

Outcome of fetuses with heart disease diagnosed in utero

Marianne Eronen

Abstract

Objective—To review the outcomes of 193 fetuses with cardiac abnormalities detected by echocardiography.

Methods—A total of 422 fetuses between 16 and 41 gestational weeks, referred to paediatric cardiologists for detailed echocardiography, were included in this study.

Results—Structural heart defects were found in 55 (28%), isolated arrhythmia in 105 (54%), and other non-structural abnormalities (dilated cardiomyopathy, hypertrophic cardiomyopathy, aneurysm of the foramen ovale, isolated pericardial effusion or echogenic foci) in 33 (17%) of 193 fetuses. Total mortality was 26%. The prognosis was poor in fetuses with structural heart defects; 37 of 55 cases (67%) died in utero or postnatally. Chromosomal abnormality was associated with structural heart defect in 38% of fetuses, of whom 38% died. Among fetuses with isolated arrhythmia survival was 95%. Poor outcome was associated with complete heart block (n=14) in 2 (14%) fetuses with hydrops and heart rate of less than 55 per minute, and with supraventricular tachycardia (n=21) in three (14%) neonates delivered prematurely at a mean gestational age of 33 weeks. Furthermore, nine of 12 fetuses (75%) with structural heart defects and arrhythmia died. Among fetuses with non-structural cardiac abnormalities, survival was 73%. Poor outcome was evident in fetuses with dilated cardiomyopathy in eight of 13 (62%) and with hypertrophic cardiomyopathy in one of eight (13%) of cases.

Conclusions—Factors associated with a poor prognosis were: structural heart defect associated with chromosomal abnormality or arrhythmia, congestive heart failure associated with supraventricular tachycardia or complete heart block, especially if delivery occurs preterm; and fetal hydrops with congestive heart failure and atrioventricular valve regurgitation.

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Keywords: Echocardiography; outcome; arrhythmia; structural heart defects

Echocardiography is the only imaging modality available for diagnosis of structural heart defect, arrhythmias, and abnormal cardiac haemodynamics in the fetus.¹⁻⁵ In trained hands, fetal echocardiography can now detect most congenital heart lesions at 16-20 weeks.⁵⁻⁶

Notable exceptions are the atrial septal defect, because of the extreme thinness and the presence of the patent foramen ovale of the atrial septum in the fetus, and the arterial duct, which is a normal finding. Some defects can also evolve or progress during fetal life, making a definitive diagnosis early in gestation difficult.⁷ Prenatal diagnosis not only allows for planned management of the heart disease prenatally and postnatally, but also allows the families to consider the option of termination of pregnancy for severe defects.

Echocardiographic assessment of fetal arrhythmias is usually accomplished using M-mode and Doppler techniques. Echocardiography not only establishes the kind of arrhythmia present but can also identify associated structural and functional heart disease as well. Fetal congestive heart failure is diagnosed when tissue perfusion of the fetus is inadequate, characterised by cardiomegaly, atrioventricular valve regurgitation, and abnormal venous velocities.⁸ The presence of fetal congestive heart failure is also characterised by fetal hydrops in association with either tachycardia or bradyarrhythmia, and results in a very poor prognosis.⁹⁻¹³

Methods

The study group comprised 422 fetuses between 16 and 41 weeks gestational age that had been referred to a paediatric cardiologist for detailed echocardiology between January 1983 and December 1995 at the Children's hospital, University of Helsinki. The indications for referral were: a strong family history of congenital heart disease; fetal heart arrhythmia; abnormal four-chamber view; extracardiac malformation; fetal hydrops; and other conditions in which the risk of cardiac abnormality was increased—for example, maternal diabetes, chromosomal abnormality, or cardiac teratogen.

The fetal heart was examined using two dimensional guided, pulsed, and continuous wave Doppler equipment (Advanced Technology Laboratories 600 MK, Toshiba SSH 65 A and Acuson 128 Color Doppler system) with a 3.5 MHz or 5 MHz transducer. Two dimensional imaging was obtained from five approaches including four-chamber, five-chamber, long axis, short axis and arch views. The studies were judged to be of adequate diagnostic quality if the following were defined: cardiac rhythm; cardiac situs; ventricular and atrial chambers; valves; and the aortic and pulmonary arterial connections to the heart and aortic and ductal arches. Colour and pulsed or

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Table 1 Indications for fetal echocardiography and results

Indication	No (%) of fetuses (n=422)	No (%) of fetuses with abnormal condition (n=193)	
		Structural congenital heart disease	Non-structural cardiac abnormality
Family history	145 (34)	3 (2)	5 (3)
Previous child	126		
Maternal	15		
Other	4		
Arrhythmia	144 (34)	7 (5)	106 (74)
Abnormal four-chamber view	40 (9)	28 (70)	2 (5)
Fetal hydrops	17 (4)	1 (6)	9 (53)
Maternal diabetes	17 (4)	0 (0)	5 (29)
Extracardiac malformations	12 (3)	4 (33)	2 (17)
Chromosomal abnormality	10 (2)	6 (60)	0 (0)
Small for gestational age	8 (2)	3 (38)	0 (0)
Pregnancy induced hypertension	7 (2)	0 (0)	0 (0)
Polyhydramnios	7 (2)	1 (14)	3 (43)
Miscellaneous	15 (4)	2 (13)	6 (40)
Twin-twin transfusion	8	0	4
Oligohydramnios	1	1	0
Thoracopagus	1	1	0
Acardiac fetus	1	0	1
Maternal hyperthyroidism	1	0	0
Exposure to ketoprofen	1	0	0
Exposure to lithium	1	0	0
Maternal HIV, antiviral medication	1	0	1
Total	422	55 (13)	138 (33)

continuous wave Doppler echocardiography were carried out when a clinically significant structural defect was suspected by imaging. The type of arrhythmia was analysed using an M-mode echocardiogram at the atrial and ventricular levels. The non-structural cardiac abnormalities were identified as dilated cardiomyopathy, hypertrophic cardiomyopathy, aneurysm of the foramen ovale, and isolated pericardial effusion or echogenic foci. All studies were performed using the standard transabdominal approach. The abnormal studies were recorded on videotape for subsequent playback and analysis.

Personal data were recorded for each mother. After counselling, she returned to the referring hospital for further care unless it was felt that the fetus would benefit from ongoing combined cardiac/obstetric management, in which case, management was continued at the Children's Hospital. The progress and outcome of each pregnancy (including those where no abnormality was detected) were followed up, and where appropriate, post mortem examinations were performed. In most of these cases the heart was examined at the Children's Hospital. Follow up lasted 12 months.

Results

Among the 422 fetuses examined by a paediatric cardiologist, 193 (46%) had cardiac abnormalities. Of the 40 mothers referred because of the obstetrician suspecting a structural heart defect, 28 (70%) were confirmed as abnormal. The reasons for the referral and the findings are shown in table 1.

The commonest indications for referral were a family history of complete heart disease (34%) and arrhythmias (34%). Three fetuses (2%) scanned for family history were found to have structural heart defects. All cases were siblings. Of the 34 families with a history of left heart hypoplasia (LHH), one (3%) fetus had the same defect. Five mothers also had fetuses with arrhythmia (three with premature atrial contractions and two with sinus bradycardia).

Among 55 fetuses with structural heart defect, poor outcome was seen in 37 cases (67%). The type of heart defect, presence or absence of chromosomal abnormality, treatment and outcome of the affected fetuses are described in table 2. Total fetal mortality was 15%; four fetuses were aborted and four fetuses died in utero. Twenty nine of the 55 (53%) died neonatally (table 2). A total of 12

Table 2 Structural fetal heart disease, treatment, and outcome

Diagnosis	No (%) of patients	Chromosomal abnormality	Fetal death (aborted)	Neonatal death	Operated		
					< 1 month of age	1-3 months of age	> 3 months of age
Ventricular septal defect	12 (22)	11	3 (1)	6	0	1	2
Left heart hypoplasia	11 (20)	2	1 (1)	10	0	0	0
Atrioventriculoseptal defect	8 (15)	5	1 (1)	0	1	1	5
Univentricular heart	7 (13)	1	2 (1)	3	2	0	0
Double outlet right ventricle	3 (5)	1	0	2	2	0	0
Truncus arteriosus	2 (4)	1	1	1	0	0	0
Tetralogy of Fallot	2 (4)	0	0	0	0	0	2
Intracardiac tumour	2 (4)	0	0	1	1	0	0
Pulmonary stenosis	2 (4)	0	0	1	1	0	0
Ectopia cordis	1 (2)	0	0	1	1	0	0
Single heart, conjoined twins	1 (2)	0	0	1	0	0	0
Ebstein's anomaly	1 (2)	0	0	1	0	0	0
Endocardial fibroelastosis	1 (2)	0	0	1	0	0	0
Mitral valve dysplasia	1 (2)	0	0	1	0	0	0
Atrial septal defect	1 (2)	0	0	0	0	0	1
Total	55	21	8 (4)	29	8	2	10

Table 3 Fetal arrhythmias, frequency of treatment, and outcome

Type of arrhythmia	No (%) of patients (n=125)	Drug treatment			Structural heart disease	Deaths	
		In utero	Postnatally	Pacemaker		Fetal	Neonatal
Premature atrial contractions	68 (54)	0	4	0	3	0	2
Supraventricular tachycardia	21 (17)	18	16	0	0	0	3
Atrioventricular block	18 (14)	2	5	13	4	2	3
Sinus bradycardia	8 (6)	0	0	0	2	1	1
Premature ventricular contractions	6 (5)	0	3	0	3	1	1
Sinus tachycardia	4 (3)	0	0	0	0	0	0
Total	125	20	28	13	12	4	10

Table 4 Non-structural fetal heart abnormalities: aetiology and outcome

Abnormality	No (%) of patients	Aetiology (n=)	Deaths	
			Fetal (aborted)	Neonatal
Dilated cardiomyopathy	13 (39)	Hydrops fetalis (5)		
		Maternal anaphylactic reaction (1)	1	
		Renal agenesis (1)		1
		Non-immunogenic (1)		1
		Rhimmunisation (1)		
		Pulmonary hypoplasia (1)		
		Pulmonary adenomatous malformation (1)	1 (1)	
		Twin-twin transfusion syndrome (1)		
		Polyhydramnios, exposure to indomethacin (1)		
		Perinatal infection (1)		1
		Chorionangioma (1)		1
		Arteriovenous fistula (1)		1
		Sacrococcygeal teratoma (1)		1
Hypertrophic cardiomyopathy	8 (24)	Acardiac fetus (1)		
		Maternal diabetes (5)		
		Twin-twin transfusion syndrome (1)		
Aneurysm of the foramen ovale	6 (18)	Non-immunogenic hydrops (1)		1
		Polyhydramnios, duodenal atresia (1)		
Isolated pericardial effusion	4 (12)	Unknown aetiology, arrhythmia (6)		
		Twin-twin transfusion syndrome (2)		
Echogenic foci	2 (2)	Maternal HIV, antiviral medication (1)		
		Non-immunogenic hydrops (1)		
Total	33	Unknown aetiology (2)	2	7

infants came to surgery at the age of 1 to 12 months with uncomplicated follow up. Chromosomal abnormality was detected in 38%; trisomy 18 (n=10), trisomy 21 (n=6), trisomy 9 (n=1), trisomy 13 (n=1), trisomy 22 (n=1), 2-P trisomy 21 (n=1) and 3 B trisomy 2 (n=1). A diagnosis of ventricular septal defect (VSD) or atrioventriculoseptal defect (AVSD) was particularly predictive of chromosomal abnormality (P=0.004). The largest group of infants who died neonatally were those with LHH (34%). During the study period no medical or surgical intervention was attempted in these infants.

Of the eight fetuses who died in utero, four (50%) were submitted to post mortem examination, including all terminations of pregnancy. Of the 29 fetuses who died as neonates, 26 (90%) were examined post mortem. The predicted structural abnormality was correct in 28 fetuses (93%) and partly correct in two fetuses (double outlet right ventricle and univentricular heart). Of the 229 fetal examinations with normal findings, two (0.9%) minor abnormalities were not diagnosed (ASD and a small VSD). Both survived. No false positive diagnoses were made.

Of the 144 fetuses referred for cardiac arrhythmia, 106 (74%) had arrhythmia. However, among the whole study population (n=422), fetal arrhythmia was detected in 125 (30%) fetuses (table 3). The most commonly noted arrhythmia was premature atrial contractions (54% of cases). Of these, poor outcome was evident in two fetuses (3%) with a

structural heart defect (one fetus with trisomy 13 and VSD and one fetus with Ebstein's anomaly). Five fetuses with aneurysm of the foramen ovale had a good outcome.

Supraventricular tachycardia (SVT) was detected in 17% of fetuses with arrhythmia with a median gestational age of 32 weeks (range 26 to 37 weeks). None of these fetuses had structural heart defects; one had aneurysm of the foramen ovale. Eighteen (86%) of 21 fetuses were treated with antiarrhythmic medication in utero: digoxin (n=8), digoxin combined with propranolol (n=4), combined triple route (intravenous, intraperitoneal, and transplacental) administration of amiodarone (n=2), digoxin combined with flecainide (n=2), digoxin combined with quinidine (n=1) and digoxin combined with verapamil (n=1). There were no intrauterine deaths. However, three infants delivered prematurely by caesarean section died neonatally (14%). The causes of death were cardiac failure accompanied by severe respiratory distress in two and severe cardiac failure in one. The mean gestational age at birth in the neonates with poor outcomes was significantly lower than in those with a good outcome (33 weeks *vs* 37 weeks; P=0.025). Of the 18 survivors, 15 (83%) were given antiarrhythmic medication postnatally.

Among 18 fetuses with complete heart block, structural heart defects were found in four (22%); two fetuses with hydrops were treated with digoxin in utero. Poor outcome was evident in five cases (28%); two fetuses

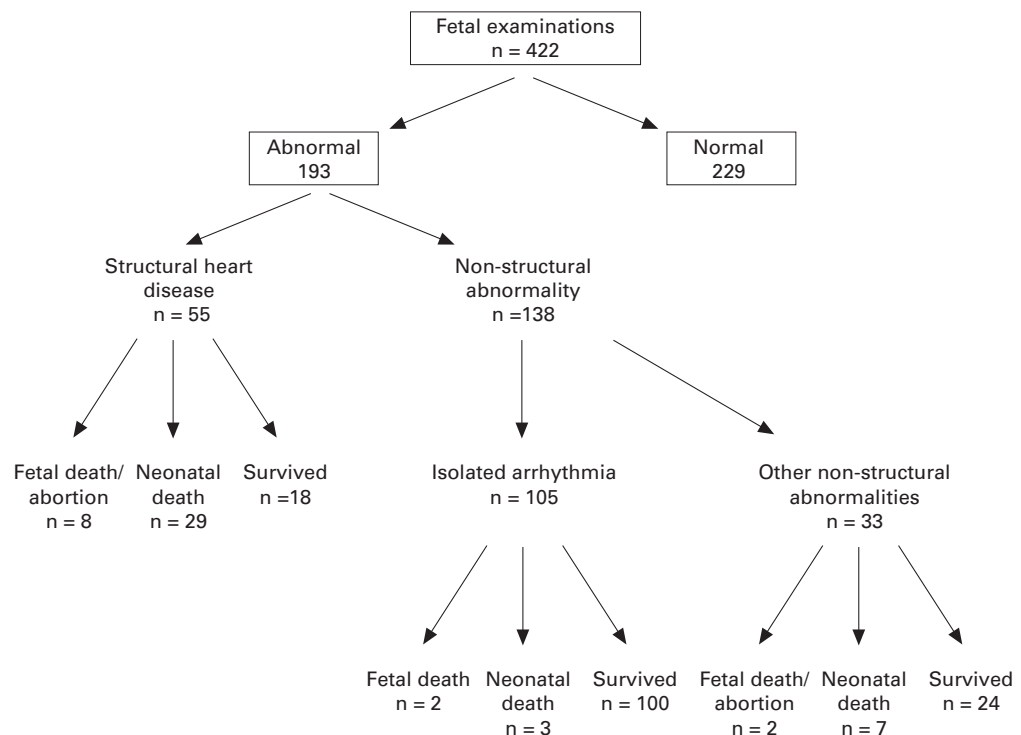


Figure 1 Outcome of 193 fetuses with heart disease diagnosed in utero.

(both with normal hearts and fetal ventricular rates of 38 to 55 per minute) and three neonates with structural heart disease died. Pacemakers were implanted in 13 of 16 (81%) neonatal survivors. The criteria for pacemaker insertion were structural heart defect or a heart rate equal to or less than 70 beats per minute.

Sinus bradycardia (heart rate of less than 100 beats per minute) was associated with poor outcome in two cases (25%) with structural heart defect. Two of six fetuses (33%) with premature ventricular contractions died; both had trisomy 18 accompanied by structural heart defects (VSD and LHH). One neonate with intracardiac myxoma in the right atrium was successfully operated on. Fetuses with sinus tachycardia (heart rate of more than 180 beats per minute) had a good prognosis.

Non-structural fetal heart abnormalities, aetiology, and outcome are presented in table 4. Congestive heart failure with dilatation of one or both ventricles, combined with atrioventricular valve regurgitation, was associated with poor outcome in eight cases (62%). Of 17 fetuses referred for maternal diabetes, five (29%) had left ventricular wall or septal hypertrophy; none had structural heart defect. One infant with non-immunogenic hydrops and severe left ventricular hypertrophy died after birth in respiratory distress syndrome. Of four pregnancies with twin-twin transfusion syndrome, two fetuses had isolated pericardial effusion, one dilated cardiomyopathy and one hypertrophic cardiomyopathy. All survived; one twin infant needed several pericardial punctures after birth. Two fetuses with echogenic foci located within the chordae tendineae in left ventricle had uncomplicated follow up.

The outcome of the whole study population is presented in fig 1.

Discussion

In this series of 193 fetuses with abnormal echocardiography poor outcome was evident in 26%, of whom 3% were aborted. Structural heart disease diagnosed in utero had the poorest prognosis; 67% of fetuses died in utero or postnatally. Chromosomal abnormality was associated with structural heart defect in 38% of fetuses, in 92% of fetuses with VSD, in 63% of fetuses with AVSD and in 18% of fetuses with LHH. A diagnosis of VSD or AVSD was therefore particularly predictive of chromosomal abnormality. In this study 38% of fetuses with an abnormal karyotype died. These findings confirm those of previous studies^{14,15} and suggest that chromosomal evaluation of any patient with an abnormal fetal echocardiogram, regardless of the gestational age, should be carried out. Knowledge of the fetal karyotype permits well defined postnatal surgical intervention that may need to be adjusted if an abnormal karyotype is present.

Antenatal diagnosis of heart disease leads to earlier recognition of treatable abnormalities which might otherwise not be recognised until after the onset of symptoms or until death.¹⁶ When chromosomal abnormalities were excluded, all fetuses with Fallot's tetralogy or univentricular heart (UVH) and 50% of fetuses with double outlet right ventricle or intracardiac tumour survived surgery. Furthermore, 88% of fetuses with AVSD, including those with trisomy 21, survived heart surgery. However, LHH was associated with a mortality of 100%, because no active intervention was attempted in these newborn infants during the study period.

The incidence of 2% structural heart defect in the same family is similar or lower than data reported by others.^{4,17,18} Although the yield of

abnormal results was low in this study group, the emotional benefit to the family of finding that their baby has a normal fetal heart, or of being prepared for delivery of a baby with an abnormal condition, is a valid reason for the fetal echocardiography.

In previous series about 15% of referrals have been for abnormalities of cardiac rhythm.^{4,17} In this study population fetal arrhythmia was detected in 30% of fetuses. As in previous reports,^{1,19,20} the most commonly noted arrhythmias were premature atrial contractions affecting 54% of fetuses with arrhythmia. Several studies have shown that premature atrial contractions are self-limiting and carry a benign prognosis.^{19,21} However, in this study 4% had concomitant structural heart defect accounting for a total mortality of 3% of fetuses with premature atrial contractions. Furthermore, 50% of fetuses with premature ventricular contractions had structural heart defects. In light of these findings, a full fetal echocardiographic evaluation should be undertaken in all patients with premature atrial or ventricular contractions.

In several studies fetal complete heart block accompanied by structural heart disease has been associated with poor prognosis.¹⁰⁻¹² The mortality of 75% in our series confirms this finding. The structural heart diseases associated with poor prognosis in this series were mitral valve dysplasia, LHH, and UVH (double inlet right ventricle). Two fetuses with isolated complete heart block died. Both had bradycardia of less than 55 beats/min. This finding is similar to that of a recent study¹³ and proves that isolated complete heart block does not always have a good prognosis.

Fetuses with SVT accompanied by cardiac failure and hydrops have been treated with a variety of different pharmacological agents.²²⁻²⁶ In this series, a total of 86% of fetuses with SVT were treated with antiarrhythmic medication in utero. However, there were three neonatal deaths (14%); all were delivered prematurely by caesarean section because of cardiac failure. Those who survived had a significantly higher gestational age at delivery. This agrees with previous studies which indicate that in utero treatment for SVT is preferable to premature delivery, especially if the lungs are immature.^{1,9,22}

A recent study showed that the prognosis is poor in fetuses with ascites, when cardiomegaly is also detected in the presence of atrioventricular valve regurgitation.²⁷ In this study congestive heart failure with cardiomegaly was associated with a mortality of 62%. In addition, four fetuses with twin-twin transfusion syndrome had decreased cardiac function or pericardial effusion and 29% of fetuses referred because of maternal insulin dependent diabetes had left ventricular wall or septal hypertrophy.^{28,29} These findings suggest that all mothers with at risk pregnancies should receive a thorough fetal echocardiographic study to evaluate heart structure and function.³⁰⁻³³

In conclusion, whenever fetal echocardiography shows a structural heart defect, the presence of chromosomal abnormality and

extracardiac malformation must be sought. When structural heart defect is associated with an abnormal karyotype, with the exception of 21-trisomy, fetal and neonatal mortality are high. When cardiac arrhythmia is associated with structural heart defect or congestive heart failure, direct or indirect medication of the fetus must be administered as effectively as possible to avoid premature delivery. Fetuses with severe hydrops associated with congestive heart failure and atrioventricular valve regurgitation have a poor prognosis. When a prenatal diagnosis of structural, rhythmic, or functional abnormality is obtained the healthcare team can outline a management strategy to maximise the care and support given to the fetus, mother, and family.

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