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Maternal Hematological Indices as Early Predictors of Adverse Fetomaternal Outcomes: A Prospective Cohort Study

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Abstract

Objective: To evaluate the association between maternal hematological indices and adverse fetomaternal outcomes.

Design: Prospective cohort study.

Setting: Tertiary care teaching hospital, India.

Population: Two hundred pregnant women followed prospectively from early gestation until delivery.

Methods: Serial hematological parameters including hemoglobin concentration, red blood cell count, and mean corpuscular volume were assessed throughout pregnancy using automated hematology analyzers. Logistic regression analysis was performed to evaluate associations between maternal hematological abnormalities and adverse fetomaternal outcomes.

Primary outcome measures: Fetal growth restriction, stillbirth, neonatal jaundice, preterm birth, and NICU admission.

Results: Second-trimester anemia was significantly associated with fetal growth restriction (OR 3.4, 95% CI 1.1–10.2; p=0.035). Low first-trimester red blood cell count was associated with a 3.4-fold increased risk of fetal growth restriction (OR 3.4, 95% CI 1.0–11.1; p=0.043). Third-trimester low mean corpuscular volume was significantly associated with stillbirth (OR 5.3, 95% CI 1.3–21.4; p=0.022), while second-trimester low mean corpuscular volume was associated with neonatal jaundice (OR 5.0, 95% CI 1.1–22.7; p=0.041). No significant associations were observed with preterm birth or NICU admission.

Conclusions: Maternal hematological abnormalities, particularly anemia and altered erythrocyte indices, are significantly associated with adverse fetomaternal outcomes and may serve as accessible markers for early pregnancy risk stratification.

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Introduction

Adverse fetomaternal outcomes remain major contributors to maternal and perinatal morbidity and mortality worldwide. Maternal physiological and hematological status plays a crucial role in placental development, fetal oxygenation, and overall pregnancy outcome.[1]

Maternal anemia is among the most common medical complications of pregnancy and has been associated with fetal growth restriction (FGR), preterm birth, low birth weight, and perinatal mortality.[2–4] Reduced maternal hemoglobin concentration impairs oxygen delivery to the placenta and fetus, contributing to chronic intrauterine hypoxia and placental dysfunction.[5]

Erythrocyte indices such as red blood cell (RBC) count and mean corpuscular volume (MCV) reflect maternal nutritional and iron status and may influence fetal growth and placental vascular adaptation.[6–8] Iron deficiency remains highly prevalent during pregnancy, particularly in low- and middle-income countries, and continues to contribute substantially to adverse pregnancy outcomes.[9,10]

Previous studies have primarily evaluated anemia as an isolated parameter; however, the longitudinal predictive role of routine hematological indices in adverse fetomaternal outcomes remains incompletely understood.[11–13] Identification of simple and routinely available hematological predictors may improve early antenatal risk stratification and permit timely intervention.

Therefore, the present study aimed to evaluate the association between maternal hematological indices and adverse fetomaternal outcomes in a prospective cohort of pregnant women.

Methods

Study design and setting

This prospective cohort study was conducted in the Department of Obstetrics and Gynecology, Jawaharlal Nehru Medical College and Hospital, Aligarh Muslim University, Aligarh, India, between May 2023 and May 2025.

Participants

A total of 200 pregnant women aged 18–36 years with singleton pregnancies were enrolled during early gestation and followed prospectively until delivery.

Inclusion criteria

Singleton pregnancy

Gestational age <14 weeks at recruitment

Maternal age 18–36 years

Exclusion criteria

Pre-existing diabetes mellitus

Chronic hypertension

Multiple pregnancy

Chronic systemic or inflammatory disease

Known hematological disorders

Data collection and laboratory analysis

Venous blood samples were collected during each trimester as part of routine antenatal assessment. Hematological parameters including hemoglobin concentration, red blood cell count, hematocrit, and mean corpuscular volume were measured using automated hematology analyzers according to standard laboratory protocols.[14]

Outcome measures

Maternal and neonatal outcomes assessed included:

Fetal growth restriction (FGR)

Stillbirth

Neonatal jaundice

Preterm birth

NICU admission

Statistical analysis

Data were analysed using SPSS software (version XX). Continuous variables were expressed as mean ± standard deviation and categorical variables as percentages. Logistic regression analysis was performed to estimate odds ratios (OR) and 95% confidence intervals (CI). A p-value <0.05 was considered statistically significant.

Results

A total of 200 pregnant women were enrolled and followed until delivery. Adverse fetomaternal outcomes observed in the cohort included fetal growth restriction, stillbirth, neonatal jaundice, preterm birth, and NICU admission.

Association between anemia and fetal growth restriction

Second-trimester anemia was significantly more common among women who subsequently developed fetal growth restriction. Women with anemia during the second trimester demonstrated an approximately threefold increased risk of FGR compared with non-anemic women (OR 3.4, 95% CI 1.1–10.2; p=0.035).

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Logistic regression analysis to determine the association of Anemia on maternal Outcomes

Maternal Outcomes		Anemia I trimester		Anemia II trimester		Anemia III trimester	
		OR	P value	OR	Pvalue	OR	P value
Mode of delivery	vaginal	1.8(0.7-5.1)	0.234	0.7(0.4-1.5)	0.474	1.9(0.9-4.2)	0.085
	C section						
Preterm		0.9(0.2-4.2)	0.868	2.0(0.6-6.7)	0.22	1.6(0.4-6.3)	0.484
PROM		0.7(0.3-2.1)	0.583	1.4(0.6-3.2)	0.393	0.8(0.2-2.4)	0.693
HDP		2.4(0.3-19.0)	0.407	0.5(0.1-1.9)	0.328	0.7(0.1-3.6)	0.743
FGR		2.2(0.3 17.6)	0.456	3.4(1.0-11.0)	0.035	1.4(0.4-5.5)	0.586
PLY		NA		0.6(0.06-6.4)	0.718	NA	
Oligo		NA		2.7(0.6-12.6)	0.194	3.7(0.8-17.6)	0.09
IHCP		NA		2.7(0.6-12.6)	0.194	0.7(0.09-6.6)	0.82
Induction of labor		1.1(0.4-2.1)		0.8(0.4-1.6)	0.545	0.6(0.21.5)	
Macrosomia		1.0(0.1-9.1)		0.3(0.03-2.7)	0.297	NA	

Logistic regression analysis to determine the association of Anemia on Fetal Outcomes

Fetal Outcomes	Anemia I trimester		Anemia II trimester		Anemia III trimester	
	OR	P value	OR	P value	OR	P value
LBW	0.7(0.2-2.6)	0.575	0.9(0.3-3.0)	0.989	0.7(0.1-3.2)	0.66
APG AT 1	1.4(0.1-13.2)	0.752	0.8(0.1-4.6)	0.756	NA	
APGAT 5	0.3(0.09-1.1)	0.079	0.9(0.3-3.4)	0.99	0.9(0.2-4.4)	0.938
Stillbirth	0.5(0.1-1.9)	0.325	1.5(0.4-4.7)	0.538	0.9(0.2-4.4)	0.939
NICU AD	2.0(0.2-16.1)	0.513	1.5(0.4-4.7)	0.538	0.9(0.2-4.4))	0.938
HYPOGLYCEMIA	NA		1.3(0.2-8.1)	0.756	NA	
RDS	NA		0.9(0.2-4.0])	0.991	NA	
NEOJAUNDICE	NA		0.8(0.1-4.1)	0.779	0.7(0.09-6.6)	0.779

Similarly, low first-trimester red blood cell count was significantly associated with fetal growth restriction. Women with reduced RBC count during early pregnancy had a 3.4-fold higher likelihood of developing FGR compared with women with normal erythrocyte counts (OR 3.4, 95% CI 1.0–11.1; p=0.043).

Logistic regression analysis to determine the association of low RBC on maternal Outcomes

MaternalOutcomes		Low RBC I trimester		Low RBC IItrimester		Low RBC IIItrimester	
		OR	P value	OR	P value	OR	P value
Modeofdelivery	Normal	1.0(0.5-2.5)	0.887	NA		NA	
	C- section						
Preterm		1.6(0.4-6.5)	0.451	NA		NA	
PROM		1.8(0.7-4.9)	0.19	NA		NA	
HDP		0.8(0.1-3.7)	0.78	NA		NA	
FGR		3.4(1.0-11.1)	0.043	NA		NA	
PLY		1.6(0.1-16.3)	0.67	NA		NA	
Oligo		NA		NA		NA	
IHCP		2.0(0.3-10.8)	0.415	NA		NA	
Macrosomia		NA		NA		NA	
Induction of labor		0.8(0.4-2.1)	0.908	NA		NA	

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Logistic regression analysis to determine the association of Low RBC on Fetal Outcomes

	Low RBC I trimester		Low RBC II trimester	Low RBC III trimester
Fetal outcomes	**OR**	**P value**		
APG AT 1	NA		NA	NA
APG AT 5	0.4(0.5-3.4)	0.423	NA	NA
LBW	1.2(0.3-4.6)	0.748	NA	NA
Stillbirth	1.6(0.4-6.5)	0.451	NA	NA
NICUAD	1.6(0.4-6.5)	0.451	NA	NA
HYPOGLYCEMIA	1.2(0.1-11.3)	0.857	NA	NA
RDS	0.6(0.07-4.9)	0.634	NA	NA
NEO JAUNDICE	0.8(0.09-6.9)	0.846	NA	NA

Association between erythrocyte indices and adverse neonatal outcomes

Low mean corpuscular volume (MCV), suggestive of microcytic anemia and iron deficiency, demonstrated significant association with adverse perinatal outcomes. Third-trimester low MCV was significantly associated with stillbirth, with affected pregnancies demonstrating more than fivefold increased risk (OR 5.3, 95% CI 1.3–21.4; p=0.022).

Logistic regression analysis to determine the association of Low MCV on maternal Outcomes

Maternal Outcomes		Low MCV I trimester		Low MCV II trimester		Low MCV III trimester	
		OR	**P value**	**OR**	**P value**	**OR**	**P value**
Mode of delivery	**Normal C-section**	0.8(0.3-2.6)	0.738	1.4(0.6-3.4)	0.386	0.7(0.2-2.9)	0.718
Preterm		0.9(0.1-7.5)	0.934	2.1(0.5-8.5)	0.268	1.2(0.1-10.2)	0.852
PROM		0.7(0.1-3.6)	0.756	1.4(0.5-4.3)	0.469	0.5(0.06-3.8)	0.48
HDP		1.7(0.3-8.6)	0.479	0.5(0.05-3.6)	0.454	1.0(0.1-8.4)	0.983
FGR		1.1(0.1-9.2)	0.919	0.4(0.06-3.9)	0.506	1.1(0.1-9.2)	0.919
PLY		NA		NA		NA	
Oligo		1.7(0.2-15.1)	0.623	1.0(0.1-8.8)	0.982	NA	
IHCP		NA		1.0(0.1-8.8)	0.983	NA	
Macrosomia		NA		1.0(0.1-8.8)	0.982	NA	
Induction of labor		1.1(0.4-3.4)	0.776	0.6(0.2-1.7)	0.35	1.2(0.4-4.0)	0.749

In addition, second-trimester low MCV was significantly associated with neonatal jaundice (OR 5.0, 95% CI 1.1–22.7; p=0.041).

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Logistic regression analysis to determine the association of Low MCV on Fetal Outcomes

Fetal outcomes	Low MCV I trimester		Low MCV II trimester		Low MCV III trimester	
	OR	P value	OR	P value	OR	P value
APGAT1	0.4(0.04-3.6)	0.401	NA		0.3(0.03-2.7)	0.278
APGAT 5	2.1(0.4-10.6)	0.349	0.5(0.06-4.3)	0.565	2.9(0.6-14.9)	0.195
LBW	1.6(0.3-7.8)	0.546	0.4(0.5-3.3)	0.409	3.9(0.9-16.1)	0.055
Stillbirth	2.1(0.4-10.6)	0.349	0.5(0.06-4.3)	0.565	5.3(1.3-22.6)	0.022
NICU AD	0.9(0.1-7.5)	0.934	0.5(0.06-4.3)	0.565	NA	
HYPOGLYCEMIA	NA		NA		NA	
RDS	NA		0.8(0.09-6.3)	0.799	NA	
NEO JAUNDICE	1.7(0.2-15.1)	0.623	5(1.0-23.8)	0.041	NA	

Other fetomaternal outcomes

No statistically significant associations were identified between maternal hematological parameters and preterm birth or NICU admission in the present study population.

Discussion

Main findings

This prospective cohort study demonstrates significant associations between maternal hematological abnormalities and adverse fetomaternal outcomes. Maternal anemia, low red blood cell count, and reduced mean corpuscular volume were associated with fetal growth restriction, stillbirth, and neonatal jaundice. These findings support the hypothesis that impaired maternal hematological status adversely influences placental development, fetal oxygenation, and neonatal adaptation.

Interpretation in context of biological mechanisms

Maternal hematological status plays a fundamental role in maintaining adequate placental perfusion and oxygen transport throughout pregnancy. Reduced maternal hemoglobin concentration compromises oxygen delivery to the fetoplacental unit, contributing to chronic intrauterine hypoxia, oxidative stress, and placental insufficiency.[2–5]

The observed association between second-trimester anemia and fetal growth restriction is biologically plausible and consistent with previous studies demonstrating impaired fetal growth among anemic mothers. Reduced oxygen-carrying capacity may impair trophoblastic invasion and uteroplacental vascular adaptation, thereby restricting fetal nutrient and oxygen supply.[5,6]

Low first-trimester RBC count was similarly associated with fetal growth restriction, suggesting that maternal hematological reserve during early placentation may be particularly important for optimal placental development. Early gestational impairment in erythropoietic function may contribute to defective placental angiogenesis and chronic placental hypoperfusion.[11]

The association between low MCV and stillbirth further highlights the potential role of iron deficiency and microcytic anemia in adverse pregnancy outcomes. Iron deficiency has been associated with placental oxidative stress, endothelial dysfunction, impaired immune adaptation, and abnormal fetal oxygenation.[7–9,12] Similarly, the association between low MCV and neonatal jaundice may reflect increased fetal erythrocyte turnover secondary to chronic intrauterine hypoxic stress. Chronic fetal hypoxia may stimulate compensatory erythropoiesis, thereby increasing bilirubin production after birth.[13]

Comparison with previous literature

The findings of the present study are consistent with prior literature linking maternal anemia with adverse perinatal outcomes. Allen et al. and Scholl et al. previously demonstrated strong associations between maternal iron deficiency and impaired fetal growth.[2,4] Steer et al. similarly reported associations between maternal hemoglobin abnormalities and altered birth outcomes.[3] Previous studies evaluating erythrocyte indices in pregnancy have also highlighted the importance of iron status and placental vascular adaptation in determining fetal growth and survival.[6–8] However, relatively few studies have prospectively evaluated trimester-specific hematological abnormalities as predictors of fetomaternal outcomes. The longitudinal prospective design of the present study therefore strengthens existing evidence by demonstrating temporal relationships between maternal

hematological abnormalities and subsequent pregnancy complications.

Strengths and limitations

The strengths of this study include its prospective cohort design, longitudinal follow-up throughout pregnancy, and assessment of routinely available hematological parameters. The use of inexpensive and universally accessible laboratory investigations increases the translational applicability of the findings. However, several limitations should be acknowledged. The study was conducted at a single tertiary care centre, which may limit generalisability to broader populations. Residual confounding due to nutritional status, dietary iron intake, socioeconomic variables, and maternal inflammatory status cannot be completely excluded. Additionally, serum ferritin and other biochemical iron markers were not evaluated, limiting precise characterization of iron deficiency states. Multivariable adjustment for all potential confounders was also limited by sample size.[15]

Clinical implications

The findings of this study have important implications for antenatal care, particularly in resource-limited settings where maternal anemia remains highly prevalent. Routine hematological parameters are inexpensive, widely available, and already integrated into standard antenatal assessment.[1,10] Early identification of women with hematological abnormalities may permit targeted nutritional intervention, iron supplementation, closer fetal surveillance, and improved risk stratification for adverse pregnancy outcomes.

Future directions

Further large-scale multicentric studies incorporating biochemical iron markers, inflammatory biomarkers, and placental vascular assessment are required to validate these findings and establish clinically useful predictive models for adverse fetomaternal outcomes.

Conclusion

Maternal hematological abnormalities, particularly anemia, reduced red blood cell count, and low mean corpuscular volume, are significantly associated with adverse fetomaternal outcomes including fetal growth restriction, stillbirth, and neonatal jaundice. These findings support the potential role of routine hematological indices as simple, accessible, and cost-effective markers for early antenatal risk stratification. Integration of hematological assessment into predictive models may improve identification of high-risk pregnancies and facilitate timely intervention aimed at reducing maternal and neonatal morbidity. Further multicentric prospective studies are required to validate these associations and determine their role in clinical obstetric practice.

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All named authors meet the ICMJE criteria for authorship for this article ,take responsibility for the integrity of the work as a whole,and have given their approval for this version to be published. Individual contributions of each author are as follows: Dr Nilofar Khan ,contributed to the study design, study planning,data acquisition, data analysis and drafting manuscript. Dr Dalia Rafat,contributed to data acquisition, data analysis and drafting manuscript.Dr Hamid Ashraf contributed to data analysis and drafting manuscript.Dr Suhailur Rehman contributed to data analysis and drafting manuscript .Dr Tabassum Nawab contributed to study planning ,data acquisition and drafting a manuscript of this article.

Declaration of competing interests

The author declare that they have no known competing financial interest or personal relationship that could have appeared to influence the work reported in this paper.

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