



ORIGINAL ARTICLE

The Prevalence of Fetal Complete Heart Block and the Role of Positive Autoantibodies in Pregnant Women with Rheumatologic Diseases

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Abstract

Introduction Complete Heart Block can lead to severe fetal complications. Maternal autoantibodies cross the placenta, increasing the risk of Fetal Complete Heart Block. This study investigates the prevalence of FCHB and the role of these autoantibodies in pregnant women with rheumatologic diseases in Bandar Abbas.

Methods This retrospective, cross-sectional study was conducted in Southern Iran between 2021 and 2023. A total of 99 pregnant women with systemic lupus erythematosus, Sjögren's syndrome, and rheumatoid arthritis, along with 4 cases of fetal bradycardia without maternal rheumatologic history, were referred for echocardiography. Among them, 63 had positive autoantibodies. Prophylactic treatment with hydroxychloroquine and/or prednisolone was administered, and treatment with dexamethasone, IVIG, and beta sympathomimetics was initiated based on echocardiographic findings and the timing of congenital heart block diagnosis.

Results Out of 103 cases, 7 fetuses (0.6%) were diagnosed with congenital heart block. Four women had no prior rheumatologic history, while three had a history of systemic lupus erythematosus and rheumatoid arthritis. Seven women tested positive for autoantibodies. A significant correlation was found between CHB and anti-Ro antibodies ($P=0.017$). Of the cases of fetal CHB, one developed hydrops fetalis, and six had CHB but normal cardiac function at birth.

Conclusion Early diagnosis and timely intervention enhance outcomes in fetal congenital heart block, but many patients still require pacemakers, highlighting the importance of careful monitoring. Advancing risk stratification, exploring novel therapies, and conducting multicenter research are essential for optimizing management and further understanding the impact of maternal autoimmunity.

Keywords Fetal complete heart block · Systemic lupus erythematosus · Sjögren's syndrome · Rheumatoid arthritis

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Introduction

Fetal heart rate typically ranges from 120 to 160

beats per minute, and any deviation or irregular rhythm is considered abnormal [1]. Fetal bradycardia is defined as a heart rate below 110 beats per minute for at least 40 min. The most common form, Complete Heart Block (CHB),

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occurs in 80% of cases, with an incidence of 1 in 15,000 to 20,000 pregnancies. First- and second-degree blocks account for 20% of cases. Structural abnormalities linked to congenital heart defects contribute to 50–55% of CHB cases, while immunologically mediated CHB in structurally normal hearts makes up 40%, with 5–10% of cases having an unknown etiology. Autoimmune-mediated atrioventricular block (AVB) is typically seen in mothers with connective tissue diseases, such as Sjögren's syndrome, systemic lupus erythematosus (SLE), or rheumatoid arthritis, or in those with positive autoantibodies without symptoms [2, 3]. The incidence of CHB in pregnancies with positive anti-SSA (Ro) and anti-SSB (La) test results ranges from 2 to 5%. These autoimmune antibodies are anti-nuclear antibodies that can cross the placenta from maternal circulation to the fetus, inducing inflammatory activities. [4, 5].

Autoantibodies SSA/Ro and SSB/La can be transferred to the fetus as early as week 11, causing damage to the cardiac conduction tissue, leading to inflammation, fibrosis, and calcification of the atrioventricular (AV) node, resulting in conduction block. This inflammatory response significantly reduces fetal heart rate to 50–70 beats per minute [6]. CHB is a life-threatening condition that can cause fetal growth restriction and intrauterine death, with a mortality rate of 20% and a 70% pacemaker requirement. CHB is typically diagnosed via fetal echocardiography between 18 and 24 weeks, though it can present as late as 29 weeks [7, 8].

Recent studies indicate that hydroxychloroquine can reduce the recurrence of fetal complete heart block (FCHB) in mothers with a history of the condition [9]. However, a study of a symptomless patient with positive anti-Ro antibodies, who had complications in two previous pregnancies, showed that FCHB still developed despite early hydroxychloroquine treatment [10]. After adding dexamethasone, the pregnancy remained stable with CHB. This study aims to investigate the prevalence of CHB in fetuses exposed to maternal anti-Ro and anti-La antibodies and examine the impact of anti-inflammatory medications, echocardiographic findings, and fetal outcomes.

Methods

This descriptive, cross-sectional, retrospective study was conducted at Dr. Shariati Hospital in Bandar Abbas (a teaching hospital affiliated with Hormozgan University of Medical Sciences) from 2021 to 2023, approved by the ethics committee.

The study population consisted of three groups: the first group included pregnant mothers diagnosed with connective tissue diseases (SLE, Sjögren's syndrome, and rheumatoid

arthritis). The second group consisted of pregnant women who, during investigations for previous miscarriage causes, tested positive for anti-Ro/La antibodies without any overt connective tissue disease. The final group included cases of fetal bradycardia diagnosed incidentally during routine pregnancy screening. The first two groups were referred by a rheumatologist or perinatologist weekly for fetal echocardiography from 16 to 26 weeks of gestation, while the last group sought confirmation and initiation of treatment at the time of suspected diagnosis. The inclusion criteria for the study were confirmed maternal rheumatologic disease and positive anti-SSA and anti-SSB antibodies, determined by the method of ELISA (Positive > 1.1 AU/ml) and CLIA (Positive > 10 IU/ml).

Maternal data collected included: age, number of previous pregnancies, gestational age at the time of echocardiography initiation or FCHB diagnosis, primary diagnosis of rheumatologic disease, onset of the disease before or during pregnancy, pre-pregnancy medications (hydroxychloroquine, prednisolone) and their dosages, medications for treating fetal complete heart block (dexamethasone, beta sympathomimetics, and IVIG), maternal autoantibody profiles, and results of fetal echocardiography obtained from the maternal medical records. Neonatal data collected included: weight, gender, Apgar score at five minutes, delivery outcomes, and need for a pacemaker obtained from the neonatal medical records.

All echocardiograms were performed by a single pediatric cardiology specialist using a Siemens Juniper model machine via transabdominal approach with a 5–7 MHz convex transducer. The fetal heart was assessed using standard echocardiographic techniques including: two-dimensional echocardiography to measure heart size in relation to the thorax, pulsed Doppler to evaluate atrioventricular valve insufficiency, and heart rhythm assessed by simultaneous monitoring of atrial and ventricular motion in M-mode or through pulsed Doppler using left ventricular input and output signals (MV-AO). The mechanical PR interval (the time between the end of atrial diastole and the beginning of ventricular systole) was measured over two to three cycles, and the average was recorded. If the PR interval exceeded 150 ms, it was considered prolonged (according to guidelines). If there was no relationship between atrial and ventricular contractions in M-mode or Doppler, complete AV block was diagnosed. Hydrops was defined by the presence of effusion in at least two fetal body organs, and endocardial fibroelastosis (EFE) was identified as areas of abnormally increased echogenicity at the endocardial surface of cardiac chambers and valves.

In cases of FCHB, if cardiac function and fetal heart rate were normal and above 50 beats per minute, dexamethasone

was administered at a dosage of 4 mg per day for two weeks, followed by 2 mg daily until the end of pregnancy. If accompanied by pericardial effusion or tricuspid regurgitation, the dexamethasone dosage was increased to 8 mg per day (4 mg twice daily) and gradually reduced to 2 mg daily until the end of pregnancy. In cases where the fetal heart rate was less than 50 beats per minute, a beta sympathomimetic such as terbutaline (5–30 mg per day) was added. Intravenous immunoglobulin was administered at a dosage of 1 g per kilogram of maternal weight every three weeks in patients diagnosed between 16 and 26 weeks.

Statistical Analysis

For data analysis, the normality of the data was assessed using the one-sample Kolmogorov–Smirnov test with the Lilliefors correction. Categorical variables were reported as proportions or percentages, while continuous variables were expressed as mean ($\pm$ standard deviation). The chi-square test was used for analyzing qualitative data. The software used for this research was IBM-SPSS v.23, and a *p*-value of less than 0.05 was considered statistically significant.

Results

From March 2021 to March 2023, a total of 1479 patients were referred to the fetal echocardiography clinic, among which 99 pregnant women were diagnosed with rheumatologic diseases, and 4 cases were referred due to fetal bradycardia detected during routine exams. Table 1 shows the demographic data and rheumatologic disease status (autoantibodies and treatment) in cases with FCHB.

Among the 99 rheumatologic patients, 52 (52.5%) were diagnosed with systemic lupus erythematosus (SLE), 12 (12.1%) with Sjögren's syndrome, and 37 (37.3%) with rheumatoid arthritis. Four pregnant women without a definitive diagnosis were referred for fetal bradycardia. The mothers' ages ranged from 19 to 46 years (mean age 32.5 ± 5.69 years). Of the 103 mothers, 42 (40.7%) tested positive for anti-SSA/Ro antibodies, 15 (14.5%) for anti-SSB/La antibodies, and 6 (5.8%) for both. A significant association was found between congenital heart block (CHB) and anti-Ro antibodies ($P=0.017$).

Among the 63 pregnant women with positive autoantibodies (61.1%), 7 (11.1%) had fetuses with complete heart block (CHB), and 2 cases of sinus bradycardia were observed, which normalized after 2–3 days. Of the patients without fetal CHB, 18 were on hydroxychloroquine alone, 54 on both hydroxychloroquine and prednisolone, and 18 on prednisolone alone before and during pregnancy. Six patients were not on medication. One patient had a mild

Table 1 Demographic data and Rheumatologic disease status (auto antibodies and treatment) in cases with FCHB

Patient number	Maternal Age (year)	Pregnancy status	Rheumatologic disease	Time of diagnosis of Rheumatologic disease	Prednisolone $\pm$ before/during pregnancy	HCQ $\pm$ before/during pregnancy	History of FCHB	Anti Ro/ SSA	Anti La/ SSB	Time of diagnosis of FCHB	DEX	Beta Sympathomimetics	IVIg
1	24	G2Ab1	-	Pregnancy	-/-	-/-	-	+	-	24w + 3d	4 mg	-	+
2	32	G3L2	RA	7Y	Stopped 2y before pregnancy	Stopped 2y before pregnancy	-	+	-	33w + 2d	4 mg	-	-
3	40	G2L1	SLE	5Y	+/+	+/+	-	+	-	34w + 4d	4 mg	-	-
4	35	G5L2Ab1	SLE	8Y	+/+	+/+	-	+	-	29w	8 mg	+ (2 weeks; discontinued due to intolerance)	-
5	28	G2Ab1	-	Pregnancy	-/-	-/-	-	+	-	20w + 1d	4 mg	-	+
6	34	G3L2	-	Pregnancy	-/-	-/-	-	+	-	22w + 2d	-	-	-
7	37	G3L1Ab1	-	Pregnancy	-/-	-/-	+	+	+	28w + 1d	8 mg	+ (3 days; discontinued due to onset of labor pain)	-

HCQ, hydroxychloroquine; DEX, dexamethasone; IVIg, intravenous immunoglobulin; RA, rheumatoid arthritis; SLE, systemic lupus erythematosus

Table 2 Pregnancies outcome with FCHB

Patient number	FHR (bpm)	PE	EFE	Hydrops Fetalis	GA at birth	Birth outcome	BW	Apgar at 5 min	Need for a pacemaker
1	50–52	–	–	–	37w + 1d	Female Live	2900gr	9/10	–
2	50–55	–	–	–	33w + 3d	Female Live	2400gr	8/10	14 month old
3	50–55	–	–	–	34w + 3d	Female Live	2560gr	8/10	–
4	40–45	+ < 5 mm	–	–	37w	Female Live	2900gr	8/10	–
5	50–55	–	–	–	39w + 1d	Female Live	3100gr	9/10	–
6	50–55	–	–	–	39w + 2d	Female Live	2650gr	9/10	18 months old
7	40–45	+ 5 mm	+	+	28w + 4d	Female Dead (6 h after birth)	2000gr	4/10	–

FHR, fetal heart rate; PE, pericardial effusion; EFE, endocardial fibroelastosis, GA, gestational age, BW, birth weight

pericardial effusion, which resolved without affecting heart function. Another case had pericardial effusion, which was treated with dexamethasone and resolved by 28 weeks. Four patients had mild tricuspid regurgitation, with three resolving by weeks 26–28. The mean age of mothers with FCHB was 32.8 years, and the average gestational age at diagnosis was 27 weeks. Among these, two had systemic lupus erythematosus (SLE), one had rheumatoid arthritis, and four had positive autoantibodies without clear symptoms. Six mothers were positive for anti-SSA/Ro and one for both anti-SSA/Ro and anti-SSB/La (Table 2).

FCHB was diagnosed in pregnant women at varying times: three without previous testing or rheumatologic history were diagnosed via ultrasound at 20, 22, and 24 weeks; one patient with rheumatoid arthritis was diagnosed at 33 weeks via NST; one SLE patient was diagnosed at 34 weeks after stopping medication; and one treated with hydroxychloroquine and prednisolone was diagnosed at 29 weeks. Among the seven FCHB cases, one had a previous history of CHB in a child, and fetal hydrops was diagnosed at 28 weeks, coinciding with positive autoantibodies. Treatment with dexamethasone was initiated for mothers with connective tissue disease, while IVIG was administered to three patients diagnosed at 20–24 weeks, though one declined treatment. Terbutaline was prescribed for two patients with a fetal heart rate < 50 bpm, but one discontinued it due to hypotension, and the other for labor pains. Monitoring for gestational diabetes and hypertension showed no new positive cases. The live birth rate for pregnancies with positive autoantibodies was 98.4%, while it was 85.7% for FCHB cases. The average gestational age at birth for CHB newborns was 35 weeks and 4 days, with an average weight of 2640 g. One newborn with fetal hydrops did not survive. The other six had normal cardiac function and heart rates but required pacemakers at 18 and 14 months, while the rest were aged 10 months to 3 years at follow-up.

Discussion

FCHB due to maternal positive autoantibodies is rare but increases the risk of fetal death and long-term complications. This study highlights its prevalence and management in pregnant women with positive autoantibodies, emphasizing the importance of understanding its pathogenesis and therapeutic approaches to reduce risks.

In our study, the prevalence of FCHB in pregnant women with positive autoantibodies was 11%, which differs from previous reports that estimated the prevalence of FCHB to be between 2 and 5%. This discrepancy may be attributed to our smaller study population. [1, 11] The mortality rate in studies has been reported to range from 6 to 20%, with a recent retrospective study noting a mortality rate of 0.3%. In our study, among the 63 pregnancies with positive autoantibodies, one case of death (1.5%) was recorded. [12, 13] The recurrence rate of CHB in this study was reported as 14.2%, which is consistent with other studies that reported recurrence rates of 12–19%. [9, 14] All mothers with FCHB had positive anti-Ro antibodies, and one patient was positive for both anti-La and anti-Ro. This consistency indicates a relationship between autoantibodies and the manifestation of neonatal lupus spectrum disorders, including FCHB. Anti-Ro antibodies are found in 35–50% of patients with lupus and 40–60% of patients with Sjögren's syndrome, while they are observed in lower frequencies in other connective tissue diseases. [15].

Anti-Ro antibodies are found in 1.7% of individuals without a history of systemic autoimmune diseases. The presence of anti-SSA/Ro and anti-SSB/La antibodies during pregnancy increases the risk of fetal heart block. [10, 15] Recent studies have suggested that in addition to maternal antibodies, additional factors may be necessary for the development of heart block. Genetic polymorphisms, including tumor necrosis factor-alpha (TNF α) and transforming

growth factor-beta (TGF β), are significantly more prevalent in children with CHB compared to healthy family members. Recent data indicate that genetic susceptibility factors in the fetus, activation of the innate immune system, type I interferon signaling, systemic inflammatory markers, and cardiac injury are present in fetuses and newborns of mothers with positive Ro/La antibodies. Factors leading to heart block are the result of the convergence of pathogenic factors and heightened sensitivity at specific time points. Changes in the interaction of these factors create variable outcomes in CHB, ranging from transient first-degree block to complete block and extensive cardiac involvement with dilated cardiomyopathy or endocardial fibroelastosis (EFE). [16].

FCHB is a life-threatening condition with no proven treatment, but current therapies aim to reduce myocarditis by administering anti-inflammatory treatments to the mother. Biochemical tests and prolonged PR intervals on echocardiography help assess the risk and diagnose FCHB [17]. Studies show that positive autoantibodies identify women at high risk, and prolonged PR intervals are essential for diagnosing fetal cardiac damage and guiding maternal treatment [7]. Preventive anti-inflammatory medications may protect against FCHB. In our study, the results of preventive anti-inflammatory medication use indicate that maternal treatment with anti-inflammatory agents during pregnancy has a protective effect against congenital heart block development. Only one patient with SLE, who was on a regimen of 200 mg daily hydroxychloroquine and 5 mg daily prednisolone both before and during pregnancy, was diagnosed with FCHB at 29 weeks. A prospective study demonstrated that hydroxychloroquine reduced the recurrence of FCHB in mothers who had experienced fetal complete heart block in previous pregnancies [18].

A case report described a 27-year-old pregnant woman without rheumatologic disease who had positive anti-Ro antibodies and was diagnosed with persistent bradycardia from week 21. She was treated with hydroxychloroquine and prednisolone, and delivery occurred at week 37, resulting in a newborn with CHB who required pacemaker placement on the sixth day after birth [19]. Managing fetal complete heart block in pregnancies with maternal positive autoantibodies is challenging due to the risk of rapid progression and serious complications. Early diagnosis, careful monitoring, and prompt treatment with corticosteroids, immunoglobulin, beta sympathomimetics, along with multidisciplinary care, are crucial for improving outcomes [6, 20].

In fetal echocardiography, an increase in the PR interval of more than 150 ms is considered a reversible biomarker, and weekly monitoring is recommended for pregnancies with positive anti-Ro antibodies from weeks 16–24. Moreover, early diagnosis of cardiac conduction abnormalities and the use of fluorinated steroids in cases of second- and third-degree block is advised due to the potential reversibility of

the damage [7]. Recently, a study reported a symptomless patient with rheumatologic disease and positive anti-Ro antibodies who experienced two complicated pregnancies. Despite prophylactic treatment with hydroxychloroquine starting from week 6 of pregnancy, the fetus developed complete heart block. Dexamethasone was subsequently added to the treatment regimen, but the pregnancy continued with stable CHB, highlighting the ambiguity in managing recurrent fetal CHB in the context of hydroxychloroquine prophylaxis [10].

A case-control study found that combining steroids and IVIG improved cardiac function in fetuses with immune-mediated endocardial fibroelastosis, with favorable outcomes attributed to the anti-inflammatory effects that reduce cardiac tissue damage [21]. However, the adverse effects of fluorinated steroids, such as hypertension, hyperglycemia, weight gain, and risks to the fetus like growth restriction, adrenal insufficiency, and oligohydramnios, should be considered [22]. A systematic review of 890 pregnant women with positive autoantibodies showed 4% perinatal mortality and 12% congenital heart block, confirming adverse pregnancy outcomes linked to positive SSA/Ro antibodies [7]. A retrospective study of 72 CHB patients found that those who received pacemakers had favorable long-term outcomes, with low complications and preserved cardiac function, though dilated cardiomyopathy remains a late concern, highlighting the need for ongoing clinical monitoring [23].

Limitations of this study include its retrospective nature, single-center design, and small sample size. Larger, multi-center studies are essential to further clarify long-term prognosis and guide clinical decision-making.

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Author Contributions NA examined the pregnant women. AD, RMG, and ZE examined the neonates. KHRK was consulted, performed fetal echocardiography and neonatal echocardiography. KHRK collected the data. FA analyzed the data. KHRK wrote the manuscript. SHR and MRKJ revised the manuscript. All authors read and approved the final manuscript.

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Data Availability The data sets used during the current study are available from the corresponding author on reasonable request.

Declarations

Conflict of interest The authors declare that they have no competing interests.

Ethical approval This study was approved by the Ethics Committee of the Hormozgan University of Medical Sciences, Children's Hospital under the code: IR.HUMS.REC. 1402.304.

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