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Original Article

Maternal iron status in mothers of infants with ventricular septal defect: A retrospective case–control study

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ABSTRACT

Objective: Maternal nutrition is increasingly recognized as a modifiable determinant of fetal cardiovascular development. Experimental and epidemiological studies suggest that iron deficiency may contribute to congenital heart disease, but data focusing on ventricular septal defect (VSD) remain limited. This study aimed to evaluate the association between maternal iron status and isolated perimembranous VSD in offspring.

Materials and Methods: This single-center retrospective case–control study included mothers of 41 infants younger than one year with isolated perimembranous VSD and 38 control mothers whose infants had no structural heart disease after echocardiographic evaluation. Maternal laboratory parameters were retrieved from electronic medical records, and the earliest available results were used for analysis. Evaluated parameters included hemoglobin, ferritin, vitamin B12, folate, and 25-hydroxyvitamin D. Group comparisons were performed using the Mann–Whitney U test or chi-square test, as appropriate. Binary logistic regression was used to assess independent associations with VSD, and receiver operating characteristic analysis was performed for ferritin.

Results: Maternal ferritin levels were significantly lower in the VSD group than in controls (median 25.8 [IQR 15.5–52.0] vs 57.9 [IQR 21.3–79.3] ng/mL, p=0.012), whereas maternal hemoglobin, vitamin B12, folate, and vitamin D levels did not differ significantly. In multivariable logistic regression adjusted for maternal age, hemoglobin, vitamin B12, and folate, ferritin remained independently associated with VSD (OR 0.83 per 10-ng/mL increase, 95% CI 0.72–0.96, p=0.012). Ferritin showed moderate discriminative ability for VSD (AUC 0.665, 95% CI 0.537–0.781).

Conclusion: Lower maternal ferritin levels were associated with isolated perimembranous VSD in offspring, independent of hemoglobin levels. These findings suggest that reduced maternal iron stores may be relevant to fetal septal development and warrant confirmation in prospective studies with measurements obtained during early pregnancy.

Keywords: Congenital heart disease, ferritin, iron deficiency, perimembranous ventricular septal defect

Introduction

Congenital heart disease (CHD) represents the most common congenital anomaly worldwide, affecting around 1–2% of live births and remaining a leading cause of infant morbidity and mortality. Within the broad spectrum of CHD, ventricular septal defect (VSD) is consistently reported as the most frequently observed subtype (1). The etiology of CHD is multifactorial, arising from complex interactions between genetic predisposition and environmental influences, among which maternal factors during early gestation are increasingly recognized as critical determinants of risk (1–4). Recent systematic review data also suggest that maternal micronutrient deficiencies may contribute to fetal CHD risk, including VSD, although the quality and consistency of the available evidence vary across nutrients and study designs (5).

Maternal nutrition is a key modifiable determinant of fetal development. Pregnancy has been described as a state of impending or existing iron deficiency, and commonly used biomarkers such as hemoglobin and ferritin may not always fully reflect tissue iron sufficiency during gestation (6). During pregnancy, iron requirements rise markedly to support maternal erythropoiesis, placental function and

fetal growth, and a large proportion of pre-pregnant and pregnant women have inadequate iron stores or iron deficiency anemia worldwide (2,7). Iron deficiency is the leading cause of anemia in pregnancy and remains highly prevalent worldwide, particularly where dietary diversity and intake of animal-source foods are low (2,8). Maternal iron deficiency anemia has been linked to adverse perinatal outcomes and impaired offspring development, underscoring the need for systematic screening and treatment in pregnancy (2,8,9). Importantly, iron deficiency may be present even before overt anemia develops, making ferritin-based assessment particularly relevant when evaluating maternal iron stores (5,10).

Experimental work in mice demonstrates that maternal iron deficiency perturbs embryonic cardiovascular morphogenesis, producing ventricular septal defects and outflow tract anomalies through mechanisms involving premature differentiation of cardiac progenitors and increased retinoic acid signaling; early iron repletion can rescue these defects (1). This biological plausibility is supported by developmental studies showing that retinoic acid signaling and second heart field regulation are essential for normal cardiac septation and outflow tract development (11,12). Emerging human data suggest that maternal anemia and low iron status may be associated with an increased risk of congenital heart defects, including ventricular septal defect, indicating that maternal iron deficiency could represent a potentially modifiable factor in VSD development (13,14).

Şırnak is one of the provinces with the highest fertility rates in Türkiye, and the Southeastern Anatolia region, where Şırnak is located, has a high proportion of households in the lowest socioeconomic welfare category (15). In clinical practice, children with congenital heart disease in this region frequently come from families with limited socioeconomic resources, and maternal nutritional deficiencies may therefore represent a potentially underrecognized risk factor. The aim of this study was to evaluate the association between maternal iron status, assessed by ferritin levels, and the presence of VSD in their children.

Materials and Methods

This study was designed as a single-center retrospective case–control study conducted at the pediatric cardiology clinic of Şırnak State Hospital, including infants evaluated between April 2022 and January 2025. The aim of the study was to evaluate the association between maternal biochemical parameters and the presence of VSD in infants younger than one year of age. The VSD group consisted of infants younger than one year of age with a confirmed diagnosis of isolated perimembranous VSD who were followed in the pediatric cardiology clinic. The diagnosis of VSD was confirmed by transthoracic echocardiography performed by a pediatric cardiologist. The control group consisted of infants younger than one year of age who were referred to the pediatric cardiology clinic for evaluation of a cardiac murmur but were subsequently confirmed to have no structural heart disease after clinical examination and echocardiographic assessment.

Infants with additional major congenital heart defects, major extracardiac anomalies, or insufficient maternal laboratory data were excluded from the study. A small number of infants in the VSD group had Down syndrome, reflecting the known association between trisomy 21 and septal defects.

Maternal laboratory parameters were retrieved retrospectively from the hospital's electronic medical record system. The earliest available maternal laboratory results were used for analysis. Because of the retrospective nature of the study, maternal laboratory measurements were not uniformly obtained during pregnancy. In most cases, the available laboratory measurements were obtained during the postpartum period, whereas first-trimester laboratory data were available for only two mothers. Among mothers with postpartum laboratory data, the median interval between delivery and maternal blood sampling was 123 days.

The evaluated maternal biochemical parameters included:

- Complete blood count
- Ferritin
- Vitamin B12
- Folic acid
- 25-hydroxyvitamin D

Maternal demographic and obstetric characteristics, including maternal age at delivery, gravidity, and interpregnancy interval, were obtained from obstetric outpatient clinic records. Information on parental consanguinity was available only for the VSD group and was recorded when accessible.

Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were assessed for normality using visual (histograms and probability plots) and analytical methods. Non-normally distributed variables were expressed as median with interquartile range (IQR). Categorical variables were summarized as frequencies and percentages. Comparisons between the VSD group and the control group were performed only for variables available in both groups, including maternal age at delivery, maternal hemoglobin level, ferritin level, vitamin B12 level, folate level, and vitamin D level. Since several biochemical parameters showed non-normal distribution, comparisons between groups were conducted using the Mann–Whitney U test for continuous variables and the chi-square test for categorical variables. Variables considered clinically relevant based on previous literature and available in both study groups, including maternal age at delivery, hemoglobin, ferritin, vitamin B12, and folate levels, were entered into a multivariable binary logistic regression model to evaluate their independent association with the presence of VSD. Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated. Receiver operating characteristic (ROC) curve analysis was performed to assess the discriminative ability of maternal ferritin levels for identifying VSD. The area under the curve (AUC) was calculated. The optimal cut-off value was determined using the Youden index, and corresponding sensitivity and specificity values were reported. A p-value <0.050 was considered statistically significant.

Results

A total of 79 participants were included in the study, consisting of 41 mothers in the VSD group and 38 mothers

Table I: Baseline maternal and infant characteristics

Variable	VSD	Control	p
Total number of patient	41	38	-
Maternal age at delivery (years)*	27.45 (23.67–33.33)	29.46 (23.70–34.67)	0.346
Maternal hemoglobin (g/dL)*	12.70 (11.90–13.30)	12.65 (11.50–13.38)	0.864
Maternal ferritin (ng/mL)*	25.80 (15.50–52.03)	57.85 (21.33–79.25)	0.012
Vitamin B12 (pg/mL)*	282.00 (236.00–426.00)	339.00 (287.00–398.25)	0.197
Folate (ng/mL)*	6.73 (4.10–10.11)	5.64 (4.79–11.00)	0.757
Vitamin D (ng/mL)*	11.05 (8.80–14.20)	13.30 (8.95–15.73)	0.407
Infant gender (Female/Male)	18 / 23	15 / 23	0.865†

**: median (IQR) (Mann–Whitney U test), †:chi-square test*

Table II: Logistic regression analysis for the presence of VSD

Variable	OR	95% CI	p
Maternal age at delivery	0.97	0.90–1.05	0.460
Maternal hemoglobin	1.15	0.80–1.66	0.459
Maternal ferritin (per 10-ng/mL increase)	0.83	0.72–0.96	0.012
Vitamin B12	0.999	0.996–1.003	0.731
Folate	1.02	0.92–1.13	0.662

Binary logistic regression analysis was performed to evaluate the association between maternal biochemical parameters and VSD. The Hosmer-Lemeshow goodness-of-fit test indicated adequate model fit (Chi-square = 9.204, df = 8, p = 0.325), and the Nagelkerke R square was 0.151.

in the control group. Among the infants in the VSD group, five patients had Down syndrome.

Baseline characteristics

Maternal age at delivery was comparable between the two groups (median 27.45 years [IQR 23.67–33.33] in the VSD group vs 29.46 years [IQR 23.70–34.67] in the control group, p=0.346). Similarly, maternal hemoglobin levels did not differ significantly between the groups (median 12.70 g/dL [IQR 11.90–13.30] vs 12.65 g/dL [IQR 11.50–13.38], p=0.864).

The distribution of infant gender was also similar between the groups (female/male: 18/23 vs 15/23, p=0.865). The baseline maternal and infant characteristics of the study population are summarized in Table I.

Maternal ferritin levels were significantly lower in the VSD group compared with the control group (median 25.80 ng/mL [IQR 15.50–52.03] vs 57.85 ng/mL [IQR 21.33–79.25], p=0.012). The distribution of maternal ferritin levels is illustrated in Figure 1.

No significant differences were observed between the groups in terms of vitamin B12 levels (median 282 pg/mL [IQR 236–426] vs 339 pg/mL [IQR 287–398], p=0.197), folate levels (median 6.73 ng/mL [IQR 4.10–10.11] vs 5.64 ng/mL [IQR 4.79–11.00], p=0.757), or vitamin D levels (median 11.05 ng/mL [IQR 8.80–14.20] vs 13.30 ng/mL [IQR 8.95–15.73], p=0.407).

Logistic regression analysis

Binary logistic regression was used to examine the relationship between maternal biochemical parameters and the occurrence of VSD, as presented in Table II.

The multivariable logistic regression model included maternal age at delivery, hemoglobin level, vitamin B12 level, folate level, and ferritin level. Maternal ferritin remained independently associated with VSD. Specifically, the odds of VSD decreased by 17% for every 10 ng/mL increase in ferritin (OR 0.83, 95% CI 0.72–0.96, p=0.012), while other maternal biochemical parameters were not independently associated

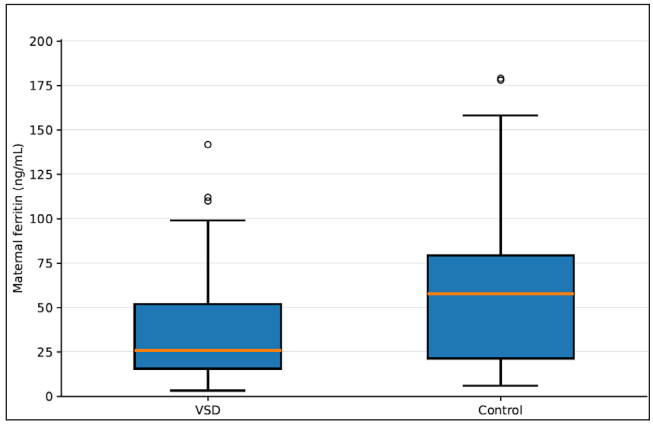

Figure 1: Distribution of maternal ferritin levels in mothers of children with ventricular septal defect (VSD) and controls. Maternal ferritin levels were significantly lower in mothers of children with VSD than in controls (median 25.8 ng/mL vs 57.9 ng/mL, p = 0.012).

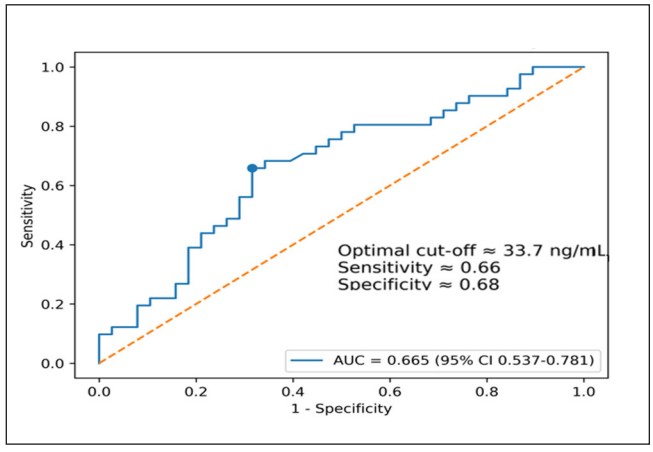

Figure 2: Receiver operating characteristic (ROC) curve of maternal ferritin levels for discrimination of ventricular septal defect (VSD). Maternal ferritin demonstrated moderate discriminative ability with an area under the curve (AUC) of 0.665 (95% CI 0.537–0.781). The optimal ferritin cut-off value determined using the Youden index was 33.7 ng/mL, yielding a sensitivity of 65.9% and specificity of 68.4%.

with VSD. A subgroup analysis was performed within the VSD cohort to evaluate whether maternal ferritin levels differed between infants requiring surgical intervention and those managed conservatively. Among the 41 patients with VSD, 13 required surgical repair. Maternal ferritin levels were numerically lower in the surgical group compared with the non-surgical group (median 19.7 ng/mL vs 27.3 ng/mL); however, this difference did not reach statistical significance (Mann–Whitney U test, p=0.230).

ROC curve analysis

ROC curve analysis was conducted to evaluate the capacity of maternal ferritin levels to differentiate VSD cases, as depicted in Figure 2. Maternal ferritin exhibited a moderate level of discriminative power, evidenced by an area under the curve (AUC) of 0.665 (95% CI 0.537–0.781). Using the Youden index, the best ferritin cut-off value was found to be 33.7 ng/mL. This value showed a sensitivity of 65.9% and a specificity of 68.4%.

Discussion

In this retrospective case–control investigation, mothers of infants diagnosed with VSD exhibited significantly reduced ferritin concentrations compared to mothers of infants without VSD. Nevertheless, hemoglobin levels and other micronutrient concentrations were comparable across both cohorts. Maternal ferritin concentrations, notably, remained independently associated with VSD, even when accounting for potential confounding factors. This observation suggests a potential association between diminished maternal iron reserves and VSD, rather than a direct causal relationship. These findings may suggest that ferritin could serve as a more sensitive marker of maternal iron status in this context. This distinction is clinically relevant because iron depletion may precede changes in hemoglobin concentration, particularly in pregnancy, where biochemical iron deficiency can exist despite apparently normal hemoglobin values (7,10).

While our study focused on isolated perimembranous VSD, whether maternal iron status is similarly associated with other VSD subtypes, such as muscular defects, remains to be determined and warrants further investigation. Receiver operating characteristic analysis demonstrated that maternal ferritin had a moderate discriminative ability for identifying VSD. However, its modest performance (AUC: 0.665) suggests that its clinical applicability as a standalone predictive biomarker may be limited and should be interpreted within a broader clinical context.

An additional consideration is the presence of Down syndrome in a subset of patients within the VSD group. Trisomy 21 is strongly associated with congenital heart defects, with approximately 40–60% of affected individuals having structural cardiac anomalies, and ventricular septal defect representing one of the most frequently observed lesions. This association has been consistently demonstrated both in large population-based cohorts and in regional studies, including data from Türkiye, where congenital heart defects are reported in nearly half of individuals with Down syndrome (16,17). Although the number of such patients in our cohort was limited, their inclusion may represent a potential source of confounding and should be considered when interpreting the observed association between maternal ferritin levels and VSD. Therefore, our findings should be interpreted with caution, and future studies incorporating detailed genetic or chromosomal stratification are warranted to better distinguish the relative contributions of maternal and genetic factors.

Experimental and clinical data together suggest that maternal iron deficiency may disrupt embryonic heart development through converging metabolic and signaling pathways. In experimental models, iron is required as a cofactor for CYP26 enzymes that regulate retinoic acid (RA) metabolism; iron deficiency may reduce CYP26 activity, leading to excess RA signaling and altered differentiation and migration of second heart field progenitors. These changes have been associated with structural heart defects, including VSD. Maternal iron deficiency may also contribute to embryonic hypoxia and altered vascular development. These mechanisms provide biologically plausible pathways that may help explain the observed association, although they do not establish causality (1,14,18).

An important consideration when interpreting our results is the timing of maternal biochemical assessment. In our cohort, most iron parameters were measured in the postpartum period and therefore cannot be assumed to capture iron status during the critical window of early cardiac morphogenesis. While iron deficiency in pregnancy is often part of a broader, chronic imbalance between increased physiological demand and insufficient intake or absorption, postpartum ferritin levels cannot be considered a precise proxy for early gestational iron status (19). In addition, postpartum ferritin levels may be influenced by delivery-related factors such as mode of delivery and peripartum blood loss, which were not systematically accounted for in this study. Furthermore, ferritin is an acute-phase reactant and may be affected by inflammatory processes, and pregnancy-specific diagnostic thresholds remain an area of ongoing refinement (10,19,20). Clinical guidelines further emphasize that a low serum ferritin is diagnostic of iron deficiency in pregnancy, and that ferritin levels <30 ng/mL are indicative of iron deficiency, although higher levels do not fully exclude iron depletion (21). Taken together, these factors warrant cautious interpretation of postpartum ferritin levels. Nevertheless, lower ferritin concentrations observed in mothers of infants with VSD may still reflect an underlying, longer-standing maternal iron vulnerability.

In line with this interpretation, population-based data have shown that maternal anemia in the first 100 days of pregnancy is associated with a higher odds of congenital heart defects in offspring, which may also be relevant to VSD (13). Complementary evidence from the Generation R cohort indicates that dysregulated maternal iron status in early pregnancy is associated with persistent alterations in cardiac structure and function in childhood, with higher maternal haemoglobin relating to smaller ventricular end-diastolic volumes in boys and maternal anemia predicting larger LV volumes and reduced systolic function, while higher ferritin in girls was associated with increased left ventricular mass (22). A recent systematic review of observational studies similarly concluded that maternal micronutrient deficiency may be relevant to fetal CHD risk, including VSD, although the available evidence remains heterogeneous and insufficient

for nutrient-specific causal inference (5). Together, these findings may indicate that both iron deficiency and iron overload could be associated with long-term, gender-specific differences in cardiovascular development. These observations are broadly in line with our findings, although causal relationships cannot be inferred. The regional context may also be relevant for interpreting our findings. Şırnak is in the Southeastern Anatolia region, which has some of the highest fertility rates in Türkiye but also some of the worst socioeconomic indicators (15). Repeated pregnancies, limited dietary diversity, and restricted access to nutritional resources may increase the risk of maternal micronutrient deficiencies, including iron deficiency. In daily pediatric cardiology practice in this region, children with CHD are frequently observed to come from families facing nutritional challenges. Although our study was not designed to directly evaluate socioeconomic determinants, our findings raise the possibility that maternal nutritional status may represent an important and potentially modifiable factor in regions with similar demographic and social characteristics.

Limitations

One important limitation of this study is the timing of maternal biochemical measurements. Maternal ferritin levels were not uniformly obtained during pregnancy, and most measurements were obtained during the postpartum period. Therefore, these values cannot be considered a direct representation of maternal iron status during early cardiac morphogenesis. In addition, the relatively small sample size and single-center design may limit the generalizability of the findings. Residual confounding factors, including dietary habits, iron supplementation during pregnancy, and broader socioeconomic variables, could not be fully accounted for. Given the retrospective design, limited sample size, and timing of measurements, these findings should be considered hypothesis-generating rather than confirmatory. Consequently, our results suggest a possible association between diminished maternal ferritin concentrations and VSD, rather than a causal relationship. Further prospective studies with measurements obtained during early pregnancy are needed to better clarify the timing and nature of this association, as well as to determine whether optimization of maternal iron status may contribute to the prevention of VSD.

Conclusion

In conclusion, lower maternal ferritin levels were associated with the presence of VSD in offspring, independent of hemoglobin levels. These findings suggest that maternal iron stores, rather than overt anemia, may be relevant for fetal cardiac development. Further prospective studies are needed to clarify causality and to determine whether optimization of maternal iron status may contribute to the prevention of congenital heart defects.

Ethics committee approval

This study was conducted in accordance with the Helsinki Declaration Principles. The study was approved by Sirnak University (07.12.2023, reference number: 83711).

Contribution of the authors

Study conception and design: MMA, VT; data collection: MMA, VÇ, ED; analysis and interpretation of results: MMA, VÇ, VT; draft manuscript preparation: MMA. All authors reviewed the results and approved the final version of the manuscript.

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Conflict of interest

The authors declare that there is no conflict of interest.

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