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A Cross-Sectional Comparative Study of Iron Nutrition and Vitamin D Levels in Pregnant and Postpartum Women with Different HIV Infection Statuses and Their Offspring

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Abstract: *Objective:* To compare differences in iron nutritional status and vitamin D levels between HIV-infected and non-infected pregnant women and their offspring, and to provide a reference for nutritional interventions during pregnancy and infancy in HIV-positive populations. *Methods:* A controlled study design was used. A total of 122 HIV-infected pregnant women and their offspring who were registered and delivered at Xichang Maternal and Child Health Hospital, Butuo County Maternal and Child Health Hospital, and Zhaojue County Maternal and Child Health Hospital between January 2024 and December 2025 were selected as the observation group. During the same period, 116 healthy pregnant women who were HIV antibody-negative and their offspring were selected as the control group. Serum iron, ferritin, total iron-binding capacity (TIBC), transferrin, and 25-hydroxyvitamin D [25(OH)D] levels were measured in the pregnant women and their offspring in both groups, and the incidence rates of iron deficiency and vitamin D insufficiency/deficiency were compared. *Results:* Maternal serum iron levels in the observation group were lower than those in the control group, while ferritin, TIBC, and transferrin levels were higher than those in the control group (all $P<0.05$). In the offspring, ferritin and serum iron levels in the observation group were higher than those in the control group, whereas TIBC and transferrin levels were lower than those in the control group (all $P<0.05$). The incidence of iron deficiency among pregnant women in the observation group was higher than that in the control group ($P<0.001$). The incidence rates of transferrin below the normal range and reduced TIBC in the observation group were higher than those in the control group (all $P<0.05$). The incidence of ferritin below the normal range in the offspring was lower than that in the control group ($P<0.001$). *Conclusion:* HIV-infected pregnant women have decreased iron reserves and a significantly increased risk of iron deficiency. Elevated ferritin levels in HIV-exposed offspring tend to reflect inflammation-mediated alterations in iron metabolism rather than true iron overload. HIV infection status did not significantly alter vitamin D levels in the mother–child population, but vitamin D insufficiency was common among pregnant women and infants in this region. Clinically, dynamic monitoring of iron nutrition during pregnancy should be emphasized for HIV-infected pregnant women, and interpretation of iron metabolism indicators in HIV-exposed children

should incorporate inflammatory markers for comprehensive assessment, thereby providing a basis for individualized nutritional supplementation for mothers and infants.

Keywords: HIV infection; Pregnant women; Offspring; Iron metabolism; Vitamin D

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1. Introduction

HIV infection remains a major public health challenge threatening the health of women of childbearing age and their offspring worldwide [1]. With the widespread use of highly active antiretroviral therapy (ART) and continuous improvements in prevention of mother-to-child transmission (PMTCT), an increasing proportion of HIV-infected pregnant women are delivering healthy offspring. Consequently, micronutrient management during pregnancy and infancy in this population has attracted growing attention in maternal and child clinical practice. Iron and vitamin D are key micronutrients during pregnancy and infant growth [2]. In addition to participating in hemoglobin synthesis, iron is involved in immune regulation. During pregnancy, women's physiological iron consumption increases markedly; iron deficiency can increase the risks of maternal anemia and postpartum hemorrhage, interfere with fetal iron reserve establishment, and potentially harm the neurocognitive development of offspring. Vitamin D regulates calcium and phosphorus metabolism and bone development and also mediates immune function; its deficiency can contribute to adverse pregnancy outcomes, infantile rickets, and recurrent respiratory tract infections. The global population of HIV-infected women of childbearing age is substantial, and micronutrient levels in pregnant women and their offspring can affect the actual effectiveness of PMTCT and children's subsequent health, making research in this area of practical public health significance [3]. HIV can disrupt micronutrient metabolic balance through chronic inflammatory responses, intestinal absorption impairment, inadequate nutritional intake, and adverse effects of antiviral drugs [4]. Most current studies focus on the overall nutritional status of HIV-infected individuals. Reports that use paired samples of HIV-infected pregnant women and their offspring and simultaneously compare a full set of iron metabolism indicators with vitamin D are relatively scarce, and existing conclusions remain inconsistent. Some studies have only examined the mother or the child as a single group, making it difficult to fully explain the nutritional changes in both mother and child under HIV exposure. Therefore, this study adopted a cross-sectional controlled design to compare iron metabolism indicators and 25(OH)D concentrations between HIV-infected and non-infected pregnant women and their offspring, and to determine the current prevalence of iron deficiency and vitamin D insufficiency. The aim was to provide a clinical reference for nutritional screening and individualized intervention in HIV-positive pregnant women and exposed offspring.

2. General materials and methods

2.1. General materials

A total of 122 HIV-infected pregnant women and their live-born offspring who were registered and delivered at Xichang Maternal and Child Health Hospital, Butuo County Maternal and Child Health Hospital, and Zhaojue County Maternal and Child Health Hospital between January 2024 and December 2025 were

selected as the observation group. During the same period, 116 healthy pregnant women who underwent prenatal care at these hospitals were HIV antibody-negative, and their offspring were selected as the control group. All subjects or their guardians signed informed consent, and the study protocol was reviewed and approved by the hospital ethics committee.

Inclusion criteria for the observation group: HIV infection diagnosed before or during pregnancy and confirmed by Western blot, regular antiretroviral therapy during pregnancy, and standardized completion of PMTCT. Inclusion criteria for the control group: negative HIV antibody test and no special complications during pregnancy. Exclusion criteria: pregnant women in both groups were excluded if they had gestational hypertension, diabetes, intrahepatic cholestasis of pregnancy, twins or multiple pregnancies, premature rupture of membranes, acute or chronic infection with fever, a history of anemia or blood transfusion, or if they had taken iron or vitamin D supplements within 1 month before the study. Offspring in both groups were excluded if they had preterm birth, low birth weight, congenital malformations, inherited metabolic diseases, acute infectious diseases, or recent supplementation with iron or vitamin D.

2.2. Detection methods

Fasting venous blood (3–5 mL) was collected in the morning, and serum was separated for immediate testing. Serum iron and TIBC were measured using an automated biochemical analyzer, ferritin was measured by chemiluminescence immunoassay, transferrin was measured by immunoturbidimetry, and 25(OH)D was measured by chemiluminescence. The reference intervals of our laboratory were as follows: serum iron 7.80–32.20 μmol/L, ferritin 12.00–135.00 μg/L, TIBC 54.00–77.00 μmol/L, and transferrin 2.00–3.60 g/L. Vitamin D was classified as follows: 25(OH)D < 30 nmol/L was defined as deficiency, 30–50 nmol/L as insufficiency, and ≥ 50 nmol/L as sufficiency.

2.3. Outcome measures

Iron metabolism indicators (serum iron, ferritin, TIBC, and transferrin) and vitamin D (25(OH)D) levels were compared between the two groups of pregnant women; iron metabolism and vitamin D levels were compared between the two groups of offspring; and the incidence rates of iron deficiency and vitamin D insufficiency/deficiency were compared.

2.4. Statistical analysis

SPSS 27.0 software was used for statistical analysis. After normality testing, continuous data were expressed as mean ± standard deviation (SD), and comparisons between groups were performed using the t-test. Categorical data were expressed as n (%), and comparisons between groups were performed using the chi-square test. $P < 0.05$ was considered statistically significant.

3. Results

3.1. Comparison of iron metabolism and Vitamin D levels between the two groups of pregnant women

Maternal serum iron levels in the observation group were lower than those in the control group, while ferritin, TIBC, and transferrin levels were higher than those in the control group (all $P < 0.05$). There was no statistically significant difference in 25(OH)D levels between the two groups of pregnant women ($P = 0.431$),

suggesting that HIV infection status did not significantly affect maternal vitamin D levels. See **Table 1**.

Table 1. Comparison of iron metabolism and vitamin D levels between the two groups of pregnant women (mean ± SD)

Indicator	Control group (n=116)	Observation group (n=122)	*t*	*P*
Age (years)	27.87 ± 4.97	31.99 ± 5.37	6.133	<0.001
Serum iron (μmol/L)	20.13 ± 5.77	19.19 ± 10.26	2.713	0.007
Ferritin (μg/L)	38.47 ± 28.00	42.07 ± 36.40	6.921	<0.001
TIBC (μmol/L)	69.62 ± 10.61	74.93 ± 18.46	7.826	<0.001
Transferrin (g/L)	2.84 ± 0.34	3.12 ± 0.79	6.703	<0.001
25(OH)D (nmol/L)	67.89 ± 31.79	55.16 ± 23.02	0.789	0.431

3.2. Comparison of iron metabolism and vitamin D levels between the two groups of offspring

Ferritin and serum iron levels in the offspring of the observation group were higher than those in the control group, while TIBC and transferrin levels were lower than those in the control group (all $P < 0.05$). There was no statistically significant difference in 25(OH)D levels between the two groups of offspring ($P = 0.455$). See **Table 2**.

Table 2. Comparison of iron metabolism and vitamin D levels between the two groups of offspring (mean ± SD)

Indicator	Control group (n=116)	Observation group (n=122)	*t*	*P*
Mean age (years)	0.62 ± 0.25	0.35 ± 0.27	8.074	<0.001
Serum iron (μmol/L)	17.18 ± 12.52	21.31 ± 9.91	3.773	0.0002
Ferritin (μg/L)	50.33 ± 53.02	225.84 ± 236.79	7.448	<0.001
TIBC (μmol/L)	77.30 ± 19.31	65.60 ± 20.25	3.401	<0.001
Transferrin (g/L)	2.94 ± 0.53	2.50 ± 1.68	2.113	0.036
Vitamin D level (nmol/L)	82.17 ± 35.70	81.17 ± 27.77	0.748	0.455

3.3. Comparison of the Incidence of Iron Deficiency and Vitamin D insufficiency/deficiency

The incidence of iron deficiency among pregnant women in the observation group was higher than that in the control group ($P < 0.001$). The incidence rates of transferrin below the normal range and reduced TIBC in the observation group were higher than those in the control group (all $P < 0.05$). The incidence of ferritin below the normal range in offspring was lower than that in the control group ($P < 0.001$). There was no statistically significant difference in the incidence of vitamin D insufficiency/deficiency between the two groups of pregnant women or offspring ($P = 0.899$). See **Table 3**.

Table 3. Comparison of the incidence of iron deficiency and vitamin D insufficiency/deficiency between the two groups (n (%))

Item	Control group (n=116)	Observation group (n=122)	χ^2	*P*
Maternal iron deficiency (ferritin <12 μg/L)	4 (3.4)	24 (19.7)	15.077	<0.001
Maternal 25(OH)D < 50 nmol/L	58 (50.0)	60 (49.2)	0.016	0.899

Item	Control group (n=116)	Observation group (n=122)	χ^2	*P*
Offspring ferritin < 12 μg/L	18 (15.5)	2 (1.6)	14.878	<0.001
Offspring transferrin < 2.0 g/L	4 (3.4)	26 (21.3)	17.224	<0.001
Offspring TIBC < 54 μmol/L	15 (12.9)	34 (27.9)	8.115	0.004
Offspring vitamin D insufficiency/deficiency	18 (15.5)	14 (11.5)	0.835	0.361

Note: Vitamin D insufficiency/deficiency was defined as 25(OH)D <50 nmol/L.

4. Discussion

Iron is an important trace element involved in hemoglobin synthesis and immune responses, and physiological changes during pregnancy further increase iron consumption [5]. HIV infection can disrupt iron metabolic balance through multiple pathways, including chronic inflammatory stimulation, impaired intestinal absorption, inadequate dietary intake, and adverse effects of antiretroviral drugs. The data in this study showed that serum iron indicators decreased in HIV-infected pregnant women, while TIBC and transferrin levels increased, and the detection rate of iron deficiency reached 19.7%, significantly higher than the 3.4% in healthy controls. This indicates that HIV-infected pregnant women constitute a high-risk group for iron deficiency during pregnancy. From a pathological perspective, persistent HIV infection induces chronic inflammatory responses, upregulates hepcidin expression, inhibits intestinal iron absorption, and causes iron accumulation in macrophages. In addition, disease-related loss of appetite and weakened gastrointestinal absorption, combined with gastrointestinal adverse effects caused by some antiretroviral drugs, ultimately deplete maternal iron reserves [6]. Iron deficiency during pregnancy not only increases the risks of maternal anemia and postpartum hemorrhage but also hinders fetal iron reserve accumulation, with potential long-term negative effects on offspring neurocognitive development. Therefore, routine iron nutritional screening and individualized iron supplementation for HIV-infected pregnant women are of practical clinical importance.

This study found divergent features in iron metabolism indicators between mothers and offspring: the mothers in the observation group had insufficient iron reserves, whereas HIV-exposed offspring had higher serum iron and ferritin levels than controls, a lower proportion of low ferritin, and decreased TIBC and transferrin levels, with increased proportions of insufficiency/deficiency in these two indicators. Ferritin not only reflects body iron reserves but is also an acute-phase reactant; under infection and inflammatory stimulation, ferritin can increase independently of iron overload. HIV-exposed fetuses are affected by intrauterine viral antigens and maternal immune activation and remain in a low-grade inflammatory environment, which may be the main cause of abnormally elevated ferritin in offspring [7]. In addition to these intrauterine inflammation-mediated biological effects, feeding patterns may also confound the differences in iron metabolism between groups. As part of PMTCT strategies, HIV-exposed infants have a higher rate of formula feeding, and the iron load of formula is significantly higher than that of human milk. Increased exogenous iron intake can elevate serum iron and ferritin levels and participate in regulating the synthesis and expression of TIBC and transferrin. This study did not perform stratified analysis by feeding pattern, so the interference of feeding exposure on the tested outcomes could not be completely eliminated. These results suggest that when clinically evaluating iron nutritional status in HIV-exposed infants, ferritin alone should not be used to judge iron overload. Elevated ferritin may result from intrauterine low-grade inflammation-mediated metabolic changes or be regulated by postnatal feeding patterns. Comprehensive assessment should

combine inflammatory markers such as C-reactive protein and feeding history to avoid misjudgment and reduce clinical problems such as excessive iron supplementation or missed diagnosis of iron deficiency.

During pregnancy, the placenta actively transports iron to the fetus to meet fetal hematopoietic developmental needs [8]. In normal late pregnancy, maternal ferritin shows a physiological decline, and HIV infection activates inflammatory pathways that further deplete maternal iron reserves. After delivery, the residual effects of intrauterine immune activation can still regulate hepcidin, leading to elevated ferritin in offspring; this phenomenon does not indicate sufficient body iron reserves. Thus, under HIV infection, inflammation differentially regulates iron metabolism in mothers and offspring. Clinical assessment of maternal and infant iron nutrition should consider their respective physiological and pathological conditions, and maternal iron deficiency should not be used to infer the iron nutritional status of offspring directly.

The vitamin D results showed no statistically significant differences in 25(OH)D levels or the incidence of vitamin D insufficiency/deficiency between the two groups of pregnant women and offspring, suggesting that in HIV-infected individuals receiving standardized ART, infection itself does not significantly affect vitamin D levels [9]. This result is not completely consistent with some literature conclusions, possibly because the subjects had stable antiviral treatment control and the local area has abundant sunlight. However, the rate of vitamin D insufficiency/deficiency among pregnant women in both groups was close to 50%, and some infants also had suboptimal vitamin D levels, highlighting a public health problem of vitamin D insufficiency among local pregnant women and infants. Vitamin D participates in calcium and phosphorus metabolism, bone mineralization, and immune regulation and is of great significance for the growth and immunity of mothers, fetuses, and infants. Even if HIV infection does not directly affect vitamin D levels, routine monitoring is still recommended for HIV-infected pregnant women and their offspring, especially for those using antiretroviral drugs that interfere with vitamin D metabolism, with dynamic measurement of 25(OH)D and supplementation as needed.

Taken together, the results of this study indicate that HIV-infected pregnant women are at high risk of insufficient iron reserves, and elevated ferritin in their offspring is mostly inflammation-mediated iron metabolic remodeling rather than true iron overload. HIV infection has limited impact on maternal and infant vitamin D levels, but vitamin D insufficiency is prominent in this population [10]. Clinically, dynamic monitoring of iron and vitamin D during pregnancy should be improved for HIV-infected pregnant women, and individualized nutritional supplementation should be implemented. Interpretation of iron metabolism indicators in HIV-exposed offspring should be comprehensively judged in combination with inflammatory markers to optimize maternal and infant nutritional outcomes.

This study has several limitations. It was a multicenter cross-sectional controlled study and can only reveal associations between variables, not infer causality. Although the samples were drawn from three medical institutions, the included institutions were relatively geographically concentrated, so extrapolation of the conclusions to other regional populations remains limited. There was an imbalance in offspring age between the two groups, which may have confounded the results of iron metabolism-related tests. This study did not perform stratified analyses based on $CD4^+$ T lymphocyte count, viral load, or specific antiretroviral regimens; the effects of these immune and treatment indicators on iron metabolism and vitamin D levels remain to be further clarified. Future larger-sample, prospective cohort studies with matched offspring age and inclusion of immune- and treatment-related stratification factors are needed to verify the current conclusions further and explore the mechanisms of micronutrient changes in HIV-infected mothers and infants.

5. Conclusion

In summary, this cross-sectional controlled study compared the iron and vitamin D nutritional status of HIV-infected and non-infected pregnant women and their offspring. The results showed that HIV-infected pregnant women had decreased serum iron and increased TIBC and transferrin, with a significantly elevated risk of iron deficiency, indicating that they are a high-risk group for iron deficiency during pregnancy. HIV-exposed offspring had markedly elevated ferritin and decreased TIBC and transferrin; these changes were mostly caused by iron metabolic remodeling triggered by chronic inflammation related to HIV exposure. There was no statistically significant difference in maternal and infant vitamin D levels between the two groups, but the proportion of vitamin D insufficiency was relatively high in both groups. Clinically, dynamic monitoring of iron and vitamin D during pregnancy should be strengthened for HIV-infected pregnant women, and individualized nutritional supplementation should be implemented. When interpreting iron metabolism indicators in HIV-exposed children, comprehensive assessment should be combined with inflammatory markers to improve maternal and child health and nutritional outcomes in this population.

Disclosure statement

The author declares no conflict of interest.

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