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Original Research Article

Incidence of congenital anomalies in newborns born to mothers with heart disease

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ABSTRACT

Background: Heart disease is seen in 1% pregnancies. Prior studies have either looked at only the prevalence of congenital heart diseases (CHD) in newborns or the congenital anomalies (CA) in babies born to those with chronic medical disorders as a whole. There is only sparse literature looking specifically at their prevalence in mothers with heart disease.
Methods: We aimed to study incidence of CAs in newborns born to mothers with heart disease and to study other relevant outcomes like prematurity, low birth weight and intrauterine growth retardation which have significant associations with CAs. The present study was a descriptive study consisting of retrospective and prospective data and consisted of pregnant women with heart disease from 2006 to 2010 and their newborns.
Results: 55 pregnant women with their 55 newborns were selected. Among the 55 newborns, 15% babies had congenital anomalies including CHD, 13% had only CHDs. 21% babies with CHDs were born to CHD mothers and none of the mother baby diagnosis was completely concordant. No statistically significant outcome of CA in newborns born to mothers with heart disease was found. No statistically significant associations among low birth weight, prematurity and CAs was found.
Conclusions: 15% had congenital anomalies including CHD. No statistically significant outcome of CA in newborns born to mothers with heart disease was found. Since the study population comprised of a small sample size, significant derivations could not be made. Further studies are required in this direction to see if associations are significant.

Keywords: Cardiac anomalies, Congenital anomalies, Congenital heart disease, Heart disease, Pregnancy outcome

INTRODUCTION

Congenital Anomalies (CAs) are significant causes of childhood deaths and disabilities, especially in developing countries.[1] They include structural, functional or metabolic anomalies that originate during intrauterine life and which can interfere with body functions and result from defective embryogenesis or intrinsic abnormalities in development process.[2] The general prevalence of CAs varies from 1.9% of live births to 2.7%.[3,4] They account for 13-16% of neonatal deaths in India.[5] They are classified as Major and Minor and as Single and Multiple defects.[6] Most common CAs are that of cardiovascular system, Central nervous system and Musculoskeletal.[7-9] Most common syndrome associated with these anomalies is Down`s Syndrome.[2] Congenital heart diseases (CHDs) are structural CAs of heart and intrathoracic vessels seen in 5-15 per 1000 live births.[10] Most common cause of mortality due to CAs is usually due to a major cardiac anomaly.[10]

Prior studies have either looked at only the prevalence of CHDs in newborns or the CAs in babies born to those with chronic medical disorders as a whole. There is only sparse literature looking specifically at the prevalence of all CAs in mothers with heart disease. So the present

study was undertaken and looked from a pediatrician's perspective on all the CAs in newborns born to mothers with heart disease. Though the study was undertaken with a small population, it is nevertheless the first of its kind by a pediatrician and we hope to extend our study to a large base too.

The incidence of CAs in mothers with heart disease is reported to be 2% and that of CHDs 10-12%.[11-13] Causes can be manifold like advanced cardiac conditions with risk factors, mothers on certain drugs, mothers who have undergone infertility treatment, advanced age of mothers, malnourished mothers, increased risk of infections, increased risk of obstetric complications and bad obstetric history of the mothers.[10-18] Heart diseases like CHD and rheumatic heart disease (RHD) complicate approximately 1% of all pregnancies.[14] They influence the outcome of pregnancy apart from being a major cause of maternal mortality especially in developing nations.[15,16] In the present era with scientific advancement, more and more women are surviving with fewer adverse outcomes.[15]

Earlier studies have shown a high feto-maternal morbidity in mothers with certain cardiac risk factors such as those with cyanosis, New York Heart Association (NYHA) 3/4, prior cardiac events like arrhythmias, stroke, failure, severe myocardial dysfunction and severe outflow obstruction.[15-18]

The incidence of severe valvular diseases (of rheumatic origin) in pregnancy also has been declining in the subcontinent because of widespread use of penicillin group of drugs and early surgeries.[19] Nevertheless, the outcome of those newborns in developing nations is gloomy compared to their western counterparts because of various environmental and genetic factors.[19] There is an increased risk of recurrence of CHDs in subsequent babies born to CHD mothers.[20] There is also a direct association between prematurity and CAs, birth defects being twice as common among preterms while 8% preterms had CAs.[21]

The present study was conducted at a referral cardiac centre with good obstetric and neonatal set up and these pregnant women with cardiac complications were followed up regularly. Owing to the same reason, the study population is a heterogeneous one with cases comprising of mothers with low and high risk cardiac disease, mothers on certain drugs and mothers who have undergone surgery.

METHODS

Aims

Primary objective was to study the incidence of CAs in newborns born to mothers with heart disease. Secondary objective was to study other relevant outcomes like prematurity, low birth weight LBW and intrauterine growth retardation IUGR which have significant associations with CAs and also to study the outcomes of CAs between operated and unoperated cases, those with or without drugs, those with or without cardiac risk factors, those with CHD or RHD, those with cyanotic and acyanotic CHDs and those in whom cardiac disease was detected early and those in whom it was detected during present pregnancy.

The present study was a descriptive study consisting of retrospective and prospective data. It was conducted in a multispeciality hospital that included a cardiac centre too.

Inclusion criteria

The inclusion criteria consisted of pregnant women with heart disease (congenital heart disease and rheumatic heart disease) enrolled at Narayana Multispeciality Hospital in Bangalore from 2006 to 2010 and their newborns who were all born by operative mode of delivery.

Exclusion criteria

Those with isolated conduction defects and cardiomyopathies were excluded.

Hence out of the total 70 cases, after exclusion, only 55 pregnant women were selected with their 55 newborns. The study received institutional ethics committee approval. Informed consent was obtained from patients before data collection. Investigations were carried out as per the hospital protocol. The retrospective study design consisted of old patient records with obstetric and cardiac details including echocardiography reports. Prospectively each pregnant woman was monitored from the first antenatal visit to the delivery regularly by obstetricians and cardiologists and a cross sectional study of the neonates was done with examination just after birth.

The following baseline characteristics were noted in all pregnant women: age, parity, weight, antenatal booking status, bad obstetric history (BOH), type of cardiac lesion, presence or absence of cardiac risk factors like prior cardiac events like stroke, arrhythmias, failure, presence of cyanosis, New York Heart Association NYHA 3/4, severe outflow tract obstruction like area of mitral valve less than 2 cm^2, aortic valve less than 1.5 cm^2 and left ventricular peak outflow gradient of more than 30 mmHg, severe myocardial dysfunction like left ventricular ejection fraction (LVEF) <40%, original cardiac status including echocardiography findings (fetal echocardiography was done for those mothers with CHD and those on anticoagulation warfarin which was concordant with neonatal echocardiography), cardiac status at pregnancy, use of cardiac medications including beta blockers, diuretics, warfarin/heparin, angiotensin converting enzyme inhibitors (ACEI), use of drugs for obstetric comorbidities like insulin, methyl dopa, progesterone and cardiac surgeries like valve

repairs and replacements, complications during pregnancy, presence of obstetric risk factors, mode of anaesthesia, and karyotyping and genetic counseling if sought (for bad obstetric history, syndromic facies, previous baby with CAs) and outcome of mothers in the form of survival or death.

Other associated relevant adverse events like low birth weight (LBW), [extremely low birth weight (ELBW) and very low birth weight (VLBW) included], prematurity, small for gestational age, (SGA) or intrauterine growth retardation (IUGR). Neonatal deaths were also noted.

These neonatal outcomes were compared between groups of newborns born to mothers with and without cardiac risk factors, born to mothers with RHD and CHD, born to cyanotic and acyanotic CHD, born to mothers with operated and unoperated heart disease and between those born to mothers with and without cardiac drugs.

SPSS v 17 software was used for statistical analysis. Descriptive analysis was used to summarize baseline characteristics of the study population using mean and standard deviation for continuous variables and proportion and percentage for ordinal variables. Chi Square test of proportion was used to assess the statistical significance of difference in proportions between the dependent neonatal outcomes and independent maternal risk factors. Multiple logistic regression was used to assess the relation between neonatal outcomes and maternal factors. Statistical level of significance was fixed at $p<0.05$.

RESULTS

Neonatal

Among the 55 newborns, 8 babies (15%) had congenital anomalies including CHD, 7 (13%) had only CHDs, 2 babies had both cardiac and non-cardiac anomalies. Most common CHD was patent ductus arteriosus (PDA) (4, 50%) followed by ventricular septal defect (VSD) (3, 37.5%) and atrial septal defect (ASD) (3, 37.5%) as single or combined defect (Table 1). Of the 7 CHDs, 5 were born to CHD mothers (21%) and none of the mother baby diagnosis was completely concordant but 3 were partially concordant (60%) (ASD, VSD, PDA) and 2 were born to RHD mothers of which 1 mother was on anticoagulation warfarin/heparin for valve replacement but did not have fetal warfarin syndrome as warfarin was switched to heparin at 6th week gestational age.

A total 5 were girls, 3 were boys. Among the 8 babies, 4 were early preterm; 1 was 27 weeker, 3 were 28-32 weeks, (50%) and rest were full term. There were 7 low birth weight babies (LBWs<2.5 kg), of which 4 were due to prematurity (1 ELBW 750 g, 1 VLBW 1.05 kg, 2 LBWs 1.2 and 1.3 kg) and 3 due to intrauterine growth restriction (IUGR) (2-2.5 kg) (Table 4). Among a total of 25 LBWs, 7 had CAs (28%); among 15 preterms, 4 had CAs (27%) and among a total of 18 IUGRs, 3 had CAs (17%). Though prematurity was independently a significant outcome of sorts (p=0.01), its association with CAs was not significant and also no significant associations between LBWs and CAs were found.

Table 1: Neonatal congenital anomalies.

Baby CA	Maternal heart disease	Maternal age	Parity	Maternal RHD/CHD
ASD with PDA	CoA with VSD (operated before)	36 years	Primi	CHD
Dextrocardia with VSD	Mitral valve stenosis (operated before pregnancy)	32 years	Primi	RHD
ASD VSD PDA	Mitral valve regurgitation repaired before pregnancy	34 years	Primi	RHD
PDA	CoA with VSD (repaired)	37 years	Primi	CHD
VSD with digeorge syndrome with bilateral cataract with cleft lip and palate	Truncus with VSD with pulmonary arterial hypertension PAH (repaired)	25 years	Second gravida	CHD
CoA with PDA with ambiguous genitalia	PDA with Eisenmenger disease	24 years	Primi	CHD
PUV	severe MS moderate MR mild AR	23 years	Second	RHD
PDA With ASD	Complete AVSD with ASD with VSD with TGA with PS	21 years	Second	Complex CHD

There were 2 neonatal deaths of which 1 had CA (50%). One of the babies who died within the neonatal period was a preterm AGA 27 weeks VLBW baby (1.03 kg) with congenital heart disease PDA with severe coarctation of aorta (CoA) with ambiguous genitalia with karyotyping 46XY, normal male type internal genitalia, normal electrolytes and BP, awaiting enzyme levels, developed culture positive sepsis and died of multiorgan dysfunction syndrome. He was also on prostaglandin E1 infusion for CoA. 1 baby with VSD had hypoparathyroidism and hypocalcemia with karyotyping suggestive of Di George syndrome, chromosome 22q.11

micro deletion and had bilateral congenital cataract for which excision was done. Genetic counselling was done.

A 1 baby had only CA [posterior urethral valves (PUV)]. His renal profile was deranged. He was operated soon after.

There was no statistically significant difference in the outcomes between operated and unoperated cases p=0.37 and 0.22, those with or without drugs p=0.33 and 0.54, those with or without cardiac risk factors p=0.06 and 0.14, those with CHD or RHD p=0.43 and 0.21, those with cyanotic and acyanotic CHDs p=0.65 and 0.53 and those in whom cardiac disease was detected early and those in whom it was detected during present pregnancy p=0.55 and 0.50.

Maternal

Among the 8 mothers with heart diseases, 5 (63%) had CHD and the remaining 3 (38%) had RHD. 2 were >35 years, 4 in between 20 to 25 years and 2 were between 25 to 35 years.

None of them had taken infertility treatment. 5 were primiparous and 3 were multiparous. There were none who had previous anomalous babies (Table 1).

A total 7 were old patients and 1 was newly diagnosed who also died. Only 1 among these had BOH (recurrent abortions and 1 still birth) and she died along with her baby who also died in early neonatal period. 1 among these 8 died due to shock. She was a case of PDA with Eisenmenger disease with severe pulmonary arterial hypertension (PAH), with 2 cardiac risk factors, who had severe pregnancy induced hypertension (PIH) too. Only 3 mothers had obstetric comorbidities like PIH, oligohydramnios, premature rupture of membranes (PROM) and gestational diabetes mellitus (GDM). 3 of the mothers had cardiac complications in pregnancy like congestive cardiac failure, atrial fibrillation, PAH, thrombosis (Table 3).

Table 2: Maternal charachteristics.

Baby CA	Maternal heart disease	Maternal cardiac risk factors- Y/N	Maternal cardiac drugs- Y/N	Maternal cardiac surgery- Y/N
ASD with PDA	CoA with VSD (operated before)	N	Y- beta blockers, ACEI	Y- CoA repaired VSD closed
Dextrocardia with VSD	Mitral valve stenosis (operated before pregnancy)	N	Y- warfarin, heparin	Y- mitral valve replacement
ASD VSD PDA	Mitral valve regurgitation repaired before pregnancy	N	N	Y-mitral valve repair
PDA	CoA with VSD (repaired)	N	Beta blockers	Y-CoA Repair with VSD closure
VSD with DiGeorge syndrome with bilateral cataract with cleft lip and palate	Truncus with VSD with pulmonary arterial hypertension PAH (repaired)	Y-failure, NYHA 4	Diuretics, ACEI	Y-truncus repaired VSD closed
CoA with pda with ambiguous genitalia	PDA with Eisenmenger disease	Y-cyanosis, NYHA 4	Diuretics, ACEI	N
PUV	Severe MS moderate MR mild AR	Y-arrhythmia, severe mitral valve obstruction, NYHA 4	Diuretics, ACEI, warfarin, heparin, beta blockers	N
ASD with PDA	Complete AVSD with ASD with VSD with TGA with PS	Y- arrhythmia, cyanosis, NYHA 4	Diuretics, ACEI	N

Y- yes, N- no

4 had cardiac risk factors of which 2 had more than 2 risk factors and 4 had no cardiac risk factors. Stepping up of NYHA class from 1/2 to 3/4 was seen in 1 mother and stepping down of NYHA class from 3/4 to 1/2 in 2 mothers (Table 2).

A total 5 were operated and 3 cases were unoperated for their cardiac disease. Only 1 mother was not on any drugs while 7 mothers were on ≥1 drug. Only 1 was on single drug while the rest were on >2 drugs. 2 were on warfarin. 1 for her mitral valve replacement and another for thrombosis, both switched to heparin at 6 weeks post

menstrual age which was changed back to warfarin at 13 weeks till around term and then later after a month. 3 were on beta blockers, 4 on diuretics, 5 on ACEI, 1 on progesterone for PROM, 3 on Methyldopa and 1 on Insulin for PIH and GDM respectively (Table 2). None of the mothers were on any known teratogens or alcohol. There was no consanguinity or family history of CAs in anybody. 4 mothers were malnourished with BM1<17. None had any other medical condition including hypertension or diabetes mellitus from before. All mothers underwent caesarean section on spinal anesthesia (5) or general anesthesia (4).

Table 3: Maternal characteristics.

Baby CA	Maternal heart disease	Obstetric comorbidities	Cardiac complications	Other drugs
ASD with PDA	CoA with VSD (operated before)	N	N	N
Dextrocardia with VSD	Mitral valve stenosis (operated before pregnancy)	N	N	N
ASD VSD PDA	Mitral valve regurgitation repaired before pregnancy	N	N	N
PDA	CoA with VSD (repaired)	N	N	N
VSD with DiGeorge syndrome (bilateral cataract with bilateral cleft lip and palate)	Truncus with VSD with pulmonary arterial hypertension PAH (repaired)	PIH	Y -PAH	Methyl dopa
CoA with PDA With ambiguous genitalia	PDA with Eisenmenger disease	PIH, GDM, BOH, PROM, oligohydramnios	Y- PAH, cardiogenic shock	Methyl dopa, progesterone and insulin
PUV	Severe MS moderate MR mild AR	PIH, oligohydramnios	Y -AF, thrombosis	Methyl dopa
PDA with ASD	Complete AVSD with ASD with VSD with TGA with PS	N	N	N

Y- yes, N- no

Table 4: Outcomes.

Baby CA	Baby sex	LBW -Y/N	Prematurity	IUGR	Neonatal death
ASD with PDA	F	Y- 2.2 kg	N	Y	N
Dextrocardia with VSD	F	Y- 2.4 kg	N	Y	N
PDA ASD VSD	F	Y- 2.7 kg	N	N	N
PDA	F	2.3 kg	N	Y	N
VSD with DiGeorge syndrome (bilateral cataract with cleft lip and palate)	M	2.3 kg	Y	N	N
CoA with PDA with ambiguous genitalia	M	1 kg	Y	N	Y
PUV	M	1.6 kg	Y	N	N
PDA with ASD	F	2.2 kg	Y	N	N

Y- yes, N- no

DISCUSSION

In our study of 55 pregnancies, 15% had CAs whereas in other studies it was 2%.[11] This relatively higher incidence of CAs as compared to other studies is seen mainly because of the small sample size. Similarly, the incidence of CHDs in newborns in this study was 13% which is concordant with other studies which showed 10-14%.[12,13] No statistically significant outcome of CA in newborns born to mothers with heart disease was found probably due to small sample size. Other studies have also shown similar outcome.[11,12,15,16]

Among the 8 babies, 5 were boys (63%) with slight male preponderance as in other studies.[11-13] 4 were early preterm (50%) and there were 7 low birth weight babies (LBWs<2.5 kg) (87.5%), of which 4 were due to prematurity (1 ELBW 750 gm, 1 VLBW 1.05 kg, 2 LBWs 1.2 and 1.3 kg) and 3 due to intrauterine growth restriction IUGR (2-2.5 kg) (37.5%). Among a total of 25 LBWs, 7 had CAs (28%) and among 15 preterms, 4 had CAs (27%) and among a total of 18 IUGRs, 3 had CAs (17%). Other studies have shown significant relationship between prematurity/IUGR with CAs, birth defects being twice as common among preterms while 8% preterms had

CAs.[21] Though prematurity was independently a significant outcome of sorts p=0.01 as in other studies too its association with CAs was not significant and also no significant associations between LBWs and CAs were found.[10-12,15,16] Our study showed higher percent because of small size. There were 2 neonatal deaths of which 1 had CA with critical CHD, CoA with PDA, was on PGE1 but the cause of death was due to prematurity, VLBW and sepsis. The baby was in cardiac intensive care unit managed by pediatric cardiologists and intensivists. Except this baby none had life threatening malformation/ CHD.

Of the 7 CHDs, 5 were born to CHD mothers (21%) and none of the mother baby diagnosis was completely concordant but 3 were partially concordant (60%) as against 21% complete and 20% partial concordance with ASD/VSD/PDA in few studies.[22] The small sample size and our centre being a tertiary referral cardiac centre with good follow up of all the mothers during their pregnancy must have improved the condition of the mothers thereby reducing their cardiac risk factors.

This is in concordance with the hereditary transmission of CHD of probable single gene origin. In other studies, incidence of CHD with maternal CHD ranged from 10-14%.[12,14,22] Most common CHD was PDA (4, 50%) followed by VSD (3, 37.5%) and ASD (3, 37.5%) as single or combined defect, whereas other studies showed VSD and ASD as the most common subtypes.[22] The prevalence of CHD in mothers of children with CHD was 19.7%.[22] This slightly higher percentage was probably because of the smaller study population. This association of maternal and fetal CHD can be attributed to single gene autosomal dominant inheritance or multifactorial inheritance.[22] There is no association quoted between maternal RHD and fetal CHD in literature. The fetal CHDs in cases with maternal RHD might be due to multifactorial inheritance.

Karyotyping was done on those babies born to mothers with bad obstetric history and those with suspected chromosomal syndromes. So here it was done for 2. Among them, one baby born to mother with bad obstetric history. This baby also had ambiguous genitalia who also had a major life threatening cardiac defect of CoA with PDA but died due to sepsis on day 5 of life. Mother was not on any teratogenic drugs. Investigations revealed normal enzyme levels required in the synthesis of sex hormones and karyotyping showed normal 46XY. Another baby born to mother with truncus repair who had bilateral congenital cataract and hypocalcemia due to hypoparathyroidism was diagnosed to have chromosome 22q.11 micro deletion (Di-George syndrome). Chromosome 22q11.23 micro deletion is one of the common microdeletion syndromes inherited as an autosomal dominant disorder with variable clinical expression. Reported incidence of 22q11.23 micro deletion is 1 in 4000 to 1 in 6000 live births. Most of the cases are sporadic. Familial inheritance is seen in 5-10% of all cases.[22] Genetic counselling was done for both of them to prevent similar outcome in next pregnancies.

The changes during pregnancy can result in complications in the mother which may have neonatal implications. Prior studies have focused on the risk stratification of women with cardiac disease so that they receive appropriate care and counselling.[11,15] These risk factors are prior events like arrhythmias, stroke, failure; presence of cyanosis, severe valvular outflow obstruction, NYHA 3 or 4 and severe myocardial dysfunction in the form of less than 40% ejection fraction.[11-15]

Hemodynamic compromise secondary to valvular stenosis and the resulting decrease in uterine blood flow are probable explanations for the high incidence of impaired intrauterine fetal growth seen in this study of patients with valvular heart disease.[10-16] Maternal arrhythmias have been shown to cause fetal distress and may be an additional mechanism leading to compromised uterine blood flow and fetal outcome.[11,12] Maternal oxygen saturation is inversely proportional to neonatal birth weight; hence cyanosis causes increased incidence of IUGR, LBW, prematurity. The neonatal outcome was significantly adverse in terms of LBW, prematurity, IUGR in those born to mothers with cardiac risk factors.[15,16] Since the birth defects are closely associated with LBW and prematurity, these can also be the likely causes of high incidence of CAs here. Prior studies have reported even worse perinatal outcomes in those babies born to mothers with cardiac and obstetric risk factors but due to our study population being small, significant derivations could not be made plus ours being a tertiary referral cardiac centre, we attempted to reduce the risk factors like stepping down of maternal NYHA class and addressing every major or minor problem judiciously with good follow up.[15] Drug dispensing was supervised too thus eliminating recall bias.

In our study, there was no statistically significant difference in the outcomes between operated and unoperated cases. This is in agreement with other studies.[11,12,15,16,23] This could partly be because of their post-operative cardiac status being compromised as a result of their original high risk cardiac condition. cardioactive drugs including diuretics, ACEI and beta blockers have been associated with impairment of uterine blood and in turn LBW, SGA and prematurity but not with CAs.[24-26] Our study did not show significant results of association of CAs with any of these drugs as did other such studies.[24-26]

There was no difference in the neonatal outcome among newborns born to mothers with RHD and CHD groups as in other studies also between mothers with cyanotic and acyanotic heart disease groups and those with or without cardiac risk factors.[11,12,15,16] The reason for this could probably be attributed to the study population being low and also to good cardiac care of the neonates. On the contrary, earlier studies have shown that outcome in

acyanotic heart diseases was better than in cyanotic heart diseases and also outcome in those with cardiac risk factors was worse than without.[15,16,23]

The study population was small but still it was the first of its kind study with complete data. Hence significant associations might not have been derived. This research was descriptive in nature with no intervention.

CONCLUSION

Our study showed 15% association of CAs and 13% CHDs with maternal heart disease. The cause for 21% babies with CHD being born to mothers with CHD could be single gene defects, chromosomal defects or environmental causes. There is a need to monitor them antenatally as early as they miss periods so that they can be advised to switch over to safer medications soon after missed periods, planned pregnancies with periconceptional FA especially in one with BOH, step down their NYHA class, judiciously manage cardiac and non-cardiac complications and improve their nutrition. Mothers with newly diagnosed disease needs a detailed cardiac examination and ECHO if any abnormality found on their first antenatal visit becomes essential. The need for neonatal USG/brain USG to detect all possible CAs and fetal followed by neonatal ECHO in those with maternal CHDs and on drugs and also karyotyping in those with BOH and those with recurrence of CAs go a long way in genetic counselling, antenatal diagnosis and their prevention. We look forward to doing similar study in a large population base to see if significant exposures could be expected with respect to CAs independently or CAs with prematurity and LBW.

Therefore, this study contributes a paediatrician's bird's eye view on the subject and reiterates that a multidisciplinary approach with cardiologist, obstetrician, neonatologist, geneticist is required to manage and counsel the mothers with cardiac disease for antepartum surveillance.

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