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Original Article

Hematological and Iron Profile Changes in Pregnant Women Across Trimesters

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ABSTRACT

Background: Iron requirements increase progressively during pregnancy, while physiological hemodilution and depletion of maternal iron reserves can alter hematological and biochemical indices across gestation. Objective: To evaluate trimester-wise changes in hemoglobin, mean corpuscular volume (MCV), serum ferritin, serum iron, total iron-binding capacity (TIBC), transferrin saturation, and anemia prevalence among pregnant women in Karachi, Pakistan. Methods: This prospective longitudinal study enrolled 150 pregnant women during the first trimester, of whom 138 completed assessments at 8–12, 20–24, and 32–36 weeks of gestation. Hematological and iron-profile parameters were measured at each assessment and trimester-wise changes were evaluated using repeated-measures statistical analysis. Results: Hemoglobin decreased from 11.8 ± 1.2 to 10.7 ± 1.1 g/dL ($p<0.001$), while MCV decreased from 84.6 ± 5.4 to 79.8 ± 6.1 fL ($p=0.002$). Ferritin declined from 38.5 ± 18.4 to 17.2 ± 11.5 ng/mL, serum iron from 86.4 ± 24.5 to 61.5 ± 20.6 µg/dL, and transferrin saturation from 28.1 ± 8.5% to 17.5 ± 7.1%, whereas TIBC increased from 312.6 ± 45.3 to 358.4 ± 52.1 µg/dL (all $p<0.001$). Anemia prevalence increased from 18.8% to 42.0%. Conclusion: Maternal hematological and biochemical iron-status indices deteriorated progressively across pregnancy, supporting trimester-sensitive assessment of iron status rather than reliance on hemoglobin alone. Keywords: Pregnancy; Iron deficiency anemia; Hemoglobin; Ferritin; Serum iron; Total iron-binding capacity; Transferrin saturation; Mean corpuscular volume; Maternal health; Pakistan.

INTRODUCTION

Pregnancy is accompanied by substantial physiological adaptations in the maternal hematological system that are required to support placental development, fetal growth, and the progressive increase in maternal circulatory demands. Expansion of plasma volume and red-cell mass alters maternal hematological indices throughout gestation, while increasing requirements for micronutrients, particularly iron, place additional demands on maternal nutritional reserves. Iron is essential for hemoglobin synthesis, oxygen transport, cellular metabolism, placental function, and fetal development. When maternal iron availability is inadequate to meet these progressively increasing requirements, iron stores become depleted and may ultimately result in iron deficiency and iron deficiency anemia (IDA), which remains an important nutritional and hematological disorder during pregnancy (1,2).

Maternal anemia represents a substantial public-health concern, particularly in low- and middle-income settings where nutritional deficiencies, socioeconomic constraints, limited access to antenatal screening, and inadequate correction of pre-existing iron deficiency may increase vulnerability during pregnancy. Global evidence demonstrates that anemia affects a considerable proportion of pregnant women and

contributes disproportionately to the burden of maternal morbidity in resource-constrained populations (3). Severe maternal anemia has been associated with adverse maternal outcomes, while poor maternal nutritional status has also been linked with unfavorable fetal and neonatal outcomes, including preterm birth and impaired fetal growth (4,5). In Pakistan, iron deficiency remains particularly relevant because women may enter pregnancy with limited iron reserves resulting from nutritional inadequacy and other socioeconomic and reproductive factors (6).

Maternal iron requirements are not constant throughout gestation. Requirements are comparatively modest during early pregnancy but increase progressively during the second and third trimesters as maternal erythropoiesis expands and fetal and placental iron utilization accelerates (7,8). At the same time, physiological plasma-volume expansion produces hemodilution and a corresponding reduction in hemoglobin concentration. Consequently, a decline in hemoglobin during pregnancy does not necessarily represent pathological iron deficiency in isolation. Interpretation of maternal hematological changes therefore requires consideration of both physiological adaptation and the progressive utilization of maternal iron stores (8–10).

Hemoglobin concentration remains the most commonly used laboratory parameter for identifying anemia during pregnancy, but it provides only an indirect assessment of maternal iron status and may remain relatively preserved during the early stages of iron depletion. Iron deficiency generally develops sequentially, beginning with depletion of storage iron before substantial impairment of erythropoiesis becomes evident. Serum ferritin is therefore particularly useful for evaluating stored iron, whereas serum iron, total iron-binding capacity (TIBC), and transferrin saturation provide complementary information regarding circulating iron availability and iron transport (11–13). Evaluating these biochemical indicators alongside hematological indices can provide a more comprehensive characterization of maternal iron status than reliance on hemoglobin concentration alone.

Mean corpuscular volume (MCV) provides additional information concerning erythrocyte morphology and the development of iron-restricted erythropoiesis. Progressive iron deficiency may eventually produce microcytic changes reflected by declining MCV; however, depletion of iron stores can precede substantial alteration in red-cell indices (9,11). For this reason, simultaneous assessment of hemoglobin, MCV, serum ferritin, serum iron, TIBC, and transferrin saturation can help characterize the sequence of hematological and biochemical changes occurring across pregnancy and differentiate declining iron availability from changes reflected only by conventional complete blood count measurements.

The magnitude and timing of maternal iron depletion may vary considerably among pregnant women. Pre-pregnancy nutritional status, dietary iron intake, parity, socioeconomic circumstances, physiological iron requirements, and adherence to iron supplementation may influence maternal hematological and biochemical profiles (6,14). Women entering pregnancy with relatively limited iron reserves may be particularly susceptible to progressive depletion as gestation advances. Consequently, evaluation at a single time point may not adequately reflect the dynamic trajectory of maternal iron status.

This issue is particularly relevant in Pakistan, where maternal nutritional deficiencies continue to represent an important healthcare concern. Pakistani studies have reported substantial variation in hematological and iron-related parameters among women of reproductive age and pregnant populations, suggesting that maternal iron status may be influenced by regional, nutritional, socioeconomic, and healthcare-related factors (6,15). Karachi represents a large and socioeconomically diverse metropolitan population, making locally derived longitudinal evidence valuable for understanding how hematological and iron indices change across gestation.

Much of the available evidence evaluates pregnant women at a single gestational stage or compares different groups of women across trimesters. Such cross-sectional assessments cannot fully characterize within-participant changes occurring as pregnancy progresses. Longitudinal follow-up of the same women from early to late pregnancy provides a stronger approach for examining the temporal pattern

of hematological adaptation and iron depletion because each participant contributes measurements at successive gestational stages. Simultaneous evaluation of conventional hematological indices and biochemical markers of iron status can therefore provide clinically relevant information regarding the period during which deterioration in maternal iron status becomes most apparent.

Accordingly, the present prospective longitudinal study aimed to evaluate trimester-wise changes in hemoglobin concentration, MCV, serum ferritin, serum iron, TIBC, and transferrin saturation among pregnant women receiving antenatal care in Karachi, Pakistan. The study further sought to characterize the progression of anemia and iron depletion from the first through the third trimester and to examine the relationship between maternal characteristics and the development of iron deficiency anemia during pregnancy.

MATERIALS AND METHODS

A prospective longitudinal observational study was conducted among pregnant women receiving antenatal care at selected tertiary-care healthcare facilities in Karachi, Pakistan. Study activities were undertaken over a 12-month period from January to December 2024. The longitudinal design was selected to evaluate changes occurring within the same participants across successive stages of pregnancy rather than comparing independent groups of women assessed at different gestational ages. A total of 150 pregnant women were enrolled during early pregnancy and scheduled for assessment during the first, second, and third trimesters. Follow-up assessments were performed at 8–12 weeks, 20–24 weeks, and 32–36 weeks of gestation, respectively. Complete longitudinal follow-up through the third trimester was available for 138 participants.

Pregnant women attending routine antenatal-care services at the participating facilities were screened for eligibility during the first trimester. Eligible participants were women aged 18–40 years with a clinically and ultrasonographically confirmed singleton pregnancy, a gestational age of 8–12 weeks at enrollment, planned regular antenatal follow-up, willingness to provide venous blood samples at each trimester assessment, and no previous history of a major hematological disorder. Women with multiple pregnancy, known hemoglobinopathies including thalassemia or sickle-cell disease, chronic kidney disease, liver disease, autoimmune disorders, malignancy, a history of blood transfusion within the preceding six months, or severe pregnancy complications requiring immediate medical intervention were excluded. Participants who entered the study but did not complete the required longitudinal follow-up were treated as losses to follow-up rather than as baseline eligibility exclusions.

After enrollment, demographic, obstetric, clinical, and relevant nutritional information was recorded using a structured data-collection form. Variables collected included maternal age, gestational age, parity, previous pregnancy history, socioeconomic status, dietary pattern, use of iron supplementation, and antenatal-care history. These characteristics were recorded to describe the study population and to permit assessment of their relationship with the development of iron deficiency anemia. Written informed consent was obtained from participants before study procedures were undertaken.

Each participant underwent repeated hematological and biochemical assessment at three predefined gestational periods: 8–12 weeks during the first trimester, 20–24 weeks during the second trimester, and 32–36 weeks during the third trimester. The same core laboratory parameters were evaluated at each assessment to characterize within-participant changes across pregnancy. Approximately 5 mL of venous blood was collected under aseptic conditions at each follow-up visit. Samples intended for complete blood count assessment were collected in ethylenediaminetetraacetic acid tubes, whereas samples intended for biochemical iron-profile assessment were collected in plain gel tubes.

Complete blood count analysis was performed using an automated hematology analyzer. Hemoglobin concentration was measured as the principal hematological indicator of anemia, while mean corpuscular volume was evaluated as an erythrocyte index relevant to the development of microcytic changes

associated with declining iron availability. The same hematological measurements were repeated during each trimester to permit longitudinal comparison of maternal blood indices.

Serum samples were used for assessment of maternal iron status. The biochemical parameters evaluated were serum ferritin, expressed in ng/mL; serum iron, expressed in µg/dL; total iron-binding capacity, expressed in µg/dL; and transferrin saturation, expressed as a percentage. Serum ferritin was evaluated as an indicator of maternal iron storage, while serum iron and TIBC were used to characterize circulating iron availability and iron-binding capacity. Transferrin saturation was calculated from the measured serum iron and TIBC values using the following formula:

Transferrin saturation (%) = (Serum iron / TIBC) × 100.

Laboratory procedures were conducted according to the routine standardized procedures applied by the participating clinical laboratories. Internal quality-control procedures were applied during laboratory analysis to maintain analytical consistency and reliability across repeated measurements. Hematological and iron-profile findings from each visit were recorded in the study database together with the corresponding demographic and clinical information.

Maternal anemia status was evaluated using pregnancy-specific hemoglobin criteria, while iron status was assessed through the combined interpretation of serum ferritin, serum iron, TIBC, transferrin saturation, and hematological indices. Evaluation of multiple parameters was used to characterize the progressive pattern of maternal iron depletion rather than relying solely on hemoglobin concentration. Trimester-wise laboratory measurements were compared to determine whether hematological indices and biochemical iron markers remained stable or changed with advancing gestation. Anemia prevalence was determined at each gestational assessment, and maternal characteristics recorded at enrollment were considered in relation to the development of iron deficiency anemia.

Continuous variables, including hemoglobin concentration, MCV, serum ferritin, serum iron, TIBC, and transferrin saturation, were summarized using means and standard deviations. Categorical participant characteristics were summarized using frequencies and percentages. Longitudinal changes in hematological and iron-profile variables were evaluated across the three trimester assessments using repeated-measures statistical analysis appropriate to the within-participant study design. Statistical comparisons were undertaken to assess variation across gestational stages, and the relationship between recorded maternal characteristics and development of iron deficiency anemia was evaluated where applicable. Statistical significance was defined as a two-sided p-value <0.05.

The analysis population for complete trimester-wise longitudinal comparisons comprised participants with measurements available across all three scheduled assessments. Of the 150 women initially enrolled, 138 completed follow-up through the third trimester and constituted the complete longitudinal dataset used for the reported trimester-wise analysis. Participants with incomplete follow-up were excluded from the complete-case longitudinal comparisons. This distinction between the enrolled cohort and the complete longitudinal analysis population was maintained when interpreting the study findings.

All participant information and laboratory findings were recorded in a secure study database and handled confidentially. Participation was voluntary, and informed consent was obtained before enrollment and repeated blood sampling.

RESULTS

Participant Characteristics

A total of 150 pregnant women were enrolled during the first trimester. Complete assessments across all three predefined gestational periods were available for 138 participants, corresponding to a complete follow-up rate of 92.0%; 12 participants (8.0%) did not complete the required longitudinal follow-up and were excluded from the complete-case trimester-wise analysis. The mean maternal age was 27.4 ± 4.6

years. Most participants were aged 21–30 years, the majority were multigravida, and approximately one-third were primigravida. Regular iron supplementation during pregnancy was reported by most participants.

Table 1. Trimester-Wise Changes in Hematological Parameters Among Pregnant Women With Complete Follow-Up (n = 138)

Parameter	First Trimester, 8–12 weeks	Second Trimester, 20–24 weeks	Third Trimester, 32–36 weeks	First-to-Third Trimester Change	p-value
Hemoglobin (g/dL), mean ± SD	11.8 ± 1.2	11.2 ± 1.1	10.7 ± 1.1	−1.1	<0.001
MCV (fL), mean ± SD	84.6 ± 5.4	82.1 ± 5.8	79.8 ± 6.1	−4.8	0.002
Anemia, n (%)	26 (18.8)	41 (29.7)	58 (42.0)	+32 cases; +23.2 percentage points	<0.001

Abbreviations: MCV, mean corpuscular volume; SD, standard deviation.

Hemoglobin concentration declined progressively across gestation, decreasing from 11.8 ± 1.2 g/dL during the first trimester to 11.2 ± 1.1 g/dL during the second trimester and 10.7 ± 1.1 g/dL during the third trimester. This represented an absolute first-to-third trimester reduction of 1.1 g/dL, with a statistically significant overall difference across the reported trimester assessments (p<0.001).

A similar progressive decline was observed in MCV. Mean MCV decreased from 84.6 ± 5.4 fL in the first trimester to 82.1 ± 5.8 fL in the second trimester and 79.8 ± 6.1 fL in the third trimester. The absolute reduction between the first and third trimester was 4.8 fL (p=0.002), demonstrating an increasing tendency toward microcytic erythrocyte indices as pregnancy advanced.

The prevalence of anemia also increased across successive gestational stages. Anemia was present in 26 of 138 women (18.8%) during the first trimester, increased to 41 of 138 (29.7%) during the second trimester, and reached 58 of 138 (42.0%) during the third trimester (p<0.001). This corresponded to an absolute increase of 23.2 percentage points between the first and third trimesters and 32 additional women meeting the applied anemia criterion by the third-trimester assessment.

Table 2. Trimester-Wise Changes in Maternal Iron Profile Parameters Among Pregnant Women With Complete Follow-Up (n = 138)

Parameter	First Trimester	Second Trimester	Third Trimester	First-to-Third Trimester Change	p-value
Serum ferritin (ng/mL), mean ± SD	38.5 ± 18.4	25.6 ± 14.7	17.2 ± 11.5	−21.3	<0.001
Serum iron (µg/dL), mean ± SD	86.4 ± 24.5	72.8 ± 22.1	61.5 ± 20.6	−24.9	<0.001
TIBC (µg/dL), mean ± SD	312.6 ± 45.3	335.8 ± 48.6	358.4 ± 52.1	+45.8	<0.001
Transferrin saturation (%), mean ± SD	28.1 ± 8.5	22.3 ± 7.9	17.5 ± 7.1	−10.6 percentage points	<0.001

Abbreviations: SD, standard deviation; TIBC, total iron-binding capacity. Note: First-to-third trimester changes were calculated directly from the reported trimester means.

Maternal iron-profile parameters demonstrated a consistent deterioration across successive trimesters. Serum ferritin showed the largest proportional decline among the measured iron-status indicators, decreasing from 38.5 ± 18.4 ng/mL in the first trimester to 25.6 ± 14.7 ng/mL in the second trimester and 17.2 ± 11.5 ng/mL in the third trimester. The absolute first-to-third trimester reduction was 21.3 ng/mL, and the reported difference across gestational assessments was statistically significant (p<0.001).

Serum iron followed a similar pattern, declining from 86.4 ± 24.5 µg/dL during the first trimester to 72.8 ± 22.1 µg/dL during the second trimester and 61.5 ± 20.6 µg/dL during the third trimester. This represented an absolute decrease of 24.9 µg/dL from early to late pregnancy (p<0.001).

In contrast to ferritin and serum iron, TIBC increased progressively with advancing gestation. Mean TIBC increased from 312.6 ± 45.3 µg/dL in the first trimester to 335.8 ± 48.6 µg/dL in the second trimester and 358.4 ± 52.1 µg/dL in the third trimester. The first-to-third trimester increase was 45.8 µg/dL (p<0.001).

Transferrin saturation declined concurrently, falling from 28.1 ± 8.5% in the first trimester to 22.3 ± 7.9% in the second trimester and 17.5 ± 7.1% in the third trimester. The corresponding absolute reduction was

10.6 percentage points ($p<0.001$). Taken together, the reduction in ferritin, serum iron, and transferrin saturation alongside the progressive increase in TIBC demonstrated a consistent trimester-dependent pattern of declining maternal iron availability.

Across the complete longitudinal cohort, advancing gestation was accompanied by progressive reductions in hemoglobin concentration, MCV, serum ferritin, serum iron, and transferrin saturation, while TIBC increased. Anemia prevalence increased from 18.8% in the first trimester to 42.0% in the third trimester. The most pronounced biochemical change was observed in serum ferritin, which declined by 21.3 ng/mL between the first and third trimesters. These findings demonstrate a consistent temporal pattern of declining hematological and biochemical iron-status indices across pregnancy.

DISCUSSION

The present prospective longitudinal study demonstrated a consistent deterioration in hematological and biochemical indicators of maternal iron status as pregnancy advanced. Among women who completed assessments across all three trimesters, mean hemoglobin and MCV progressively decreased, serum ferritin and serum iron declined substantially, transferrin saturation decreased, and TIBC increased. These changes were accompanied by an increase in anemia prevalence from 18.8% during the first trimester to 42.0% during the third trimester. Considered collectively rather than as isolated laboratory measurements, these findings indicate that advancing gestation was associated with progressively reduced maternal iron availability and increasing evidence of iron-restricted erythropoiesis.

The decline in hemoglobin concentration from 11.8 ± 1.2 g/dL in the first trimester to 10.7 ± 1.1 g/dL in the third trimester is compatible with the well-recognized hematological adaptations of pregnancy. Expansion of maternal plasma volume exceeds the proportional increase in red-cell mass, resulting in physiological hemodilution and a reduction in measured hemoglobin concentration as gestation progresses (7–10). Hemodilution alone, however, is unlikely to explain the complete pattern observed in the present cohort because declining hemoglobin occurred simultaneously with marked reductions in ferritin, serum iron, and transferrin saturation and an increase in TIBC. Pregnancy substantially increases iron utilization for maternal erythropoiesis, placental development, and fetal growth, particularly during the second and third trimesters, and inadequate replenishment of these requirements can progressively exhaust maternal iron reserves (7,8,16).

Anemia became increasingly frequent with advancing gestation, rising from 26 of 138 women (18.8%) in the first trimester to 41 (29.7%) in the second trimester and 58 (42.0%) in the third trimester. This pattern is consistent with the broader epidemiology of gestational anemia, in which the burden commonly increases later in pregnancy as physiological requirements intensify and pre-existing nutritional deficiencies become more apparent (3–5). Severe maternal anemia has been associated with increased maternal morbidity and mortality, while inadequate maternal nutritional status is also associated with unfavorable fetal and neonatal outcomes (4,5). The increasing anemia frequency observed in this cohort is particularly relevant in Pakistan, where nutritional and socioeconomic determinants contribute substantially to iron deficiency among women of reproductive age and where regional studies have documented considerable variation in maternal hemoglobin, MCV, and ferritin concentrations (6,15,23).

MCV declined from 84.6 ± 5.4 fL during the first trimester to 79.8 ± 6.1 fL during the third trimester. Although MCV remains within or near conventional reference ranges in many women during earlier stages of iron depletion, a progressive decline can reflect increasingly iron-restricted erythropoiesis. Hematological indices change throughout normal pregnancy, but biochemical depletion of stored iron generally precedes pronounced microcytosis or overt anemia (9,11,12). This temporal relationship is clinically important because reliance on hemoglobin or MCV alone can fail to identify declining iron reserves before hematological consequences become established. The simultaneous assessment of

biochemical and hematological parameters therefore provides a more informative representation of maternal iron status than any single marker considered independently (11–13).

Serum ferritin demonstrated the most pronounced reduction among the measured biochemical indicators, decreasing from 38.5 ± 18.4 ng/mL in the first trimester to 17.2 ± 11.5 ng/mL in the third trimester, an absolute decline of 21.3 ng/mL. Ferritin reflects maternal storage iron and commonly declines as stored iron is mobilized to meet increasing maternal, placental, and fetal requirements (8,11–13). The trajectory observed in this study therefore supports progressive depletion of maternal iron stores over gestation. However, ferritin is also an acute-phase reactant and may be influenced by inflammatory states; interpretation of individual ferritin concentrations should consequently consider the wider clinical and biochemical context rather than applying the marker in isolation (11,13,17).

Changes in circulating iron transport were similarly consistent with diminishing iron availability. Serum iron decreased from 86.4 ± 24.5 µg/dL to 61.5 ± 20.6 µg/dL, while transferrin saturation declined from 28.1 ± 8.5% to 17.5 ± 7.1%. Conversely, TIBC increased from 312.6 ± 45.3 µg/dL during the first trimester to 358.4 ± 52.1 µg/dL during the third trimester. Increased transferrin production and iron-binding capacity can occur as available iron declines, while lower transferrin saturation reflects reduced circulating iron available for erythropoiesis. These complementary changes strengthen the interpretation of progressive reduction in maternal iron availability beyond that indicated by hemoglobin alone (7,8,16,17).

The longitudinal pattern should also be considered in relation to maternal nutritional and clinical characteristics. Iron status during pregnancy is influenced by pre-pregnancy iron reserves, parity, dietary intake, socioeconomic circumstances, gestational demands, and the use and adherence to iron supplementation (6,13,14). Most participants in the present study reported regular iron supplementation, yet hematological and biochemical iron indices continued to decline. This observation should not be interpreted as evidence that supplementation was ineffective because dose, preparation, initiation time, adherence, absorption, baseline iron status, and therapeutic indication were not incorporated into the available longitudinal outcome analysis. Evidence from supplementation studies indicates that antenatal iron supplementation can reduce iron deficiency and maternal anemia, although the magnitude of benefit and effects on broader maternal and infant outcomes vary according to baseline status, treatment regimen, adherence, and population characteristics (18,19,25). Continuity of antenatal care and appropriate identification and management of iron deficiency remain particularly relevant in settings where nutritional and healthcare-access barriers coexist (22).

The findings have practical implications for antenatal assessment. Hemoglobin measurement is inexpensive and widely available but identifies only one component of iron-related hematological change. The marked decline in ferritin and transferrin saturation observed before the end of pregnancy supports the clinical value of considering biochemical iron assessment in women with suspected depletion or an increased risk of iron deficiency. Established clinical guidance and reviews emphasize early recognition of iron deficiency, assessment of iron stores where clinically indicated, and appropriate oral or parenteral replacement according to severity, gestational stage, tolerance, and response to treatment (1,16,17,20,21). Recent Pakistani studies have similarly highlighted the importance of systematic antenatal identification and management of iron deficiency anemia, including both continuity-of-care approaches and treatment of established deficiency (22–24). The present study did not evaluate therapeutic effectiveness and therefore does not establish that routine biochemical screening improves pregnancy outcomes; rather, it demonstrates that substantial changes in iron-related laboratory indices can occur across gestation even when hemoglobin is considered concurrently.

A principal strength of this study was its prospective longitudinal design, which permitted hematological and iron-profile changes to be observed repeatedly within the same women at predefined gestational stages. This approach reduces the between-participant variation inherent in comparing independent cross-sectional trimester groups and provides a clearer representation of temporal changes

during pregnancy. The study also evaluated several complementary markers rather than relying exclusively on hemoglobin, allowing the observed hematological changes to be considered alongside indices of iron storage, circulating iron, iron-binding capacity, and transferrin saturation.

Several limitations should nevertheless be considered when interpreting the findings. Twelve of the 150 enrolled participants did not complete all three assessments, resulting in an 8.0% loss to complete longitudinal follow-up. Complete-case analysis may introduce attrition bias if women who did not complete follow-up differed systematically from those retained. Participants were recruited from selected tertiary-care facilities in Karachi; consequently, the findings may not fully represent pregnant women receiving care in primary-care, rural, or other regional settings. Maternal characteristics including diet, socioeconomic status, parity, previous pregnancy history, and iron supplementation were collected, but adjusted estimates quantifying their independent relationships with longitudinal iron changes were not available in the reported analysis. Iron supplementation may therefore have introduced clinically important heterogeneity that could not be accounted for. In addition, inflammatory biomarkers were not included among the reported laboratory measurements, limiting assessment of potential inflammation-related variation in ferritin. These factors warrant caution when generalizing the observed mean trajectories to all pregnant women or interpreting the biochemical changes as equivalent in magnitude across individual participants.

Overall, the study demonstrates that maternal hematological and iron-profile parameters undergo substantial and directionally consistent changes across pregnancy. The parallel decline in hemoglobin, MCV, ferritin, serum iron, and transferrin saturation, together with increasing TIBC and anemia prevalence, supports assessment of maternal iron status as a dynamic process rather than a single laboratory event. Longitudinal evaluation integrating conventional hematological indices with clinically appropriate biochemical assessment may provide a more complete characterization of maternal iron status as gestation progresses.

CONCLUSION

Among pregnant women completing longitudinal follow-up, advancing gestation was associated with progressive reductions in hemoglobin, MCV, serum ferritin, serum iron, and transferrin saturation and a corresponding increase in TIBC, while anemia prevalence increased from 18.8% in the first trimester to 42.0% in the third trimester. The concurrent deterioration in hematological and biochemical indices indicates that hemoglobin alone provides an incomplete representation of changes in maternal iron status across pregnancy. These findings support trimester-sensitive interpretation of maternal hematological and iron-profile measurements and demonstrate the value of considering markers of iron storage and availability alongside routine hematological indices when clinically assessing pregnant women.

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