Original Article

Perinatal and Neonatal Outcomes of the Prenatal Diagnosis of Congenital Heart Disease in Ramathibodi Hospital

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Objective: The primary objective was to determine the incidence and the types of congenital heart disease (CHD), and its association with chromosomal and syndromic abnormalities diagnosed prenatally at Ramathibodi Hospital. The secondary objective was to determine the perinatal outcome and its associated factors.

Materials and Methods: Antenatal care information of pregnant women carrying CHD fetuses including maternal characteristics, ultrasound findings, diagnosis, genetic results, and pregnancy outcomes were recorded retrospectively. After delivery, the neonatal diagnosis was made by a neonatologist, pediatric cardiologist, and geneticist if suspected of genetic or syndromic abnormalities.

Results: Among neonates delivered at Ramathibodi Hospital between January 2013 and December 2017, CHD was diagnosed in 180 pregnancies with the incidence of CHD at 15.62 per 1,000 births. Common diagnoses included a ventricular septal defect, atrioventricular septal defect, and pulmonary stenosis. Genetics studies were performed in 61.18%. The most identified chromosomal abnormalities were trisomy 18 (47.73%), trisomy 21 (20.45%), and monosomy X (13.64%). Delivery outcomes were live birth (55.26%), termination of pregnancy (38.82%), and stillbirth (5.92%). Early neonatal mortality was 226.19 deaths per 1,000 live birth. Chromosomal abnormality in fetuses with CHD varied significantly and associated with the termination of pregnancy and stillbirth while the severity of defects and preterm births notably associated with early neonatal mortality.

Conclusion: CHD is one of the most common congenital defects that affects perinatal and neonatal outcomes, and prenatal diagnosis remains challenging. In case of suspected cardiac defect, accurate ultrasonographic diagnosis of cardiac and extracardiac malformations are fundamental steps in antenatal care. Chromosomal analysis is mandatory whereas genetic laboratory and personnel are available. Current individual data in terms of treatment and prognosis should be carefully discussed. Delivery and neonatal care plan by the multidisciplinary team can provide optimal delivery time and appropriate treatment for CHD neonates.

Keywords: Congenital heart defect, Chromosomal abnormality, Pregnancy outcomes, Neonatal mortality

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Congenital heart disease (CHD) is the most common congenital anomaly worldwide. The incidence is about 4 to 13 per 1,000 livebirth[1], and it is the leading cause of neonatal mortality[2]. In modern obstetrical practice, ultrasonography is generally performed in every pregnant woman[3]. After the diagnosis of CHD, family counseling in terms of prognosis, intrauterine complication, and association with chromosomal or syndromic abnormality should carefully be discussed. However, the research about prenatally diagnosed CHD in Thai pregnant women is limited. The present study aimed to determine the prevalence of the individual type of CHD diagnosed prenatally, associated abnormalities, pregnancy outcomes, and early neonatal mortality. The knowledge might be useful for obstetricians, genetic counselors, and health care providers to understand the many aspects of prenatal diagnosis of CHD and be able to counsel pregnant women and their families thoroughly.

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Materials and Methods

Data of prenatal diagnosis was retrospectively

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Table 1. Demographic data of pregnant women with the prenatal diagnosis of congenital heart disease

Demographic data	Mean±SD (range) or n (%)
Maternal characteristic (n=152)	
Maternal age (years)	31.86±6.45 (18 to 45)
Gestational age at diagnosis (weeks)	23.70±6.10 (13 to 39)
Chromosomal study; n (%)	93 (61.18)
• Chorionic villus sampling	5 (5.37)
• Amniocentesis	51 (54.84)
• Cordocentesis	29 (31.18)
• Postnatal	8 (8.60)
Delivery; n (%)	
• Termination of pregnancy	59 (38.82)
• Livebirth	84 (55.26)
• Stillbirth	9 (5.92)
Fetal characteristic (n=152)	
Sex; n (%)	
• Male	57 (37.50)
• Female	95 (62.50)
Chromosomal abnormality; n (%)	44 (28.95)
• Aneuploidy	41 (93.18)
Trisomy 21	9 (20.45)
Trisomy 18	21 (47.73)
Trisomy 13	3 (6.82)
Monosomy X	6 (13.64)
Trisomy 9	2 (4.55)
• Other	3 (6.82)
Syndromic abnormality; n (%)	7 (4.61)
Gestational age at delivery (weeks)	36.67±3.11 (25 to 40)
Preterm birth; n (%)	32 (34.41)
Birthweight (g)	2571.61±829.24 (600 to 4,505)
Apgar score	
• 1-minute	6.39±2.64 (0 to 9)
• 5-minute	7.75±2.38 (1 to 10)
Early neonatal mortality; n (%)	19 (22.62)

SD=standard deviation

collected between January 2013 and December 2017. After approval by the Committee of Human Rights Related to Research Involving Human Subjects of Faculty of Medicine, Ramathibodi Hospital, Mahidol University, the present study population consisted of pregnant women carrying CHD fetuses. Antenatal care information including maternal characteristics, ultrasound findings, diagnosis, genetic results, pregnancy outcomes, and neonatal outcomes were recorded. The pregnancy outcomes included the termination of pregnancy, which means the therapeutic abortion in pregnancy at less than 24 weeks of gestation, the preterm birth, which means delivery at the gestational age of less than 37 completed weeks; and the stillbirth, which means intrauterine fetal death at gestational age of more than 24 weeks or birth weight of more than 500 grams. Early neonatal mortality means neonatal death within 28 days after delivery. All CHD were diagnosed by maternal fetal medicine specialists. The ultrasound machines used during the study were Voluson E8 (GE Medical Systems, Zipf, Austria) with 2-5 MHz two-dimension convex transducer and 1-7 MHz wide band convex volume transducer. After delivery, the neonatal diagnosis was made by a neonatologist, pediatric cardiologist, and pediatric geneticist if there were suspected genetic or syndromic abnormalities. Patients with incomplete antenatal and delivery records were excluded.

The present study was performed at the referral university hospital. Congenital malformation cases consisted of referral cases from many parts of Thailand and in-house cases from prenatal ultrasound screening in low-risk pregnancy.

Statistical analysis

Demographic data and outcome of pregnancy are shown in Table 1. The incidence of each CHD, associations with chromosomal or syndromic abnormalities and gestational age at detection are presented in Table 2. Additionally, CHDs found in each chromosomal and syndromic abnormalities are shown in Table 3. The information is presented in range, mean with standard deviation, and in number with percentage. In Table 2, gestational age at detection was compared by student t-test. The factors associated with birth outcomes and early neonatal mortality were analyzed by multivariate logistic regression analysis. Stata Statistical Software, version 15 (StataCorp LLC, College Station, TX, USA) was used for statistical analysis in the present study.

Results

Among 11,527 births in the 5-year study period, there were 180 cases of fetus with CHD. The incidence rate was 15.62 per 1,000 births. After excluding 28 cases who lost to follow-up or delivered at other hospitals (Figure 1), the remaining 152 cases were analyzed. Demographic data are shown in Table 1.

The incidence of complex heart defect was 3.12 per 1,000 births. Mean maternal age of the study

Table 2. Congenital heart diseases: type, number, associated abnormality, termination of pregnancy, and gestational age at detection

Type	n	Incidence (/1,000 births)	Association with other CHDs n (%)	Association with a chromosomal or syndromic abnormality n (%)	TOP n (%)	GA at detection (weeks) Mean±SD (range)
Total CHD	152	-	-	51 (33.55)	59 (38.82)	23.70±6.10 (13 to 39)
VSD	54	4.68	40 (74.07)	24 (44.44)	21 (38.88)	23.24±6.17 (16 to 37)
AVSD	24	2.08	10 (41.67)	8 (33.33)	11 (45.83)	23.29±6.40 (18 to 38)
TA/VSD	6	0.35	1 (16.67)	0 (0.00)	0 (0.00)	26.17±4.17 (22 to 31)
TOF or TOF/PA	16	1.39	1 (6.25)	9 (56.25)	7 (43.75)	24.75±6.39 (17 to 37)
DORV	18	1.56	15 (83.33)	6 (33.33)	8 (44.44)	22.78±6.04 (16 to 35)
PS	20	1.73	19 (95)	5 (25.00)	6 (30.00)	25.1±6.26 (16 to 35)
AS	3	0.26	2 (66.67)	1 (33.33)	1 (33.33)	22.33±4.93 (19 to 28)
Coarctation of aorta	7	0.61	4 (57.14)	1 (14.29)	2 (28.57)	24.29±6.47 (15 to 33)
Interrupted aortic arch	3	0.26	2 (66.67)	0 (0.00)	1 (33.33)	23.67±8.50 (24 to 35)
TVD	4	0.35	1 (25.00)	2 (50.00)	3 (75.00)	24.5±7.93 (18 to 36)
Ebstein anomaly	10	0.87	0 (0.00)	0 (0.00)	4 (40.00)	22.8±6.00 (14 to 33)
HLHS	10	0.87	4 (40.00)	7 (70.00)	7 (70.00)	18.2±1.81 (17 to 21)*
TGA	8	0.69	8 (100)	0 (0.00)	3 (37.50)	21.13±1.13 (20 to 23)
Truncus arteriosus	4	0.35	2 (50.00)	0 (0.00)	1 (25.00)	23.75±5.12 (19 to 31)
Abnormal aortic arch	4	0.35	2 (50.00)	0 (0.00)	1 (25.00)	24.25±4.57 (21 to 31)
Dextrocardia	8	0.69	7 (87.50)	1 (12.50)	2 (25.00)	25.13±5.30 (16 to 30)
Cardiomyopathy	20	1.74	3 (15.00)	6 (30.00)	4 (20.00)	26.39±6.66 (18 to 39)

TOP=termination of pregnancy; GA=gestational age; CHD=congenital heart disease; VSD=ventricular septal defect; AVSD=atrioventricular septal defect; TA/VSD=tricuspid dysplasia with VSD; TOF/PA=tetralogy of Fallot with pulmonary atresia; DORV=double outlet of right ventricle; PS=pulmonary stenosis; AS=aortic stenosis; TVD=tricuspid valve dysplasia; HLHS=hypoplastic left heart syndrome; TGA=transposition of the great artery

* p-value less than 0.05 compared with other CHDs

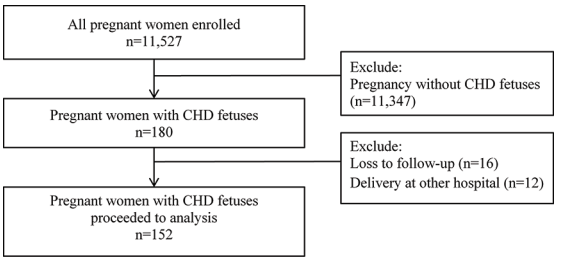

Figure 1. Flow diagram of prenatal congenital heart disease participant in this cohort.

population was 32 years. Average gestational age at diagnosis was 23.7 weeks. About 56% of these chose to have the invasive diagnostic chromosomal study. The type of prenatal procedures mostly performed were amniocentesis (54.84%), cordocentesis (31.18%), and chorionic villus sampling (5.37%), respectively. The postnatal chromosomal study, another option to identify the genetic abnormality in fetus after delivery, was used in eight cases. KaryoLite BACs-on-Beads™ (BoBs™), molecular cytogenetic test using comparative genomic hybridization, was the postnatal genetic test chosen in non-viable fetus.

The chromosomal abnormality was found in 44 cases (29.61%), including 41 cases of aneuploidy and three cases of other chromosomal abnormalities, which were pentasomy X, unbalanced Robertsonian translocation, and chromosomal duplication. The syndromic anomaly was found in seven cases, which were tuberous sclerosis, fetal warfarin syndrome, otopalatodigital syndrome type 2, 22q11 microdeletion syndrome, Noonan syndrome, Rubinstein-Taybi syndrome, and Beckwith-Wiedemann syndrome.

Almost 40% of the pregnancies with fetal CHD chose to terminate the pregnancy while the rest continued until delivery. Ninety percent of the latter group delivered livebirth neonates. Unfortunately, the other 10% ended up in stillbirth. Mean gestational age of delivery was late preterm, nearly 37 weeks of gestation, with mean birth weight of 2,571.61 grams. Among livebirth infant, 22.89% died within

Table 3. Chromosomal and syndromic abnormalities with associated congenital heart disease

Abnormalities	Associated congenital heart diseases	No. of termination of pregnancy n (%)
Trisomy 21 (n=9)	• Cardiomegaly with/without pericardial effusion (n=3) • Aberrant right subclavian artery (n=3) • AVSD (n=1) • PA/IVS (n=1) • HLHS (n=1)	8 (88.89)
Trisomy 18 (n=21)	• Isolated VSD (n=7) • AVSD (n=4) • TOF (n=4) • AVSD with DORV (n=2) • DORV (n=2) • HLHS (n=2)	16 (76.19)
Trisomy 13 (n=3)	• TOF (n=1) • HLHS (n=1) • Isolated VSD (n=1)	3 (100)
Trisomy 9 (n=2)	• TOF (n=1) • AS with VSD (n=1)	2 (100)
Monosomy X (n=6)	• HLHS (n=3) • AS with VSD (n=1) • AVSD (n=1) • Coarctation of aorta (n=1)	6 (100)
49,XXXXX (n=1)	• Isolated VSD (n=1)	1 (100)
45,XX,der(15,18)(q10,q10) (n=1)	• DORV with PS (n=1)	1 (100)
46,XX,dup(8)(p23.1p11.2) (n=1)	• TOF (n=1)	1 (100)
Beckwith-Wiedemann syndrome (n=1)	• Hypertrophic cardiomyopathy (n=1)	0 (0.00)
DiGeorge syndrome (n=1)	• TOF (n=1)	1 (100)
Fetal warfarin syndrome (n=1)	• Isolated VSD (n=1)	1 (100)
Noonan syndrome (n=1)	• TOF (n=1)	1 (100)
Otopalatodigital syndrome type 2 (n=1)	• TVD with PS (n=1)	1 (100)
Rubinstein-Taybi syndrome (n=1)	• TVD (n=1)	0 (0.00)
Tuberous sclerosis (n=1)	• Multiple cardiac rhabdomyoma (n=1)	0 (0.00)

VSD=ventricular septal defect; AVSD=atrioventricular septal defect; PA/IVS=pulmonary atresia with intact ventricular septum; HLHS=hypoplastic left heart syndrome; TOF=tetralogy of Fallot; DORV=double outlet of right ventricle; AS=aortic stenosis; PS=pulmonary stenosis; TVD=tricuspid valve dysplasia

the first month of life. Gender distribution among the population was 95 females (62.5%) and 57 males (37.5%).

After delivery, postnatal echocardiogram or pathological examination was sent to confirm CHD. Ten cases had a normal heart scan after birth. These comprised of three cases of occasional premature atrial contraction (PAC), two cases of coarctation of aorta, two cases of ventricular septal defect (VSD), and two cases of mild pericardial effusion.

The incidence of common CHD is detailed in Table 2. VSD was the most common diagnosis in the present series. The prevalence was 4.68 per 1,000 births, which included VSD with other heart defects. However, the prevalence of isolated VSD was only 1.21 per 1,000 births. Pulmonary stenosis (PS) was highly associated with other CHD resulting in less prevalence of isolated PS, with only 0.09 per 1,000 births. In comparing within isolated CHD, the five most common defects detected prenatally would be atrioventricular septal defect (AVSD), double outlet of right ventricle (DORV), tetralogy of Fallot (TOF), Ebstein anomaly, and hypoplastic left heart syndrome (HLHS), respectively.

Some of the CHDs are strongly associated with a chromosomal or syndromic abnormality such as

HLHS, TOF, and tricuspid valve dysplasia (TVD). While atrial septal defect, tricuspid dysplasia with VSD (TA with VSD), interrupted aortic arch, Ebstein anomaly, transposition of the great artery (TGA), truncus arteriosus (TA), and abnormal aortic arch did not have any association in this population. The details of common aneuploidy and type of CHD are shown in Table 3. Fetal cardiomyopathy, the disorder of diseased and malfunctioned myometrium, had a prevalence of 1.74 per 1,000 births. Besides genetic abnormality (27.78%), other common etiologies were infant of diabetic mother (IDM) and fetal anemia.

Option to terminate the pregnancy was discussed with family after the diagnosis of poor prognosis CHD, which included CHD with a genetic disorder, CHD with multiple anomalies, or severe type CHD. Most family of the fetus with TVD and HLHS chose termination of the pregnancy, while most of TA with VSD, TA, and abnormal aortic arch decided to continue.

Aneuploidy is the most common chromosomal abnormalities presented as an abnormal number of chromosomes, either an extra (trisomy) or missing of chromosome (monosomy). Fetuses with aneuploidy typically have multi-organ anomalies such as heart, brain, and kidney. In the present study, the types of cardiac abnormalities mostly found in trisomy 21 were cardiomegaly with or without hydrops fetalis (33.33%) and aberrant right subclavian artery (ARSA) (33.33%). Isolated VSD was found in one-third of trisomy 18, followed by AVSD with or without DORV (28.57%) and TOF (19.05%). Mainly cardiac finding in monosomy X was left-sided abnormality (83.33%), whether it is HLHS (50%), coarctation of the aorta (16.67%), or aortic stenosis (16.67%).

Discussion

In the era of high-resolution ultrasonography, prenatal diagnosis of a fetal anomaly by ultrasound is an important part of antenatal care. According to the guideline for fetal anatomy scan, all pregnant women should be assessed between 18 to 22 weeks of gestation[3]. In terms of cardiac anomaly screening, four-chamber view, heart size, position, left and right ventricular outflow tract, three-vessel view, and three-vessel trachea views should be obtained[4]. Average gestational age in the diagnosis of CHD was 23.7 weeks, which is almost two weeks later than the recommendation. Referral time from the outside hospital may explain this gap.

Mean gestational age of detection of HLHS was significantly lower than others. Ebstein anomaly was the only defect found since the first trimester in the present study. Both could be apparently detected from the four-chamber view, a principle view for cardiac scan that was easily obtained in all fetal position[5]. HLHS is a left-sided cardiac abnormality that shows ventricular disproportion. Hypoplasia and non-functioning left side of the heart is key to diagnosis[6]. Ebstein anomaly is suspected when tricuspid valve displacement is observed. It may accompany cardiomegaly, marked right atrial dilatation, and holosystolic tricuspid valve regurgitation[7]. There is a difference from the other studies[8], our AVSD cases were recognized quite late. However, the gestational ages of detection in conotruncal defects were comparable. The mean gestational age of detection was almost 24 weeks of gestation. This could raise the concern for complex CHD that considers termination of pregnancy because the procedure should be done before 24 weeks of gestation according to in-house policy.

In contrast, the gestational age in the diagnosis of cardiomyopathy was approximate 26 completed weeks, which is comparable with other studies[9,10]. Besides aneuploidy, many of the non-genetic, extracardiac factors can affect the size and function of the heart. Described in a previous study[11], anemia from hemoglobin Bart's disease and hereditary red cell membrane defect, high cardiac output condition in chorioangioma, metabolic caused from maternal diabetes mellitus, Beckwith-Wiedemann syndrome, non-compaction of left ventricle, and idiopathic cause[10] were also found to be etiologies of fetal cardiomyopathy in this study as well.

Almost 40% of CHD proceeded to termination of pregnancy. The majority of these (72.88%) were fetuses with chromosomal or syndromic abnormality. After the diagnosis of CHD was made, prompt scanning for extracardiac abnormality is recommended[12]. Appropriate and available genetic testing should be discussed with the family based on current knowledge of the association between cardiac and genetic disease. The present study showed that advanced maternal age had a strong association with a chromosomal abnormality (adjusted odds ratio [aOR] 5.14, 95% CI 2.38 to 11.07), but not with the syndromic abnormality (aOR 0.25, 95% CI 0.03 to 2.43). Complex CHD was defined as a combination of more than one cardiac defect[13] and did not increase the risk of chromosomal abnormality (aOR 0.43, 95% CI 0.27 to 1.74).

The significant association between chromosomal abnormalities and termination of pregnancy (aOR

Table 4. Multiple logistic regression analysis of factors affected birth outcomes in pregnancy with CHD fetuses

Variables	TOP (n=59)				Preterm birth (n=32)				Stillbirth (n=9)			
	n	aOR	95% CI	p-value	n	aOR	95% CI	p-value	n	aOR	95% CI	p-value
Chromosomal abnormality (n=44)	36	15.79	6.11 to 40.80	<0.001	4	2.95	0.58 to 14.83	0.19	4	24.07	3.54 to 163.54	0.001
Syndromic abnormality (n=7)	3	3.32	0.65 to 17.00	0.15	1	0.77	0.07 to 9.13	0.84	N/A			
Complex CHD (n=36)	13	1.05	0.40 to 2.74	0.92	10	1.46	0.54 to 3.90	0.45	3	1.51	0.28 to 8.26	0.63

aOR=adjusted odds ratio, also adjusted for advanced maternal age and fetal gender; CI=confidence interval; CHD=congenital heart disease; N/A=not available

Table 5. Multiple logistic regression analysis of factors for early neonatal mortality in CHD neonates (n=19)

Variables	n	aOR	95% CI	p-value
Advanced maternal age	8	3.53	0.91 to 13.78	0.28
Syndromic abnormality	1	3.65	0.22 to 60.04	0.34
Male neonatal gender	6	0.44	0.11 to 1.75	0.12
Complex heart disease	8	4.07	1.05 to 15.82	0.04
Preterm birth	12	9.65	2.66 to 35.01	0.001

aOR=adjusted odds ratio; CI=confidence interval

15.79, 95% CI 6.11 to 40.80) is shown in Table 4. It could be interpreted that genetic information is an influencing factor for choosing to discontinue their pregnancy. For those who continue their pregnancy, genetic aberrations also significantly increased the risk of stillbirth (aOR 24.07, 95% CI 3.54 to 163.54) but not for preterm birth (aOR 2.95, 95% CI 0.58 to 14.83). Cytogenetic testing generally used in the present study was G-banding chromosome analysis to detect numerical and many structural abnormalities. For conotruncal heart defect, 22q11 microdeletion diagnosis by fluorescent in situ hybridization is recommended[14] and was tested substantially in the present study. Interestingly, the prevalence of DiGeorge syndrome (about 1 in 12,000 births) is lower than in other report[15].

The cohort of the fetus with complex CHD did not associate with termination of pregnancy (aOR 1.05, 95% CI 0.40 to 2.74), preterm birth (aOR 1.46, 95% CI 0.55 to 3.90), or stillbirth rate (aOR 1.51, 95% CI 0.40 to 2.74). However, early neonatal mortality of complex CHD group was significantly more than isolated heart disease (aOR 4.07, 95% CI 1.05 to 15.82) in the neonatal period as shown on Table 5. Delivery is a critical transition period of a fetus. After birth, some types of CHD need immediate intervention such as in ductal dependent pulmonary stenosis and intravenous prostaglandin E2 should be started as soon as possible[16]. Despite intensive care, these patients still have a relatively high early neonatal mortality rate at 226.19 deaths per 1,000 live birth.

Several types of heart disease have high right atrial pressure that could cause hydrops fetalis or tachyarrhythmia and might follow by intrauterine fetal demise[17]. These include Ebstein anomaly and TA with VSD. The pathophysiology of hydrops fetalis is increased hydrostatic pressure of the precordial venous system and low cardiac output leading to hypoxia and increase capillary permeability[18]. Beside of abnormal chromosome and multiple anomaly fetuses, stillbirths in the present study had Ebstein anomaly, cardiomyopathy with hydrops fetalis, and AVSD.

Apparently, preterm birth was a significant factor influencing the rate of early neonatal mortality. Preterm birth can lead to multiple complications including low birth weight, respiratory distress syndrome, intraventricular hemorrhage, and necrotizing enterocolitis[19]. These might be a direct cause of death or indirectly caused by complications of intervention. Without indication for preterm birth, congenital heart fetus should deliver at term. However, some types of severe CHD may progress in utero. Result in hydrops fetalis or non-reassuring fetal status could be the indication for urgent delivery.

Limitation of the present study was the selected population. The data were compared among the fetuses with CHD, so relative risk of these variables compared to pregnancy with normal fetus was not obtained. In further study, perinatal outcomes between CHD fetuses and normal fetuses should be scrutinized. Moreover, the genetic testing used in the present study might affect the study outcomes. Nowadays microarray-based comparative genomic hybridization (aCGH) is the recommended genetic test for fetal anomaly[20], while in the present study conventional karyotype and FISH for 22q11 microdeletion were mainly used because aCGH was not available and unaffordable for patients.

Conclusion

CHD is one of the most common congenital

defects that affects perinatal and neonatal outcomes. Prenatal diagnosis remains challenging. In case of suspected cardiac defect, detailed scanning for accurate diagnosis and extracardiac malformation are fundamental steps in antenatal care. Chromosome analysis is mandatory if genetic laboratory and personnel are available. Current individual data in term of treatment and prognosis should be carefully discussed. Delivery and neonatal care plan by a multidisciplinary team can provide optimal delivery time and appropriate treatment to CHD neonates.

What is already known on this topic?

- The incidence of CHD and complex CHD in Thailand.

- Common chromosomal and syndromic abnormalities associated with CHDs are trisomy 21, trisomy 18, trisomy 13, Turner syndrome, and 22q11 microdeletion syndrome.

What this study adds?

- The pregnancy outcomes include termination of pregnancy, preterm birth, stillbirth, livebirth, birth weight, and neonatal mortality of pregnancy with CHD fetuses in tertiary care hospital in Thailand.

- Chromosome abnormality in CHD fetuses increased the rate of termination of pregnancy and stillbirth.

- Among these patients, neonatal mortality was influenced by preterm birth and severity of CHD.

Conflicts of interest

The authors declare no conflict of interest.

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