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# Prenatal Diagnosis, Management, and Clinical Outcomes of Fetal Arrhythmias: A 4-Year Experience from a Single Tertiary Center

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**ABSTRACT**

Fetal arrhythmias occur in 1-2% of all pregnancies and can be categorized into three types: rhythm irregularities. tachyarrhythmias and bradyarrhythmias. The rate, duration and severity of the arrhythmia typically determine its hemodynamic consequences. The aim of this study was to evaluate prenatal management strategies and clinical outcomes of fetal arrhythmias in a tertiary perinatal center over a 4 year period.

We retrospectively reviewed 46 fetuses diagnosed with arrhythmia between October 2020 and December 2024. Maternal characteristics, type and of arrhythmia, prenatal treatment, birth parameters and neonatal outcomes were collected.

Forty-six cases of fetal arrhythmia were included in the study. The participants were divided into three groups based on arrhythmia type: rhythm irregularities (n=21, 46%), bradyarrhythmias (n=14, 30%), and tachyarrhythmias (n=11, 24%). Most patients in the bradyarrhythmia group (n=7, %50) were diagnosed with complete atrioventricular block (CAVB), all of whom tested positive for maternal autoantibodies. Supraventricular tachycardia was the most common type of tachyarrhythmia observed, followed by 2:1 atrial flutter.

Fetuses with rhythm irregularities generally have favorable outcomes. The course of fetal CAVB associated with congenital heart defects, particularly left atrial isomerism, appear to be worse than that of immune-mediated CAVB. Persistent tachyarrhythmias should be treated in utero due to the risk of rapid progression to cardiac failure. Once fetal arrhythmias are diagnosed, close follow-up and parental counseling is recommended, using a multidisciplinary team approach involving pediatric cardiology and maternal-fetal medicine specialists.

**Keywords:** Fetal arrhythmia, fetal tachyarrhythmia, fetal bradyarrhythmia, echocardiography, antiarrhythmic therapy, hydrops fetalis, prenatal diagnosis

## Introduction

Fetal arrhythmias occur in approximately 1-2% of pregnancies and compose 10-20% of all referrals to fetal medicine and cardiology units (1). The normal fetal heart rate (FHR) ranges between 110-160 beats per minute (bpm) and fetal arrhythmia is defined as an abnormality in the heart rate or rhythm (2). Clinically, fetal arrhythmias are classified into three subgroups: rhythm irregularities; tachyarrhythmias (FHR above 160 bpm) such as supraventricular tachycardia (SVT), atrial flutter (AF) and ventricular tachycardia; and bradyarrhythmias (traditionally defined as FHR below 110 bpm or below 3 percentile for gestational age) such as sinus node dysfunction, atrioventricular blocks (AVB) and long-QT syndrome (3). Fetal arrhythmias can also be classified as sustained or intermittent depending on whether they are present for more than 50% of examination time (4). The rate, duration and degree of the irregularity typically determine the severity of the arrhythmia and its hemodynamic consequences (2). The diagnosis and assessment of fetal arrhythmias can be performed using echocardiographic modalities such as color flow, M-mode, and pulsed wave Doppler imaging by evaluating the relationship between atrial and ventricular contractions (5, 6).

The most common cause of fetal arrhythmias is premature atrial contractions (PACs), also known as atrial extrasystoles, which present as an

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irregular rhythm with missed or skipped beats in anatomically normal hearts (7, 8). They have generally good prognosis, do not require treatment or further investigation and resolve spontaneously during pregnancy or in the first year of life (7). Although clinically insignificant in most cases, about 1% of PACs can progress to SVT, highlighting the importance of close follow-up during pregnancy (4, 9). In approximately 10% of pregnancies complicated by fetal arrhythmias, the condition can be life-threating with the most likely diagnoses being sustained SVT and atrial flutter (AF) for tachyarrhythmias, and sinus bradycardia, second-degree AVB and complete AVB (CAVB) for bradyarrhythmias (10). In such cases, sustained arrythmia may lead to low cardiac output, hemodynamic decompensation, hydrops, and even intrauterine fetal demise (7). Arrhythmias associated with hemodynamic compromise require treatment to improve fetal survival rate, and timely detection is essential for planning appropriate prenatal and postnatal clinical management (1, 11). Transplacental therapy remains the mainstay of perinatal management, especially in cases of sustained tachyarrhythmias, and digoxin, sotalol, and flecainide are the most commonly preferred antiarrhythmic agents (12). CAVB, the most common type of fetal bradyarrhythmia, may be associated with cardiac malformations or the presence of maternal autoantibodies including anti-Ro/SSA or anti-La/SSB (5). In immune-mediated fetal heart block, anti-inflammatory treatment with fluorinated steroids may be an effective therapeutic option (13). Additionally, the use of β-mimetics has also been shown to help maintain a fetal heart rate >55 bpm (13).

Although several reports and reviews have previously investigated fetal arrhythmias, few have focused on clinical outcomes, and studies providing data from routine clinical practice remain limited. In this retrospective study, we evaluated the prenatal diagnosis, management strategies and perinatal outcomes of pregnancies complicated by fetal arrhythmias in our tertiary perinatal center over a four-year period.

## Materials and Methods

All cases of fetal arrhythmia diagnosed and managed at our maternal-fetal medicine unit, between October 2020 and December 2024 were included in this retrospective study. Patients with incomplete medical records and lacking postnatal follow-up data were excluded.

The study was approved by our institutional ethics committee (approval number: 2024/306) and conducted in accordance with the Helsinki Declaration. Informed consent was obtained from all patients prior to enrollment. Prenatal ultrasound scans were conducted by experienced maternal-fetal medicine specialists using ARIETTA 850 system (Hitachi Medical Corporation, Tokyo, Japan) equipped with a 3.5 mHz abdominal transducer. A comprehensive echocardiographic assessment was performed in each case, including M-mode, color flow imaging, and pulsed wave Doppler studies to measure atrial and ventricular heart rates, atrioventricular (AV) and ventriculo-atrial (VA) intervals. The type of fetal arrhythmia and the presence of structural cardiac malformations were confirmed by a pediatric cardiologist, either on site or during follow-up examinations. After birth, all neonates underwent a detailed cardiac examination by pediatric cardiologists to confirm the diagnosis. The following data were retrieved from hospital database or through follow-up phone calls: maternal characteristics, gestational age at diagnosis, fetal initial atrial and ventricular heart rate, type of arrhythmia, the presence of a structural cardiac or extracardiac malformation, fetal hydrops maternal autoimmune diseases, maternal autoantibodies such as anti-Ro/SSA or anti-La/SSB, intrauterine treatment, gestational age at delivery, admission to neonatal intensive care unit (NICU), neonatal outcomes and postnatal interventions.

Fetal ventricular heart rate above 160 bpm was defined as fetal tachyarrhythmia. Tachyarrhythmias were classified as sustained or intermittent, depending on whether the abnormal rhythm persisted for more than 50% of the examination time. Sinus tachycardia (ST), SVT and AF are the main subtypes of fetal tachyarrhythmia. ST was diagnosed with an atrial heart rate 160-200 bpm and 1:1 atrioventricular (AV) conduction. SVT was defined as a ventricular heart rate 200-260 bpm with 1:1 AV conduction (Figure 1). AV re-entry tachycardia (AVRT), which accounts % 90% of fetal SVT, is characterized by a short VA interval (VA<AV). Other forms of SVT, such as atrial ectopic tachycardia and permanent junctional reciprocating tachycardia (PJRT), typically present with a long VA interval. AF was diagnosed with an atrial heart rate of 400-500 bpm and a ventricular heart rate of 200-240 bpm due to variable AV conduction, most commonly 2:1. Fetal bradyarrhythmia was defined as a sustained ventricular heart rate below the third percentile

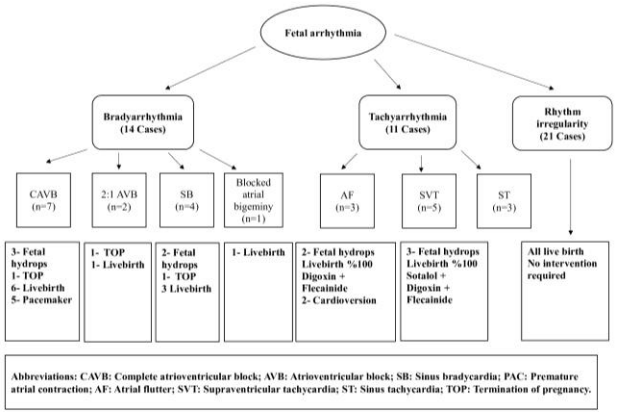


**Fig. 1.** Management and outcomes of prenatally diagnosed fetuses with arrhythmia

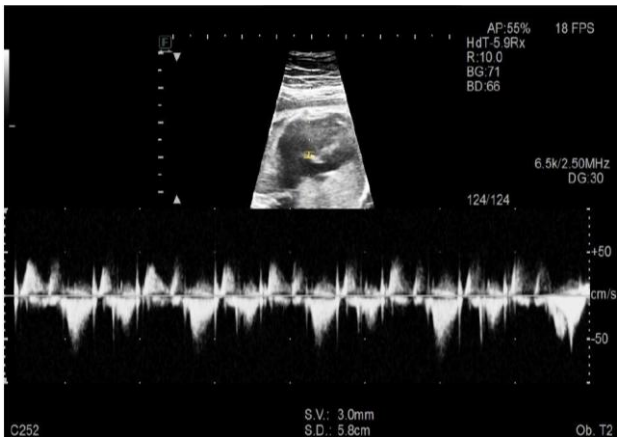


**Fig. 2.** Premature atrial contractions (PACs) presenting as conducted atrial bigeminy

for gestational age. The main causes of fetal bradyarrhythmia include sinus bradycardia, blocked ectopic beats, and AVB. First-degree AVB was defined as delayed conduction from the atria to the ventricles, characterized by a prolonged PR interval. Second-degree AVB was diagnosed by a regular atrial rate with a constant 2:1 AV conduction rate. CAVB was identified by the complete dissociation between atrial and ventricular activity (Figure 2). Irregular rhythm was defined as any deviation from a regular rhythm with a normal heart rate, including premature atrial contractions (PACs) and premature ventricular contractions (PVCs) (Figure 3). Hydrops fetalis was defined as the accumulation of fluid in at least two fetal compartments, typically presenting with generalized skin edema, ascites, pleural effusion, or pericardial effusion.

Digoxin was used as the first-line therapy in cases of sustained fetal tachyarrhythmias, irrespective of the presence of hydrops. A total oral loading dose of 1-2 mg digoxin was given in three equally

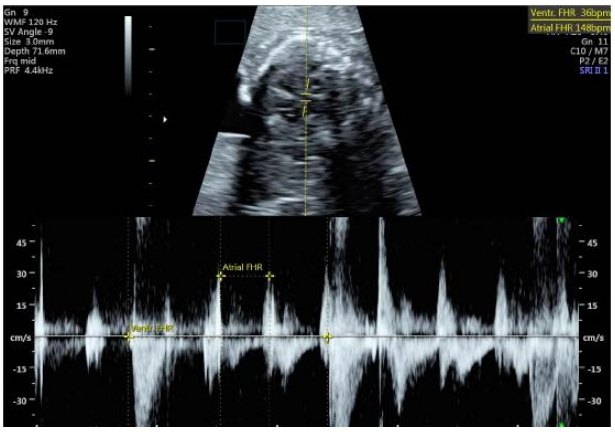


**Fig. 3.** Complete atrioventricular block (CAVB). Atrial beats are regular; however, ventricular beats are slower and completely dissociated from the atrial beats

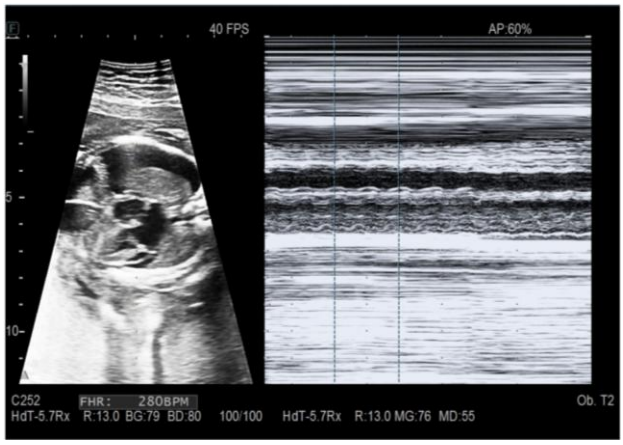


**Fig. 4.** M-mode Doppler showing fetal supraventricular tachycardia with 1:1 atrioventricular conduction

divided doses, followed by measurement of serum digoxin levels, with a target therapeutic range of 1-2 ng/ml. Once therapeutic levels were confirmed, the maintenance therapy was initiated at a total daily dose of 0.5-0.75 mg, administered in divided doses. The fetal response was evaluated 48-72 hours after the first dose. If no improvement was observed despite therapeutic digoxin levels, second-line therapy agents including sotalol and flecainide, were added to the treatment protocol. Flecainide treatment was initiated at a dose of 100 mg in every 8 hours, up to a maximum of 400 mg per day. Sotalol was similarly administered at 80 mg in every 8 hours. All patients underwent a baseline cardiology evaluation prior to the initiation of antiarrhythmic therapy and maternal cardiac function was monitored daily with electrocardiography (ECG).

In patients with anti-Ro/SSA or anti-La/SSB positivity, the PR interval was routinely assessed after 18 weeks of gestation. If the PR interval was

above 150 msec, 4 mg of dexamethasone was initiated. Salbutamol, a β-agonist agent, was given to increase fetal heart rate if it was <50-55 bpm, at a dose of 10 mg every 8 hours, up to a maximum of 40 mg per day.

**Statistical Analysis:** All statistical analyses were conducted using IBM SPSS Version 24.0 (Chicago, IL, USA). Initially, the distribution of continuous data was assessed for normality by examining histogram plots, evaluating kurtosis and skewness values, and performing the Shapiro-Wilk test. Normally distributed continuous variables were expressed as mean ± standard deviation and compared using one-way ANOVA. Tukey's and Games-Howell post-hoc tests were used for pairwise comparisons. Variables that were not normally distributed are expressed as median (minimum-maximum) and were compared using Kruskal-Wallis H test and post hoc pairwise comparison was performed using Mann-Whitney U test. A p-value of <0.05 was considered statistically significant. For the comparison of categorical variables, the Chi-Square test was initially performed. For group-to-group comparisons, pairwise Chi-Square tests with Bonferroni correction were applied to identify which groups differed significantly. According to the Bonferroni correction, a *p*-value of <0.016 was considered statistically significant. Descriptive statistics were performed to depict the clinical characteristics and perinatal outcomes of fetuses diagnosed with brady and tachyarrhythmia.

## Results

Forty-six pregnant women diagnosed with fetal arrhythmia were included in the study. The patients enrolled in the study were divided into three groups based on the type of arrhythmia: bradyarrhythmia, tachyarrhythmia, and rhythm irregularity. The demographic characteristics and clinical parameters of the pregnant women are presented in Table 1. The mean maternal age of the patients and gestational age at diagnosis in the study groups were 28.9 (±4.71), 29 (±5.42), and 29.8 (±4.99), respectively.

Comparisons of clinical characteristics and perinatal outcomes of the patients are presented in Table 2. According to our comparison of three study groups, no statistically significant differences were observed in terms of age ($F_{2,43}$=0.12, p=0.880), BMI ($F_{2,43}$=2, p=0.140), gravida (p=0.060), parity (p=0.050) and mode of delivery (p=0.210) among the bradyarrhythmia, tachyarrhythmia and rhythm irregularity groups. However, statistically significant differences were observed among the three study groups in terms of the duration of neonatal intensive care unit (NICU) stay (p=0.020), gestational age at diagnosis (Welch's $F_{2,27}$=10.35, p=0.001), gestational age at delivery (Welch's $F_{2,22.3}$=4.16, p=0.002), and birth weight ($F_{2,43}$=5.61, p=0.007). The mean gestational age at diagnosis in the three study groups was 23.9 (± 4.18), 30.9 (± 3.62), and 28.8 (± 5.58) weeks, respectively, while gestational age at delivery was 33.6 (± 5.86), 37.1 (± 1.27), and 37.9 (± 1.43) weeks, respectively. Games–Howell post-hoc tests for gestational age at diagnosis and gestational age at delivery revealed that both parameters were significantly lower in the bradyarrhythmia group than in the rhythm irregularity and tachyarrhythmia groups (p=0.001), while no significant difference was observed between the tachyarrhythmia and rhythm irregularity groups (p=0.406). Tukey's post-hoc test performed for birth weight indicated that birth weight was significantly lower in the bradyarrhythmia group than in the rhythm irregularity and tachyarrhythmia groups (p=0.008); however, no statistically significant difference was observed between the tachyarrhythmia and rhythm irregularity groups (p=0.982). According to the results of the Mann–Whitney U test performed for post-hoc analysis of neonatal intensive care unit (NICU) stay, the duration of NICU stay was significantly longer in both the bradyarrhythmia and tachyarrhythmia groups than in the rhythm irregularity group (p=0.022 and p=0.016, respectively), while no statistically significant difference was observed between the tachyarrhythmia and bradyarrhythmia groups (p=0.580).

Statistically significant differences were observed among the three groups in terms of maternal antibody positivity, termination of pregnancy, fetal hydrops, and live births. According to the results of the post-hoc pairwise Chi-Square tests conducted to determine which group differed significantly, maternal antibody positivity was significantly higher in the bradyarrhythmia group than in the other groups (*p* <0.016).

The clinical characteristics of fetuses diagnosed with bradyarrhythmia are illustrated in Table 3. Five patients diagnosed with fetal bradyarrhythmia in the study population had congenital heart defects, including congenitally corrected transposition of the great arteries (cTGA), left isomerism, double-outlet right ventricle (DORV), and atrioventricular septal defect (AVSD). Most

**Table 1:** The Demographic Characteristics and The Perinatal Outcomes of The Pregnancies With Fetal Arrhythmia

| | Brady-arrhythmias (n=14) | Tachyarrhythmias (n=11) | Rhythm irregularities (n=21) | Total (n=46) |
|---|---|---|---|---|
| Age (years) [a] | 28.9 (±4.71) | 29 (±5.42) | 29.8 (±4.99) | 29.3 (±4.90) |
| Gravida [b] | 1 (1-4) | 3 (1-7) | 1 (0-5) | 1 (0-5) |
| Parity [b] | 0 (0-2) | 1 (0-4) | 1 (0-5) | 1 (0-5) |
| BMI (kg/m$^2$) [a] | 28.6 (±4.21) | 26.1 (±2.75) | 26.2 (3.78) | 26.9 (±3.79) |
| GA at diagnosis (weeks)[a] | 23.9 (±4.19) | 30.9 (±3.62) | 28.8 (±5.58) | 27.8 (±5.43) |
| Heart rate (bpm) [b] | 65 (46-150) | 200 (138-255) | 144 (100-195) | 144 (46-255) |
| Structural heart defect [c] | 7 (50) | 1 (9.1) | 4 (19) | 12 (26.1) |
| Fetal hydrops [c] | 5 (35.7) | 5 (45.5) | 0 (0) | 10 (21.7) |
| TOP [c] | 3 (21.4) | 0 (0) | 0 (0) | 3 (6.5) |
| Maternal antibody positivity [c] | 7 (50) | 0 (0) | 0 (0) | 7 (15.2) |
| Live birth [c] | 11 (78.6) | 11 (100) | 21 (100) | 43 (93.5) |
| Delivery mode [c] SVB | 3 (21.4) | 0 (0) | 5 (23.8) | 8 (17.4)) |
| C/S | 11 (78.6) | 11 (100) | 16 (76.2)) | 38 (82.6) |
| GA at delivery (weeks) [a] | 33.6 (±5.86) | 37.1 (±1.28) | 37.9 (±1.43) | 36.4 (±3.84) |
| Birth weight (gr) [a] | 2242 (±1133) | 3023 (±570) | 3075 (±494) | 2809 (±834) |
| NICU duration (days) [b] | 8 (0-120) | 6 (0-7) | 0 (0-35) | 4 (0-120) |
| Postnatal intervention [c] | 2 (14.3) | 8 (72.7) | 1 (4.8) | 11 (23.9) |

BMI: body mass index; GA: gestational age; SVB: spontaneous vaginal birth; C/S: caesarean section; NICU: neonatal intensive care unit; TOP: termination of pregnancy; bpm: beat per minute;
[a]Continuous variables normally distributed were expressed as mean and standard deviation.
[b]Variables not normally distributed were expressed as median (minimum-maximum)
[c] Categorical variables were presented as percentages (%) and counts (n)

Patients in the bradyarrhythmia group (%50) were diagnosed with complete AV block. Other types of bradyarrhythmia were 2:1 AV block, sinus bradycardia, and premature atrial contraction (PAC).

The clinical features of patients with fetal tachycardia are shown in Table 4. Consistent with the literature, SVT was the most prominent tachyarrhythmia observed in our cohort. All cases of SVT had short VA intervals,

The management and overall outcomes of fetuses with prenatally diagnosed arrhythmia are shown in Figure 4.

## Discussion

In our retrospective 4-year study at a tertiary maternal-fetal unit, the most common fetal arrhythmias identified were rhythm irregularities, accounting for 46% (21/46) of all cases, most frequently diagnosed as PACs, either conducted or non-conducted, consistent with the literature (5, 14) (Figure 2). PACs are usually detected in the third trimester and in our study, the median gestational age at diagnosis was 28.8±5.58 weeks, aligning with previous reports (8). Most PACs are caused by the physiological immaturity of fetal conduction system or by stimulation of the right

**Table 2:** Comparison of The Clinical Characteristics and The Perinatal Outcomes of The Pregnancies With Fetal Arrhythmia

| | Brady-arrhythmias (n=14) | Tachyarrhythmias (n=11) | Rhythm irregularities (n=21) | P Values [a] |
|---|---|---|---|---|
| Age (years) [b] | 28.9 (±4.71) | 29 (±5.42) | 29.8 (±4.99) | 0.880 |
| Gravida [c] | 1 (1-4) | 3 (1-7) | 1 (0-5) | 0.060 |
| Parity [c] | 0 (0-2) | 1 (0-4) | 1 (0-5) | 0.050 |
| BMI (kg/m$^2$) [b] | 28.6 (±4.21) | 26.1 (±2.75) | 26.2 (3.78) | 0.140 |
| GA at diagnosis(weeks)[b] | 23.9 (±4.19) | 30.9 (±3.62) | 28.8 (±5.58) | 0.001 |
| Structural heart defect [d] | 7 (50) | 1 (9.1) | 4 (19) | |
| Fetal hydrops [d] | 5 (35.7) | 5 (45.5) | 0 (0) | 0.001 |
| TOP [d] | 3 (21.4) | 0 (0) | 0 (0) | 0.020 |
| Maternal antibody positivity [d] | 7 (50) | 0 (0) | 0 (0) | 0.001 |
| Live birth [d] | 11 (78.6) | 11 (100) | 21 (100) | 0.020 |
| Delivery mode [d] SVB C/S | 3 (21.4) | 0 (0) | 5 (23.8) | 0.210 |
| | 11 (78.6) | 11 (100) | 16 (76.2)) | |
| GA at delivery (weeks) [b] | 33.6 (±5.86) | 37.1 (±1.28) | 37.9 (±1.43) | 0.002 |
| Birth weight (gr) [b] | 2242 (±1133) | 3023 (±570) | 3075 (±494) | 0.007 |
| NICU duration (days) [c] | 8 (0-120) | 6 (0-7) | 0 (0-35) | 0.023 |
| Postnatal intervention [c] | 2 (14.3) | 8 (72.7) | 1 (4.8) | 0.001 |

BMI: body mass index; GA: gestational age; SVB: spontaneous vaginal birth; C/S: caesarean section; NICU: neonatal intensive care unit; TOP: termination of pregnancy; bpm: beat per minute.

[a]Level of significance $P < 0.05$

[b]Continuous variables that were normally distributed are expressed as mean ± standard deviation and were compared using One-way ANOVA test with Tukey's post-hoc and Games-Howell post-hoc tests for pairwise comparison.

[c]Variables that were not normally distributed are expressed as median (minimum-maximum) and were compared using Kruskal-Wallis H test and post hoc pairwise comparison was performed using Mann-Whitney U test.

[d]Categorical variables were presented as percentage (%) and count (n) and were compared using Chi-Square and Pairwise Chi-Square test

atrial wall by the foramen ovale flap, and they usually follow an uneventful course, resolving spontaneously (15). However, a small number of cases may be associated with an underlying congenital heart defect (CHD) or genetic condition, such as Costello syndrome (1). In rare instances, PACs may also trigger episodes of tachycardia and progress to SVT (7). Therefore, once PACs are detected, a detailed assessment of fetal cardiac anatomy is recommended, along with weekly fetal heart rate monitoring throughout pregnancy (5, 15). In our cohort, all cases with PACs resolved either by the end of pregnancy or shortly after the birth, and none progressed to SVT during follow-up. No major cardiac defects were observed; however, additional anatomic findings included muscular ventricular septal defects (VSD) in two cases and an aneurysmatic flap valve of the foramen ovale in another two cases.

**Table 3:** The Clinical Characteristics of Fetuses Diagnosed With Bradyarrhythmia

| | | Brady-arrhythmias (n=14) |
|---|---|---|
| Type of arrhythmia | Complete AV block | 7 (50) |
| | 2:1 AV block | 2 (14) |
| | Sinus bradycardia | 4 (28) |
| | PAC | 1 (7) |
| Congenital heart defect | c-TGA | 1 ((7) |
| | DORV + Left isomerism | 1 (7) |
| | Aortic stenosis | 1 (7) |
| | AVSD + Left isomerism | 1 (7) |
| | Left isomerism + PLSVC | 1 (7) |
| Heart rate (bpm) | | 75.7 (± 28.51) |

BMI: body mass index; GA: gestational age; SVB: spontaneous vaginal birth; C/S: caesarean section; NICU: neonatal intensive care unit; TOP: termination of pregnancy; bpm: beat per minute; DORV: double outlet right ventricle; cTGA: corrected transposition of great arteries; PAC: premature atrial contractions; AVSD: atrioventricular septal defect; PLSVC: persistent left superior vena cava

The most common types of fetal bradyarrhythmia are sinus bradycardia, blocked atrial bigeminy/trigeminy and AVBs (13). Transient sinus bradycardia due to vagal stimulation during examination is usually benign; however, persistent bradycardia warrants further evaluation to identify the underlying etiology (8, 16). Blocked atrial bigeminy is a form of non-conducted PACs, characterized by every other atrial extrasystole failing to conduct to the ventricles (17). It is typically benign, well-tolerated, and resolves spontaneously (17). Postnatal 24-hour Holter monitoring is usually sufficient for follow-up (16). Fetal AVB can result from congenital displacement of the sinoatrial node in the presence of CHD such as congenitally corrected transposition of great arteries (cTGA) or heterotaxy syndromes, or from progressive inflammation and/or fibrosis of the conduction system due to the transplacental passage of maternal autoimmune antibodies (9, 11). In our series, 30% (14/46) of all cases were diagnosed with fetal bradyarryhtmia, including 7 with CAVB, 2 with 2:1 AVB, 4 with sinus bradycardia and 1 with blocked atrial bigeminy, with CAVB being the most common form, consistent with previous reports (18) (Figure 3). In the literature, left atrial isomerism is the most common cardiac defect associated with fetal bradyarrhythmias, followed by cTGA and atrioventricular septal defect (AVSD) (18). Similarly, in our series, 50% (7/14) of fetuses with bradyarrhythmia had associated CHD, including left atrial isomerism in three cases, cTGA in one, AVSD in one, aortic stenosis in one and DORV in one. Of these, four presented with sinus bradycardia and one with 2:1 AVB. In two cases with left atrial isomerism, the parents opted for TOP, while the remaining case died in the neonatal period. Additionally, one case with aortic stenosis also resulted in neonatal death. Our findings are consistent with the literature, which suggests less favorable outcomes and reports up to 80% adverse perinatal events in cases of fetal bradyarrhythmias with CHD (18, 19). Jaeggi et al. reported that the prognosis is particularly poor in the presence of left atrial isomerism, which is in accordance with our findings (20). In our series, all cases of CAVB were immune-mediated, with positive maternal anti-Ro/SSA and anti-La/SSB autoantibody results, consistent with previous studies reporting a high prevalence of maternal anti-Ro/SSA positivity in this group (18, 21). In immune-mediated CAVB, anti-inflammatory treatment with fluorinated steroids such as dexamethasone has been shown to improve outcomes related to myocardial inflammation, although it does not alter cardiac rhythm (10, 22). Furthermore, the use of β-mimetics may help maintain a fetal heart rate >55 bpm (23). Jaeggi et al. reported

improvement in one-year survival and reduced neonatal morbidity rates in fetuses with CAVB treated with dexamethasone and β-mimetics (22). However, conflicting evidence exists, with some studies reporting no significant benefit from prenatal corticosteroid therapy in fetuses with CAVB (24). In our series, all fetuses with maternal autoimmune disease and CAVB received a combination of dexamethasone and terbutaline, due to a fetal heart rate <55 bpm. Among the seven fetuses that received maternal treatment, all survived, except for one diagnosed with Down syndrome via karyotype analysis, for which TOP was elected. Although CAVB persisted, cardiac function improved, and hydrops regressed in five of the affected fetuses. This favorable survival rate in fetuses with CAVB may be attributed to timely initiation of maternal dexamethasone and terbutaline therapy, but it may also be explained by the absence of congenital heart defects. 5 cases with CAVB, in which fetal heart rate remained >60 during follow-up and no hemodynamic compromise was observed, underwent permanent pacemaker implantation during the neonatal period.

The tachyarrhythmia group in our cohort mainly consisted of cases with SVT with a short VA interval (8/11, 72.8%), which aligns with the literature suggesting that SVT makes up about 75% of all fetal tachyarrhythmias. (7, 25) (Figure 4). In total, 40% of fetuses with SVT present with hydrops due to cardiac failure (17). The risk of hydrops increases with higher ventricular rates, the presence of structural cardiac anomalies, and onset of tachycardia before 32 weeks of gestation (16). In cases of fetal tachyarrhythmia, hydrops is a major prognostic determinant and is strongly associated with fetal demise (17). In our series, the incidence of hydrops fetalis in tachyarrhythmia group was 45.5% (5/11), which was significantly higher than in the bradyarrhythmia group. According to existing literature, congenital heart defects are found in 1-5% of fetuses with tachyarrhythmia, and the presence of additional cardiac anomalies is associated with more unfavorable outcomes (6, 7). In our tachyarrhythmia group, only one fetus (9.1%) had an additional cardiac defect, namely a VSD, and we did not find a significant influence of this on perinatal outcomes. A sustained fetal tachyarrhythmia or evidence of hemodynamic compromise requires urgent treatment to improve fetal survival (7). Currently, the combinations of flecainide plus digoxin and sotalol plus digoxin are the most preferred treatment modalities for restoring sinus rhythm (26). In our cohort, all patients, including eight SVT and three 2:1 atrial flutter, received in utero treatment: four were treated with digoxin alone, four with digoxin plus flecainide, and three with digoxin plus sotalol. Antiarrhythmic treatment was effective in converting the SVT or AF to sinus rhythm in six patients and in reducing hydrops in four patients; however, eight fetuses (%72.7) required antiarrhythmic therapy during the neonatal period as well. Our findings are consistent with the literature that suggests most of the infants diagnosed with SVT prenatally also present with SVT postnatally (25, 27). One fetus (1/11, 9.1%) died on the 10$^{th}$ postnatal day despite undergoing electrical cardioversion and antiarrhythmic therapy. Our results indicate similar outcomes in fetuses with tachyarrhythmia compared with previous reports (11, 27). Springer et al. reported a live birth rate of 92.2% in fetuses with tachyarrhythmia and a neonatal death rate of 8.5%, whereas O'Leary et al. reported a lower neonatal mortality rate of 3% (11, 27).

When fetal bradyarrhythmia and tachyarrhythmia groups were compared in terms of gestational age at delivery, delivery occurred significantly earlier in the bradyarrhythmia group, likely reflecting the early onset of fetal compromise, consistent with the literature (11). Accordingly, NICU stay was also prolonged, possibly due to prematurity, the need for pacemaker evaluation, and extended cardiac monitoring. On the other hand, as reported in the literature, successful transplacental antiarrhythmic treatment can be achieved in most cases of fetal tachyarrhythmia, helping to postpone delivery (14). In our series, fetal bradyarrhythmias were diagnosed at an earlier gestational age (23.9 ± 4.19 weeks) compared to tachyarrhythmias (30.9 ± 3.62 weeks). This finding is consistent with the literature, which suggests that the onset of AVB, the predominant bradyarrhythmia in our cohort, typically occurs between 18-24 weeks of gestation and rarely develops after 28 weeks, whereas fetal SVT and AF are primarily diagnosed in third trimester (3).

The strength of our study lies in the inclusion of all types of fetal arrhythmias, as well as the availability of data on birth parameters and neonatal outcomes. The main limitations of this study are its retrospective design with a relatively short study period, small sample size and the lack of long-term cardiac outcomes in the infants.

In conclusion, our findings underscore the importance of accurate classification, timely diagnosis, and individualized management of fetal

arrhythmias. Fetuses with rhythm irregularities generally present more favorable outcomes than bradyarrhythmias and tachyarrhythmias. The course and the survival of fetal CAVB associated with congenital heart defects, particularly left atrial isomerism, appear to be worse than of immune-mediated CAVB. Persistent tachyarrhythmias should be treated in utero due to the risk of rapid progression to cardiac failure. Favorable neonatal outcomes can be achieved following the administration of maternal antiarrhythmic agents. Once fetal arrhythmias are diagnosed, close follow-up and parental counseling is recommended using a multidisciplinary team approach involving pediatric cardiology and maternal-fetal medicine to discuss pregnancy management, intrauterine treatment options, possible prognosis and postnatal interventions. Our findings align with previous reports and may assist clinicians in the decision-making process for managing fetal arrhythmias. Further studies with larger cohorts are required to define the relationship between clinical parameters and outcomes, and to optimize intrauterine treatment strategies.

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