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Original article

Predicting postpartum cardiac events in pregnant women with complete atrioventricular block

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ABSTRACT

Background: Women with complete atrioventricular block (CAVB) can tolerate hemodynamic changes during pregnancy; however, the incidence of cardiac events in women with CAVB may increase after delivery. The aim of this study was to investigate predictive factors for postpartum cardiac events in pregnant women with CAVB.

Methods and results: Pregnant women with CAVB who received perinatal management at a tertiary cardiac center from 1981 to 2015 were retrospectively reviewed. Univariate and multivariate logistic analyses of postpartum cardiac events were performed. Postpartum cardiac event was defined as cardiopulmonary arrest, cardiac failure, or the need for permanent pacemaker implantation (p-PMI) within 3 months after delivery. A total of 63 pregnancies in 36 women with CAVB were included in this study; 25 had undergone p-PMI before pregnancy. Regardless of p-PMI status, women with CAVB had no further increases in heart rate during the second and third trimesters. No heart failure was found during pregnancy and delivery. Postpartum cardiac events occurred in 9 pregnancies (14.3%) in 8 women with CAVB; 3 had cardiac failure and p-PMI, 3 had cardiac failure, 2 required p-PMI, and 1 had cardiopulmonary arrest. Multivariate analysis showed that perinatal ventricular pause (odds ratio 11.60, 95% confidence interval 1.90–82.18, $p < 0.01$) and family history of CAVB (odds ratio 10.59, 95% confidence interval 1.36–90.56, $p = 0.03$) were associated with postpartum cardiac events.

Conclusions: All cardiac events occurred during the postpartum period among women with CAVB, and ventricular pause during the perinatal period and a family history of CAVB were predictors of postpartum cardiac events. Close follow-up should be considered during the postpartum period for women with high-risk CAVB.

Introduction

Complete atrioventricular block (CAVB) is a type of bradyarrhythmia that results from complete absence of atrioventricular conduction [1]. The incidence of congenital CAVB without congenital heart defect (CHD) is approximately 1 in 20,000 births, mainly due to circulating maternal antibodies such as anti-SSA/Ro antibodies [2,3]. Some types of CHD such as heterotaxy and corrected transposition of the great arteries often lead to discontinuity between the atrioventricular node and ventricular conduction tissues, resulting in CAVB [4]. Moreover, CAVB is also known to be a complication of surgery for CHD in approximately 2–3% of patients [5]. CAVB of all causes is irreversible. When patients with CAVB do not have enough cardiac output due to bradycardia, symptoms such as syncope, dizziness, and dyspnea emerge. Notably, sudden death and late-onset dilated cardiomyopathy are also concerns [6,7]. Not all patients with CAVB have hemodynamic instability; however, permanent pacemaker implantation (p-PMI) is required for high-risk patients with CAVB [8].

Some women with CAVB present for the first time during pregnancy or the postpartum period with congestive heart failure

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as a result of the additional hemodynamic burden associated with pregnancy [9,10]. Although p-PMI has become more common in young women with symptomatic CAVB before pregnancy, prophylactic p-PMI in asymptomatic women is not usually indicated. Actually, many pregnant women with CAVB maintain hemodynamic stability; thus, pacing is not always required [9,11]. A previous review demonstrated that most pregnant women with CAVB do not have any specific obstetrical problems during pregnancy and can be safely managed during labor without temporary pacemaker insertion (t-PMI) [12]. Conversely, some women experience postpartum cardiac events and eventually need p-PMI, even when pregnancy and delivery were uneventful [12]. Taken together, most women with CAVB can tolerate hemodynamic changes during pregnancy and delivery; however, the incidence of cardiac events may be higher during the postpartum period. To date, it remains unclear which factors increase the risk for postpartum cardiac events.

In the present study, we investigated predictive factors for postpartum cardiac events in women with CAVB. A single-center retrospective review of pregnant women with CAVB by PMI status was performed.

Materials and methods

A single-center retrospective exploratory study was conducted with institutional review board approval (M28-105). Pregnant women with CAVB who received perinatal management at the National Cerebral and Cardiovascular Center of Japan from 1981 to 2015 were included in this study. Patients with sick sinus syndrome and first or second degree AVB were excluded.

At our center, we assessed heart rate and cardiac function using 12-lead and 24-h Holter electrocardiography and echocardiography once during each trimester. For patients with p-PMI, heart rates were set to from 60 to 70 bpm prior to pregnancy and were increased up to 90 bpm according to their symptoms or degree of cardiomegaly during pregnancy. The protocol during delivery in pregnant women with CAVB from 1997 to 2008 was described in our previous reports [11,12]. Since 2009, intrapartum t-PMI, epidural anesthesia, and mechanical delivery were not routinely used during delivery. We reassessed the heart rate and cardiac function within 1 week and 1 month after delivery.

We collected the following data from medical records: maternal age, parity, etiology of CAVB, family history of CAVB (in parents and siblings), history of syncope, gestational age at delivery, mode of delivery, induction, epidural anesthesia, total blood loss, infant birth weight, umbilical cord pH, heart rates, total heart beats of Holter electrocardiography, presence of ventricular pause, radiographic cardiothoracic ratio (CTR), and echocardiographic data (left ventricular end-diastolic diameter, left ventricular end-systolic diameter, and fractional shortening) from the first trimester to the postpartum period. Perinatal ventricular pause was defined as an increase in the RR interval of 3 s or more from the first trimester to 1 week after delivery [13]. Postpartum cardiac event was defined as cardiopulmonary arrest, congestive cardiac failure, or p-PMI within 3 months after delivery. Congestive cardiac failure was defined as the emergence of symptoms such as dyspnea, dizziness, or pulmonary edema that required diuretic treatment [14]. To investigate heart rate trends during pregnancy and the postpartum period, we compared the data from pregnant women with CAVB with the data from normal pregnancy defined as healthy pregnant woman without maternal and obstetrical complications [15].

Statistical analysis was performed using JMP 11 (SAS Institute, Cary, NC, USA). Data are presented as means ± standard deviation or number of patients. Student's *t*-test was used to compare continuous variables between groups. Categorical variables were evaluated using the chi-square test or Fisher's exact test as appropriate. We also conducted univariate and multivariate logistic analyses. Each cut-off value was calculated using receiver operating characteristic analysis. The best prediction model was selected using stepwise backward elimination with $p \geq 0.10$ as the criterion for exclusion, which was adjusted for baseline variables. Values of $p < 0.05$ were considered significant in all analyses.

Results

Study cohort and baseline characteristics

A total of 63 pregnancies in 36 women with CAVB were enrolled in the present study. Sixteen women became pregnant only once, 12 women experienced pregnancy twice, 7 women 3 times, and 1 woman 4 times. Thirty-two pregnancies in 23 women with CAVB from 1981 to 2008 were described in our previous report [12]. Of 63 pregnancies with CAVB, 25 had undergone p-PMI before pregnancy, 23 had a temporary pacemaker insertion during delivery, and 15 did not need a pacemaker. There were no complications relating to either the pacing systems or the leads such as battery depletion and infection during pregnancy and delivery. No heart failure was found during pregnancy. All pregnancies who were enrolled after the previous report [12] had no trouble during delivery without temporary pacing. Mean heart rate trends during pregnancy and the postpartum period in women with and without CAVB are shown in Fig. 1 [15]. Women with CAVB that did not have p-PMI had significantly higher heart rates during pregnancy and the postpartum period than prior to pregnancy ($p < 0.01$). The largest increase in heart rate in pregnant women with CAVB was only 10 bpm, whereas the increase in heart rate during the third trimester was more than 20 bpm in normal pregnant women [15]. Regardless of the presence or absence of p-PMI, no change in heart rate was observed during pregnancy and the postpartum period in women with CAVB. No differences were found in echocardiographic and radiographic findings based on p-PMI status in women with CAVB (Supplementary Table 1).

Postpartum cardiac events in women with CAVB

Postpartum cardiac events were observed in 9 pregnancies (14.3%) in 8 women with CAVB; 3 of 25 pregnancies with p-PMI

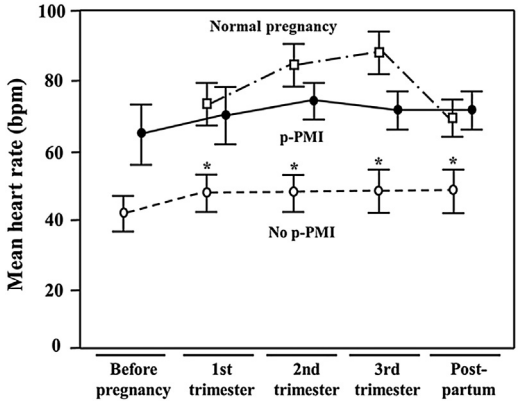

Fig. 1. Trends in mean heart rate during pregnancy and postpartum in women with and without CAVB. Women with CAVB who did not have p-PMI had higher heart rates during pregnancy and the postpartum period than before pregnancy (*$p < 0.01$). However, unlike normal pregnant women without CAVB, further increases in heart rate were not observed during the second and third trimesters in pregnant women with CAVB. Black circles indicate women with CAVB who had p-PMI before and during pregnancy ($n = 25$). White circles indicate women with CAVB who did not have p-PMI ($n = 38$). White squares indicate normal pregnant women [15]. CAVB, complete atrioventricular block; p-PMI, permanent pacemaker implantation.

Table 1
Pregnancies in women with CAVB who experienced postpartum cardiac events ($n = 9$).

Patient	p-PMI before pregnancy	Age during pregnancy, years	Family history of CAVB	Perinatal ventricular pause	Symptoms during pause	Postpartum cardiac event
A	No	26	No	3 s (3rd trimester) 10 s (postpartum)	Syncope	p-PMI
B	No	33	Yes	6 s (postpartum)	None	p-PMI
C (1)	No	34	Yes	No	–	Cardiac failure p-PMI
D	No	26	No	5 s (postpartum)	None	Cardiac failure p-PMI
E	No	38	No	No	–	Cardiac failure p-PMI
F	DDD	29	No	No	–	Cardiac failure
G	VDD	36	No	No	–	Cardiac failure
C (2)	DDD	38	Yes	No	–	Cardiac failure
H	No	36	No	5 s (3rd trimester) 5 s (postpartum)	None	CPA

CAVB, complete atrioventricular block; CPA, cardiac pulmonary arrest; p-PMI, permanent pacemaker implantation.

before pregnancy, and 6 of 38 pregnancies without p-PMI. Of 9 pregnancies with postpartum cardiac events, 3 had cardiac failure and p-PMI, 3 had cardiac failure, 2 required p-PMI, and 1 had cardiopulmonary arrest (Table 1). Two women had a sister with CAVB and p-PMI. Patient A had 3 s of ventricular pause and syncope at 36 weeks of gestation and 10 s of ventricular pause 1 week after delivery; p-PMI was performed 1 month later. Although patient B had no symptoms during pregnancy, 6 s of ventricular pause occurred 1 week after delivery; she underwent p-PMI 2 weeks later. Patient C had p-PMI due to progression of cardiomegaly 2 months after her first pregnancy ended in spontaneous abortion and congestive cardiac failure was observed during the postpartum period of her next pregnancy. Patients D and E had exertional dyspnea and dizziness that began in the third trimester and continued until the postpartum period; thus p-PMI was performed. Patients F and G had decreased left ventricular contraction and developed congestive cardiac failure 3 months and 2 weeks after delivery, respectively. Patient H had cardiopulmonary arrest 1 month after an uneventful elective cesarean delivery and could not be rescued. Five seconds of ventricular pause were detected during the third trimester and ventricular pause was also recorded during the postpartum period. Each ventricular pause occurred during sleep at night and did not accompany any symptoms.

Univariate and multivariate analyses of postpartum cardiac events

We compared the baseline characteristics of pregnancies with CAVB by postpartum cardiac event status (Table 2). Fourteen pregnancies had underlying CHD, including ventricular septal defect, atrial septal defect, pulmonary stenosis, and patent ductus arteriosus. Since there were no cases of complex CHD or cardiomyopathy in this study cohort, all CHDs were included in the subsequent analyses. Maternal age during pregnancy was significantly higher in pregnancies with postpartum cardiac events than those without (32.9 ± 4.4 years vs. 29.0 ± 4.7 years, $p = 0.02$). Family history of CAVB was more common in pregnancies with postpartum cardiac events than those without (33.3% vs. 5.6%, $p = 0.01$). Perinatal ventricular pause was also more common in pregnancies with postpartum cardiac events than those without (44.4% vs. 9.3%, $p = 0.02$). CTR during the first trimester was larger in pregnancies with postpartum cardiac events than those without ($55.7 \pm 3.2\%$ vs. $46.0 \pm 2.6\%$, $p = 0.02$) (Table 3). There were no cases of sustained ventricular tachycardia. No differences were found in echocardiographic and radiographic findings based on postpartum cardiac event status. Multivariate analysis showed that perinatal ventricular pause (odds ratio 11.60, 95% confidence interval 1.90–82.18, $p < 0.01$) and family history of CAVB (odds ratio 10.59, 95% confidence interval 1.36–90.56, $p = 0.03$) were associated with postpartum cardiac events (Table 4).

Discussion

Our study confirmed postpartum cardiac events in 14.3% of pregnancies with CAVB and demonstrated that perinatal ventricular pause and family history of CAVB are associated with postpartum cardiac events in pregnant women with CAVB. We also found specific heart rate trends in women with CAVB; regardless of p-PMI status, they had no further increases in heart rate during the second and third trimesters.

Ventricular pause during the perinatal period could predict postpartum cardiac events such as cardiopulmonary arrest, cardiac failure, and the need for p-PMI. Notably, all perinatal ventricular pause except one were asymptomatic during sleep. We showed heart rate trends during pregnancy and the postpartum period in women with CAVB in this study. Women with CAVB that did not have p-PMI had slightly increased heart rates during pregnancy; however, further increases in heart rate were not observed during the second and third trimesters. To maintain sufficient cardiac output during pregnancy, pregnant women with CAVB had greater increase in stroke volume, with more compensatory enlargement of the heart compared to pregnancy in women without CAVB [9,10]. A previous report showed that grand multiparity was associated with increase in midlife atrial conduction time [16]. The period of pregnancy and the peripartum are characterized by hormonal changes that affect both cardiovascular hemodynamics and adaptive myocardial remodeling [17]. Prolonged increases in stroke volume and enlargement of the heart may develop into cardiomyopathy and conduction disorders and lead to the emergence of ventricular pauses and cardiac events during the postpartum period in women with CAVB [18]. Interestingly, increases in heart rate were not observed during the second and third trimesters even among women with CAVB that had p-PMI. Since left ventricular end-diastolic diameter and CTR during the second and third trimesters were similar based on p-PMI status in women with CAVB, women with p-PMI were also presumed to increase stroke volume with compensatory enlargement of the heart. As a result, congestive cardiac failure during the postpartum period was observed in some women with CAVB that had p-PMI. Some investigators have recommended the use of rate-responsive pacemakers, because the pacing rate can be adjusted as required during pregnancy and delivery [19]. Although not all women with CAVB require pacing, just like physiological heart rate changes observed in normal pregnant women, intensive pacing rate control

Table 2
Baseline characteristics of pregnancies with CAVB by postpartum cardiac event status ($n = 63$).

Variable	No event ($n = 54$)	Postpartum cardiac event ($n = 9$)	p
Age during pregnancy, years	29.0 ± 4.7	32.9 ± 4.4	0.02[a]
Primipara status	24 (49.0)	5 (55.6)	0.92
Congenital heart defect	12 (22.2)	2 (22.2)	1.00
Ventricular septal defect	8	1	
Atrial septal defect	1	0	
Pulmonary stenosis	3	0	
Patent ductus arteriosus	0	1	
Etiology of CAVB			
Maternal antibodies	1 (1.8)	1 (11.1)	0.14
History of surgery for heart disease	7 (13.0)	2 (22.2)	0.46
Unknown	46 (85.2)	6 (66.7)	0.18
Family history of CAVB	3 (5.6)	3 (33.3)	0.01*
History of syncope	6 (11.1)	1 (11.1)	1.00
p-PMI before pregnancy	22 (40.7)	3 (33.3)	1.00
DDD	13	2	
VDD	5	1	
VVI	2	0	
VAT	2	0	
Perinatal ventricular pause	5 (9.3)	4 (44.4)	0.02*
Abortion	8 (14.8)	1 (11.1)	1.00
Gestational age at delivery, weeks[a]	38.4 ± 1.3	36.8 ± 3.9	0.25
Preterm delivery[a]	5/46 (10.9)	1/8 (12.5)	0.89
Mode of delivery[a]			
Vaginal	42/46 (91.3)	6/8 (75.0)	0.21
Cesarean section	4/46 (8.7)	2/8 (25.0)	0.21
Induction of labor[a]	33/46 (71.7)	4/8 (50.0)	0.24
Epidural anesthesia at delivery[a]	33/46 (71.7)	3/8 (37.5)	0.10
Total blood loss, mL[a]	675 ± 431	919 ± 482	0.15
Birth weight, g[a]	2711 ± 484	2437 ± 633	0.26
Umbilical artery pH[a]	7.30 ± 0.08	7.34 ± 0.09	0.54

Data are shown as means ± SD or n (%).
* $p < 0.05$.
[a] Parameters at delivery were analyzed after excluding abortion.
CAVB, complete atrioventricular block; p-PMI, permanent pacemaker implantation.

by the gestational ages may be needed for women with CAVB at high risk for postpartum cardiac events.

A previous investigator reported that pregnancy can be thought of as a physiological stress test, and complications during pregnancy identify women at high risk for subsequent events [20]. Adverse cardiac events during pregnancy are associated with an increased risk of subsequent cardiac events, and women with such events should receive closer surveillance both during pregnancy and after delivery [21]. Our previous report on pregnant women with an implantable cardioverter-defibrillator also showed that additional caution might be required in the postpartum period, as well as during pregnancy and labor [22]. Although unproven, the increase in atrial stretch might be sufficient to provoke conduction disturbance in some previously unaffected women or unmask conduction disturbance in patients with a preexisting substrate or subclinical disease [9,10]. Structural changes related to atrial and ventricular remodeling might also contribute to an increased conduction delay or atrial and ventricular arrhythmia during pregnancy. These changes might also explain the resolution of atrioventricular conduction delay observed during the postpartum period when the physiological changes would be expected to regress. Bradycardia during the postpartum period may be affected by a progressive decrease of sympathetic nervous system activity [23]. Taken together, women with CAVB who have ventricular pauses during the perinatal period are at high risk for postpartum cardiac events, thus close follow-up should be considered. Our findings may suggest that at least once during the third trimester and once again during the early postpartum period, we should perform Holter electrocardiography in CAVB women without p-PMI.

A family history of CAVB was also a predictor of postpartum cardiac events in pregnant women with CAVB. The association between CAVB and genetic factors is poorly understood. Only two distinct forms of familial heart block have been identified. Brink et al. reported a genetic link between progressive CAVB and a genetic locus at chromosome 19q13 [24]. Schott et al. mapped CAVB to chromosome 3p21, where the SCN5A cardiac sodium channel is encoded, and identified two *SCN5A* mutations [25]. In the future, we plan to perform genetic examinations to provide a method for genome-based diagnosis in the families studied and a foundation for cloning studies to identify the causative genes. On the other hand, high levels of maternal anti-SSA antibody are associated with the risk of fetal cardiac complications [2]. Since each woman had a sister with CAVB and p-PMI in this study, the possibility of undetected maternal antibodies involvement could not be denied. Regardless of whether the cause is genetic or acquired, our results suggest that pregnant women with a family history of CAVB should be managed as a group at high risk for postpartum cardiac events.

The strengths of this study are that our institution is one of the largest tertiary cardiac centers and the study cohort included a variety of CAVB with and without p-PMI. We confirmed relatively high incidence of postpartum cardiac events in pregnant women with CAVB, even though no heart failure was found during pregnancy. On the basis of multivariate statistical analysis, we demonstrated for the first time that pregnant women with CAVB who have perinatal ventricular pause and a family history of CAVB are at higher risk for postpartum cardiac events. Furthermore, we showed the trends in heart rate during pregnancy in women with CAVB. We identified specific heart rate trends; regardless of p-PMI

Table 3
Physiological examinations and radiography findings in pregnancies with CAVB by postpartum cardiac event status ($n = 63$).

Variable	No event ($n = 54$)	Postpartum cardiac event ($n = 9$)	p
Holter electrocardiography			
Total HR, 1000 beats/day			
First trimester	82.7 ± 19.2	78.7 ± 25.9	0.76
Second trimester	83.5 ± 23.6	94.5 ± 36.5	0.56
Third trimester	84.4 ± 23.9	74.3 ± 25.5	0.46
Postpartum	86.3 ± 22.5	89.3 ± 28.4	0.82
Mean HR, beats/min			
First trimester	55.4 ± 14.5	54.7 ± 18.5	0.94
Second trimester	59.1 ± 16.9	66.7 ± 26.8	0.54
Third trimester	59.1 ± 17.2	52.5 ± 18.5	0.51
Postpartum	61.2 ± 15.9	62.8 ± 19.9	0.87
Ventricular premature contraction, /day			
First trimester	281.6 ± 682.9	0	0.22
Second trimester	123.8 ± 276.6	4.3 ± 4.9	0.16
Third trimester	77.9 ± 154.8	27.7 ± 44.5	0.30
Postpartum	34.5 ± 46.0	3.0 ± 1.7	0.01*
Ventricular Couplets, /day			
First trimester	0.5 ± 1.1	0	0.18
Second trimester	0.7 ± 1.2	0	0.07
Third trimester	0.8 ± 1.4	1.7 ± 2.9	0.66
Postpartum	0.4 ± 0.7	0	0.06
Echocardiography and chest radiography			
LVDD, mm			
First trimester	50.4 ± 5.9	51.5 ± 4.7	0.70
Second trimester	51.4 ± 5.5	53.3 ± 4.6	0.64
Third trimester	52.5 ± 5.1	53.7 ± 4.8	0.66
Postpartum	51.4 ± 5.4	52.3 ± 4.7	0.73
FS, %			
First trimester	35.4 ± 9.6	38.4 ± 2.7	0.37
Second trimester	35.4 ± 7.4	34.0 ± 0.2	0.52
Third trimester	35.3 ± 7.2	35.2 ± 2.3	0.99
Postpartum	32.5 ± 8.0	31.7 ± 5.6	0.84
CTR, %			
First trimester	46.0 ± 2.6	55.7 ± 3.2	0.02*
Second trimester	52.8 ± 7.9	55.4 ± 6.6	0.82
Third trimester	50.2 ± 7.4	50.6 ± 4.2	0.95
Postpartum	51.1 ± 5.7	49.3 ± 5.5	0.66

Data are shown as means ± SD.
* $p < 0.05$.
CAVB, complete atrioventricular block; CTR, cardiothoracic ratio; FS, fractional shortening; HR, heart rate; LVDD, left ventricular diastolic diameter.

Table 4
Univariate and multivariate analyses of postpartum cardiac events in pregnancies with CAVB.

Variable	Univariate			Multivariate (stepwise)		
	OR	95% CI	p	OR	95% CI	p
Maternal age ≥33 years	4.30	1.04–17.75	0.05			
Epidural anesthesia	0.29	0.07–1.27	0.10			
Perinatal ventricular pause	9.00	1.70–47.60	0.02	11.60	1.90–82.18	<0.01
Family history of CAVB	7.67	1.25–46.96	0.04	10.59	1.36–90.56	0.03

Cut-off values were calculated using receiver operating characteristic analysis. The best prediction model was selected using stepwise backward elimination with $p \geq 0.10$ as the criterion for exclusion, which was adjusted for baseline variables.
CAVB, complete atrioventricular block; CI, confidence interval; OR, odds ratio.

status, no change in heart rate was observed during pregnancy and the postpartum period. Thus, we speculated that these non-physiological heart rate changes during pregnancy might be associated with the relatively high incidence of postpartum cardiac events even among women with CAVB that had p-PMI.

There were several limitations in this exploratory study. First, progression of CAVB may be affected by the etiology; however, unknown etiology of CAVB was the most common in this study cohort, partially because maternal antibodies were rarely detected. Maternal antibodies might not be fully examined. However, a previous nationwide survey of fetal congenital CAVB showed that maternal antibody-positive rate was only 66% in the Japanese population, which was lower than in Caucasians [2,3]. Second, due to the retrospective nature and relatively small sample size, it is difficult to analyze excluding all biases. Notably, p-PMI before pregnancy has the potential to result in a selection bias. Women with CAVB who undergo p-PMI before pregnancy were presumed to be higher risk in baseline, whereas p-PMI might have prevented postpartum cardiac events. Moreover, abortion was also included in this study. However, hemodynamic changes occurred even at the first trimester, and 1 of 8 pregnancies with CAVB ended in abortion had postpartum cardiac events. Third, it was difficult to analyze postpartum cardiac events with complete separation of cardiopulmonary arrest, cardiac failure, and the need for p-PMI, because these events often overlapped. Fourth, there were insufficient clinical data such as plasma natriuretic peptide levels or exercise test to investigate whether these factors can predict postpartum cardiac events. It is possible changes in heart rate

during exercise could mirror some of those seen during pregnancy. Lastly, the oldest data in this study were from approximately 40 years ago, and the management of pregnant women with CAVB has changed over time. However, this study provides relevant information on the single-center experience of a rare condition with a mean follow-up period after p-PMI of 21 years (range, 1–36 years), and we showed no major complications associated with pacemaker during pregnancy and delivery.

Conclusions

All cardiac events occurred during the postpartum period among women with CAVB, and ventricular pause during the perinatal period and a family history of CAVB were predictors of postpartum cardiac events. Close follow-up should be considered during the postpartum period for women with high-risk CAVB. In the future, larger multicenter prospective studies stratified by etiologies of CAVB and p-PMI before pregnancy are needed to clarify whether prophylactic p-PMI before or during pregnancy and intensive pacing rate control can prevent postpartum cardiac events in women with high-risk CAVB.

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Conflict of interest

The authors declare that there is no conflict of interest.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.jjcc.2019.04.002.

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