Fetal Conduction Abnormalities: Retrospective Analysis of 40 Cases from a South Indian Tertiary Care Centre with Management Insights and Brief Review

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Abstract

Background: Fetal cardiac rhythm disruptions pose significant challenges to prenatal and postnatal well-being. These disruptions, encompassing various arrhythmias, necessitate timely diagnosis and precise management. Transabdominal fetal echocardiography has become a crucial diagnostic tool for evaluating fetal arrhythmias, enabling tailored interventions. **Objectives:** This retrospective study aims to examine 40 cases of fetal arrhythmias at our institution, shedding light on common abnormalities, diagnostic intricacies, and therapeutic strategies. **Methods:** A seven-year retrospective analysis was conducted at a tertiary-level hospital in South India. Cases with sustained fetal arrhythmias were reviewed, considering gestational age, types of arrhythmias, associated anomalies, and maternal factors. The diagnostic process involved transabdominal fetal echocardiography, emphasizing M-Mode and pulsed wave Doppler measurements. **Results:** Fetal bradyarrhythmias were predominant (57.5%, 23/40), with complete heart blocks being a primary cause. Hydrops was observed in 20% (8/40) of cases. Structural cardiac anomalies were present in 27.5%(11/40) of cases, with maternal antibody positivity noted in 22.5% (9/40). Management varied, including intrauterine and postnatal interventions based on the gestational period, severity of rhythm disturbance, and structural abnormalities. Few ectopic cases exhibited spontaneous regression. Fetal tachyarrhythmias(20%; 8/40) included supraventricular tachycardia (62.5%; 5/8), atrial flutter (12.5%, 1/8), junctional ectopic tachycardia (12.5%, 1/8), and ventricular tachycardia (12.5%, 1/8). Combined digoxin and flecainide showed success, especially in hydrops-associated cases. **Conclusion:** This study provides insights into the diverse presentations, diagnostic challenges, and therapeutic approaches in managing fetal arrhythmias. The findings underscore the critical role of accurate prenatal diagnosis for tailored therapeutic interventions. While advancements have been made, persistent challenges necessitate ongoing innovation. Call for further research to refine treatment strategies and collaboration among multidisciplinary teams remains paramount for these uncommon conditions.

Introduction

The intricate development of the fetal conduction system is essential for the establishment of a functional cardiovascular system. Disruptions in the conduction pathways during fetal life can lead to serious consequences, impacting both prenatal and postnatal well-being. Disturbances of fetal cardiac rhythm are reported to occur in 1 to 3 % of all the pregnancies and 10% of these can be life-threatening.[1] While most are benign, certain types like supraventricular or ventricular tachycardia, atrial fibrillation, and atrioventricular (AV) block may lead to severe complications.[2] Transient fetal arrhythmias are more common, but persistent severe bradycardia and sustained tachycardia can result in fetal hydrops, preterm delivery, and increased morbidity and mortality.[3] Hence, early diagnosis before progression to hydrops, is both crucial and challenging and requires a specialized team with expertise in fetal echocardiography. The etiology of fetal conduction disturbances is diverse, encompassing ischemia, inflammation, electrolyte imbalances, structural defects, and inherited genetic conditions.[4] Understanding the underlying causes and effective management strategies for

these abnormalities is vital for improving prenatal care and neonatal outcomes. In this study, we present a retrospective analysis of a cohort comprising 40 cases of fetal arrhythmias diagnosed and managed in our institute with an aim to contribute valuable insights into the diverse challenges encountered.

Aims and Objectives

Aims:

To identify the common fetal conduction abnormalities observed in our institution and understand the clinical implications associated with these abnormalities.

To enhance understanding of the diverse presentations, diagnostic challenges, and therapeutic approaches in managing fetal arrhythmias.

Objectives:

Provide an update on the diagnosis and management of fetal arrhythmias by reviewing current protocols.

Materials and Methods:

A retrospective study was conducted at the Prenatal Diagnostic and Intervention center in the Department of Obstetrics and Gynecology at Jawaharlal Institute of Postgraduate Medical Education and Research (JIPMER), a tertiary referral Institute of National Importance in Puducherry, South India, from June 2016 to July 2022. During the hospital visit, patients are required to sign a form granting consent for the analysis of their pregnancy data for scientific purposes, in accordance with the existing ethical guidelines. As this study involved a retrospective review of data collected for clinical purposes, formal approval from an ethics committee was not sought. A total of 55 cases of reported intermittent or sustained fetal arrhythmias were identified during the study period of 7 years, with review of the medial records and Fetal Echocardiography images and cine-loops. The cases of non-sustained rhythm abnormalities were excluded and the 40 cases with sustained rhythm abnormalities were included in the study. Pregnancy and cardiac outcomes were acquired from the system data base and case files.

The analysis involved the examination of fetal arrhythmia types, clinical characteristics, and outcomes, as well as outlining a management approach. Fetal arrhythmias were categorized into irregular rhythms, tachyarrhythmia, and bradyarrhythmia, with definitions[5] provided in **Table 1** . A fetal medicine expert re-evaluated the images and cine loops to confirm and assign final diagnoses. The study utilized detailed transabdominal fetal echocardiography, performed by a single professional using the Voluson E8 from General Electronics. M-mode and Doppler studies complemented 2-D assessments. Management decisions were made considering gestational period, type, and severity of rhythm disturbance, and the presence of structural abnormalities. Descriptive analysis was presented for numerical variables as mean, standard deviation, or median, while categorical variables were expressed in numbers and percentages. Due to the small sample size, statistical and multivariate analyses were not conducted.

Results

We included a total of 40 cases with prenatally detected fetal cardiac rhythm disturbances. Bradyarrhythmia accounted for the majority at 57.5% (n=23/40) among all types of fetal arrhythmias, followed by irregular rhythm at 22.5% (n=9/40) and tachyarrhythmia at 20% (n=8/40). Complete heart blocks (CHB) emerged as the primary cause of fetal bradyarrhythmia. At the time of diagnosis, hydrops was observed in 20% of all fetal arrhythmia cases (n=8/40). Structural anomalies were present in 30% of cases (n=12/40) of which 27.5% (n=11/40) were cardiac anomalies and in 2.5% (n=1/40) was lymphatic anomaly. Maternal antibody positivity for anti-Ro, anti-La was noted in 22.5% (n= 9/40); anti-Ro positive in 8 cases, both positive in one case and SLE was seen in 39.139/23. The clinical characteristics and outcomes are summarized in **Table 2 and 3** .

Ectopies

We identified 8/40 cases of fetal ectopy, with 6/8 premature atrial contractions (PAC) and 2/8 premature ventricular contractions (PVC). The clinical characteristics and outcomes of these cases are summarized in**Figure 1** . Two among these exhibited a bigeminal pattern (**Figure 2)** , one showed a trigeminal pattern (**Figure 2)** , and one displayed a mixed bigeminal and trigeminal pattern.

Bradyarrhythmias

We identified 23/40 cases of bradyarrhythmia, comprising 16/23 cases of CHB, 4/23 cases of AV block, and 3/23 cases of sinus bradycardia. Of these, 17/23 fetuses exhibited normal cardiac and extracardiac anatomy, while 6/23 had associated anatomical abnormalities (5 cardiac and 1 cystic hygroma). The cardiac anomalies included pulmonary stenosis with cardiomegaly (n=1/5), right ventricle hypoplasia, atrioventricular septal defect (n=2/5), and heterotaxy (n=1/5). None had chromosomal abnormalities or congenital infections. **Figure 3** illustrates few cases diagnosed employing M-mode and pulsed wave Doppler.

Among the CHB cases, 9/16 were associated with Maternal Systemic Lupus Erythematosus (SLE), with 1/9 case also linked to secondary antiphospholipid syndrome (APS) and maternal hypothyroidism, and another 1/9 associated with Sjögren's syndrome. There were 2/16 cases of Type 2 diabetes mellitus (DM) noted in the mother. We offered antibody testing (anti-Ro, anti-La) to all mothers with fetuses exhibiting bradyarrhythmia. Anti-Ro alone tested positive in 5/16 cases, both antibodies were positive in 1/16 patient, 10/16 mothers had negative antibodies and remained negative throughout pregnancy.

Among the cases of CHB associated with SLE and positive maternal antibodies (n=9/16), a combined treatment of hydroxychloroquine (HCQ) and dexamethasone was administered until delivery. Weekly follow-ups were done to monitor fetal AV intervals, cardiac function, fetal development, and maternal side effects of dexamethasone and HCQ. There were 2/16 intrauterine fetal demises: one at 36 weeks among the SLE cases and another at 30 weeks with fetal heterotaxy. Additionally, 3/16 neonatal deaths occurred among the two fetuses with atrioventricular septal defects and one with right ventricle hypoplasia on postnatal days 10, 1 month, and 5th day, respectively. All these cases had exhibited persistent bradycardia with irregular rhythm. Termination of pregnancy (TOP) was chosen by 4/16 patients at an earlier gestation. There were 7/16 liveborn infants who have not been prescribed any medication and are currently under cardiology with expectant management.

Among the AV block cases, there were 3 second degree block and one first degree block case. Among the second-degree block cases, 1/4 case was complicated with fetal growth restriction (FGR) with absent end-diastolic flow (AEDF) and had later on progressed to CHB. An emergency lower segment caesarean section (LSCS) was performed at 32 weeks of gestation. The neonate-maintained heart rate between 50-55 beats per minute (bpm) and was started on beta adrenergic, postnatally. There were 2/3 cases of seronegative second-degree AV block. Among them, one case was associated with FGR and severe oligohydramnios and was delivered vaginally at 34 weeks due to preterm labor. The neonate, maintained a heart rate between 60-70 bpm and was on expectant management without the need for medical or surgical intervention. The other case had atrial septal defect (ASD) and total anomalous pulmonary venous connection (TAPVC) with hydrops fetalis, and the patient opted for termination at 21 weeks. The remaining 1/4 case had a first-degree AV block with SLE and positive anti-Ro antibody and hydrops fetalis. However, antenatal steroid therapy for six weeks resulted in the resolution of hydrops, and the fetal heart rhythm reverted to normal. Postnatally, the neonate-maintained sinus rhythm.

Among the 3/23 cases of sinus bradycardia, there were no associated cardiac abnormalities or positive maternal antibodies and 2/3 cases reverted to normal sinus rhythm. Postnatal ECG and echo for these cases were normal. In 1/3 fetus, the prenatal diagnosis of sinus bradycardia changed to CHB on postnatal evaluation with HR <40bpm with minimal response to beta sympathomimetics and required pacing which was done 24 hours after birth. However, severe pericardial effusion was noted on PND 3 and the baby expired.

Tachyarrhythmias

There were 8/41 cases of tachyarrhythmias. Among them, we identified 5/8 cases of fetal supraventricular

tachycardia (SVT) (**Figure 2)**and 1/8 case each of ventricular tachycardia (VT), atrial flutter, and junctional ectopic tachycardia (JET). Among the SVT cases, 3/5 fetuses had presented with hydrops. In these cases, a combined treatment approach involving oral digoxin and flecainide was started. While successful restoration to sinus rhythm was observed in all cases (3/3), one patient opted for termination of pregnancy at 24 weeks due to persistent hydrops. The other 2/3 cases, with resolved hydrops and restored sinus rhythm, underwent regular follow-ups through ultrasound examinations. Elective LSCS was performed for one fetus at 39 weeks, and postnatal evaluations, including ECG and echocardiography, revealed normal results. The child was under cardiology follow up till one year and remained in good health. The second fetus was delivered vaginally at 38 weeks, and postnatal assessments showed normal cardiac function.

In the remaining 2/5 cases, the initial treatment involved the administration of oral digoxin. When the initial intervention did not produce a positive response, a secondary antiarrhythmic agent (flecainide) was introduced. In one case, sinus rhythm was successfully restored after the addition of flecainide, with one episode of relapse before stabilization. The baby was delivered by LSCS at 38 weeks, maintaining a normal cardiac rhythm postnatally. However, in the other case, despite the combined use of flecainide and digoxin, persistent tachycardia was observed. Subsequent echocardiography revealed moderate pleural effusion with cardiomegaly. Emergency LSCS was performed at 36 weeks due to impending heart failure, and adenosine was administered postnatally to manage persistent tachycardia. Despite an initial positive response, subsequent relapse and deterioration occurred, leading to death on postnatal day 3.

Additionally, we encountered one case of JET with features of hydrops fetalis, referred at 24 weeks for fetal tachycardia (Fetal heart rate (FHR) 170–180 bpm) The fetal heart exhibited structural normalcy, with regular rhythm and 1:1 AV conduction, yet minimal heart rate variability. Spectral Doppler revealed increased retrograde flow during atrial systole in the ductus venosus and hepatic and pulmonary veins. Transplacental digoxin and flecainide were administered, resulting in the conversion to sinus rhythm after 6 weeks of treatment. Hydrops fetalis and retrograde flow in veins resolved, although ventricular filling remained monophasic. The fetus was delivered by LSCS at 38 weeks, with postnatal ECG and echocardiography showing normal results and infant maintaining sinus rhythm till postnatal day 5 and was discharged.

In one case, atrial flutter with an atrial rate of 460bpm and ventricular rate of 230 bpm was identified at 36 weeks of pregnancy in a patient with normal prenatal history. Although there was mild tricuspid valve regurgitation and minimal pericardial effusion, no evident cardiac structural anomaly, atrial enlargement, or hydrops was observed. After discussing management options, prognosis, and postnatal outcomes, the patient opted for medical management and was initiated on digoxin, resulting in a reversion to sinus rhythm after 3 days. An emergency LSCS was performed at 37 weeks due to breech presentation and premature rupture of membranes (PROM). The patient delivered a vigorous baby weighing 2.8 kg, but the infant experienced moderate pulmonary hypertension, requiring continuous positive airway pressure (CPAP). Postnatal echocardiography showed mild tricuspid regurgitation and a patent foramen ovale. The infant is currently in sinus rhythm and undergoing cardiology follow-up.

In another instance, ventricular tachycardia was diagnosed at 37 weeks of gestation in a patient who presented with decreased fetal movements for one day but otherwise uneventful antenatal period. Fetal echocardiogram showed VT with a ventricular rate of 240 bpm compared to an atrial rate of 130 bpm. The ECG displayed a polymorphic pattern with a prolonged QT interval. Maternal Anti-Ro/La antibodies were negative, and both parents had normal echocardiograms. An intravenous magnesium sulfate infusion with a loading dose of 4 gm followed by a maintenance dose of 1g/h was initiated to gain rapid control of the fetal arrhythmia and continued for 48 hours. The fetal VT resolved after 10 hours of magnesium sulfate commencement. Propranolol was administered after 48 hours at a dose of 80 mg three times daily, with intermittent relapses noted before the rhythm ultimately reverted to normal. Spontaneous labor occurred, and the patient delivered at 38 weeks of gestation. The newborn, however, exhibited poor Apgar scores, leading to NICU admission. The baby experienced severe encephalopathy, hyperkalemia, and acute kidney injury, ultimately succumbing 3 hours after birth. Genetic testing was not pursued as parental consent was not obtained.

Discussions

The evolution of prenatal diagnosis and treatment for fetal arrhythmias embarked on its journey in the 1980s, marked by Kleinman et al.'s pioneering report on prenatal diagnosis using M-mode echocardiography. Since then, various diagnostic methods, from fetal ultrasonography to fetal magnetocardiography, have emerged, each contributing to our understanding of fetal cardiac rhythm abnormalities. By employing a combination of these diagnostic modalities healthcare professionals can effectively classify rhythm abnormalities in fetuses, enabling tailored interventions. However, despite these advancements, diagnosing fetal arrhythmias remains a challenging task. At present, transabdominal fetal echocardiography has emerged as one of the mainstays for diagnosing fetal arrhythmias.[7]

Main findings and interpretations:

Bradyarrhythmias and ectopies:

Bradyarrhythmias emerged as the predominant fetal arrhythmia (23/40) in our study, followed by irregular rhythms (9/40) and fetal tachyarrhythmias (8/40). This finding diverges from previous studies[8-10] where fetal arrhythmias manifested commonly as irregular rhythms and were mostly deemed benign with minimal impact on fetal hemodynamics and often resolve spontaneously without intervention. However, among the cases of irregular rhythms also we found 5/9 cases associated with SHD, leading to adverse outcomes such as one IUD, one MTP for complex cardiac anomaly and one infant mortality. We also saw a case of PAC with a bigeminal pattern which progressed to SVT. This highlights the need for careful monitoring and proactive management of fetal cardiac rhythm abnormalities, even when irregular rhythms are present.

Our study reveals that only 26.1% (6/23) of fetal bradyarrhythmia cases were associated with structural cardiac anomalies, contrasting the literature reports[11,12] where such associations were seen in approximately half of the cases. Moreover, both maternal SLE and anti-Ro/La antibody positivity was observed in 39.1% (9/23) of fetal bradyarrhythmia cases , emphasizing the need for heightened awareness of maternal health conditions impacting fetal cardiac rhythm and the importance of screening and early intervention in at-risk populations.

Management of fetal bradyarrhythmia poses unique challenges, especially in cases of CHB where an expectant approach is primarily employed, as there is no documented evidence of reversing an established third-degree block.[5,16] Options like beta-adrenergic agents, immunoglobulin, and plasmapheresis are not preferred due to unclear efficacy and potential maternal risks.[17-19,21,22] A study by Jaeggi et al.[20]suggested a survival rate above 90% for antibody-related CHB if maternal high-dose dexamethasone was initiated at the time of anomaly diagnosis and maintained throughout pregnancy. Another meta-analysis in 2018, including five different observational studies with second-degree immune-mediated CHB, concluded that the use of fluorinated glucocorticoids should not be discouraged unless more robust evidence is available.[16] In our study, all cases with CHB (16/40) and AV block (4/40) received treatment with dexamethasone until delivery, and HCQ was added for cases with SLE. There were in total 14/20 live births among these cases, supporting existing literature that emphasizes the positive role of dexamethasone in improving outcomes in such cases. Similarly, in cases of isolated atrioventricular (AV) block, early initiation of treatment and close monitoring contribute to favorable outcomes, as illustrated by successful reversion to sinus rhythm in cases managed expectantly.

Tachyarrhythmias

Fetal tachyarrhythmias, occurring in 0.4 to 0.6% of pregnancies,[23] can lead to severe complications if left untreated, including nonimmune hydrops, premature delivery, fetal demise, and perinatal morbidities. However, the requirement for therapeutic intervention in this condition remains a subject of ongoing debate, centered on the natural progression of the disease. Divergent opinions range from advocating non-intervention approach to aggressive pharmacotherapeutic intervention. Transplacental treatment by indirect drug administration to the mother appears to be the most preferred approach.[23,24]

SVT stands out as the most common contributor, accounting for 70-75% of cases, followed by atrial flutter, which contributes to 25-30%.[25] Our study aligns with this pattern, with SVT being the predominant

contributor (62.5%, 5/8). While fetal arrhythmias are typically isolated findings, around 5% of cases may also involve congenital heart diseases.[26,27] In our study, all identified tachyarrhythmias were isolated without associated SHD or tumors.

The presence of hydrops also significantly influences management and outcomes, as it indicates an impact on the cardiovascular system, reducing ventricular filling and cardiac output.[28] In this study, hydrops fetalis was observed in 50% (4/8) of fetal tachyarrhythmia cases. The primary goal of fetal therapy is to achieve the prevention or resolution of hydrops either through conversion to sinus rhythm or ventricular rate control.[29,30]Transplacental administration of antiarrhythmic drugs is a commonly employed and effective approach, with the primary aim of preventing or resolving hydrops.[23,24] Although no standardized protocol exists for drug selection or doses, successful outcomes have been documented. However, the decision on in utero or postnatal treatment is challenging, with the effectiveness of both approaches well-established.[31] While intrauterine treatment is highly effective, postnatal treatment remains a viable option particularly in cases when imminent delivery is crucial.

Digoxin is widely accepted as a first-line antiarrhythmic drug.[24] Sotalol, flecainide, and amiodarone serve as secondary options when digoxin proves ineffective in achieving conversion to sinus rhythm.[32] However, studies indicate that for fetuses with hydrops, digoxin's limited placental transfer hampers its effectiveness.[33] Therefore, for cases involving hydrops, especially in fetal treatment, it is recommended to initiate sotalol or flecainide, both of which exhibit good placental transfer abilities.[31,34] In our study, the combination of digoxin and flecainide was introduced in the cases which had associated hydrops, encompassing three instances of SVT and one case of JET. All these cases reverted to sinus rhythm along with resolution of the hydrops, accounting for 50% of the case of fetal tachyarrhythmias. Digoxin monotherapy was initiated in two isolated cases of SVT and once case of atrial flutter. Successful reversion to sinus rhythm was achieved in the case of atrial flutter. However, in SVT cases, monotherapy with digoxin did not result in reversion, necessitating the addition of other antiarrhythmic agents in both instances.

Thus, the study underscores the prevalence of SVT in fetal tachyarrhythmias and the pivotal role of hydrops in treatment decisions. While much of the literature often compares digoxin versus flecainide and explores stepwise approaches to managing SVT, there are limited case studies delving into the initiation of combined digoxin and flecainide as a first line treatment, particularly in cases involving hydrops.[35,36] The successful application of this combined therapy in our study suggests a potential shift in the treatment paradigm and proposes it as a viable first-line option for SVT with hydrops. However, further studies with larger sample sizes are necessary to validate the findings and it is crucial to exercise caution and make thoughtful selections of antiarrhythmics tailored to the unique characteristics of each case.

VT is a rare fetal arrhythmia, constituting only 1–2% of cases.[25] Optimal therapy remains unclear due to its rarity. Current literature suggests maternal intravenous magnesium therapy as a first-line treatment for sustained VT.[37]Additional measures, such as intravenous lidocaine, oral propranolol, or oral mexiletine, are recommended alongside magnesium.[32,37,38] In cases related to isoimmunization or myocarditis, dexamethasone and intravenous immunoglobulin infusion have been employed.[39] In this study, we encountered one case of VT with prolonged QT, where treatment with IV magnesium sulphate followed by oral propranolol was initiated. While the rhythm was controlled with small recurring episodes, the baby succumbed to neurological complications 3 hours after birth. Hence, the study highlights the rarity of fetal VT, emphasizing the challenges in therapeutic strategies and the need for further research to establish standardized protocols. Beyond clinical aspects, the study suggests delving into the genetic and molecular factors associated with fetal VT, potentially leading to personalized management strategies based on identified markers. The impact of early VT diagnosis, evaluation of maternal and fetal risk factors, and a detailed analysis of neurological complications constitute key facets of this multifaceted approach.

Strengths and limitations

The strength of our study lies in its single-center design, contributing to decreased interobserver variability and ensuring data consistency and inclusion of all three types of fetal arrhythmia. However, notable limita-

tions include lack of long term follow up, and the small sample size derived from a single center, potentially limiting the generalizability of findings.

Conclusion

In summary, the management of fetal arrhythmias plays a pivotal role in enhancing outcomes, emphasizing the significance of accurate prenatal diagnosis to guide appropriate treatments. Challenges persist in addressing both fetal tachycardia and bradycardia, necessitating ongoing exploration of more effective strategies. The evolution of prenatal care has brought advancements, yet a multidisciplinary approach is crucial in navigating existing challenges. Moving forward, the need for multicenter prospective clinical studies is fundamental to shape the future treatment landscape for these uncommon conditions, fostering a more comprehensive understanding and improving outcomes for affected pregnancies.

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Data availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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