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A study of Haemoglobin and serum ferritin levels in newborn babies born to anaemic women

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ABSTRACT

Background: This study aimed to assess the association between mode of delivery and the haemoglobin and serum ferritin levels of newborns, by comparing these parameters in vaginally delivered and caesarean-delivered neonates of non-anaemic mothers and mothers with iron deficiency anaemia (IDA). **Methods:** A total of 120 full-term pregnant women aged 18–40 years, admitted through the OPD or labour room of the Department of Obstetrics and Gynaecology, were recruited. All participants had full-term newborns without prenatal or perinatal complications. The study was conducted at a tertiary care hospital in eastern India. **Results:** Eighty percent of the cases had not taken iron–folic acid (IFA) supplementation during pregnancy, compared with 20% among the controls, and this difference was statistically significant. A strong correlation was observed between maternal and neonatal haemoglobin and serum ferritin levels. As the data were not normally distributed, the Spearman-Rho correlation test was applied, yielding significant results. Newborns of mothers with IDA had markedly lower haemoglobin and ferritin levels compared to those born to non-anaemic mothers, indicating that maternal IDA adversely affects neonatal iron stores. An independent t-test confirmed statistically significant differences in haemoglobin levels between cases and controls**. Conclusion:** Maternal IDA appears to negatively influence the iron reserves of newborns. Optimal placental iron transfer is essential for fetal development and for ensuring adequate iron stores at birth to support early infancy. When maternal iron stores are depleted, fetal iron requirements may be only partially met. Further research is warranted to

explore how lower cord serum ferritin levels relate to iron status and developmental outcomes later in infancy.

Keywords: Serum Ferritin Levels, Pregnancy, Iron Deficiency Anaemia, Newborn Iron Status

Introduction

Anaemia is characterized by haemoglobin levels that are quantitatively or qualitatively lower than normal.[1] Iron deficiency anaemia (IDA) is the most common type of anaemia among pregnant women, particularly in developing countries.[2] According to ICMR, anaemia in pregnancy is classified as: mild (haemoglobin 10–10.9 g/dL), moderate (7–9.9 g/dL), severe (<7 g/dL), and very severe (<4 g/dL).[3] The CDC defines anaemia in pregnancy as haemoglobin <11 g/dL in the first and third trimesters, and <10.5 g/dL in the second trimester. Similarly, the WHO defines anaemia in pregnancy as haemoglobin <11 g/dL.[4]

During pregnancy, disproportionate increases in plasma volume and red cell mass lead to physiological anaemia. The rise in red cell mass is due to increased erythropoiesis, which heightens the demand for iron—mobilized primarily from body stores in the form of ferritin.[5] Pregnant women are especially vulnerable to iron deficiency because of increased metabolic demands related to the growing fetus, placenta, and maternal tissues, combined with dietary inadequacies. In developing countries, many women begin pregnancy with depleted iron reserves due to poor dietary intake, menstrual blood loss, and closely spaced pregnancies. The severity of anaemia is therefore inversely related to iron stores.[6]

Maternal iron deficiency significantly influences fetal iron stores. Iron is transferred from mother to fetus via the placenta through a unidirectional mechanism. Maternal iron binds to transferrin, which delivers iron to the placenta; although maternal transferrin does not cross the placenta, iron is subsequently bound to fetal transferrin for transport to fetal tissues.[7-8] Serum ferritin is considered a sensitive and reliable biomarker of iron stores and provides a more accurate indication of iron status than haemoglobin.

Iron deficiency anaemia is a major preventable cause of perinatal complications, including preterm delivery, intrauterine growth restriction, and neonatal or perinatal mortality. Adequate iron is critical for early brain growth and neurological function, as it supports neuronal and glial metabolism, neurotransmitter synthesis, and myelination. Deficiency adversely affects cognitive, emotional, motor, and neuropsychological development.[9] A pregnant woman requires approximately 1000 mg of iron to meet the demands of pregnancy, while the fetus accumulates iron at a rate of about 1.35 mg/kg/day during the third trimester—a critical period of rapid brain development.[10]

Placental iron transport adjusts according to maternal iron status. In conditions of iron deficiency, placental transferrin receptor expression increases to enhance iron uptake. However, once maternal stores are severely depleted, this compensatory mechanism becomes insufficient, resulting in reduced fetal iron pools. Consequently, fetuses of

iron-deficient mothers may have decreased cord serum ferritin and, eventually, reduced haemoglobin levels at birth.[11] Although term infants generally have adequate iron reserves for the first three months of life, iron stores are subsequently mobilized to meet the expanding haemoglobin mass. Because breast milk alone cannot meet increasing postnatal iron requirements, plasma ferritin levels naturally decline. Infants born with lower iron stores are at higher risk of early depletion and subsequent IDA.[12]

Iron plays a crucial role in neurotransmission, energy metabolism, and myelination in the developing brain. The human brain growth spurt begins in the last trimester and continues through the first two years of life. Studies have demonstrated persistently lower cognitive and motor scores in infants with IDA, even after iron supplementation. Biochemical evidence, such as slowed nerve conduction velocity, further highlights the neurological impact of early iron deficiency.[13]

Disruption of maternal–fetal iron homeostasis due to maternal IDA may contribute to adverse outcomes, including preterm birth, intrauterine growth restriction, and increased neonatal mortality.[14] Understanding the relationship between maternal iron status and fetal iron stores is essential for preventing iron deficiency during pregnancy and infancy, thereby improving maternal and neonatal outcomes. Therefore, this study was undertaken to evaluate the effect of maternal iron deficiency anaemia on newborn iron stores.

Materials and Methods

This observational case–control study was conducted in the Department of Obstetrics and Gynaecology at a tertiary care hospital in eastern India. A total of 120 full-term pregnant women aged 18–40 years, admitted through the OPD or labour room, were recruited. All participants were in labour and delivered newborns with a birth weight ≥2500 g, without any prenatal or perinatal complications.

Of the 120 mothers, 60 were selected as controls (non-anaemic, haemoglobin >11 g/dL and serum ferritin >12 μg/L), and 60 were classified as cases with iron deficiency anaemia (IDA) (haemoglobin <11 g/dL and serum ferritin <12 μg/L).

Umbilical cord clamping was performed one minute after delivery for all participants. Five millilitres of maternal venous blood and 5 ml of cord blood were collected. Each sample was divided into two aliquots: one aliquot of whole blood was immediately sent to the haematology laboratory for haemoglobin estimation, while the second aliquot was allowed to clot at room temperature. Serum obtained from the clotted sample was used for ferritin analysis. Serum ferritin concentration was measured using an electrochemiluminescence immunoassay (ECLIA), based on a sandwich assay principle with two-point calibration and an assay time of 18 minutes. The ECLIA technique employs a ruthenium complex and tripropylamine (TPA) as key components.

Ethical approval for the study was obtained from the Institutional Ethics Committee, and written informed consent was provided by all participants.

Inclusion Criteria

- Primigravida or multigravida at term (≥37 weeks)
- Non-anaemic pregnant women (controls)
- Pregnant women with IDA (cases)
- Newborns of mothers with or without IDA delivered vaginally or by caesarean section

Exclusion Criteria

- Preterm deliveries
- Multifetal gestation
- Eclampsia
- Antepartum haemorrhage
- Diabetes mellitus
- Cardiac, renal, respiratory, or haematological disorders
- Newborns with birth asphyxia, neonatal sepsis, or elevated inflammatory markers such as C-reactive protein >5 mg/L

Statistical Analysis

Data analysis was performed using SPSS version 24. A p-value <0.05 was considered statistically significant. Statistical tests applied included the independent t-test, Mann–Whitney U test, Pearson correlation coefficient, Spearman's rho correlation test, and chi-square test, depending on the nature and distribution of the variables.

RESULTS

Table no.1: Distribution of Iron Intake during Pregnancy among Cases and Controls

SES	**Case**	**Control**	**X^2 - Value**	**P-Value**
Low	28	42	15.9	0.00008
Middle	28	13		
High	4	5		
Total	60	60		
Socioeconomic Status Distribution				
Iron Intake during Pregnancy	**Cases (%)**	**Control (%)**	**X^2- Value**	**P-Value**
Yes	24 (40)	48 (80)	60	0.00001
No	36 (60)	12 (20)		
Total	60 (100)	60 (100)		

The chi-square test yielded a statistically significant p-value of 0.00008 when the p-values of the two groups-the cases and the controls-were compared.

Table no.2: Correlation between Maternal and Newborn Serum Ferritin Levels

Hb Status(gm/dl)	**Mean**	**SD**	**Correlation Co-Efficient (r)**	**P-Value**
Maternal HB	10.5	1.1	0. 478	0.00001
Newborn HB	12.9	1.8		

Ferritin Levels(µg//L).				
Maternal S. Ferritin	20.4	4.1	0.478	0.0001
Newborn S. Ferritin	105.5	6.5		

Using Pearson's correlation test, the p-value of the aforementioned parameters was determined to be 0.00001, indicating a very significant statistical result.

Table no.3: Association between Maternal and Newborn Haemoglobin Status in Cases and Controls

Hb Status of Pregnant Women (gm/dl)	**Newborn Hb**		**P-Value**
	Mean	**SD**	
Cases (Hb <11)	11.1	1.3	0.0001
Controls (Hb >11)	14.6	2.2	

The p-value was determined using the information in the above table, and the result was 0.0001, which indicates statistical significance.

Table no. 4: Association between Maternal and Newborn Serum Ferritin Status in Cases and Controls

Ferritin Status (µg//L) of Pregnant Women	**Newborn Ferritin**		**P-Value**
	Mean	**SD**	
Cases (S.Ferritin<12)	58.3	3.5	0.0001
Control (S.Ferritin>12)	152.9	6.8	

We performed the Mann-Whitney U-Test to determine a correlation between them because of non parametric distributions of datas. Using the aforementioned information and parameters, the p-value was determined to be 0.0001, indicating a statistically significant link between the blood ferritin levels of mothers and newborns.

DISCUSSION

Out of the 120 participants, 60 were enrolled as controls (non-anaemic, Hb >11 g/dL and serum ferritin >12 µg/L) and 75 as cases with IDA (Hb <11 g/dL and serum ferritin <12 µg/mL). Most participants in both groups were between 23–32 years of age (60% of cases and 72% of controls). More than 60% of women in both groups were primigravidae, and the majority belonged to low socioeconomic backgrounds. During pregnancy, 80% of the women in the case group did not take iron–folic acid (IFA) supplementation, compared with only 20% in the control group. This difference was statistically significant. Although both groups had mixed modes of delivery, most delivered vaginally; however, delivery mode was not related to the primary objectives of this study.

A highly significant positive correlation (p = 0.00001) was found between maternal and neonatal haemoglobin concentrations. Similarly, a strong positive correlation was observed between maternal and neonatal serum ferritin levels. Because the data were not normally distributed, the Spearman-Rho correlation test was used, showing

consistent correlation in both groups. Newborns of mothers with IDA had significantly lower haemoglobin ($p = 0.00001$) and serum ferritin levels ($p = 0.0001$) compared to newborns of non-anaemic mothers, indicating that maternal iron status directly influences neonatal iron stores. These findings align with earlier studies.[15–18]

Table 2 demonstrates the relationship between maternal and neonatal haemoglobin levels in both groups. Cord blood analysis showed that neonates born to mothers with low haemoglobin levels were at the highest risk for anaemia ($p = 0.0001$). An independent t-test confirmed this significance. Cord serum ferritin was also lower in newborns of iron-depleted mothers, an observation consistent with previous reports.[19–20] These findings further support the concept that maternal–fetal iron transfer is most affected when maternal iron stores are suboptimal. Some studies, however, did not observe such relationships.[21–23] Methodological differences, including variations in serum ferritin cut-off values (<12 µg/L), failure to exclude infection (which may falsely elevate ferritin), and maternal iron supplementation during pregnancy, may account for the discrepancies. Serum ferritin is a reliable marker of body iron stores.[24] The significantly lower ferritin concentrations among neonates born to IDA mothers in our study suggest that these infants begin life with diminished iron reserves. Similarly, neonatal haemoglobin levels were also significantly lower in the IDA group.

The absence of a statistically significant difference in the prevalence of overt anaemia between groups is not unexpected, as differences in haemoglobin may not be clinically apparent at birth. Hay et al.[25] noted that cord serum ferritin is a good predictor of iron status during the first two years of life. Other studies also suggest that the influence of maternal iron deficiency becomes more evident later in infancy rather than at birth.[26–28] Therefore, infants of IDA mothers may be at increased risk of developing iron deficiency anaemia during early childhood due to reduced iron stores at birth. This may compromise cellular immunity, cognitive development, and overall neurological function.

Our findings also showed that neonatal ferritin and haematological markers were significantly higher than maternal levels, a trend supported by previous studies.[29–30] This is explained by the active, preferential transfer of iron from mother to fetus via the placenta.[31] In iron deficiency states, the placenta appears to compete more effectively with maternal erythropoiesis for circulating transferrin-bound iron. This may occur through increased placental transferrin receptor synthesis.[32]

As iron deficiency becomes more pronounced, the relationship between maternal and fetal iron status changes. Placental iron transport is primarily regulated by fetal demand via fetal transferrin binding and transferrin receptor saturation on the basal (fetal-facing) syncytiotrophoblast layer.[33–35] Placental transferrin receptor (TfR) expression increases with both maternal iron deficiency and heightened fetal iron demand.[37] This enhances maternal–placental iron uptake.[38] However, when maternal iron stores become severely depleted, upregulation of placental TfR cannot fully compensate, leading to reduced fetal iron pools and decreased ferritin and haemoglobin levels at birth.[39]

Despite the high prevalence of maternal iron deficiency in pregnancy, many neonates do not present with overt anaemia. This may be explained by the unique

characteristics of iron absorption and placental transfer. Some studies suggest that the placenta transfers iron independent of maternal–fetal concentration gradients and may have a higher affinity for circulating maternal iron.[40] Iron absorbed from the maternal gastrointestinal tract contributes more significantly to fetal iron supply than maternal iron stores.[41] After 30 weeks of gestation, when maternal iron absorption peaks, the majority of iron transfer to the fetus occurs.[42-43] Neonates born to mothers with low iron stores have been shown to incorporate higher levels of iron tracer from maternal oral…

CONCLUSION

The findings of this study suggest that maternal iron-deficiency anaemia (IDA) may adversely affect the iron reserves of newborns. Optimal placental function is essential for adequate fetal growth and the establishment of sufficient iron stores at birth, which are crucial for early infancy. Although fetal iron needs may be met to some extent even when maternal stores are depleted, they are not fully compensated. Further research is warranted to evaluate how varying levels of reduced cord serum ferritin influence infant iron status and subsequent developmental outcomes.

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