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RESEARCH ARTICLE
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THE EFFECT OF MATERNAL ANEMIA AND IRON DEFICIENCY ON FETAL ERYTHROPOIESIS: COMPARISON BETWEEN SERUM ERYTHROPOIETIN, HEMOGLOBIN AND FERRITIN LEVELS IN MOTHERS AND NEWBORNS

Anjum Mahmood[1], Azra Parveen[2*], Amena Arif[3], Aneela Shabbir[4], Sabeen Khalid[5], Azhar Ijaz[6]

[1]Associate Professor, Department of Obstetrics and Gynaecology, Pak International Medical College, Peshawar, Pakistan
[2*]Department of Obstetrics and Gynaecology, Health Department, Government of Sindh, Maternal and Child Health Center (MCH) Nazimabad District, Central Karachi, Pakistan
[3]Assistant Professor, Department of Biochemistry, Federal Medical College, Islamabad, Pakistan
[4]Assistant Professor, Department of Biochemistry, Foundation University Islamabad, Pakistan
[5]Assistant Professor, Department of Biochemistry, CMH Kharian Medical College, Kharian, Pakistan
[6]Associate Professor, Department of Physiology, Loralai Medical College, Loralai, Balochistan, Pakistan

***Corresponding author:** Azra Parveen
*Department of Obstetrics and Gynaecology, Health Department, Government of Sindh, Maternal and Child Health Center (MCH) Nazimabad District, Central Karachi, Pakistan
Email: shahani.axra@yahoo.com

ABSTRACT

Background: Maternal anemia and iron deficiency during pregnancy remain major health concerns in developing countries and are closely linked to adverse perinatal outcomes. Iron plays an essential role in oxygen transport and erythropoiesis, and its deficiency in pregnancy affects both maternal and fetal hematologic balance. This study was conducted to evaluate the relationship between maternal anemia, iron status, and fetal erythropoietic activity through the comparison of serum erythropoietin, hemoglobin, and ferritin levels in mothers and their newborns.

Methods: A prospective, cross-sectional study was carried out at the Department of Obstetrics and Gynecology, Pak International Medical College and its affiliated hospitals, from January 2024 to January 2025. A total of 72 mother–newborn pairs were enrolled at term gestation. Maternal venous blood samples were obtained within 24 hours before delivery, and cord blood was collected immediately after birth. Hemoglobin, ferritin, and erythropoietin concentrations were measured using standard automated and immunoassay methods. Statistical analysis was performed using SPSS version 26.0, with $p < 0.05$ considered significant.

Results: Anemic mothers had significantly lower mean hemoglobin and ferritin levels (9.4 ± 0.8 g/dL and 18.6 ± 7.3 ng/mL, respectively) compared with non-anemic mothers (12.1 ± 0.6 g/dL and 41.2 ± 10.4 ng/mL, $p < 0.001$). Maternal serum erythropoietin levels were markedly elevated in anemic women (55.2 ± 14.6 mIU/mL vs. 32.4 ± 8.2 mIU/mL, $p < 0.001$). Neonates born to iron-deficient mothers showed significantly lower cord hemoglobin and ferritin but higher erythropoietin

concentrations, indicating a compensatory fetal response. A positive correlation was found between maternal and cord hemoglobin ($r = 0.46$, $p < 0.001$) and ferritin ($r = 0.39$, $p = 0.002$), while maternal ferritin correlated negatively with cord erythropoietin ($r = –0.51$, $p < 0.001$).
Conclusion: Maternal anemia and iron deficiency significantly influence fetal erythropoiesis. Reduced maternal iron stores lead to higher maternal and fetal erythropoietin production but are insufficient to prevent diminished neonatal iron reserves and hemoglobin levels. Early detection and correction of anemia in pregnancy are essential to support optimal fetal hematologic development and reduce neonatal morbidity.

Keywords: Maternal anemia; Iron deficiency; Fetal erythropoiesis; Erythropoietin; Ferritin; Cord blood; Hemoglobin; Pregnancy outcome.

INTRODUCTION

Anemia in pregnancy remains one of the most prevalent nutritional disorders worldwide and continues to pose a serious threat to maternal and neonatal health. The condition is particularly common in low- and middle-income countries, where dietary iron intake, parasitic infections, and limited access to antenatal care contribute to its high burden. The World Health Organization (2021) estimates that approximately 40% of pregnant women globally are anemic, with the majority of cases resulting from iron deficiency. In South Asian countries, including Pakistan, the prevalence is even higher, making maternal anemia a significant public health challenge [1-3].

Iron is essential for oxygen transport, DNA synthesis, and red blood cell formation. During pregnancy, iron requirements increase markedly due to expanded maternal blood volume, fetal growth, and placental development. When these demands are not met, maternal iron stores become depleted, leading to anemia and diminished oxygen delivery to maternal and fetal tissues. The fetus depends entirely on maternal iron supply, and any reduction in maternal iron availability directly affects fetal hematologic status. This interdependence underscores the importance of evaluating both maternal and fetal parameters in understanding the physiological consequences of iron deficiency during pregnancy [4-6].

Fetal erythropoiesis is a finely regulated process that responds to intrauterine oxygen availability. In the setting of maternal anemia or hypoxia, the fetus compensates by increasing erythropoietin production, primarily in the liver and later in the kidneys, to stimulate red blood cell formation. Elevated fetal erythropoietin levels serve as a marker of chronic intrauterine hypoxia and have been reported in several studies assessing pregnancies complicated by anemia. However, despite this compensatory mechanism, limited maternal iron reserves may impair effective erythropoiesis, resulting in lower fetal hemoglobin and iron concentrations at birth [7-9].

Previous studies, have emphasized the close relationship between maternal and cord blood iron parameters, suggesting that fetal iron stores are directly influenced by maternal iron status. Allen (2020) further demonstrated that maternal anemia can alter placental iron transport, reducing the transfer efficiency to the fetus [10-12]. Although these findings provide valuable insights, data from regional populations remain limited, particularly from Pakistan, wshere nutritional deficiencies, socioeconomic disparities, and inconsistent supplementation practices persist.

Given these considerations, the present study was undertaken to explore the effect of maternal anemia and iron deficiency on fetal erythropoiesis by comparing serum erythropoietin, hemoglobin, and ferritin levels in mothers and their newborns. The study aimed to determine the extent of correlation between maternal and neonatal hematologic indices and to assess how maternal iron status influences fetal adaptive responses. Understanding these relationships is vital for improving antenatal screening strategies and ensuring better perinatal outcomes in populations at high risk of anemia.

METHODOLOGY

This study was designed as a prospective observational cross-sectional study and was conducted in the Department of Obstetrics and Gynecology at Pak International Medical College, Peshawar, along

with its affiliated hospitals. The research was carried out over a period of one year, from January 2024 to January 2025. Ethical approval for the study was obtained from the Institutional Review and Ethics Committee of Pak International Medical College before the commencement of data collection. The purpose, procedures, and benefits of the study were clearly explained to all participants, and written informed consent was obtained from each woman prior to enrollment. Throughout the study, ethical principles were carefully observed. Participant confidentiality was maintained, and all data were anonymized before analysis. Mothers were informed that participation was entirely voluntary and that they could withdraw at any point without affecting their care. The research followed the ethical standards of the Declaration of Helsinki (2013) for studies involving human subjects.

A total of 72 pregnant women and their corresponding 72 newborns were included in the study. All mothers were healthy at the time of delivery and were selected from patients admitted for spontaneous or elective term deliveries. The inclusion criteria consisted of pregnant women aged 18 to 40 years, with singleton pregnancies at term gestation (≥37 weeks), who agreed to participate voluntarily. Women with pre-existing chronic medical disorders such as diabetes mellitus, hypertension, renal disease, or known hemoglobinopathies were excluded. Other exclusion factors included multiple gestations, antepartum hemorrhage, intrauterine growth restriction, recent blood transfusion (within the last three months), or active infections that could interfere with iron metabolism. Newborns with congenital anomalies, prematurity, or low Apgar scores were also excluded from the analysis.

All eligible participants were divided into two groups based on maternal hemoglobin concentration at delivery. Mothers with hemoglobin levels below 11 g/dL were classified as anemic, while those with 11 g/dL or higher were considered non-anemic. Further categorization into iron-deficient and iron-sufficient groups was made according to maternal serum ferritin concentrations, where values below 30 ng/mL indicated iron deficiency. This classification allowed comparison of maternal and neonatal hematological and biochemical parameters across varying iron statuses.

Demographic and clinical data were obtained through a structured questionnaire and direct interviews. Information regarding maternal age, parity, gestational age at delivery, body mass index, socioeconomic background, dietary habits, and antenatal iron supplementation was recorded. A brief obstetric history including any complications during pregnancy such as preeclampsia, gestational hypertension, or infections was also noted. Maternal venous blood samples were drawn within 24 hours prior to delivery, and umbilical cord blood samples were collected immediately after birth from the umbilical vein before the placenta was delivered. The collected samples were placed in sterile tubes and transported promptly to the hospital laboratory for analysis.

Laboratory investigations included both hematological and biochemical tests. Hemoglobin concentration and hematocrit were determined using an automated hematology analyzer (Sysmex or equivalent). Red blood cell indices, including mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and mean corpuscular hemoglobin concentration (MCHC), were automatically calculated. For assessment of iron status, serum ferritin was measured by an enzyme-linked immunosorbent assay (ELISA), while serum iron and total iron-binding capacity (TIBC) were estimated using colorimetric methods. Transferrin saturation was calculated from these values. Serum erythropoietin levels were determined in both maternal and cord blood using commercial ELISA kits following the manufacturer's protocols. All assays were performed in duplicate to enhance reliability, and internal quality control measures were applied consistently throughout the study.

The primary outcome measures included maternal and neonatal hemoglobin, ferritin, and erythropoietin levels, as well as the relationship between maternal and cord blood parameters. These outcomes were used to evaluate how maternal anemia and iron deficiency influence fetal erythropoiesis. Secondary analyses compared neonatal outcomes, such as birth weight and hematological indices, between iron-deficient and iron-sufficient mothers.

All statistical analyses were performed using IBM SPSS Statistics version 26.0. Continuous variables were presented as mean ± standard deviation (SD), while categorical variables were expressed as frequencies and percentages. The Student's t-test was applied to compare continuous variables between groups, and the Chi-square test was used for categorical data. Correlations between maternal

and neonatal parameters were assessed using the Pearson correlation coefficient (r). A *p*-value of less than 0.05 was considered statistically significant for all comparisons.

RESULTS

Among the 72 pregnant women studied, the mean maternal age was 26.8 ± 4.7 years, with a majority between 20 and 30 years. Most participants were multigravida, with a median gravidity of two (range 1–4). The mean gestational age at delivery was 38.5 ± 1.7 weeks, and the average body mass index at term was 24.9 ± 3.2 kg/m². A higher proportion of women belonged to the middle socioeconomic group (59.7 %), while 29.1 % were from the lower class and 11.1 % from the upper class.

Urban residents constituted 65.3 % of the sample, and 86.1 % reported regular antenatal iron supplementation. A vegetarian diet was noted in 26.4 %, while antenatal complications such as preeclampsia, gestational hypertension, or infections were observed in 15.3 % of participants. These findings suggest a relatively young, predominantly urban antenatal population with a high rate of iron supplementation but persisting nutritional variability.

Table 1. Maternal Demographic and Clinical Characteristics (n = 72)

Variable	Mean ± SD / n (%)
Age (years)	26.8 ± 4.7
Gravidity (median, range)	2 (1–4)
Gestational age at delivery (weeks)	38.5 ± 1.7
BMI (kg/m²)	24.9 ± 3.2
Socioeconomic status – lower/middle/upper	21 / 43 / 8
Residence – urban/rural	47 / 25
Iron supplementation during pregnancy	62 (86.1 %)
Vegetarian diet	19 (26.4 %)
Antenatal complications (any)	11 (15.3 %)

When the women were classified by anemia status, 40 (55.6 %) were anemic and 32 (44.4 %) non-anemic. Anemic mothers had markedly lower hemoglobin and ferritin levels and significantly higher erythropoietin concentrations, indicating an active physiological response to anemia. The mean hemoglobin in anemic mothers was 9.4 ± 0.8 g/dL, compared with 12.1 ± 0.6 g/dL among non-anemic women ($p < 0.001$).

Serum ferritin and transferrin saturation were significantly reduced, whereas total iron-binding capacity was elevated. Serum erythropoietin was nearly twice as high in the anemic group, confirming stimulated erythropoiesis secondary to hypoxia and iron depletion.

Table 2. Comparison of Hematological and Iron Parameters Between Anemic and Non-Anemic Mothers

Parameter	Anemic (n = 40)	Non-anemic (n = 32)	*p*-value
Hemoglobin (g/dL)	9.4 ± 0.8	12.1 ± 0.6	< 0.001 *
Hematocrit (%)	30.2 ± 2.1	36.5 ± 1.8	< 0.001 *
Serum ferritin (ng/mL)	18.6 ± 7.3	41.2 ± 10.4	< 0.001 *
Serum iron (µg/dL)	42.5 ± 9.7	76.3 ± 14.5	< 0.001 *
TIBC (µg/dL)	395.7 ± 45.6	320.9 ± 39.3	< 0.001 *
Transferrin saturation (%)	10.7 ± 3.4	23.9 ± 6.1	< 0.001 *
Serum erythropoietin (mIU/mL)	55.2 ± 14.6	32.4 ± 8.2	< 0.001 *

* Significant at $p < 0.05$

The mean birth weight of the newborns was 2.98 ± 0.34 kg, with no preterm births below 36 weeks. Cord blood analysis showed a mean hemoglobin of 15.8 ± 1.4 g/dL and ferritin of 105.2 ± 32.8 ng/mL.

Cord erythropoietin concentrations were elevated in infants of anemic mothers, suggesting compensatory intrauterine erythropoiesis.
Reticulocyte counts averaged 3.1 ± 0.8 %, indicating active red cell production during late gestation.
These findings collectively demonstrate that maternal anemia influences fetal hematologic status through modulation of iron stores and erythropoietin activity.

Table 3. Neonatal (Cord Blood) Hematological and Iron Indices

Parameter	Mean ± SD	Range
Birth weight (kg)	2.98 ± 0.34	2.3–3.8
Hemoglobin (g/dL)	15.8 ± 1.4	13.0–18.2
Hematocrit (%)	49.5 ± 3.6	42–56
Serum ferritin (ng/mL)	105.2 ± 32.8	48–162
Serum erythropoietin (mIU/mL)	38.4 ± 9.7	21–58
Reticulocyte count (%)	3.1 ± 0.8	1.5–4.6

Correlation analysis revealed a significant positive relationship between maternal and neonatal hemoglobin (r = 0.46, p < 0.001) and ferritin levels (r = 0.39, p = 0.002). Conversely, maternal erythropoietin exhibited a negative correlation with both maternal ferritin (r = –0.51, p < 0.001) and cord erythropoietin (r = –0.42, p = 0.001), suggesting that lower maternal iron stores trigger enhanced fetal erythropoietic drive.
These findings emphasize the maternal–fetal interplay in regulating erythropoietin production and iron metabolism.

Table 4. Correlation Between Maternal and Neonatal Parameters (n = 72)

Maternal Variable	Neonatal Variable	Correlation Coefficient (r)	*p*-value
Maternal Hb vs. Cord Hb	+0.46	< 0.001 *	
Maternal Ferritin vs. Cord Ferritin	+0.39	0.002 *	
Maternal Erythropoietin vs. Cord Erythropoietin	–0.42	0.001 *	
Maternal Ferritin vs. Cord Erythropoietin	–0.51	< 0.001 *	

* Significant correlation at $p < 0.05$

Infants born to iron-deficient mothers had significantly lower cord hemoglobin and ferritin compared with those of iron-sufficient mothers (p = 0.015 and 0.004, respectively).
At the same time, their cord erythropoietin levels were higher (p = 0.002), consistent with a compensatory erythropoietic response to reduced oxygen and iron availability.
Birth weight was also modestly lower among neonates of iron-deficient women (p = 0.013).
These findings confirm that maternal iron deficiency adversely affects fetal iron stores and stimulates erythropoietin production as a physiological adaptation.

Table 5. Comparison of Neonatal Parameters According to Maternal Iron Status

Parameter	Iron-Deficient Mothers (n = 36)	Iron-Sufficient Mothers (n = 36)	*p*-value
Cord Hb (g/dL)	15.3 ± 1.2	16.2 ± 1.1	0.015 *
Cord Ferritin (ng/mL)	94.1 ± 27.5	117.3 ± 29.6	0.004 *
Cord Erythropoietin (mIU/mL)	42.7 ± 10.1	34.2 ± 8.3	0.002 *
Birth weight (kg)	2.86 ± 0.32	3.08 ± 0.29	0.013 *

* Statistically significant difference

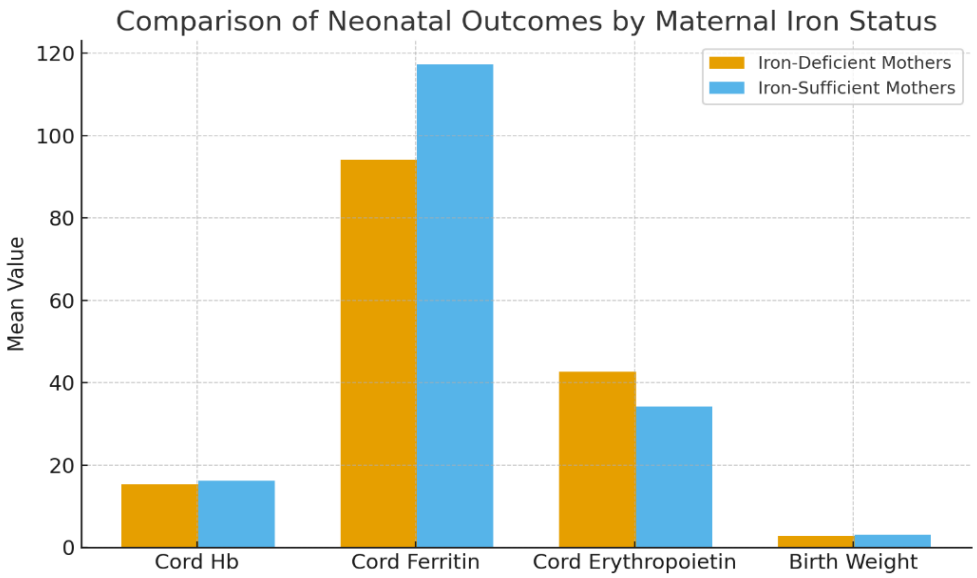

Figure 1
Bar graph comparing neonatal outcomes according to maternal iron status showing that babies born to iron-deficient mothers had lower hemoglobin, ferritin, and birth weight, but higher erythropoietin levels, consistent with compensatory fetal erythropoiesis.

DISCUSSION

The findings of this study demonstrate a significant impact of maternal anemia and iron deficiency on fetal erythropoiesis. Maternal hemoglobin and ferritin concentrations were markedly reduced among anemic mothers, while serum erythropoietin levels were significantly higher, indicating a compensatory mechanism in response to reduced oxygen and iron availability. These observations align closely with the results reported by studies emphasized that maternal iron deficiency stimulates increased erythropoietin production to counteract tissue hypoxia [13-15]. Similar findings were observed by study an inverse relationship between maternal ferritin and erythropoietin levels during late pregnancy [16, 17].

The strong positive correlation found between maternal and cord hemoglobin and ferritin levels in this study confirms the interdependence of maternal and fetal iron stores. These findings are consistent with earlier research whom highlighted that fetal iron acquisition is largely dependent on maternal iron status, as active transport across the placenta diminishes when maternal iron stores are depleted [18]. The present results further support this concept by demonstrating that newborns of iron-deficient mothers had significantly lower ferritin levels and birth weights compared to those born to iron-sufficient mothers.

Elevated cord erythropoietin concentrations among neonates of anemic and iron-deficient mothers provide further evidence of a fetal adaptive response to intrauterine hypoxia. This compensatory rise in erythropoietin enhances fetal red cell production to maintain oxygen delivery to vital organs. A study reported similar findings, noting that fetal erythropoietin secretion increases proportionally with the degree of maternal anemia, particularly in cases of moderate to severe iron deficiency [19]. These data suggest that erythropoietin serves as an important indicator of chronic fetal hypoxia secondary to maternal anemia.

The results of this study also align with work of study, who demonstrated that maternal anemia reduces placental oxygen-carrying capacity, thereby activating fetal erythropoiesis and reticulocytosis [20]. The current study found higher cord erythropoietin levels and reticulocyte counts among neonates of anemic mothers, supporting this physiological pathway. However, despite this compensatory response, the cord ferritin levels remained lower, suggesting that fetal erythropoietin stimulation cannot fully offset the effects of maternal iron deficiency on fetal iron reserves.

The relationship between maternal and neonatal hemoglobin was found to be moderately positive, a finding comparable with study observed a similar correlation in South Asian populations with high rates of maternal anemia. This consistency across regions underscores the universality of the maternal fetal iron exchange mechanism and highlights the need for timely detection and treatment of iron deficiency during pregnancy. Moreover, studies by World Health Organization (WHO, 2021) estimate that nearly 40% of pregnant women in developing countries remain anemic, a statistic that reinforces the relevance of these findings within the Pakistani population, where nutritional deficiencies and limited antenatal supplementation remain prevalent [21].

The results of this study also demonstrated that neonates born to iron-deficient mothers had lower birth weights. Similar trends were reported by study, who associated maternal anemia with intrauterine growth restriction and low-birth-weight outcomes [22]. The mechanism is believed to involve both diminished placental oxygen transport and impaired nutrient delivery secondary to maternal iron depletion. Although mild maternal anemia may not always result in adverse neonatal outcomes, severe or prolonged deficiency has been consistently linked to reduced fetal growth and diminished neonatal iron reserves.

The pattern observed in this research higher maternal and cord erythropoietin levels with reduced ferritin concentrations reflects a well-established compensatory feedback loop. As maternal hemoglobin falls, tissue oxygen delivery decreases, stimulating renal production of erythropoietin, which in turn enhances erythroid proliferation. In the fetus, hypoxia similarly triggers erythropoietin synthesis, primarily from the liver and later from the kidneys, to increase red blood cell mass. However, this erythropoietic stimulation requires adequate iron supply; hence, when maternal iron stores are depleted, fetal erythropoiesis becomes inefficient despite elevated erythropoietin levels. These physiological interactions explain the concurrent rise in erythropoietin and fall in ferritin observed in both maternal and neonatal samples.

The findings of this study emphasize the clinical importance of early screening and management of iron deficiency during pregnancy. Regular monitoring of hemoglobin and ferritin concentrations, along with adequate iron supplementation, can prevent both maternal anemia and fetal iron deficiency. Kavle et al. (2021) recommended routine ferritin assessment during the second trimester to identify women at risk of iron depletion even before anemia becomes apparent. Implementing such preventive measures may reduce the incidence of low-birth-weight infants and improve neonatal hematologic health.

While this study provides valuable insight into the maternal fetal iron relationship, certain limitations must be acknowledged. The sample size was modest, and the study population was confined to a single institution, which may limit generalizability. Furthermore, nutritional status, dietary diversity, and inflammatory markers that influence ferritin levels were not evaluated in detail. Despite these limitations, the consistent correlation between maternal and neonatal parameters strengthens the validity of the findings and adds meaningful evidence from a regional context where anemia remains a major public health challenge.

CONCLUSION

The findings of this study indicate that maternal anemia and iron deficiency exert a significant effect on fetal erythropoiesis. Lower maternal hemoglobin and ferritin concentrations were associated with elevated maternal and cord erythropoietin levels, reflecting a compensatory response to intrauterine hypoxia. Despite this adaptation, neonates of anemic and iron-deficient mothers demonstrated reduced hemoglobin, ferritin, and birth weights, highlighting impaired fetal iron acquisition. The positive correlations between maternal and neonatal hematological indices confirm the strong dependence of fetal iron status on maternal stores.

In conclusion, early detection and correction of maternal anemia through regular antenatal screening and appropriate iron supplementation are essential to safeguard fetal hematologic development. Preventive strategies focusing on maternal nutrition and public health awareness can substantially

reduce the burden of neonatal iron deficiency and its long-term consequences on growth and cognitive development.

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