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Pregnancy Outcomes in Women With Congenital Heart Disease and Right Ventricular Dysfunction: An Underrecognized Cardio-Obstetric Risk

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Abstract

Background

The number of reproductive-age women living with congenital heart disease (CHD) is increasing. Although many right-sided congenital lesions have traditionally been considered compatible with pregnancy, the prognostic impact of right ventricular (RV) dysfunction on maternal and fetal outcomes remains incompletely characterized. Recent ESC 2025 guidelines for cardiovascular disease in pregnancy specifically recognize significantly impaired subpulmonary RV function as Intermediate risk of maternal mortality or moderate-to-severe increase in morbidity, requiring individualized assessment. In this context, the present study aimed to evaluate maternal cardiovascular, obstetric, and perinatal outcomes in pregnant women with CHD involving RV dysfunction.

Methods

We conducted a cross-sectional study at a tertiary referral cardio-obstetric clinic in Isfahan, Iran. Forty-two pregnant women with CHD-associated RV systolic dysfunction were enrolled and followed by a multidisciplinary pregnancy heart team. Clinical status, modified World Health Organization (mWHO) risk class, New York Heart Association (NYHA) functional class, echocardiographic indices of RV function, and maternal and fetal outcomes were recorded. Outcomes were compared according to RV dysfunction severity.

Results

The most common CHD lesion was atrial septal defect (ASD) in 21 patients (50%), followed by tetralogy of Fallot (TOF) in 9 patients (21.4%), Ebstein anomaly in 7 patients (16.6%), pulmonary stenosis in 4 patients (9.5%), and Fontan circulation in 1 patient (2%). RV dysfunction was mild in 22 patients (52.4%), moderate in 15 patients (35.7%), and severe in 5 patients (11.9%). Moderate-to-severe RV dysfunction was associated with a higher frequency of heart failure symptoms during pregnancy (45.0% vs. 18.2%; OR 3.6, 95% CI 0.9–14.2; P = 0.06). Pregnancy-induced hypertension or preeclampsia occurred in 7 patients (17.5%), preterm birth in 14 patients (35%), small-for-gestational-age infants in 11 patients (27.5%), and fetal/neonatal mortality in 2 patients (2.4%).

Conclusions

Pregnancy in women with CHD involving RV dysfunction is associated with clinically important maternal and fetal complications, even when left ventricular systolic function is preserved. Severe RV dysfunction appears to identify a subgroup at increased risk for heart failure and adverse perinatal outcomes. These

findings support structured multidisciplinary cardio-obstetric care, serial echocardiographic surveillance, and individualized risk assessment consistent with contemporary ESC pregnancy guidance.

Introduction

Advances in pediatric cardiac surgery, interventional cardiology, and lifelong congenital heart disease care have increased survival among patients with CHD, creating a growing population of women with repaired or unrepaired congenital lesions who reach reproductive age [1–3]. Pregnancy is often possible in this population, but it imposes major circulatory demands and may unmask limited cardiovascular reserve, especially in women with ventricular dysfunction, residual valvular lesions, prior surgical repair, cyanosis, arrhythmia, or pulmonary vascular disease [1, 4, 15].

Normal pregnancy is associated with systemic vasodilation, plasma volume expansion, increased heart rate, and a substantial rise in cardiac output beginning early in gestation. These changes are accompanied by enlargement of all four cardiac chambers and increased susceptibility to premature beats and re-entrant supraventricular tachycardia [1, 4–6]. In women with structural heart disease, the ability to adapt to these hemodynamic changes is determined not only by left ventricular performance but also by right ventricular size, systolic function, loading conditions, and the presence of tricuspid or pulmonary valve dysfunction.

Compared with left ventricular dysfunction, RV dysfunction in pregnancy has been less extensively studied. This is clinically important because several common congenital lesions primarily affect RV loading and remodeling. These include ASD with chronic right-sided volume overload, repaired TOF complicated by pulmonary regurgitation and RV dilation, pulmonary stenosis or insufficiency, Ebstein anomaly with tricuspid regurgitation and RV myopathy, and Fontan circulation [11–13]. RV dysfunction may remain subclinical before pregnancy but become clinically relevant when the volume load of pregnancy increases.

Maternal cardiovascular function may also influence placental development. Previous studies have shown that maternal RV function can be associated with uteroplacental artery Doppler patterns in the first trimester and pregnancy outcome in women with CHD [7]. Impaired maternal cardiac output, venous congestion, and abnormal uteroplacental perfusion may negatively affect trophoblastic invasion and spiral artery remodeling, thereby contributing to placental insufficiency, preeclampsia, fetal growth restriction, and preterm birth [7–9, 17, 18].

Risk stratification in pregnant women with cardiovascular disease has traditionally relied on the modified World Health Organization (mWHO) classification [10, 15]. The 2025 ESC Guidelines for the management of cardiovascular disease and pregnancy further emphasize individualized assessment, the Pregnancy Heart Team, the expanded mWHO 2.0 framework, shared decision-making, pre-pregnancy evaluation, and lesion-specific monitoring throughout pregnancy and the postpartum period [32]. These concepts are particularly relevant for women with CHD and RV dysfunction, in whom conventional risk categories may not fully capture functional reserve, lesion-specific physiology, and placental consequences.

Given the scarcity of focused data on RV dysfunction in pregnancy, we evaluated maternal and perinatal outcomes in 42 pregnant women with CHD-associated RV dysfunction followed at a tertiary cardio-obstetric clinic. We hypothesized that RV dysfunction, particularly when severe, is associated with higher rates of maternal cardiovascular complications and placental-mediated fetal outcomes despite preserved left ventricular systolic function in many patients.

Methods

Study design and setting

This cross-sectional study was conducted at the Cardio-Obstetric Clinic of Alzahra Hospital, Isfahan, Iran, a tertiary referral center for pregnant women with cardiovascular disease. Pregnant women with CHD involving RV dysfunction were evaluated between 2022 and 2024. The clinic receives referrals from governmental and private obstetric centers across Isfahan province according to the regional healthcare policy of Isfahan University of Medical Sciences.

Multidisciplinary assessment

Initial evaluation and follow-up were performed by a multidisciplinary pregnancy heart team, including a cardiologist experienced in cardiovascular disease during pregnancy, an echocardiography specialist, and an obstetrician-gynecologist experienced in high-risk pregnancy. This structure is aligned with contemporary ESC guidance emphasizing specialized Pregnancy Heart Team involvement for women with moderate-to-high cardiovascular risk [32].

Eligibility criteria

Eligible diagnoses included Ebstein anomaly, severe pulmonary stenosis or pulmonary insufficiency, ASD without prior repair and RV dysfunction, repaired TOF with clinically relevant RV dysfunction, and Fontan circulation.

Patients with moderate-to-severe pulmonary hypertension, defined as estimated systolic pulmonary artery pressure > 50 mmHg, were excluded to reduce heterogeneity because of the distinct hemodynamic profile, right ventricular remodeling, and well-established high-risk pregnancy outcomes associated with pulmonary hypertension.

Data collection and definitions

NYHA functional class presented in Table 1 was based on the worst documented functional status during pregnancy follow-up rather than longitudinal change in NYHA class.

Demographic data, gravidity, parity, maternal age, BMI, CHD diagnosis, mWHO class, NYHA functional class, history of valve replacement, and echocardiographic findings were collected. RV systolic function was assessed using standard echocardiographic indices, including tricuspid annular plane systolic excursion (TAPSE), right ventricular fractional area change (FAC), and tricuspid annular S' velocity by tissue Doppler imaging, and 3D RVEF, consistent with published chamber quantification recommendations [14].

Maternal cardiovascular events included heart failure symptoms, arrhythmia, and thromboembolic events. Obstetric outcomes included pregnancy-induced hypertension or preeclampsia, gestational diabetes mellitus, PROM/PPROM, and mode of delivery. Perinatal outcomes included preterm birth before 37 weeks, small-for-gestational-age infant, neonatal respiratory distress syndrome, and fetal or neonatal death. Patients were followed monthly or bimonthly depending on maternal cardiovascular risk, symptoms, and fetal status. Hospitalization was recommended for worsening heart failure, arrhythmia, or fetal compromise.

Ethical considerations

The study protocol was approved by the Ethics Committee of Isfahan University of Medical Sciences, Isfahan, Iran (IR.MUI.MED.REC.1401.370). Written informed consent was obtained from all participants.

Statistical analysis

Data were analyzed using SPSS software version 26. Continuous variables are reported as mean +/- standard deviation when normally distributed or median with interquartile range when appropriate. Categorical variables are presented as number and percentage. Comparisons between mild/moderate RV dysfunction and severe RV dysfunction groups were performed using the chi-square test or Fisher exact test for categorical variables and the independent t-test or Mann-Whitney U test for continuous variables, as appropriate. A p-value < 0.05 was considered statistically significant. Results

A total of 42 pregnant women met the eligibility criteria. The mean maternal age was 29.81 +/- 6.29 years and 12 patients (28.5%) were primigravida. The baseline characteristics and echocardiographic findings are summarized in Table 1.

Table 1
Baseline demographic, clinical, and echocardiographic characteristics of the study population

Variable	Category	n (%) or mean +/- SD	Notes
Age, years	Mean +/- SD	29.81 +/- 6.29	
BMI	Mean	25.84	
Gestational age at delivery, weeks	Mean +/- SD	36.1 +/- 1.0	
Parity	0	12 (28.5%)	Primigravida
	1	16 (38.0%)	
	>=2	14 (33.5%)	
CHD lesion	Ebstein anomaly	7 (16.6%)	
	Severe Pulmonary stenosis	4 (9.5%)	
	Tetralogy of Fallot	9 (21.4%)	All repaired in childhood
	ASD - unrepaired	21 (50%)	
	Fontan circulation	1 (2.3%)	
NYHA class	I	19 (45.2%)	
	II	17 (40.5%)	
	III	6 (14.3%)	
mWHO class	I	0 (0%)	
	II	20 (47.6%)	
	II- III	15 (35.7%)	
	III	4 (9.5%)	
	IV	3 (7.1%)	Termination recommended but declined
RV dysfunction severity	Mild	22 (52.4%)	
	Moderate	15 (35.7%)	
	Severe	5 (11.9%)	
Significant Valvular findings	Severe TR	7 (16.6%)	
	Severe PS	3 (7.1%)	

Variable	Category	n (%) or mean +/- SD	Notes
	Severe PI	3 (7.1%)	
Valve replacement	Biologic PVR	3 (7.1%)	
	Mechanical TVR	1 (2.3%)	
	Biologic TVR	1 (2.3%)	
LVEF, %	Mean +/- SD	52.4 +/- 10.23	
PAP, mmHg	Mean	27	
TAPSE, mm	Mean	16.3	
RV FAC, %	Mean	32	
S' velocity, cm/s	Mean	9	
3D RVEF	Mean	41 +/-7.6%	

The most common lesion was unrepaired ASD, observed in 21 patients (50%), TOF was present in 9 patients (21.4%), all with previous total correction surgery and residual pulmonary valve dysfunction or pulmonary valve replacement with RV impairment. Pulmonary stenosis was present in 4 patients (9.5%), including one case of severe valvular pulmonary stenosis who remained clinically stable in early pregnancy but developed exertional cyanosis during exercise testing, indicating limited cardiopulmonary reserve and one case of severe peripheral pulmonary stenosis. Ebstein anomaly was present in 7 patients (16.6%). One patient had tricuspid atresia with Fontan circulation and LVEF of 42%, categorized as mWHO class III.

According to mWHO classification, 20 (47.6%) class II, 15 (35.7%)class II- III,4 (9.5%) class III, and 3 (7.1%) class IV or considered extremely high-risk by the local heart team.

Pregnancy termination was recommended in three patients because of severe RV-related risk. One case involved a patient with severe valvular pulmonary stenosis who remained clinically stable in early pregnancy but developed exertional cyanosis during exercise testing, indicating limited cardiopulmonary reserve. This patient accepted therapeutic pregnancy termination. Another case involved a patient with tetralogy of Fallot associated with severe pulmonary insufficiency, significant right ventricular dilation, and impaired biventricular function. Given the high-risk clinical profile, early pregnancy termination and pulmonary valve replacement were recommended; however, the patient chose to continue the pregnancy under close follow-up in the cardio-obstetric clinic. A third patient had tricuspid atresia with Fontan circulation and an LVEF of 42%. Pregnancy termination was also recommended, but the patient opted to continue the pregnancy under multidisciplinary surveillance in the cardio-obstetric clinic.

Maternal cardiovascular, obstetric, and perinatal outcomes are shown in Table 2. Heart failure symptoms were more frequent among patients with moderate-to-severe RV dysfunction (45.0% vs. 18.2%), demonstrating a strong trend toward significance (P = 0.06). Arrhythmia occurred in 3 patients (7.1%), and thromboembolic events occurred in 1 patient (2.38%).

Table 2
Maternal, obstetric, and perinatal outcomes according to RV dysfunction severity

Outcome	**Total cohort n (%)**	**Mild RV dysfunction n = 22 (52.3%)**	**Moderate /Severe RV dysfunction n = 20 (47.6%)**	**P-value**	**OR(95%CI)**
Primary composite maternal cardiac outcome	**16 (38.1%)**	5 (22.7%)	11 (55%)	0.03	4.2(1.1–15.6)
Heart failure symptoms	13 (30.9%)	4 (18.2%)	9 (45%)	0.06	3.6(0.9–14.2)
Arrhythmia	3 (7.1%)	1 (4.5%)	2 (10%)	0.59	2.3(0.2–26.1)
Thromboembolic event	1 (2.38%)	0	1 (5%)	0.99	1.1(0.06–20.3)
Pregnancy-induced hypertension/preeclampsia	7 (17.5%),n = 40	2 (9.1%)	5 (27.8%)	0.24	3.3(0.6–17.2)
Gestational diabetes mellitus	4 (9.7%)	1 (4.5%)	3 (15%)	0.33	3.7(0.3–38.8)
PROM/PPROM	4 (10%), n = 40	1 (4.5%)	3 (15%)	0.33	3.7(0.3–38.8)
Normal vaginal delivery	26 (65%),n = 40	17 (77.3%)	9 (50%)	0.08	0.29 (0.07–1.16)
Cesarean section	14 (73.8%),n = 40	5 (22.7%)	9 (50%)	0.08	3.4 (0.86–13.2)
Small-for-gestational-age infant	11 (27.5%),n = 40	3 (13.6%)	8 (44.4%)	0.04	5.1(1.1–22.8)
Preterm birth	14 (35%),n = 40	4 (18.2%)	10 (55.6%)	0.02	5.6(1.3–24.1)
Neonatal respiratory distress syndrome	4(10%) ,n = 40	1 (4.5%)	3 (15%)	0.33	3.7(0.3–38.8)
Fetal/neonatal mortality	2 (4.8%)	1(2.4%)	1(2.4%)	1	1(0.07–14.2)

Maternal cardiac outcomes (heart failure symptoms, arrhythmia, thromboembolic events, gestational diabetes mellitus, and fetal/neonatal mortality) were evaluated in the total study population (n = 42). Obstetric and neonatal outcomes requiring continuation of pregnancy beyond 20 weeks of gestation,

including pregnancy-induced hypertension/preeclampsia, PROM/PPROM, mode of delivery, preterm birth, and small-for-gestational-age infants, were analyzed among 40 pregnancies after exclusion of two pregnancies ending before 20 weeks.

Composite maternal cardiac outcome included heart failure symptoms requiring treatment or hospitalization, clinically significant arrhythmia requiring therapy, thromboembolic events, and maternal cardiac death. † Fisher's exact test was used due to small cell counts and zero-event distribution; odds ratios were calculated using Haldane–Anscombe correction where appropriate.

Hypertensive disorders of pregnancy were analyzed among pregnancies progressing beyond 20 weeks of gestation.

One elective termination was excluded from the final analysis population (n = 41). Due to the very small number of events, no statistical comparison between RV dysfunction groups was performed for fetal outcomes.

Obstetric complications included pregnancy-induced hypertension/preeclampsia occurred in 7 pregnancies (17.5%) and was more frequent among patients with moderate-to-severe RV dysfunction compared with those with mild dysfunction (27.8% vs. 9.1%; OR 3.3, 95% CI 0.6–17.2; P = 0.24), although the difference did not reach statistical significance. GDM in 4 patients (9.7%) [26–28], and PROM/PPROM in 4 patients (10%).

Fetal outcomes were assessed in 41 pregnancies (after excluding one elective termination). There were two cases of fetal demise (4.8%): one at 13 weeks of gestation and one intrauterine fetal death at 27 weeks. All patients were managed within a multidisciplinary cardio-obstetrics program at a tertiary referral center. In these cases, adverse fetal outcomes appeared to be mainly related to progression of maternal hemodynamic burden during pregnancy rather than severe RV failure itself.

Among these, one case occurred in the mild RV dysfunction group and one in the moderate RV dysfunction group. Given the small number of events, no meaningful statistical comparison between groups was performed.

The first case involved a patient with sinus venosus atrial septal defect associated with moderate RV dysfunction and right ventricular volume overload, who developed intrauterine fetal demise at 27 weeks of gestation without preceding obstetric complications. The clinical course was suggestive of progressive maternal–fetal hemodynamic imbalance in the setting of unrepaired congenital shunt physiology.

The second case involved a patient with peripheral pulmonary stenosis confirmed by CT angiography, preserved right ventricular systolic function, and moderate right ventricular hypertrophy. Fetal loss occurred at 13 weeks of gestation.

Another notable maternal complication occurred in a 27-year-old patient with unrepaired Ebstein anomaly. Pre-pregnancy transthoracic echocardiography demonstrated severe tricuspid regurgitation, a large patent foramen ovale with interatrial shunting, and moderate right ventricular systolic dysfunction. Exercise stress testing before pregnancy showed a functional capacity of 6 METs without exercise-induced oxygen desaturation. Following multidisciplinary evaluation, pregnancy was allowed with close follow-up. The patient remained clinically stable until 26 weeks of gestation, when she developed sudden unilateral visual loss. At hospital admission, arterial oxygen saturation was 85% on room air. The presentation was considered highly suggestive of a paradoxical embolic event due to worsening right-to-left shunting during pregnancy. Therapeutic anticoagulation with enoxaparin was initiated, and the pregnancy was subsequently continued under close multidisciplinary surveillance.

A further high-risk pregnancy involved a 25-year-old patient with tetralogy of Fallot associated with severe pulmonary insufficiency, marked right ventricular dilation, and impaired right ventricular systolic function. Because of the high-risk clinical profile, early pregnancy termination and pulmonary valve replacement were recommended; however, the patient declined intervention and continued the pregnancy under close cardio-obstetric follow-up. The pregnancy was subsequently complicated by preterm premature rupture of membranes at 33 weeks of gestation, resulting in preterm delivery.

Mode-of-delivery analysis was performed in 40 pregnancies after exclusion of one therapeutic abortion and one first-trimester spontaneous miscarriage. Overall, vaginal delivery was achieved in 26 pregnancies (65%), while cesarean section was performed in 14 cases (35%).

Patients with mild RV dysfunction underwent vaginal delivery more frequently compared with those with moderate-to-severe RV dysfunction (77.3% vs. 50.0%, respectively). In contrast, cesarean delivery was more common among patients with moderate-to-severe RV dysfunction (50.0% vs. 22.7%). Although this trend did not reach statistical significance, likely due to the limited sample size, it demonstrated a clinically relevant association between worsening RV function and increased likelihood of operative delivery.

The overall predominance of vaginal delivery in the cohort was consistent with the high proportion of patients with atrial septal defect physiology and preserved functional status, in whom vaginal delivery was considered feasible following multidisciplinary evaluation.

Discussion

This study evaluated pregnancy outcomes in women with CHD-associated RV dysfunction and demonstrates that RV impairment is clinically important in cardio-obstetric risk assessment. The principal findings are: first, heart failure symptoms were common and significantly associated with severe RV dysfunction; second, placental-mediated and perinatal complications, including preeclampsia, preterm birth, SGA, and fetal/neonatal mortality, occurred at clinically meaningful rates; and third, adverse outcomes were observed even though many patients were categorized as mWHO II and most had preserved or only mildly impaired right ventricular systolic function.

The high rate of heart failure symptoms in this cohort is notable. Pregnancy requires a major increase in preload and cardiac output, and a chronically dilated or pressure-loaded RV may be unable to accommodate these demands. This may be particularly relevant in repaired TOF with pulmonary regurgitation, Ebstein anomaly with severe tricuspid regurgitation, pulmonary stenosis or insufficiency, and RV volume overload in ASD [11–13]. Although statistical significance was not reached, worsening RV dysfunction was associated with a clinically meaningful increase in heart failure symptoms, likely reflecting limited right-sided hemodynamic reserve during pregnancy. Our findings support the concept that RV functional reserve, rather than LVEF alone, should be incorporated into pregnancy counseling and surveillance. Cardiorenal interactions may also contribute to clinical deterioration in advanced heart failure physiology during pregnancy [16].

Our results are consistent with previous studies indicating that women with CHD are at increased risk of cardiovascular events during pregnancy, although many prior reports have focused on overall CHD populations rather than RV dysfunction specifically [1–3, 15]. The 2025 ESC Guidelines highlight adult congenital heart disease as one of the most frequent cardiovascular disease categories encountered during pregnancy and recommend pre-pregnancy evaluation including ECG, transthoracic echocardiography, functional assessment when indicated, optimization of residual lesions, and Pregnancy Heart Team management for women at mWHO 2.0 class II-III or higher [32]. In the present cohort, three women were considered extremely high risk, and termination was recommended by the local heart team; however, two patients elected to continue pregnancy. This underscores the importance of shared decision-making, transparent risk communication, and respect for patient autonomy, which are emphasized in the updated ESC framework [32].

A stepwise increase in maternal cardiac adverse events was observed with worsening RV dysfunction, underscoring the prognostic significance of right ventricular performance in risk stratification of pregnancies complicated by congenital heart disease. A central observation of this study is the association between severe RV dysfunction and adverse fetal outcomes. The elevated rates of SGA and preterm birth may reflect placental dysfunction related to impaired maternal cardiovascular adaptation. Prior work has shown that maternal RV function may be associated with uteroplacental Doppler indices in the first trimester and pregnancy outcomes among women with CHD [7]. Similarly, maternal cardiac output and fetal Doppler indices have been shown to predict adverse neonatal outcomes in pregnant women with heart disease [18]. These findings support a mechanistic model in which impaired RV reserve leads to suboptimal uteroplacental perfusion, abnormal spiral artery remodeling, and increased susceptibility to placental insufficiency. Previous studies have also investigated biochemical markers associated with prediction of small-for-gestational-age outcomes during pregnancy [22].

A higher incidence of pregnancy-induced hypertension/preeclampsia was observed in patients with more advanced RV dysfunction, suggesting a possible association between impaired right ventricular reserve and adverse placental vascular adaptation during pregnancy [23, 24]. While preeclampsia is multifactorial, impaired placentation and maternal vascular dysfunction are central mechanisms [23–25]. RV dysfunction may contribute indirectly through reduced forward flow, increased venous pressure,

neurohormonal activation, and impaired uteroplacental perfusion. The higher incidence of SGA in patients with advanced RV dysfunction may reflect impaired maternal hemodynamic adaptation and reduced uteroplacental perfusion associated with right-sided circulatory compromise.

Advanced RV dysfunction was associated with a substantially increased risk of preterm delivery, potentially reflecting both maternal cardiovascular instability and increased rates of medically indicated early delivery in high-risk pregnancies.

The coexistence of preeclampsia, SGA, and preterm birth in this population suggests that maternal cardiac dysfunction and placental disease should be interpreted as interconnected processes rather than separate complications.

In this cohort, fetal loss occurred in two pregnancies, with a similar distribution between mild and moderate-to-severe RV dysfunction groups. Although both cases of fetal demise were observed across different severity categories, the extremely small number of events precludes any inference regarding a potential association between right ventricular dysfunction severity and fetal mortality.

Fetal outcomes in women with structural or functional cardiac disease are known to be influenced by multiple maternal and placental factors rather than isolated ventricular dysfunction alone. In line with prior reports in congenital and acquired heart disease during pregnancy, fetal loss is more closely related to the overall maternal hemodynamic burden, presence of cyanosis, arrhythmias, or combined lesions rather than a single echocardiographic parameter of RV function. Therefore, while fetal demise was observed in this cohort, these findings should be interpreted as descriptive rather than indicative of a causal relationship between RV dysfunction severity and adverse fetal outcomes

Neonatal respiratory distress syndrome may occur more frequently in pregnancies complicated by maternal cardiovascular disease, hypertensive disorders, and preterm birth [30, 31].

Arrhythmias occurred in 7.1% of participants. Pregnancy is associated with increased sympathetic tone, higher circulating volume, and increased myocardial stretch, all of which may facilitate arrhythmia in women with structural heart disease [6, 29]. Women with repaired TOF, Ebstein anomaly, and prior cardiac surgery may have additional arrhythmogenic substrates related to chamber dilation, surgical scar, and valvular regurgitation. Although arrhythmia rates in this study were not significantly different between RV dysfunction groups, the small sample size limits interpretation.

The findings also have practical implications for antenatal monitoring. In women with CHD and RV dysfunction, echocardiographic assessment should not be limited to LVEF. RV size, TAPSE, FAC, S' velocity, tricuspid regurgitation, pulmonary regurgitation, pulmonary pressures, and functional status should be evaluated serially. The 2025 ESC pregnancy guideline emphasizes individualized follow-up based on diagnosis, functional status, medication, comorbidities, and obstetric factors [32]. Our findings suggest that RV dysfunction severity should be included in such individualized risk assessment and may

help identify women requiring closer fetal growth surveillance, placental Doppler evaluation, and coordinated delivery planning.

In the present cohort, vaginal delivery was feasible in the majority of pregnancies and represented the predominant mode of delivery overall. This finding is consistent with current cardio-obstetrics recommendations and ESC pregnancy guidelines, which generally favor vaginal delivery in women with congenital heart disease in the absence of severe hemodynamic compromise or obstetric contraindications [32].

The relatively high rate of vaginal delivery observed in our study may be partly explained by the substantial proportion of patients with atrial septal defect physiology and preserved baseline functional status. In these patients, maternal hemodynamic tolerance during labor is often acceptable, particularly in the absence of significant pulmonary hypertension or advanced ventricular dysfunction.

Nevertheless, patients with moderate-to-severe RV dysfunction demonstrated a higher tendency toward cesarean delivery. Although the difference was not statistically significant, likely because of the small cohort size, this pattern probably reflects individualized multidisciplinary decision-making in higher-risk pregnancies complicated by impaired RV reserve, cyanotic physiology, or progressive maternal hemodynamic deterioration.

Importantly, our findings support the concept that the route of delivery in congenital heart disease should be determined primarily by maternal functional and hemodynamic status rather than by cardiac diagnosis alone.

However, the high cesarean rate in our cohort may reflect referral-center practice, clinician concern regarding maternal hemodynamics, fetal compromise, or local obstetric protocols. Future studies should evaluate whether structured delivery planning by a Pregnancy Heart Team can reduce unnecessary cesarean delivery while maintaining maternal and fetal safety.

This study has several limitations. First, it was conducted at a single tertiary referral center and included a relatively small number of patients, which limits statistical power and generalizability. Second, lesion-specific subgroup analysis was not feasible because of the small sample size. Third, placental Doppler indices, biomarkers, and longitudinal RV remodeling data were not systematically integrated into the analysis. Fourth, the cross-sectional design precludes causal inference. Nevertheless, the study provides clinically relevant data on a focused and underrepresented subgroup of pregnant women with CHD, namely those with RV dysfunction.

Future prospective multicenter studies should combine maternal echocardiography, cardiopulmonary exercise capacity, natriuretic peptide levels, uteroplacental Doppler assessment, fetal growth surveillance, and postpartum cardiovascular follow-up. Such studies may clarify whether RV dysfunction is an independent predictor of placental-mediated complications and whether early intervention or intensified surveillance can improve outcomes.

Conclusions

Pregnancy in women with CHD involving RV dysfunction is associated with clinically significant maternal cardiovascular and fetal complications, including heart failure symptoms, preeclampsia, preterm birth, SGA, and fetal/neonatal mortality. Severe RV dysfunction appears to identify a subgroup at particularly increased risk, even when LV systolic function is preserved. These findings emphasize the need for individualized pre-pregnancy counseling, serial echocardiographic surveillance, fetal growth and placental assessment, and multidisciplinary management by a Pregnancy Heart Team in accordance with contemporary ESC guidance.

The abbreviations used throughout the manuscript are summarized in Table 3.

Table 3
Abbreviations

Abbreviation	Definition
ASD	Atrial septal defect
CHD	Congenital heart disease
C/S	Cesarean section
DM	Diabetes mellitus
EF	Ejection fraction
FAC	Fractional area change
GDM	Gestational diabetes mellitus
HTN	Hypertension
LV	Left ventricle/left ventricular
LVEF	Left ventricular ejection fraction
mWHO	modified World Health Organization
NRDS	Neonatal respiratory distress syndrome
NYHA	New York Heart Association
PAP	Pulmonary artery pressure
PI	Pulmonary insufficiency
PPROM	Preterm premature rupture of membranes
PROM	Preterm rupture of membranes
PS	Pulmonary stenosis
PVR	Pulmonary valve replacement
RV	Right ventricle/right ventricular
SGA	Small for gestational age
SVT	Supraventricular tachycardia
TAPSE	Tricuspid annular plane systolic excursion
TDI	Tissue Doppler imaging
TFTC	Tetralogy of Fallot total correction
TOF	Tetralogy of Fallot
TR	Tricuspid regurgitation

Abbreviation	Definition
TVR	Tricuspid valve replacement
UtA	Uteroplacental artery
VSD	Ventricular septal defect

Declarations

Ethics approval and consent to participate: The protocol for this study was approved by the Ethics Committee of Isfahan University of Medical Sciences, Isfahan, Iran (IR.MUI.MED.REC.1401.370).

Consent for publication: The patients provided written informed consent to participate in the study and for publication of their medical information.

Availability of data and material: The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

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