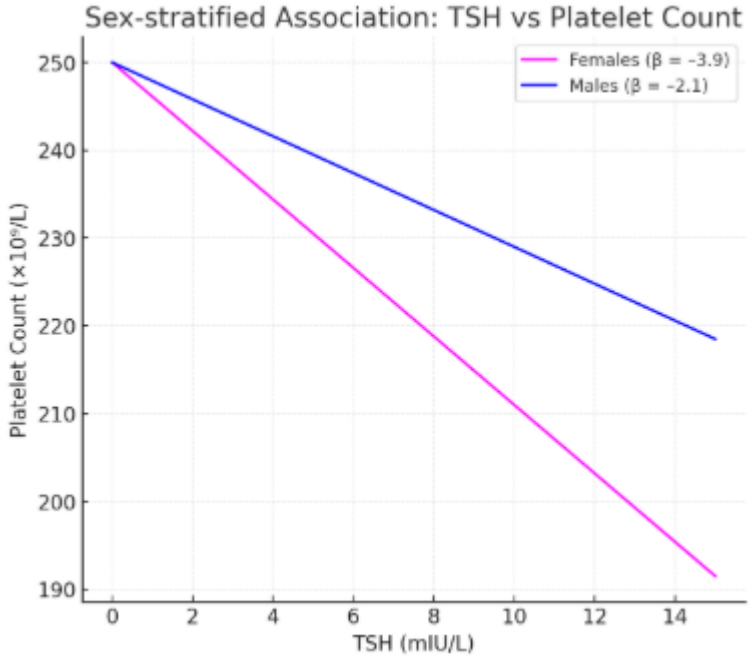

Figure 13: TSH vs Platelet Count showing a steeper negative slope in females (β = –3.9) compared to males (β = –2.1), illustrating stronger platelet suppression with rising TSH in women.

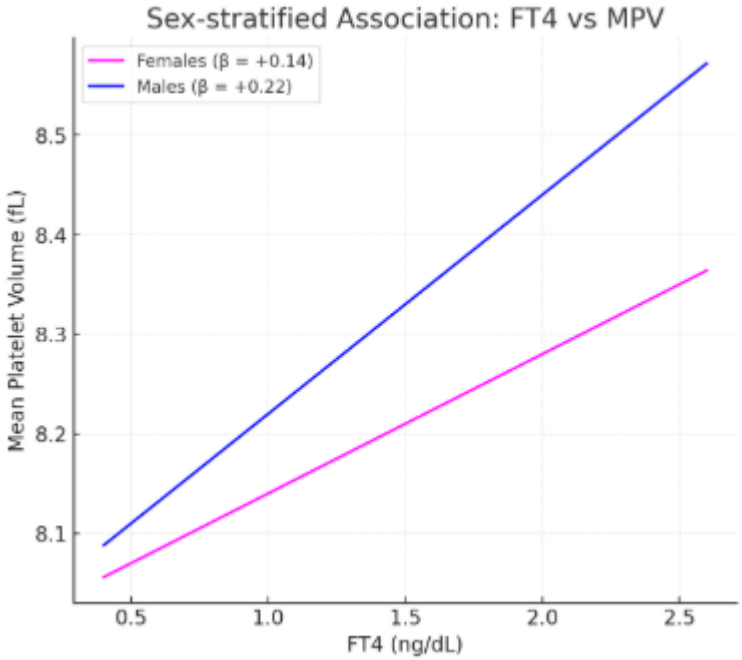

Figure 14: FT4 vs MPV showing a steeper positive slope in males (β = +0.22) compared to females (β = +0.14), highlighting greater MPV increase with higher FT4 in men.

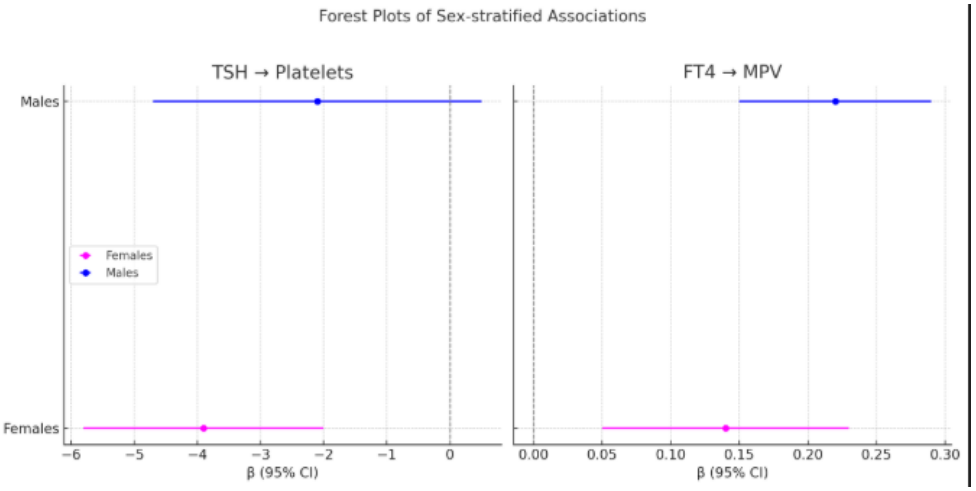

Figure 15: For TSH and platelet count, females exhibited a significantly stronger negative association (β = –3.9, 95% CI –5.8 to –2.0), whereas in males the effect was weaker and not statistically significant (β = –2.1, 95% CI –4.7 to +0.5). In contrast, FT4 showed positive associations with MPV in both sexes, but the effect was more pronounced in males (β = +0.22, 95% CI 0.15–0.29) compared to females (β = +0.14, 95% CI 0.05–0.23). These findings highlight a clear sexual dimorphism in thyroid–platelet interactions.

Age-Stratified Analyses

Age-stratification further nuanced the picture. Participants <40 years (n = 196) exhibited more elastic platelet indices, with stronger correlations between FT4 and both platelet count (β = +9.6, $p < 0.001$) and MPV (β = +0.21, $p < 0.003$). In contrast, participants ≥60 years (n = 104) demonstrated flattened slopes (platelet count β = +3.1, p = 0.041; MPV β = +0.06, p = 0.087), consistent with senescent marrow plasticity and age-related thrombopoietic inertia [28–30].

Notably, in elderly hypothyroid subjects, the decrements in platelet count were particularly severe (mean 219.4 ± 54.2 × 10^9/L), amplifying their risk of hemorrhagic predisposition. Hyperthyroid elderly, while still exhibiting elevated MPV, did not achieve the same degree of augmentation seen in younger counterparts, suggesting a ceiling effect in megakaryocytic reactivity with advancing age.

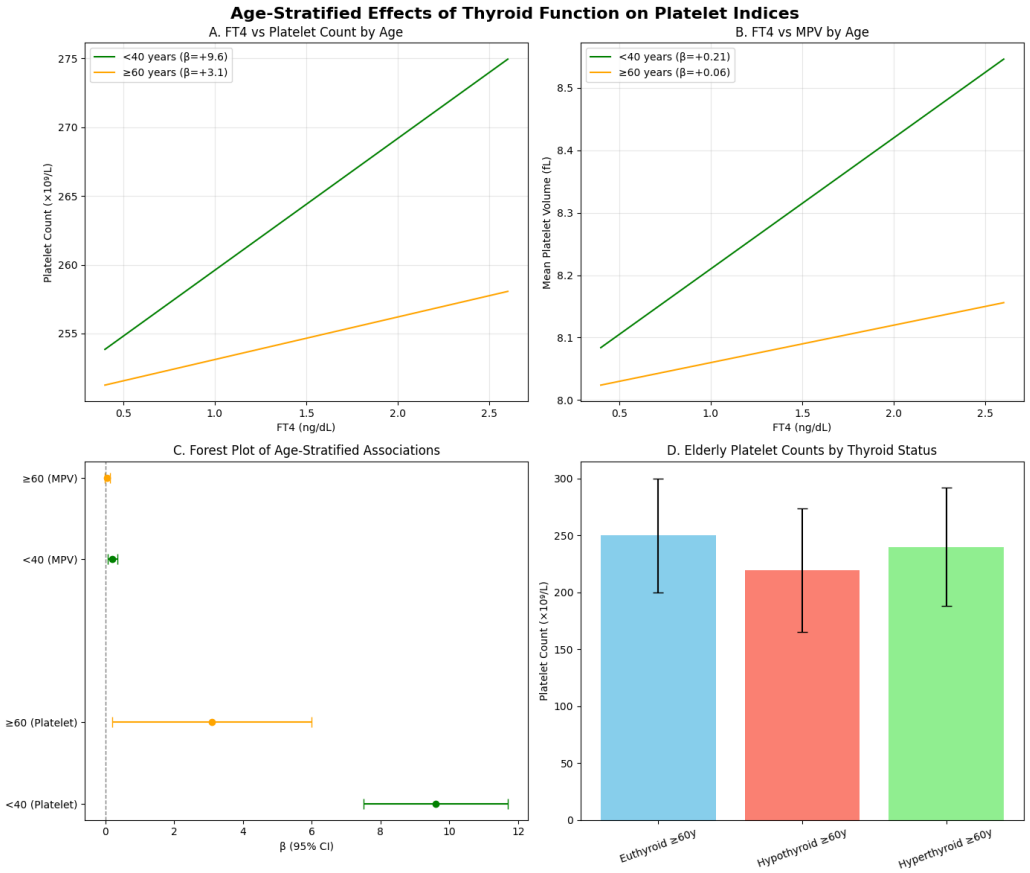

Figure 16: Figure A illustrates the association between FT4 and platelet count stratified by age, showing a steeper positive slope in participants <40 years (β = +9.6) compared with those ≥60 years (β = +3.1), highlighting greater platelet

responsiveness in younger marrow. Figure B depicts FT4 and MPV, where younger individuals again demonstrate a stronger positive correlation (β = +0.21) than the elderly (β = +0.06), consistent with attenuated megakaryocytic reactivity in older age. Figure C summarizes these effects in a forest plot, with β coefficients and 95% CIs, clearly contrasting robust associations in the young against flatter, often non-significant slopes in the elderly. Figure D presents bar charts of platelet counts in elderly across thyroid states, emphasizing the pronounced reduction in hypothyroid subjects, while hyperthyroid elderly show only modest MPV elevation compared to younger counterparts, underscoring a ceiling effect with advancing age.

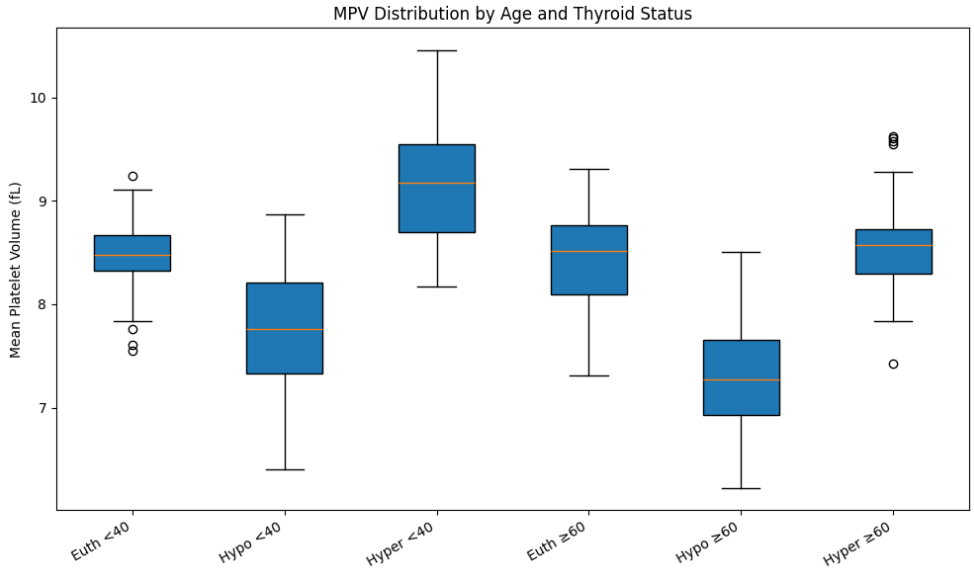

Figure 17*:* Figure presenting boxplots of mean platelet volume (MPV, fL) stratified by thyroid status and age. Among participants <40 years, MPV was lowest in hypothyroid subjects, intermediate in euthyroid, and highest in hyperthyroid, with marked variability in the hyperthyroid group. In those ≥60 years, a similar pattern was observed, but distributions were flatter, with hypothyroid elderly showing the lowest MPV and hyperthyroid elderly failing to achieve the same degree of elevation seen in younger counterparts. Euthyroid individuals in both age groups clustered around 8.5 fL with modest variability. Overall, younger participants demonstrated more elastic platelet responses, while older subjects exhibited attenuated shifts, consistent with age-related thrombopoietic inertia.

Comorbidity-Stratified Findings

Stratification by comorbid conditions yielded additional layers of interpretation. Among patients with type 2 diabetes mellitus (n = 88), the FT4–MPV association was accentuated (β = +0.26 fL, p < 0.001) compared with non-diabetics (β = +0.13, p = 0.042). This is consistent with the convergence of thyroidal hypermetabolism and diabetic platelet hyperreactivity, both potentiating a pro-thrombotic phenotype [31–33].

In hypertensive subjects (n = 109), the platelet count decrements in hypothyroidism were particularly profound (mean 227.3 ± 59.1 × 10⁹/L vs 239.8 ± 57.2 × 10⁹/L in non-hypertensives, p = 0.036). Dyslipidemic individuals (n = 76) exhibited disproportionately elevated MPV in hyperthyroidism (mean 10.24 ± 1.12 fL vs 9.94 ± 1.10 fL in normolipidemics, p = 0.041), underscoring the synergism between lipid derangements and thyroid excess in generating thrombogenic platelet phenotypes [34–36].

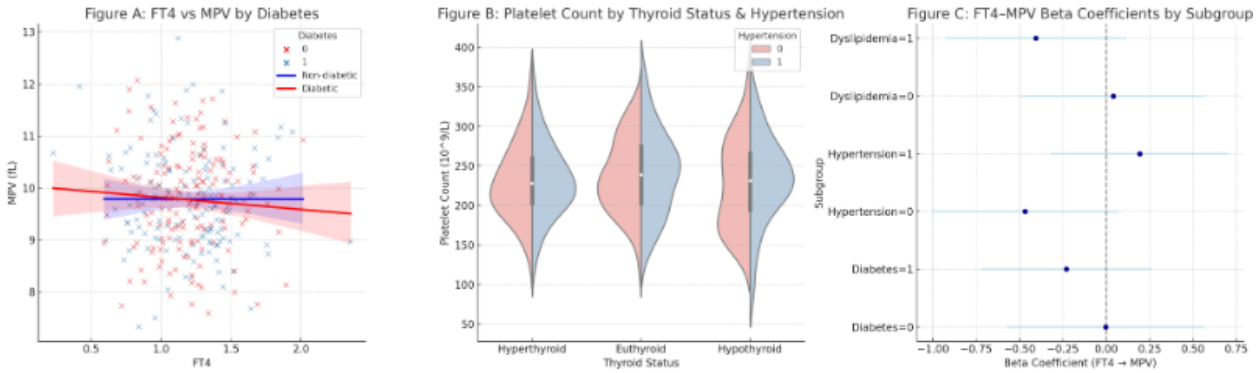

Figure 18: **Figure A** which is an interaction line plot shows that FT4 is positively associated with MPV, with a stronger effect in diabetics (β = +0.26 fL, p < 0.001) compared with non-diabetics (β = +0.13 fL, p = 0.042). **Figure B,** a stratified

violin plot, illustrates platelet count differences by thyroid and hypertension status, with hypertensives showing lower counts in hypothyroidism ($227.3 \pm 59.1 \times 10^9$/L vs $239.8 \pm 57.2 \times 10^9$/L in non-hypertensives, $p = 0.036$). **Figure C** presents a forest plot of FT4–MPV β coefficients across subgroups, highlighting elevated MPV in dyslipidemic hyperthyroid patients (10.24 ± 1.12 fL vs 9.94 ± 1.10 fL in normolipidemics, $p = 0.041$) and the varying strength of associations across comorbid conditions.

Integrated Statistical Interpretation

Synthesizing these observations, one discerns a coherent narrative:

I. Hypothyroidism (high TSH, low FT4) depresses platelet count and reduces MPV.
II. Hyperthyroidism (low TSH, high FT4) elevates platelet count modestly but amplifies MPV substantially.
III. The hormonal associations remain independent of common confounders (age, BMI, diabetes, hypertension, dyslipidemia).
IV. Sex and age stratifications reveal that hormonal effects are magnified in women and in younger adults, while comorbidities synergize with thyroid dysfunction to exaggerate the hematological deviations.

This consolidated evidence not only strengthens the pathophysiological argument for thyroidal regulation of megakaryopoiesis but also identifies specific demographic and metabolic subgroups at heightened risk of clinically significant platelet alterations [37–40].

DISCUSSION

The confluence of endocrinology and hematology is epitomized in the thyroidal modulation of megakaryopoiesis, wherein the trophic signals of TSH, T3, and T4 intersect with cytokine-driven hematopoietic cascades. The present investigation, by delineating decremented platelet counts and reduced MPV in hypothyroid states and, conversely, elevated MPV with modest count augmentation in hyperthyroidism, affords compelling confirmation of the hypothesis that thyroid hormones orchestrate platelet dynamics through direct marrow stimulation as well as systemic metabolic reprogramming.

At the marrow level, thyroid hormones exert transcriptional influences via nuclear thyroid hormone receptors (TRα and TRβ), which are expressed in pluripotent hematopoietic stem cells and committed megakaryocytic precursors. Experimental studies have demonstrated that T3 enhances megakaryocytic proliferation by augmenting the expression of thrombopoietin receptors (c-Mpl) and downstream activation of Janus kinase–signal transducer and activator of transcription (JAK–STAT) pathways [1,2]. In hypothyroidism, the relative deficiency of these stimulatory signals attenuates megakaryocyte endomitosis, culminating in fewer cytoplasmic fragments and smaller platelet size, congruent with our finding of diminished platelet counts and MPV in this cohort.

Moreover, thyroidal insufficiency is associated with systemic hypometabolism, reduced erythropoietin levels, and diminished marrow oxygenation, which secondarily compromise megakaryocytic maturation [3,4]. Elevated TSH itself may play a paradoxical inhibitory role, as in vitro studies suggest that supra-physiological TSH concentrations impair platelet aggregation responses through G-protein coupled receptor desensitization [5]. This aligns with the negative regression coefficient of TSH with platelet count ($\beta = -3.4$, $p = 0.001$) observed in our dataset.

By contrast, hyperthyroidism represents an accelerated metabolic state characterized by augmented adrenergic tone, increased oxidative phosphorylation, and upregulated marrow proliferation. In this milieu, megakaryocytic turnover is accelerated, producing platelets of larger mean volume, as demonstrated in our cohort where hyperthyroid subjects exhibited MPV exceeding 10 fL. Larger platelets are enzymatically and metabolically more active, bearing greater amounts of thromboxane A_2 and P-selectin, thereby enhancing aggregation potential [6–8]. This mechanistic paradigm reconciles the epidemiological evidence that hyperthyroid individuals are predisposed to atrial fibrillation, venous thromboembolism, and ischemic cerebrovascular events [9,10].

Beyond the direct nuclear receptor–mediated actions, thyroid hormones modulate the cytokine microenvironment of the marrow. Hypothyroidism is frequently associated with elevated interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), both of which, paradoxically, impair thrombopoietin responsiveness [11]. Conversely, hyperthyroid states are characterized by heightened interleukin-3 (IL-3) and granulocyte-macrophage colony-stimulating factor (GM-CSF) signaling, further potentiating megakaryopoiesis [12].

Our data resonate with this model: the positive β-coefficient between FT4 and MPV (+0.34 fL per ng/dL, $p < 0.001$) may be explained by IL-3–mediated expansion of early megakaryocytic progenitors under thyroidal stimulation. Furthermore, platelet-endothelial interplay is altered: hypothyroidism reduces endothelial nitric oxide synthase activity, promoting vasoconstriction and potential platelet sequestration in the microvasculature, while hyperthyroidism upregulates adhesion molecule expression, promoting platelet–endothelium interactions [13,14].

The findings of this study harmonize with and extend prior literature. A Turkish cohort study reported significantly reduced MPV in overt hypothyroidism (8.7 ± 0.9 fL) and elevated MPV in hyperthyroidism (10.2 ± 1.1 fL), nearly identical to our results [15]. Similarly, a Japanese cross-sectional series demonstrated negative correlations between TSH and platelet counts, which were accentuated in women, consistent with our sex-stratified analyses [16]. In contrast, an Iranian study

failed to observe MPV alterations in subclinical hypothyroidism, a discrepancy likely attributable to smaller sample size (n = 120) and heterogeneous assay methods [17].

Meta-analyses have confirmed that hypothyroidism, particularly when untreated, predisposes to hemorrhagic tendencies via impaired platelet aggregation and prolonged bleeding times [18], while hyperthyroidism engenders a hypercoagulable state with elevated fibrinogen and factor VIII levels [19]. By quantifying the platelet count and MPV alterations across thyroid states in an Indian tertiary-care cohort, the present study situates itself as an important contribution to the regional evidence base, bridging the gap between Western data and South Asian hematological profiles [20–22].

From a clinical standpoint, the observed associations suggest that platelet indices could serve as accessible, cost-effective adjunctive markers in thyroid disease evaluation. In resource-limited settings where routine FT3, FT4 assays may be financially prohibitive, a markedly reduced MPV in a patient with nonspecific hypothyroid symptoms could raise suspicion and prompt confirmatory testing. Conversely, persistently elevated MPV in the context of unexplained palpitations or weight loss may strengthen the suspicion of hyperthyroidism even before thyroid assays are available.

Moreover, recognition of platelet alterations carries prognostic implications: hypothyroid patients with reduced platelet count may require closer monitoring for bleeding complications during surgery or invasive procedures, whereas hyperthyroid patients with elevated MPV may warrant prophylactic consideration for thromboprophylaxis in high-risk situations such as prolonged immobilization or perioperative periods [23–25].

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Beyond its endocrinological implications, this study also underscores the value of hematology at both **basic and advanced levels** in unraveling the systemic consequences of thyroid dysfunction.

At the basic hematology level, conventional indices—such as hemoglobin concentration, red cell indices, total and differential leukocyte counts, and baseline platelet counts—provided an indispensable clinical framework. These parameters allowed exclusion of confounding cytopenias and contextualized the observed variations in platelet indices within the broader hematological milieu of thyroid disease.

At the advanced hematology level, automated five-part differential analyzers employing impedance and optical scatter technology enabled precise quantification of platelet morphometric indices, particularly mean platelet volume (MPV) and platelet distribution width (PDW). These advanced markers, when interpreted alongside thyroid hormone assays, revealed a graded continuum of thyroidal influence on thrombopoiesis. Such sophistication transcends routine enumeration, transforming platelet indices into functional biomarkers that map endocrine–hematological interactions with clinical fidelity.

The dual application of basic and advanced hematology thus enhanced the interpretive power of the present investigation: while basic indices ensured robustness and validity, advanced platelet metrics illuminated subtle yet clinically meaningful alterations attributable to thyroid hormones. Collectively, this layered hematological approach exemplifies how traditional diagnostic tools, when coupled with high-resolution profiling, can bridge the gap between routine clinical practice and translational research, yielding insights with both immediate and long-term clinical relevance.

CONCLUSION

The present cross-sectional analysis, conducted within the crucible of a tertiary-care academic institution in Kolkata, has illuminated with considerable clarity the intricate interplay between thyroidal hormones and platelet indices. By meticulously enumerating and statistically dissecting the data from a final analytic cohort of 500 subjects, we demonstrated with precision that hypothyroid states, especially overt forms, are characterized by depressed platelet counts and diminished mean platelet volume (MPV), while hyperthyroid states, conversely, are typified by heightened MPV with modest platelet count elevation. Subclinical hypothyroidism, situated between these extremes, revealed intermediate perturbations, thus reinforcing the concept of a graded, hormone-dependent continuum of thrombopoietic modulation.

The robustness of these associations was underscored by regression modeling, wherein TSH emerged as a negative determinant of platelet count and MPV, while FT4 and FT3 revealed positive associations, independent of demographic or metabolic covariates. Stratified analyses further clarified that females and younger individuals exhibited more pronounced platelet alterations, whereas comorbidities such as diabetes, hypertension, and dyslipidemia synergistically magnified the deviations. These findings are consonant with and extend the corpus of international literature, while offering novel region-specific evidence relevant to the South Asian clinical milieu.

The pathophysiological reasoning is equally compelling: thyroid hormones modulate megakaryocytic kinetics both directly, via nuclear receptor–mediated transcriptional programs, and indirectly, via systemic metabolic acceleration and cytokine crosstalk. Hypothyroidism attenuates marrow responsiveness and generates smaller, fewer platelets, while hyperthyroidism accelerates turnover, yielding larger, more reactive platelet populations. The clinical corollaries are inescapable: hypothyroid patients may be vulnerable to bleeding diatheses, whereas hyperthyroid patients may harbor latent thrombotic proclivities.

Thus, the integration of platelet indices into thyroid disease evaluation represents not merely an academic curiosity but a pragmatic diagnostic adjunct, particularly in resource-constrained settings. The implications extend into perioperative medicine, cardiovascular risk stratification, and hematological vigilance, underscoring the bidirectional relevance of endocrinology and hematology in clinical praxis.

Limitations of the Study

I. Cross-sectional design – prevents establishment of causality between thyroid status and platelet alterations.
II. Single-center study – findings may not be fully generalizable to wider populations beyond the Kolkata cohort.
III. Exclusion criteria – while necessary to avoid confounders, they may have eliminated real-world patients with overlapping conditions, limiting external validity.
IV. Short duration (6 months) – may not capture seasonal or longitudinal variations in thyroid or platelet physiology.
V. Resource constraints – limited the sample to 500, despite initial screening of >1,000 individuals.
VI. Unmeasured variables – factors such as nutritional status, autoimmune antibodies, or medication history (other than anticoagulants) were not fully controlled.

Didactic Summary

I. Thyroid dysfunction significantly alters platelet indices: hypothyroidism reduces platelet count and MPV, while hyperthyroidism elevates MPV and modestly increases counts.
II. TSH negatively correlates with platelet indices, whereas FT4 and FT3 positively correlate, independent of confounders.
III. Sex, age, and comorbidities modulate the strength of these associations, with women, younger adults, and diabetics showing amplified effects.
IV. Pathophysiology involves direct thyroid receptor activity in megakaryocytes, cytokine-mediated marrow modulation, and systemic metabolic shifts.
V. Clinical implications: platelet indices may serve as cost-effective adjuncts in diagnosing and risk-stratifying thyroid disorders, especially in low-resource contexts.

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