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Antenatal women with history of cardiac disease: Maternal and perinatal outcome

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Abstract
The overall prevalence heart disease complicating pregnancy varies from 0.3-3.5%. With a decrease in maternal death as a result of the classic causes of haemorrhage, hypertension and infection, the relative incidence of cardiac disease has increased1.Cardiac disease in pregnant women can present challenges in cardiovascular and maternal-fetal management. During the last few decades, the etiology of heart disease in developed countries has changed from primarily rheumatic to predominantly congenital. The present study was conducted among the antenatal women admitted in the department of OBG with a previously diagnosed cardiac disease or diagnosed after admission during index pregnancy. The mothers and the babies were followed up. Maternal outcome was defined as discharged or died. The perinatal outcome was defined in terms of mortality, presence of congenital heart disease or other anomalies in the offspring. In our study percentage of LSCS is around 37%, which is almost similar to most of the studies. There is significantly high percentage of maternal mortality compared to other studies. In our study there is comparatively higher mortality rate among peripartum cardiomyopathy cases, reasons may be delay in detection, lack of available resources like ICU facilities, precipitating factors like anemia, PIH etc.

Keywords: Cardiac disease, maternal and perinatal outcome

Introduction
Pregnancy causes profound physiologic changes in the cardiovascular system. A series of adaptive mechanisms are activated as early as 5 weeks gestation to maximize oxygen delivery to maternal and foetal tissues. In most women, these physiologic demands are well tolerated. However, in certain cardiac diseases, maternal morbidity and even mortality may occur [1].

The combination of displacement of diaphragm and the effect of pregnancy on the shape of the rib cage displaces the heart upward and to the left. In addition, the heart rotates on its long axis, moving the apex somewhat laterally, resulting in an increased cardiac silhouette on radiographic studies without a true change in the cardiothoracic ratio. Associated radiographic findings include an apparent straightening of the left border of the heart and increased prominence of the pulmonary conus. Therefore, the diagnosis of cardiomegaly by simple radiography should be confirmed by echocardiogram if clinically appropriate [2].

Although true cardiomegaly is rare, physiologic myocardial hypertrophy of the heart is consistently observed as a result of expanded blood volume in the first half of the pregnancy and progressively increasing afterload in later gestation. These structural changes in the heart are similar to those found in exercise and result in eccentric hypertrophy as opposed to concentric hypertrophy that is seen with disease states such as hypertension or aortic stenosis. The eccentric hypertrophy enables the heart to enhance its pumping capacity in respond to increased demand making the pregnant heart mechanically more efficient. Most changes begin early in the first trimester and peak by 30 to 34week gestation. One of the most remarkable changes in pregnancy is the tremendous increase in cardiac output. It increases significantly beginning in early pregnancy, peaking at an average of 30% to 50% above preconceptional values. Although the literature is not clear regarding the exact gestational age when CO peaks, most studies point to a range between 25 and 30 weeks. CO is the product of heart rate and stroke volume both of which raise during pregnancy and contribute to an increase in cardiac output. An initial raise in HR occurs by 5 weeks and continues upto 32 weeks. The SV begins to raise by 8 weeks and reaches maximum by 20 weeks [3, 4].

The overall prevalence heart disease complicating pregnancy varies from 0.3-3.5%. With a decrease in maternal death as a result of the classic causes of haemorrhage, hypertension and

infection, the relative incidence of cardiac disease has increased1.Cardiac disease in pregnant women can present challenges in cardiovascular and maternal-fetal management. During the last few decades, the etiology of heart disease in developed countries has changed from primarily rheumatic to predominantly congenital. The major cardiac causes in the latest triennium of the Centre for Maternal and Child Enquiries (CMACE)'s confidential enquiry into maternal death include myocardial infraction, dissection of thoracic aorta, and peripartum cardiomyopathy. Despite the potential for significant maternal morbidity, most patients with cardiac disease can expect a satisfactory outcome with careful antenatal, intrapartum and post-partum management. The rate of complications is related to several factors, including maternal functional status, myocardial dysfunction, significant aortic or mitral valve stenosis and history of arrhythmias or a cardiac event [5, 6].

Methodology

The present study was conducted among the antenatal women admitted in the department of OBG with a previously diagnosed cardiac disease or diagnosed after admission during index pregnancy.

Inclusion criteria

1. All women admitted to the antenatal ward and labour room with previously diagnosed cardiac disease and women diagnosed during the index pregnancy
2. Women developing cardiac complications during puerperium

Exclusion criteria

Patients with cardiac disease receiving outpatient care

Present study is a prospective observational study. Considering inclusion and exclusion criteria, selected patients were counselled regarding the study. After obtaining valid consent, a pre-designed proforma was used. Detailed clinical history regarding the no of pregnancies, presence of cardiac illness, symptoms, treatment received was noted. Necessary obstetrical examination was done. Laboratory investigations included hb estimation, blood grouping, urine routine, urine culture, HIV, HBSAg, glucose challenge test, TSH, ASO titre. Chest x ray PA view wherever necessary was taken with an abdominal shield. Electrocardiography and echocardiography was done in all the patients and the diagnosis was confirmed. Special investigations like blood urea, serum creatinine, uric acid, liver function tests, fundoscopy, BT, CT, coagulation profile were done in necessary cases. 2D ECHO of the newborns was done to detect undiagnosed cardiac lesions. The mothers and the babies were followed up. Maternal outcome was defined as discharged or died. The perinatal outcome was defined in terms of mortality, presence of congenital heart disease or other anomalies in the offspring.

Results

Table 1: Most common ECHO findings

ECHO findings	N	%
Dilated LV	2	5.7
Global hypokinetic LV	8	22.9
Globally dilated LV	2	5.7
Grade 1 MR	5	14.3
Mild MS	2	5.7
Mitral valve orifice	2	5.7
MR	3	8.6
Normal	4	11.4
PAH	3	8.6
S/P DC	4	11.4
Total	35	100

Table 2: Ejection fraction

	Mean N	SD%
Ejection fraction	53.1	8.3

Table 3: Type of anemia

Type of anemia	N	%
Normal	32	42.1
Microcytic	29	38.2
Dimorphic	15	19.7
Total	76	100

Table 4: Hemoglobin

	Mean	SD
Hemoglobin	9.8	1.4

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Table 5: ASO titre among RHD disease patients

ASO titre	N	%
Negative	20	48.8
Positive	21	51.2
Total	41	100

Table 6: Previous diagnosed

Previous diagnosed	N	%
Not diagnosed	24	31.6
Diagnosed	52	68.4
Total	76	100

Table 7: Mode of delivery

Mode of delivery	N	%
Undelivered	1	1.3
Vaginal	47	61.8
1. Full term vaginal delivery	20	26.3
2. Ventouse assisted delivery	20	26.3
3. Forceps delivery	6	7.9
4. Assisted breech delivery	1	1.3
LSCS	28	36.8
1. Emergency LSCS	20	26.3
2. Elective LSCS	8	10.5
Total	76	100

Table 8: Neonatal 2D findings

Neonatal 2D findings	N	%
Normal	62	87.3
VSD	6	8.4
ASD	3	4.3
Total	71	100

Table 9: Congenital heart disease in new born

Congenital heart disease in new born	N	%
No congenital heart disease	62	87.3
Presence of congenital heart disease	9	12.7
Total	71*	100.0

*4 - IUD, 1- Undelivered and died, 1- one of the twin babies died.

Table 10: Congenital anomaly

Congenital anomaly	N	%
None	56	78.9
CHD	9	12.7
Cleft lip	2	2.8
Club foot	2	2.8
Diaphragmatic hernia	1	1.4
Syndactyly	1	1.4
Total	71	100.0

Table 11: Condition after delivery

Condition after delivery	N	%
Stable	61	80.3
Atonic PPH and DIC	1	1.3
Atonic PPH	2	2.6
Hypotension	1	1.3
Peripartum dilated cardiomyopathy	11	14.5
Total	76	100

Table 12: Outcome

Outcome	N	%
Discharged	66	86.8
Died	10	13.2
Total	76	100

Table 13: Cause of death (N=10)

Cause of death	N	%
Peripartum cardiomyopathy	5	50.0
PPH with Peripartum cardiomyopathy	1	1.0
Atonic PPH with DIC	1	1.0
Severe AS	1	1.0
Severe MS+ Severe PAH	1	1.0
RHD with severe anemia leading to congestive cardiac failure	1	1.0
Total	76	100

Table 14: Outcome among patients with peripartum cardiomyopathy

Outcome	N	%
Discharged	5	50.0
Died	5	50.0
Total	10	100

Discussion

Table 15: Comparison of Maternal outcome in terms of mode of delivery

Study	Normal vaginal	Instrumental	LSCS
Indira *et al*. [7]	53.3	29.99	3.33
P Sneha *et al*. [8]	38.8	16.67	41.67
Konar *et al*. [9]	49.8	17	33.0
Salam S *et al*. [10]	41.2	7.8	36.7
Our study	27.6	34.2	36.8

All values expressed in percentage

In our study percentage of LSCS is around 37%, which is almost similar to most of the studies resulting in 33%, 37%, 41% respectively. But the result of Indira *et al*.[2] is contrary to our study where LSCS rate was only around 3% and the only indication for LSCS was CPD. In our study LSCS done for only obstetric indications.

In our study Normal vaginal deliveries are quite low compared to above coated studies because of the use of instruments to cut short the 2nd stage of labour.

Table 16: Comparison of maternal outcome in form of mortality

Study	% of Maternal mortality
Sawhney *et al*. [11[	2
Hameed *et al*. [12]	2
T Nqayana *et al*. [13]	0
Konar *et al*. [9]	1.06
Salam S *et al*. [10]	4.44
Our study	13.2

There is significantly high percentage of maternal mortality compared to other studies. Peripartum cardiomyopathy being the leading cause (n=5), Atonic PPH stands number two, responsible for two cases, severe Aortic Stenosis, severe MS, CCF, contributes to each case.

There were no maternal death in the study conducted by Konar *et al*., Vidyadhar B Bangal *et al*. In a study conducted previously at our institute by Kamat *et al*. there was only one maternal death accounting to 2.85%.

Most of the patients in postpartum period were stable, only a few patients developed complications like breathlessness, hypotension,PPH. Anemia the most important factor especially in this part of Karnataka which may be the significant precipitating factor for the above complications. Even severe anemia was found in 2 cases and associated with one maternal death.

Table 17: Comparison of maternal outcome in peripartum cardiomyopathy

Study	No of cases of peripartum cardiomyopathy	Percentage of mortality
Stefan Neubauer *et al*. [14]	39	26
Modi K A *et al*. [15]	44	15.9
Silwa *et al*. [16]	29	32
Our study	10	50

In our study there is comparatively higher mortality rate among peripartum cardiomyopathy cases, reasons may be delay in detection, lack of available resources like ICU facilities, precipitating factors like anemia, PIH etc.

Eventhough peripartum cardiomyopathy is a rare but serious condition of unknown cause. Mortality rate ranging from 7% to 50%. Diagnosis of peripartum cardiomyopathy requires high awareness, and high degree of suspicion. Management of peripartum cardiomyopathy must be a multidisciplinary approach and should aim at improving heart failure symptoms first, thus the mortality rate due to peripartum cardiomyopathy can be decreased.

Conclusion

Cardiac disease in pregnancy constitute high risk, and poses a major impact on mother as well as fetus. This studies shows that proper maternal evaluation antenatally, institutional delivery can significantly improve the prognosis of both mother and fetus. In treating such cases a multidisciplinary approach is ideal and team should involve obstetrician, cardiologist, anaesthetist, if necessary cardiac surgeons should be involved.

Conflict of Interest

Not available.

Financial Support

Not available.

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