The Incidence and Pattern of Congenital Heart Disease Among Infants of Diabetic Mothers

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Abstract
Background: One of the most potent and consistently suggested risk factors for congenital heart disease (CHD) in the newborn is maternal diabetes. Diabetes is prevalent and increasing in Iraq, and babies born to diabetic mothers (IDM) are a large, identifiable, and screenable high-risk group.
Objectives: To estimate the rate of CHD in infants of diabetic women as compared to infants of non-diabetic women, describe the pattern of defects, and identify maternal risk factors.
Methods: The prospective hospital-based cohort with a non-diabetic control group was conducted in a maternity and children's hospital in Najaf. Within their first week of life, 300 neonates of diabetic mothers underwent transthoracic echocardiography, as did 300 neonates of non-diabetic mothers. The risk ratios and multivariable logistic regression were used.
Results: CHD was found in 11.7% of IDMs versus 2.7% of controls (relative risk ≈ 4.4; 95% CI 2.1–9.3; $p < 0.001$). Ventricular septal defect (31.4%), atrial septal defect (22.9%), patent ductus arteriosus (20.0%), and hypertrophic cardiomyopathy (14.3%) were the most common defects observed in IDMs. For diabetic women and pregestational diabetes (failure to achieve good glycemic control in the first trimester (HbA1c >7%) was found to be an independent risk factor for CHD.
Conclusion: Diabetic mothers' infants have approximately four times the risk of CHD, the majority of which has been attributed to pregestational diabetes and poor periconceptional glucose control. This report suggests the need for preconception counseling, strict glycemic regulation in the first trimester, and the routine fetal/neonatal echocardiographic screening of diabetic pregnancies in Iraq.

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1. Introduction

In the general population, CHD occurs at approximately 7–8 per 1000 live births [1]. Infants of diabetic mothers have a classic risk of about 5%, but it is reported anywhere from 2.5% to 12% depending on the type and control of the diabetes and method of screening [2].

The diabetes–CHD connection is international and big in scope: The prevalence of CHD was 80 per 10,000 births in the non-diabetic group, while CHD prevalence was 318 per 10,000 births in the group with pregestational diabetes, with a relative risk

of 4.0; approximately three-quarters of CHD cases in the pregestational diabetes group were attributed to the diabetes itself [3].

In the USA, an analysis of 22.6 million live births revealed that pregestational and gestational diabetes were associated with a 4.33x and 1.47x increase in risk for critical CHD, respectively [4].

A French nationwide study reported CHD in 0.8% of non-diabetic pregnancies, compared with ~3.0% (type 1) and ~2.7% (type 2) [5], and the Japan Environment and Children's Study reported an overall odds ratio of 1.81, which was higher for pre-pregnancy diabetes [6].

The incidence of IDM CHD on neonatal echocardiography is around 11.8%, with patent ductus arteriosus, patent foramen ovale, and atrial/ventricular septal defects being most common in regional cohorts within the Gulf [7, 8].

The biology is consistent with these numbers: Maternal hyperglycemia during the first-trimester window of cardiac organogenesis disrupts fetal cardiac development; hence, uncontrolled hyperglycemia from the onset of pregnancy (pregestational diabetes) is much more harmful than hyperglycemia that develops later in pregnancy (gestational diabetes) [9].

What significance does this have for Iraq? The burden of diabetes is very high and increasing in Iraq, and the proportion of pregnancies at risk is high. But IDMs are a recognized population, and the main mechanisms – glucose control before conception and targeted echocardiographic screening – are achievable. Specific data on diabetic pregnancies in Iraq and pairing with systematic neonatal echocardiography is still limited, thus filling in some gaps.

2. Objectives

1. Estimate CHD incidence in IDMs compared to the controls.
2. Outline the range/pattern of the subtypes of CHD.
3. Recognize risk factors in the mother for CHD (type of diabetes, glycemic control, obesity).

Interpret the results in terms of screening and prevention recommendations.

3. Methods

Design: Prospective cohort with a concurrent non-diabetic control group in a hospital setting.

Setting and period: Over a 12-month period (January to December 2025) at a maternity and children's hospital in Najaf, you collected the following data.

Participants

1. **Exposed group:** 300 neonates with mothers who have diabetes (pregestational type 1/type 2 or gestational).
2. **Control Group:** 300 neonates of non-diabetic mothers, Frequency Matched for Maternal age.
3. **Exclusions:** recognized teratogen exposure, maternal age <18 or >40, multiple congenital-anomaly syndromes, known chromosomal/genetic syndrome.
4. **Sample size:** To detect a difference between an expected incidence rate of 12% for IDMs and 3% for controls with a power of 80% with $\alpha = 0.05$, ~160 per group were recruited, with 300 per group being recruited to allow for subgroup analysis.
5. **Assessment:** Early transthoracic echocardiogram (within 1st 7 days of life) performed by a pediatric cardiologist in all newborns.
6. **Maternal information:** Type of diabetes, periconceptional/first trimester HbA1c, BMI, parity.
7. **Neonatal data:** Sex, birth weight, gestational age, NICU admission.
8. **Definitions:** Echocardiographically normal heart vs structural cardiac defect. Expected to close PFOs (small isolated PFOs) were recorded as transitional findings and not as CHD. High blood sugar = HbA1c > 7% in the first trimester of pregnancy.
9. **Statistics:** Incidence and relative risk (95% CI); categorical comparison (chi-square); independent predictors (multivariable logistic regression). Significance at $p < 0.05$.
10. **Ethics:** IRB approval from Al Zahraa teaching hospital for maternity and child health; written informed parental consent.

4. Results

4.1. Sample characteristics

Diabetic group (n = 300): gestational diabetes 195 (65%), pregestational 105 (35%). Mean maternal age: 30.6 ± 5.1 years. Control group (n = 300) comparable for age.

4.2. Primary outcome — CHD incidence

Table 1

Group	n	CHD cases	Incidence	Relative risk (95% CI)	p
Infants of diabetic mothers	300	35	11.7%	4.4 (2.1–9.3)	<0.001
Infants of non-diabetic mothers	300	8	2.7%	Reference	

4.3. The pattern of CHD in infants of diabetic mothers (n = 35)

Table 2

Defect	n	% of CHD cases
Ventricular septal defect (VSD)	11	31.4
Atrial septal defect (ASD)	8	22.9
Patent ductus arteriosus (PDA, persistent)	7	20.0
Hypertrophic cardiomyopathy	5	14.3
Transposition of the great arteries (TGA)	2	5.7
Tetralogy of Fallot (TOF)	2	5.7

The septal defects were the most common, followed by hypertrophic cardiomyopathy — a defect that is characteristically overrepresented in IDMs — which occurred in roughly one in seven cases.

4.4. Maternal risk factors

Within the diabetic group:

Table 3

Subgroup	CHD incidence
Pregestational diabetes	20.0% (21/105)
Gestational diabetes	7.2% (14/195)
First-trimester HbA1c > 7%	22.0%
First-trimester HbA1c ≤ 7%	6.0%

According to the multivariable logistic regression analysis, the independent risk factors for CHD:

- Maternal diabetes (gestational or chronic) — the adjusted OR is 4.1 (95% CI 1.9–8.8), $p < 0.001$
- Pregestational vs gestational diabetes — the adjusted OR is 2.6 (1.2–5.6), $p = 0.012$
- Poor first-trimester glycemic control (indicated by HbA1c >7%) — the adjusted OR is 3.8 (1.7–8.4), $p = 0.001$
- Maternal obesity (indicated by BMI ≥30) — the adjusted OR is 1.9 (1.0–3.6), $p = 0.047$

4.5. Neonatal outcomes

IDMs had higher rates of macrosomia (18% vs 6%) and NICU admission (24% vs 9%) than controls (both $p < 0.001$).

5. Discussion

The excess modelled at ~4-fold and the IDM incidence at ~12% fall squarely within the published range and are similar to the US (4.33-fold) and Danish (4.0-fold) cohorts, as well as the ~11.8% figure for Gulf neonates [3, 4, 7]. The IDM signature is also expected to be present with septal defects first, with a significant proportion of hypertrophic cardiomyopathy [7, 8].

The two that convey the practical message are the diabetes-type gradient and glycemic-control gradient. The risk of CHD from PGDM was about three times as high as that from GDM, while the lack of optimum control during the first trimester of pregnancy conferred independent risk. Both refer to the same fact: the damage occurs during organogenesis, in early pregnancy, sometime before the woman realizes she is expecting [9]. That's a new mindset from a neonatal problem to a preconception problem.

This provides a clear and local action plan:

1. **Prevention before conception:** Identify and tightly control diabetes in women of childbearing age before conception to near normal HbA1c.
2. **Detection in pregnancy:** Fetal echocardiography for diabetic pregnancies particularly those considered as pregestational or poorly-controlled.
3. **Post natal:** All IDM neonates should have neonatal echocardiographic screening as several defects (and hypertrophic cardiomyopathy) are clinically silent early.

No infrastructure outside of the already existing antenatal/neonatal services is required to implement any of these, making them achievable in Iraqi maternity hospitals.

6. Limitations

Referral patterns could skew towards higher-risk diabetic pregnancies and overestimate incidence (single center/hospital-based). The boundary between "defect" and "transitional finding" (small PFO/PDA) in the first week before they may close is a factor in the count, and these reports are separated out here. Some mothers did not have HbA1c results in their first trimester. No long-term follow up, therefore natural history and clinical significance of minor lesions is not known. Lengthier follow-up and a multicenter design would boost estimates.

7. Conclusion and Recommendations

CHD was observed in about 4 times more infants of diabetic women compared to infants of nondiabetic women; these infants were mostly pregestational diabetic and those with poor glucose control during the first trimester of pregnancy; septal defects and hypertrophic cardiomyopathy were the most common types of CHD.

We recommend:

1. Optimizing glycemic control for all women of reproductive potential: the single most effective prevention intervention.
2. Routine fetal echocardiography of diabetic pregnancies, especially pregestational and poorly controlled pregnancies.
3. Screening of all IDM with an echocardiogram at birth prior to discharge.
4. Incorporate these steps into the Iraqi national maternal child health policies, and validate with a multicenter prospective study with follow-up.

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