A DISSERTATION ON

MATERNAL AND FETAL OUTCOME IN HEART DISEASE COMPLICATING PREGNANCY COIMBATORE GOVT. MEDICAL COLLEGE HOSPITAL COIMBATORE

DISSERTATION SUBMITTED TO

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI - 600 032

with partial fulfillment of the regulations for the award of the degree of

M.S (BRANCH – II)

(OBSTETRICS & GYNAECOLOGY)

MAY 2022

COIMBATORE MEDICAL COLLEGE HOSPITAL

COIMBATORE

REGISTER NUMBER : 221916304

DECLARATION BY THE CANDIDATE

I hereby declare that, this dissertation entitled **"MATERNAL AND FETAL OUTCOME IN HEART DISEASE COMPLICATING PREGNANCY"** has been prepared by me under the able guidance of **Dr.R. MANONMANI,** M.D.,D.G.O., Professor and HOD, Department of Obstetrics and Gynecology, Coimbatore Medical College Hospital, Coimbatore.

This dissertation is submitted to the Dr. MGR Medical University, Tamilnadu in partial fulfillment of the University regulation for the award of the **M.S. Degree (OBSTETRICS AND GYNAECOLOGY)** examination to be held in May 2022.

This work has not been submitted previously for the award of any degree or diploma by me to any other university.

Place : Coimbatore

Date : **(Dr. M.R.DINESH)**

CERTIFICATE BY THE GUIDE

This is to certify that this dissertation titled **"MATERNAL AND FETAL OUTCOME IN HEART DISEASE COMPLICATING PREGNANCY"** has been prepared by **Dr. M.R.DINESH** under my direct guidance and supervision and is being submitted to the Dr. MGR Medical University, Chennai in partial fulfillment of the university regulation for the award of the **M.S. Degree (OBSTETRICS AND GYNAECOLOGY)** examination to be held in May 2022 and her dissertation is a bonafide work.

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CERTIFICATE OF APPROVAL

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Dear **Dr.DINESH. M.R.,**

The Institutional Ethics Committee of Coimbatore Medical College, reviewed and discussed your application for approval of the proposal entitled **"MATERNAL AND FETAL OUTCOME IN HEART DISEASE COMPLICATING PREGNANCY IN COIMBATORE MEDICAL COLLEGE AND HOSPITAL."** No.0570/2021.

The following members of Ethics Committee were present in the meeting held on 22.03.2021 conducted at MEU Hall, Coimbatore Medical College Hospital Coimbatore-18.

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14	Mrs.D.Stella.,M.Com.,	Lay-Person

We approve the Proposal to be conducted in its presented form.

Sd/ Member Secretary & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Chairman, Ethics Committee

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This is to certify that the dissertation entitled **"MATERNAL AND FETAL OUTCOME IN HEART DISEASE COMPLICATING PREGNANCY"** is a bonafide research work done by **Dr. M.R.DINESH** under guidance of **Dr. R.MANONMANI,** M.D.,D.G.O Professor and HOD, Department of Obstetrics and Gynaecology, Coimbatore Medical College Hospital, Coimbatore.

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I owe my thanks to all the women who have willingly participated & consented to their personal details for the study & helped in completing it.

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INTRODUCTION

Heart disease in pregnancy is one of the leading cause of maternal morbidity, mortality and adverse perinatal outcome in medical conditions complicating pregnancy. Heart diseases complicating pregnancy ranks third among the leading causes of maternal mortality. The leading causes being antepartum and postpartum haemorrhage, followed by severe preeclampsia and complications. Physiological changes during pregnancy involve many hemodynamic changes which causes increases in intravascular volume and dilutional anaemia which inturn overloads the already diseased heart and results in adverse outcomes. Thus the importance of pre-conceptional counselling, special antenatal follow up, decision regarding mode and time of termination and postpartum follow up in pregnant mothers with heart disease needs to be emphasised.

The prevalence of heart disease in developing countries is different compared to developing countries as rheumatic heart disease is the most common cardiac disease in pregnant women.

Pregnancy is unique as it is the only non-pathologic state that causes dramatic alterations in physiology. The integral components of pregnancy has physiology that promote fetal growth and development are cardiovascular adaptations. The maternal heart undergoes dramatic structural and functional changes which begins soon after conception and it evolves as the pregnancy progresses to serve the changing demands of the developing fetus and in anticipation of blood loss at delivery. Disruptions in this physiologic system can lead to both maternal and fetal morbidity which can occur at all stages of pregnancy. In the intrapartum period, increased perfusion of the uterus, pain-related tachycardia, and fluid shifts will lead to additional hemodynamic variation. Almost all of these cardiovascular adaptations of pregnancy return to baseline in the postpartum period; however, some may persist for up to 6 months after delivery, emphasizing the need for maternal care in the "fourth trimester."

Many heart diseases remain undiagnosed until later in pregnancy. Hence early diagnosis and comprehensive obstetric and cardiac care by obstetricians, cardiologists, intensivists, anaesthetists and paediatricians can prevent complications leading to a better prognosis.

Because many of the cardiovascular adaptations of pregnancy are marked departures from the nonpregnant physiology, pregnancy may unmask previously unrecognized cardiac disease such as rheumatic mitral stenosis that can lead to significant morbidity and mortality.Cardiovascular disease remains the leading cause of maternal mortality in the United States . Normal pregnancy symptoms may overlap with those of cardiovascular disease. An understanding of the maternal adaptations that may elicit or exacerbate cardiovascular disease is essential for any obstetric provider. Appropriate triage and follow-up is essential in women with cardiovascular disease during pregnancy and also the postpartum period.

AIMS AND OBJECTIVES

1. To assess the prevalence of heart disease in pregnancy in the population.

2. To study the presentation of heart disease in pregnancy.

3. To study the maternal and fetal outcome, adverse events.

4. To assess the clinical profile of the pregnant patients with different cardiac conditions, specifically looking for

    - Maternal outcome: Maternal death, heart failure and cardiogenic shock, thromboembolic manifestations, cerebro-vascular accidents, arrhythmia.

    - Fetal outcome: Live births, IUGR / Low birth weight, still birth, spontaneous abortion, therapeutic abortion

REVIEW OF LITERATURE

1. Hamilton and Thompson conducted the study on heart diseases in pregnancy which was the first one at the Boston Lying in Hospital back in 1921-38.

2. A study on heart disease complicating pregnancy during 1950 was conducted by Sudhir Bose Calcutta and Masoni Bombay. They concluded the incidence ranging from 0.2 to 0.98%

3. Szekely and Smith conducted a study between 1942 and 1971 among 1000 women admitted in a general hospital in New Castle. Rheumatic heart disease had a declining trend in this study. Mitral stenosis was the commonest rheumatic heart disease in 90% cases. 15.5% of the women developed pulmonary congestion and 1.6% of women developed pulmonary edema. Maternal mortality was 1.62% and was mainly found to be due to heart failure and pulmonary edema.

4. Bitch and Mc Faul found 51% of heart diseases to be congenital among pregnant women. Tan and De Sueit in 1998 reported the incidence of rheumatic heart disease to be 12.1%.

5. Sach et al found the maternal mortality to be 0.3 - 5.6 per lakh live births from a study done from 1954-1985.

6. Heart disease has been reported as the cause of maternal death in 8.5% cases in a study conducted between 1968 and 1993 by Ayhan et al (1994).35,36 A study

conducted by Tan and De Sweit in 199837 reported that only 12% had rheumatic heart disease.

7. Brickner delineated that progress in surgical methods and development in medical management will cause an increase in the number of women with congenital heart disease reaching pregnancy.

8. Martin in 1999 described that heart disease remains to be the third most common cause of maternal death

9. Silversides et al. in 2018 reported that arrhythmia and heart failure are the most common maternal cardiac complications, but there has been a reduction in the frequency of pulmonary edema.

10. A study conducted by Small in 2012 reported that cardiac disease was responsible for a significant amount of maternal morbidity and obstetric intensive care unit admission. Fryar in 2012 that almost 50% of adults aged more than 20 and above have at least one risk factor for cardiovascular diseases like obesity, Hypertension and Diabetes. Cardiac disease complicates >1% of all pregnancies and it is the leading indirect cause of maternal death in 20% of all cases described by Simpson and Berg .

11. From Brazil and Avilaand and co-workers in 2003 reported the maternal mortality rate to be 2.7% in 1000 pregnancies complicated by heart disease. According to

CDC- Heart disease is the main cause of death in women who are 25-44 years as described by Kung and colleagues in 2008.

12. Callaghan & co-workers in 2008 conducted a study in the US reported that 7.6% of severe obstetric morbidities are caused by cardiac disease.

13. Mahesh Koregol, Nina Mahale studied 110 cases of heart disease complicating pregnancy found rheumatic heart disease to be the most common type of heart disease which is in contrast to the western studies. Mitral stenosis was seen in 44.4% of women and mitral regurgitation was seen in 20.8% of mothers. Small for gestational age, SGA was noted in 45.3%, still births were found in 6.9% babies and neonatal deaths was noted in 2.9% of patients. 30.2% of babies needed NICU admission. Maternal and perinatal complications strongly correlated with maternal cardiac functional classification.

14. Suman Puri, Aman Bhatia et al that enrolled 97 women, the prevalence of heart disease was 4.3%. RHD accounted for 70.2%. Congenital heart disease was seen in only 9% of women. Mitral stenosis was found in nearly 19% patients.. Heart failure was observed in 6% of the women. Myocarditis and thromboembolism was also observed in a few women. Pre-term deliveries and low birth weight babies were found to be the common perinatal complications. Stillbirths and very low birth weight babies were 14.1 % and 21.2% respectively. The severity of the symptoms of heart disease in pregnancy was found to be a better indicator of perinatal outcome than the duration of the disease.

15. In a study in Andhra Pradesh involving 60 women with heart diseases, the maternal mortality rate was reported as 6.66%, which was slightly higher. Acute pulmonary edema was found to be the main cause of death. The prevalence of heart disease as per this study was 0.433%. 80% of women were found to have rheumatic heart disease. Anaemia and gestational hypertension were recorded the common complications.

16. Malhotra et al found a high rate of preterm low birthweight babies and increased incidence of forceps delivery among cardiacmothers compared with non cardiac mothers.

17. Nagamani et al reported the incidence of heart diseaseamong pregnant mothers to be at 1.23% from a prospective cohort study. The ratio of rheumatic heart disease with congenital heart disease was reported as 60:40. Maternal mortality rate was 5.1% and the intra uterine death rate was 5%. Severe mitral stenosis was seen to be the cause of 2 maternal deaths. Major determinants of the poor maternal outcome in their study were NYHA functional classification at the time of admission and ejection fraction.

HEART DISEASES IN PREGNANCY

HEMODYNAMIC CHANGES IN PREGNANCY

The hemodynamic parameters that adapt during pregnancy include heart rate (HR), systemic vascular resistance (SVR), blood volume, cardiac output (CO), and blood pressure (BP).Most changes begin to occur in the first trimester, peak in the late second or early third trimester, and then plateau for the remainder of pregnancy with a return to pre-pregnancy values during the postpartum period

Heart Rate

Maternal HR increases steadily throughout pregnancy and has been shown in some studies to be the first hemodynamic parameter to change after conception as early as 5 weeks of gestation . Unlike most other hemodynamic parameters, which will change at rapid rates until a plateau point in the second trimester, HR increases steadily throughout pregnancy . Peak increase in HR is generally between 10–20 beats per minute (bpm), or 20%– 24%, above pre-pregnancy values . Maternal position may cause variations in HR, with slightly lower rates found in the left lateral position than in the supine position, most likely due to alterations in preload with position changes. HR typically returns to pre-pregnancy values in the immediate postpartum period, that is, within 6 hours postpartum.

Systemic vascular resistance

The decrease in SVR occurs as early as 5 weeks of gestation and occurs prior to full development of the placenta . In the first trimester, SVR decreases by about 11%,

mainly due to the vasodilatory effects of progesterone, estrogen, prostaglandins, and relaxin . Increased nitric oxide production which occurs in pregnancy also has a role in the decrease in SVR. As the placenta develops, it will further decreases the SVR by adding a high-flow, low-resistance component to the maternal circulation. Decreased SVR leads to a decrease in BP, more specifically diastolic BP, and a widening of pulse pressure.

Blood volume

Increased blood volume in pregnancy is evident from studies . Both plasma and red blood cell (RBC) expansion contribute to the total blood volume increase of pregnancy. Plasma volume expansion is primarily mediated by the upregulation of the RAA system, which begins as early as 6 weeks' gestation and continues to increase rapidly throughout pregnancy, with the greatest increase in the second trimester. After 28 weeks, the rate of plasma volume increase slows; plasma volume reaches its peak at approximately 33 weeks gestation. The degree of blood volume expansion is proportional to birth weight, and diminished plasma volume increase is associated with preeclampsia and growth restriction. Fetal growth restriction has been found to be preceded by inadequate volume expansion even as early as 8 weeks of pregnancy.

Cardiac Output

CO is the product of stroke volume and HR that reflects the capacity of the heart to respond to the perfusion requirement of the body. Typically, the peak rise in CO occurs by 8 weeks, with continued increase into the second trimester and a peak between 25–35 weeks . In some studies, CO has been shown to be increased by 51% during pregnancy.But, there is no data in the literature regarding the precise magnitude and pattern of change in CO.This increase in cardia output was found to be steady, though non-linear, until its peak at 31 weeks, with a subsequent plateau. In early pregnancy, stroke volume was the major contributor to the increase in CO, and in late pregnancy, HR became increasingly influential.Maternal position also affects CO, which has implications during obstetric procedures, imaging studies, and during labor and delivery. In the supine position, the gravid uterus compresses the inferior vena cava, decreasing venous return to the heart and causing a significant decrease in CO.

Hemodynamic Changes in Labor

An increase in cardiac output during labor has been long reported in the literature. Cardiac output increases by an average of 30.9% during contractions, with pain and anxiety contributing to this increase . 35% increase in cardiac output over the course of prolonged labor.

Delivery and the Postpartum Period

The most marked hemodynamic changes occur at the moment of delivery itself and in the immediate postpartum period. CO increases up to 82% at delivery, then begins to decline to pre-labor values within approximately 1 hour following delivery. Increased CO may be secondary to the return of blood supply from the uterus to the systemic circulation that leads to an greater increase in stroke volume . Other factors that may contribute to this increase include increased venous return after the relief of the weight of the gravid uterus and auto transfusion which occurs from uterine involution. An increase in CO may combat the blood loss experienced at delivery. CO rapidly decreases for the first 2 weeks following delivery, and over the course of the postpartum period returns to pre-pregnancy values. Complete normalization of cardiac output can take up to 6 months postpartum .

Heart rate has been shown to decrease in the first 48 hours postpartum .By 2 weeks after delivery, heart rate reaches its nadir and does not decrease thereafter. Blood pressure tends to remain stable throughout this period, except in those patients who have lost a clinically significant amount of blood.

In recent literature, CO was found to increase throughout cesarean section, peak within 2 minutes after delivery of newborn and placenta, and reach levels lower than baseline at 24–36 hours postoperatively . This is likely due to the rapid increase in venous return and preload after delivery of the fetus and placenta .

THE HEMODYNAMICS OF UNCOMPLICATED PREGNANCIES: ANTEPARTUM, INTRAPARTUM, AND POSTPARTUM CHANGES

Heart Rate	Systemic Vascular Resistance	Plasma Volume	RBC Volume		Cardiac Output	Blood Pressure
1st trimester	Increase	Decrease	Increase	Increase	Sharp increase	Decrease
2nd trimester	Steady increase	Rapid decrease	Rapid increase	Steady increase	Slower increase	Continued decrease, nadir
3rd trimester	Steady increase	Slight increase after 32 weeks	Slower increase after 28 weeks	Steady increase	Plateau	Increase
Intrapartum	Continues to increase	No significant change	Decrease due to blood loss	Decrease due to blood loss	Increase	Increase
Postpartum	Return to pre-pregnancy within 6–48 hours	Return to pre-pregnancy values soon after delivery	Vaginal delivery: decrease over 10 days Cesarean delivery: no change	Vaginal delivery: decrease over 10 days Cesarean delivery: no change	Rapid decrease in first 2 weeks, slower decrease for up to 6 months	Return to pre-pregnancy, unknown time frame

Earliest change	5 weeks	5 weeks	6 weeks	8–10 weeks	Before 8 weeks	7 weeks
Peak/nadir above/ below pre-pregnancy values	+20%–25%	−30%	+45%	+20%–30% (with iron), +15%–20% (no iron)	+30%–50%	−5–10 mmHg (systolic); up to −15 mmHg (diastolic)
GA at peak/nadir	Intrapartum	32 weeks	32 weeks	Term	25–35 weeks	20–24 weeks

Preconception Counseling

The task of advising females regarding the safety of pregnancy has been challenging until recently. After almost three decades, the body of literature in this field has moved from expert opinion and case reporting to cumulative data reported from multicenter

Advances in medical and surgical arenas have created a larger pool of women of childbearing age with heart disease

Congenital heart disease is the most frequently encountered type of cardiac disorder in pregnancy and must be managed by a multidisciplinary team with appropriate expertise in adult congenital heart disease

The modified WHO risk classification is the more widely used and is the simplest risk scoring scale

Pregnancy is not recommended for women in modified WHO risk category IV

While CHD is more prevalent, those with acquired heart disease carry a higher risk of mortality during childbirth. These include women with ischemic heart disease, aortopathies, various cardiomyopathies, hypertensive disorders, and valvular heart disease, particularly requiring anticoagulation. In addition, over the past 2–3 decades, the number of children undergoing heart transplant has risen and consequently the number of these young women now and in future will be seeking to become pregnant. Today, transplanted female patients represent approximately 20% of overall cardiac transplants population, 25% of which are reported to be between the ages of 18–39 years .While successful pregnancies have been reported, the majority experience complications such as hypertensive disorders of pregnancy, prematurity, graft rejection, and/or infection .

Modified WHO Classification of Maternal Cardiovascular Risk		
Pregnancy Risk Category	**Risk Description**	**Maternal Risk Factors**
WHO I	No detectable increase in maternal mortality and no/ mild increase in morbidity risk Maternal cardiac event rate 5.7%–10.5%	Uncomplicated small/mild pulmonary stenosis, PDA, mitral valve prolapse Successfully repaired simple lesions (ASD, VSD, PDA, anomalous pulmonary venous drainage)

		Atrial or ventricular ectopic beats, isolated

WHO II	Small increase in maternal mortality and moderate increase in morbidity risk Maternal cardiac event rate 2.5%–5%	(If otherwise well and uncomplicated) Unoperated ASD, VSD Repaired TOF Most arrhythmias
WHO II–III	Moderate increase in maternal mortality morbidity risk Maternal cardiac event rate 10%–19%	Mild LV impairment Hypertrophic cardiomyopathy Native or tissue valvular disease (not considered risk category I or IV) Marfan syndrome without aortic dilation Aortic dilation <45 mm in bicuspid aortic valve aortopathy Repaired coarctation
WHO III	Significantly increased maternal mortality or severe morbidity risk Expert counseling required In the event of pregnancy, intensive specialist cardiac and obstetric monitoring needed throughout pregnancy, childbirth and the puerperium	Mechanical valve Systemic RV Fontan circulation Cyanotic heart disease (unrepaired) Other complex CHD Aortic dilation 40–45 mm in Marfan syndrome

	Maternal cardiac event rate 9%–27%	Aortic dilation 45–50 mm in bicuspid aortic valve aortopathy
WHO IV	Extremely high maternal mortality or severe morbidity risk Pregnancy is contraindicated In the event of pregnancy, termination should be discussed If pregnancy continues, care should follow class III recommendations Maternal cardiac event rate 40–100%	Pulmonary arterial hypertension (of any cause) Severe systemic ventricular dysfunction (LV ejection fraction <30%, NYHA class III–IV) Previous peripartum cardiomyopathy with any residual impairment of LV function Severe mitral stenosis, severe symptomatic aortic stenosis Aortic dilation >45 mm in Marfan syndrome Aortic dilation >50 mm in bicuspid aortic valve aortopathy Native severe coarctation

DIAGNOSIS OF PREGNANCY:

Chest Radiograph

The chest radiograph is a commonly used diagnostic modality in pregnancy and it provides important information about the lungs, airways, blood vessels, and size of the heart and bones of the spine and chest. Indications for obtaining a chest radiograph in pregnancy are no different from those in nonpregnant patients and should be considered in any pregnant patient who presents with new-onset dyspnea to evaluate for pulmonary edema, cardiomegaly, and atrial enlargement.

Electrocardiogram

The electrocardiogram (ECG) is often one of the initial diagnostic modalities performed in pregnant women with suspected heart disease. The ECG is helpful in identifying conditions such as myocardial infarction, arrhythmia, and PE. During normal pregnancy there may be some subtle changes in the ECG, such as shortening of the PR and QT intervals, left axis deviation , and nonspecific ST-T wave changes in the left precordial leads.

Echocardiography

Transthoracic echocardiography can be used to evaluate ventricular function, valvular abnormalities, and pericardial disease. It uses high-frequency sound waves to image cardiac structures. Ultrasound waves are harmless to the tissues at the intensities used in diagnostic imaging. Echocardiography should be obtained in pregnant women who complain of chest pain, syncope, shortness of breath out of proportion to pregnancy, and palpitations. Furthermore, an echocardiogram should be performed on women with documented arrhythmia during pregnancy and those with known heart disease, stroke, or prior history of chemotherapy or radiation . Serial echocardiography may be indicated during pregnancy based on the underlying cardiac disease.

Exercise Stress Test

In stable women with symptoms of coronary disease but no acute features, it is reasonable to perform exercise stress testing. Studies have shown that exercise stressing in pregnancy is safe. Stress testing can be either ECG-only, or if imaging is needed, exercise echocardiography can be performed. Studies have shown that

exercise stress testing in inactive, regularly active, and vigorously active healthy women between 28 and 32 weeks of gestation showed no abnormal fetal bradycardic responses and there were no adverse neonatal outcomes.

Cardiac Magnetic Resonance Imaging

Cardiac MRI is indicated when echocardiography cannot provide adequate diagnostic information and better imaging is required to optimize management. Cardiac MRI enables noninvasive evaluation of cardiac chambers, great arteries, and veins, and provides excellent evaluation of both the left and right ventricle, including ventricular size, thickness, wall motion, and ejection fraction. Cardiac MRI can be most helpful in the assessment of complex congenital heart disease and aortic pathology.

Cardiac MRI has been used safely in pregnancy for over 25 years, especially in the second and third trimester. There are no precautions or contraindications specific to the pregnant woman.

CLINICAL CLASSIFICATION

The first ever classification for the assessment of functional capacity was published in1928 by the New York Heart and it has been revised for the

Classification of the functional capacity recommended by the New York Heart Association (used for classification of dyspnoea due to heart failure)

Class I: Patients with cardiac disease, but without resulting limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, dyspnoea, or anginal pain.

Class II: Patients with cardiac disease resulting in slight limitation of physical activity. They are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnoea, or anginal pain. They are comfortable at rest. Less than ordinary activity causes fatigue, palpitation, dyspnoea, or anginal pain.

Class III: Patients with marked limitation of physical activity.

Class IV: Patients with cardiac disease resulting in inability to carry on any physical activity without discomfort. Symptoms of heart failure or of the anginal syndrome may be present even at rest. If any physical activity is undertaken, discomfort is increased.

RISK FOR MATERNAL MORTALITY CAUSED BY VARIOUS HEART DISEASES

GROUP 1 (MINIMAL RISK)

Atrial septal defect

Ventricular septal defect

Patent ductusarteriosus

Pulmonary or Tricuspid disease

Tetralogy of Fallot corrected

Bioprosthetic valve

Mitral stenosis NYHA class I & II

MORTALITY - 0.1%

GROUP 2 (MODERATE RISK)

Mitral stenosis NYHA class III & IV

Aortic stenosis

Coarctation of aorta without valvular involvement

Tetralogy of Fallot uncorrected

Previous myocardial infarction

Marfan’s syndrome with normal aorta

MORTALITY - 5 -15%

GROUP 2B

Mitral stenosis with atrial fibrillation

Artificial valve

GROUP 3 (MAJOR RISK)

Pulmonary hypertension

Coarctation of aorta with valvular involvement

Marfan’s syndrome with aortic involvement

Eisenmenger’s syndrome

Peripartum cardiomyopathy

MORTALITY - 25 -50%

CONGENITAL HEART DISEASES IN PREGNANCY:

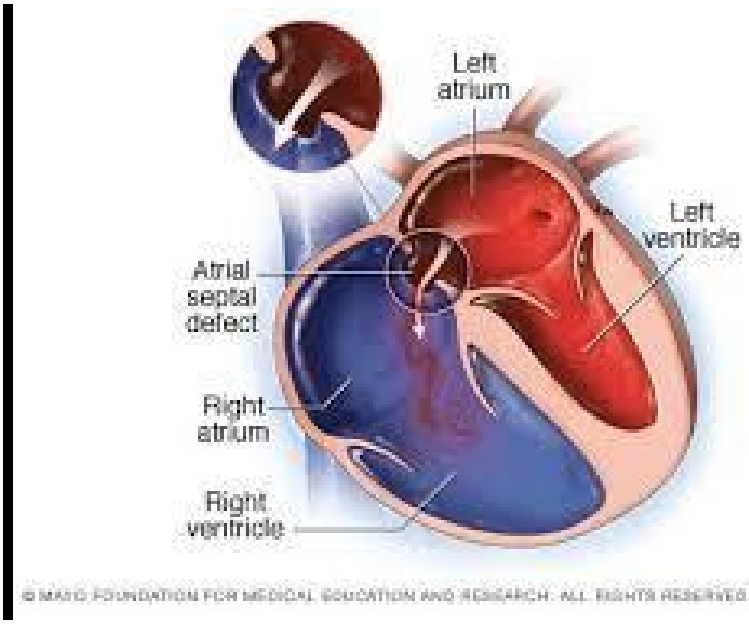
Congenital heart disease is the most common congenital defect, affecting approximately 0.8% of live births

- Over 80% of patients born with congenital heart disease will survive to adulthood. As more women with CHD reach childbearing age, care of these patients requires a multidisciplinary approach with specialists in maternal-fetal medicine, adult CHD, and obstetric anesthesia with expertise in care of this patient population.
- Aside from bicuspid aortic valves, atrial septal defects are the most common type of congenital heart defect, accounting for 11%–20% of congenital heart defects.
- A delivery plan readily accessible to all care providers should be outlined for patients who are at moderate or high risk for maternal cardiac complications.
- Patients with Eisenmenger syndrome are at high risk for maternal morbidity and mortality and therefore pregnancy is contraindicated.

Atrial Septal Defects

Aside from bicuspid aortic valves, atrial septal defects are the most common type of congenital heart defect, accounting for 15%–20% of congenital heart defects . Ostium secundum ASDs and primum ASDs are the most common, accounting for 75% and 20% of ASDs, respectively. Sinus venous defects and unroofed coronary sinus defects are, strictly defined, not ASDs as they do not involve the primum or

secundum septum, but nonetheless are included here as the result in the same physiology as ASDs . Sinus venosus defects occur at the juncture of the superior vena cava or inferior vena cava and the right atrium, and account for 15% of ASDs. Unroofed coronary sinus defects result from a fenestration in the roof of the coronary sinus as it passes posterior to the left atrium in the atrioventricular groove, allowing oxygenated blood to shunt from the left atrium into the coronary sinus, which then drains into the right atrium . Unroofed coronary sinus defects are rare, estimated to account for 1% of atrial level shunts.



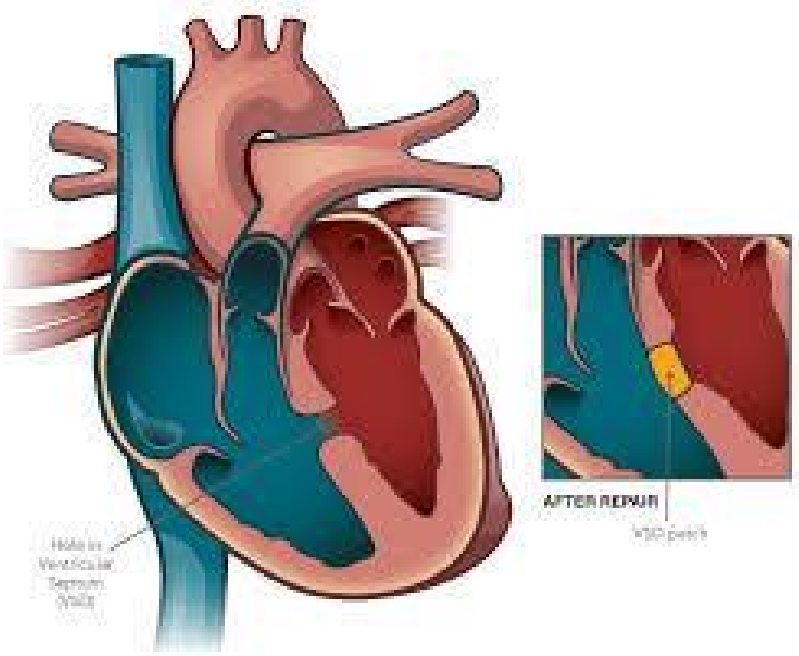
Ventricular Septal Defects

The incidence of ventricular septal defects varies widely, as many patients are asymptomatic, and many small defects close spontaneously but are among the most common forms of CHD in childhood. Approximately 10,000–11,000 isolated VSDs

are diagnosed in infants in the United States annually . Ventricular septal defects are divided into four types, based on their location within the interventricular septum:

1. Perimembranous VSDs account for 80%
2. Muscular VSDs account for 50%–10%
3. Inlet VSDs account for 5%
4. Supracristal VSDs account for 5%–7%

VSDs result in pressure loading of the right ventricle and pulmonary artery due to exposure of the right heart to systemic pressures. VSDs also result in volume loading of the left atrium and left ventricle, as the oxygenated blood shunted from the left ventricle to the right ventricle through the VSD is recirculated through the lungs and back to the left heart.



Aortic Coarctation

Aortic coarctation is a focal narrowing of the aorta, almost always at the junction of the distal aortic arch and descending aorta, just below the origin of the left subclavian artery. It is associated with a generalized arteriopathy and hypertension. CoA accounts for 5%–7% of congenital cardiac disease and is associated with bicuspid aortic valves in approximately 72% of patients, ventricular septal defects, and mitral valve abnormalities. Intracranial aneurysms, typically berry aneurysms of the circle of Willis, are seen in 3%–5% of patients with CoA.

Left Ventricular Outflow Tract Obstruction

Subvalvar, Valvar, and Supravalvar Aortic Stenosis

Subvalvar aortic stenosis accounts for nearly 30% of congenital left ventricular outflow tract obstruction lesions, and can be caused by a spectrum of malformations, from a discrete fibrous membrane to a tunnel-like fibromuscular band encompassing the circumference of the left ventricular outflow tract. It can be progressive and cause a significant pressure load on the left ventricle, with resultant hypertrophy, diastolic dysfunction, and eventual systolic dysfunction if left untreated. Surgical treatment depends on the type of obstruction and may range from resection of a discrete fibrous membrane to a septomyectomy to enlarge the left ventricular outflow tract in patients with tunnel-like obstruction .

Right Ventricular Outflow Tract Obstruction

Pulmonary Stenosis

Pulmonary valve stenosis accounts for 7%–11% of congenital heart defects, and typically occurs in isolation. The pulmonary valve may be fused and have an appearance of doming on the transthoracic echocardiogram or may be dysplastic with thickened leaflets. Pulmonary stenosis results in right ventricular pressure overload. The treatment of choice for pulmonary stenosis is balloon valvuloplasty.

Management of Congenital Heart Disease in Pregnancy, Peripartum, and Postpartum

Frequency of cardiology visits should be individualized based on level of risk as assessed by the risk stratification schemes, understanding of the severity of the patient's hemodynamic burden from residual lesions and physiological stage of CHD, which accounts for common residua and sequelae. Those who are at physiological stage A, such as a patient with a bicuspid aortic valve with normal valve function and no aortic dilation, may only require a single cardiology visit in the late second or early third trimester. Those at physiological stage B, such as a patient with a bicuspid aortic valve with normal valve function but mild aortic dilation, may benefit from cardiology visits and surveillance echocardiograms every trimester. Those in physiological stage C, such as a patient with bicuspid aortic valve and moderate aortic stenosis and/or regurgitation may require visits every 1 or 2 months. Those in physiological stage D, such as a patient with bicuspid aortic valve and severe aortic enlargement, may require more frequent visits, including the possibility of weekly visits for close monitoring and medication titration.

For severe cases a multidisciplinary meeting including the obstetric, cardiology, and anesthesia teams should be convened once the fetus reaches viability at 23−24 weeks.

The rate of fetal loss in CHD patients is approximately 15%–25%, slightly higher than that of the general population . Rate of premature delivery (10%–12%) was

more common in patients with CHD compared with the general population, particularly in patients with complex CHD . Neonatal events such as small for gestational age, respiratory distress syndrome, interventricular hemorrhage, and neonatal death also occur higher frequency of 27.9%. Maternal cyanosis, subaortic ventricular outflow tract obstruction, and low cardiac output are known predictors of adverse perinatal events .

Due to the increased incidence of CHD in the offspring of patients with CHD, a fetal echocardiogram should be performed between 18 and 23 weeks' gestation to evaluate the fetus for congenital heart defects if either biological parent has CHD.

VALVULAR HEART DISEASES IN PREGNANCY:

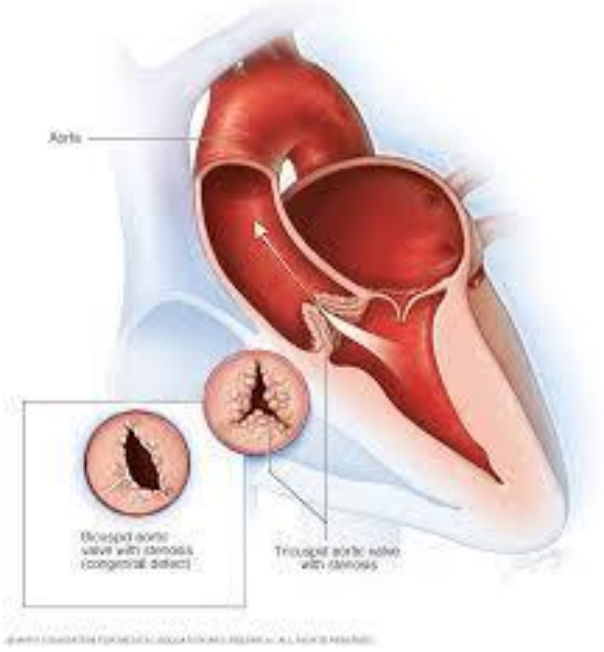
Hemodynamic Effects of Valvular Heart Disease in Pregnancy

Hemodynamic changes of pregnancy can lead to clinical decompensation of women with VHD due to increases in cardiac output , intravascular volume expansion, and a drop in systemic vascular resistance . Early in pregnancy, there is a 35%–50% increase in CO largely attributable to rises in circulating blood volume and heart rate. During the later stages of pregnancy, increases in heart rate contribute more to increased CO. Pregnancy is also accompanied by physiologic anemia with a greater

increase in plasma volume than red blood cell volume, resulting in increased flow and increased gradients across preexisting valvular lesions

Mitral Stenosis

MS is the most common acquired VHD in pregnant women .MS is considered clinically important if the valve area is ≤1.5 cm2 .The gold standard method of estimating MS severity on TTE is direct planimetry, however, this method can be technically challenging and is dependent on optimal image quality. Mild MS is usually well tolerated during pregnancy . Heart failure occurs in up to 1/3 of pregnant women with moderate MS and up to 1/2 with severe MS, even in previously asymptomatic patients, and most often during the second or third trimesters . Sustained AF occurs in up to 10% of pregnant women with moderate/severe MS and can predispose to symptomatic HF and thromboembolism . Maternal mortality from MS is reported to vary from 0%–3.2% in the West but may be higher in other parts of the world.



Aortic Stenosis

The leading causes of aortic stenosis in pregnant women are bicuspid aortic valve disease, often associated with dilation of the ascending aorta, followed by RHD . Most with mild or moderate AS can tolerate pregnancy .Heart Failure is infrequent, with an estimated prevalence of <10% in women with moderate AS who were asymptomatic pre-pregnancy. Even in patients with severe AS, maternal mortality is reported to be <1% in presence of close follow-up and treatment. Maternal and fetal adverse events are directly proportional to the severity of AS. Approximately one-third of women with severe AS require hospitalization during pregnancy. Fetal adverse events including growth restriction, low birth weight, and preterm birth have been reported in 20%–24% of babies of mothers with moderate or severe AS .

Bicuspid aortic valve is one of the most common congenital heart defects and is present in about 1% of the population. BAV is associated with histopathologic abnormalities of the ascending aorta leading to dilation and aneurysm . Among young patients <40 years with aortic dissection, BAV was present in 9% . Patients with BAV should be screened for the coexistence of aortic coarctation by clinical examination and imaging, since coarctation can compound the risk of aortic aneurysms.

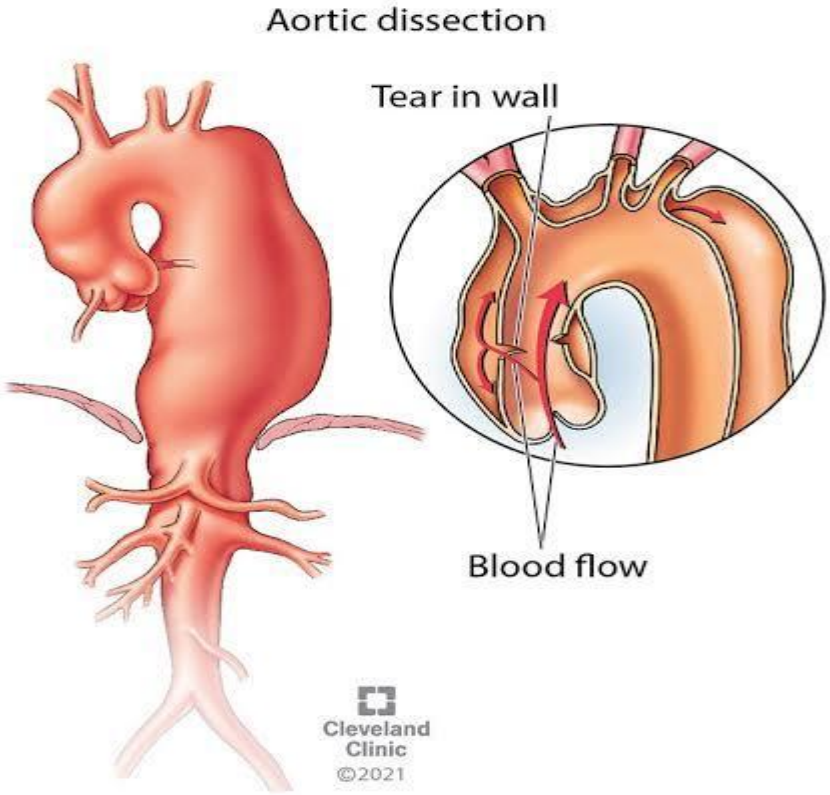
For patients who must undergo valve replacement prior to pregnancy, preconception counseling may include discussion of bioprosthetic vs. mechanical valves. While mechanical valves are highly durable, they also require consistent therapeutic anticoagulation and pose a higher risk of major cardiac events during pregnancy than bioprosthetic valves. In contrast, bioprosthetic use at a younger age risks structural valve deterioration during the reproductive years of life.

Aortic Dissection

Aortic dissection occurs when an interruption in the medial layer of the aorta allows intramural hemorrhage and propagation of blood, resulting in a sudden, severe, tearing type of pain with radiation to the back. The dissection can propagate to the aortic valve causing aortic regurgitation, or blood may enter the pericardial space

causing cardiac tamponade. If branch vessels are involved, ischemia of the coronary, carotid, spinal, or visceral arteries can occur.

Women with aortopathy should undergo MRI (or CT) of the entire aorta prior to conception. Additional risk factors include family history of aortic dissection or sudden death, the rate of growth (increase in diameter is ≥5 mm/year), and/or worsening aortic regurgitation . Even after aortic replacement, risk for dissection in the distal aorta or other vascular beds remains . Elective surgery could be considered during pregnancy if the aorta is >45 mm and increasing .If the fetus is viable, cesarean section can be performed followed directly by cardiothoracic surgery. If the fetus is not yet viable, surgical treatment for the mother is recommended.



CARDIOMYOPATHIES :

Adaptation to the physiologic requirements of pregnancy can challenge women with Cardiomyopathy. Women with baseline reduced cardiac reserve may not be able to accommodate demands to increase cardiac output by 35%–50%. Pregnancy is a state of volume overload and so increased volume load may exacerbate associated valve lesions such as mitral regurgitation or increased ventricular filling pressure precipitating overt Heart Failure. Cardiomyopathy was present in nearly 7% . Dilated cardiomyopathy was seen in 32%, Postpartum cardiomyopathy in 25%,

hypertrophic nonobstructive cardiomyopathy in 16%, hypertrophic obstructive cardiomyopathy in 11%,]. Heart failure was the most common cardiovascular event during pregnancy PPCM is a form of DCM in association with pregnancy in the absence of structural heart disease or another explanation for DCM. Criteria for diagnosis include LV enlargement and dysfunction (typically an ejection fraction <45%) presenting toward the end of pregnancy or in the months post-delivery in a woman without previously known structural heart disease . Diagnosis is confirmed by transthoracic echocardiography.

Risk factors for development of PPCM are well known. Preeclampsia and hypertension (chronic or gestational), age greater than 30, multiple gestations, and higher gravidity and parity are other risk. Current research suggests that an imbalance in angiogenic factors promotes PPCM in susceptible individuals. Both prolactin and soluble FMs-like tyrosine kinase have been implicated in its development

Maternal prognosis is variable but may be better than many other forms of cardiomyopathy. Mortality is higher, presence of cardiac arrest or shock, and length of stay are significantly longer in patients with PPCM than normal pregnancy. PPCM deliveries were more likely to be by cesarean . Factors suggesting worse prognosis include degree of LV dilatation, worse ejection fraction at presentation,

associated RV dysfunction, abnormal cardiac biomarkers, family history of heart failure, and low cholesterol . Most women improve in the first 6 months postpartum but delayed recovery has also been reported .

Neonatal outcomes in PPCM are also worse. Babies were born earlier, smaller, more likely to be small for gestational age, and APGAR scores were lower . For women with PPCM, it is unknown if early delivery will diminish progression or prevent development of LV dysfunction, but earlier delivery should be considered in the setting of worsening heart function. Otherwise timing of delivery should be determined by obstetric factors, such as fetal growth or development of preeclampsia, using a team approach. Vaginal delivery is preferred with spinal/epidural delivery.

PULMONARY HYPERTENSION:

Pulmonary hypertension is one of the highest risk medical conditions encountered in pregnancy. This has a prevalence of 9.7 cases per 100,000 that preferentially affects young women of child-bearing age .Pulmonary hypertension is defined as an elevated mean pulmonary arterial pressure (mPAP) ≥25 mmHg at rest confirmed by right heart catheterization, and is considered severe when mPAP is ≥50 mmHg.

Pulmonary hypertension encompasses a group of diseases with various pathophysiologies, including pulmonary arterial hypertension (PAH), pulmonary

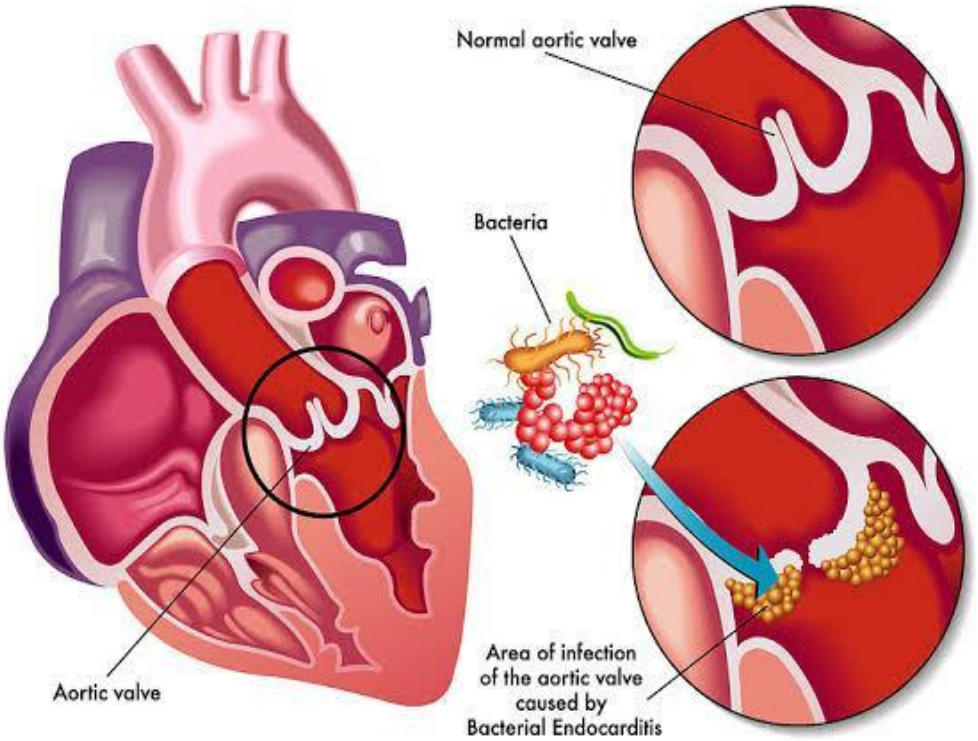
hypertension related to left heart disease, pulmonary hypertension related to lung disease, chronic thromboembolic pulmonary hypertension, and pulmonary hypertension with unclear and/or multifactorial mechanisms Pulmonary hypertension in pregnancy carries a high maternal mortality rate estimated at 18–33%, which represents an improvement in recent years, particularly in patients with severe pulmonary hypertension and Eisenmenger syndrome . Mortality is mostly the result of the inability of the right ventricle to accommodate the increased pulmonary artery pressure, leading to right heart failure The risk of right heart failure is particularly high during the intrapartum and postpartum periods due to the changes in intravascular volume and pressure during these stages.

Both vaginal and cesarean deliveries pose potential risks for the patient to deteriorate. Elective preterm cesarean delivery (between 32–36 weeks) has been advocated in patients with severe pulmonary hypertension .

INFECTIVE ENDOCARDITIS:

Infective endocarditis is defined as an infection of a heart valve (native or prosthetic), the endocardial surface, or an indwelling cardiac device. The yearly incidence of IE is estimated at 3-9/100,000 in the general population, similar to that reported in pregnancy. While the majority of cases of IE during pregnancy are identified antepartum, it can also present up to 6 weeks postpartum or after an

abortion .Previously, rheumatic heart disease was a major contributing factor for IE, the prevalence of which has fallen significantly in developed countries and its contribution to IE is negligible. In pregnancy, the most common risk factors are intravenous (IV) drug use and congenital heart disease that constitute 15%–43% and 12%–38% of cases of IE, respectively .If prophylaxis is warranted, antibiotic choice should be governed by clinical factors including the patient's relevant allergies and current medication and antibiotic use. Prophylaxis should be given as a single dose 30–60 minutes prior to the anticipated procedure but may be given up to 2 hours after a procedure if needed



CARDIAC SURGERIES IN PREGNANCY:

The ideal surgical timing depends partially on the urgency of the procedure.

In the setting of life-threatening conditions requiring urgent surgical intervention, such as aortic dissection or mechanical valve thrombosis, surgery should be undertaken as soon as possible.

For less urgent procedures, the procedure may be delayed for fetal benefit, as it has been shown that fetal mortality declines as gestational age progresses .

Prior to fetal viability around 24 weeks' gestation, preterm labor or delivery would lead to demise of the fetus, thus delay until closer to viability may be of some benefit for fetal survival. Subsequent to fetal viability, preterm labor or fetal distress requiring delivery will be associated with increased infant morbidity and mortality proportionate to degree of prematurity

For patients in the third trimester, cesarean delivery immediately followed by initiation of cardiopulmonary bypass for cardiac surgery is usually the preferred strategy, as the fetus is typically developed well enough for reasonably good survival after delivery

MATERIALS AND METHODS

The study of maternal and perinatal outcome in heart disease complicating pregnancy was conducted in the Department of Obstetrics and Gynaecology, Coimbatore medical college and hospital, Coimbatore. This study was performed during the period between July 2020 to June 2021 for 12 months and a total of 130 cases of heart disease complicating pregnancy were included in the study.

METHODOLOGY

Methodology included

(i) Meticulous history taking including signs and symptoms of heart disease and significant history of Rheumatic Fever

(ii) History of cardiac complications and decompensation in previous pregnancies

(iii) Details of the heart disease

(iv) Details of medical and surgical treatment of Heart Disease

(v) A methodical clinical examination with cardiac evaluation

All the cases that were available up to the study period have been taken for the purpose of study. All the cases were followed up, the presentation, course of disease, interventions needed, mode and gestational age of delivery, antenatal and

postnatal complications, maternal and fetal outcome were observed and documented, taken for analysis.

STUDY DESIGN

Prospective observational study

STUDY POPULATION

Patients who are admitted in Obstetrics and gynecology ward at Coimbatore medical college hospital with Heart disease complicating pregnancy for a period of one year. These patients were followed up till discharge and in the postpartum period.

INCLUSION CRITERIA:

All pregnant women with various Heart Disease (Rheumatic, Congenital, Valvular) who are admitted for safe confinement.

Pregnant patients with heart disease who got admitted with signs and symptoms caused by heart disease and complications.

EXCLUSION CRITERIA:

Pregnant women without heart disease

Pregnant women with heart disease who had spontaneous abortions, blighted ovum and molar pregnancy.

Pregnant women with heart disease who are diagnosed in the postnatal period.

Pregnant women who are not known cases of heart disease but presenting with symptoms and signs indicating heart disease were taken up to meticulous history taking and clinical examination and cardiologist assessment was made and those who did not have heart disease were excluded and the patients newly diagnosed to have a heart disease were included in the study.

OBSERVATION AND RESULTS

The present study was conducted in the DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY COIMBATORE MEDICAL COLLEGE AND HOSPITAL for a period of 12 months from July 2020 to June 2021 among the antenatal patients with heart disease. During the study period, the total number of patients who were included in the study with heart disease complicating pregnancy were 148 patients. Out of these 148 patients 138 patients delivered vaginally and by caesarean section and 10 underwent Medical termination of pregnancy.

TABLE 1:

TOTAL NO. OF CARDIAC PATIENTS	TOTAL NO. OF DELIVERIES	TOTAL NO. OF MTP
148	138	10

TABLE 2:

There were a total of 9648 deliveries in the department out of which 148 deliveries were of patients with heart disease.

TOTAL NO. OF DELIVERIES	HEART DISEASE DELIVERIES	INCIDENCE OF HEART DISEASE
9648	138	1.43 %

DISTRIBUTION OF CASES AS PER PURPOSE OF ADMISSION

TABLE 3:

PURPOSE OF ADMISSION	TOTAL NO. OF PATIENTS	PERCENTAGE
DELIVERY	138	93.3%
MTP	10	6.7%

URBAN – RURAL DISTRIBUTION

The distribution of the patients according to their residential address was studied. 62.16% were from urban areas and 37.83% were from rural areas.

TABLE 4:

TOTAL NO. OF PATIENTS	NO. OF PATIENTS FROM URBAN AREAS	INCIDENCE
148	92	62.16%

TABLE 5:

TOTAL NO. OF	NO. OF PATIENTS FROM RURAL	INCIDENCE

PATIENTS	AREAS	
148	37	37.83%

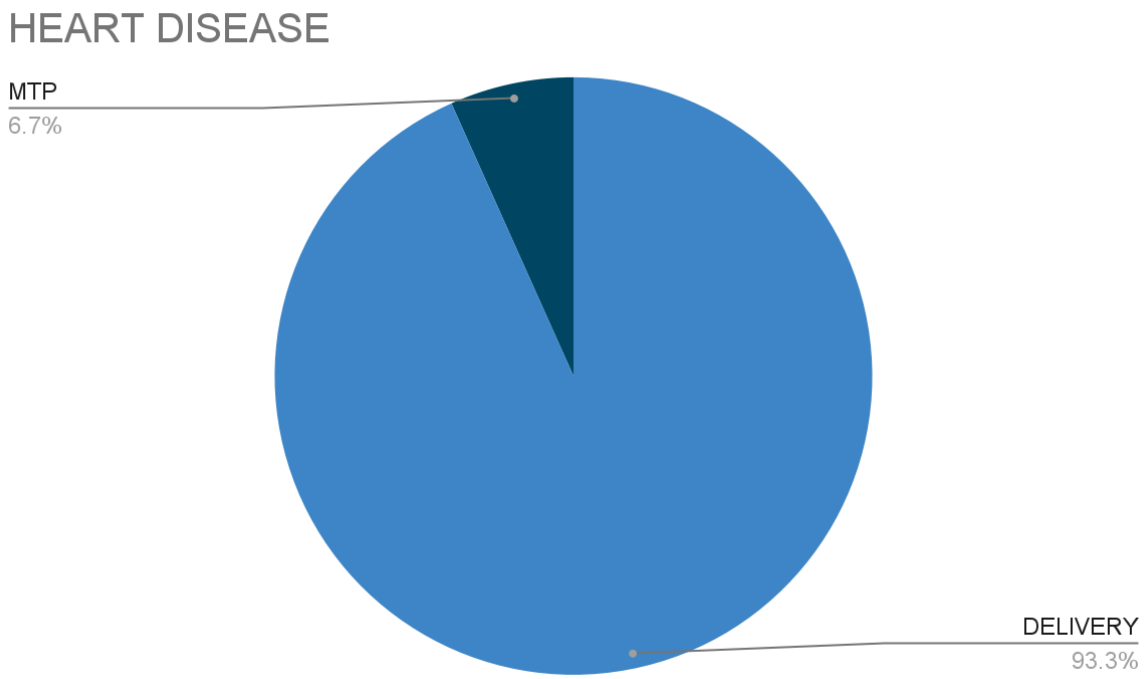


CHART 1: DISTRBUTION OF HEART DISEASE COMPLICATING PREGNANCY BASED ON ADMISSION

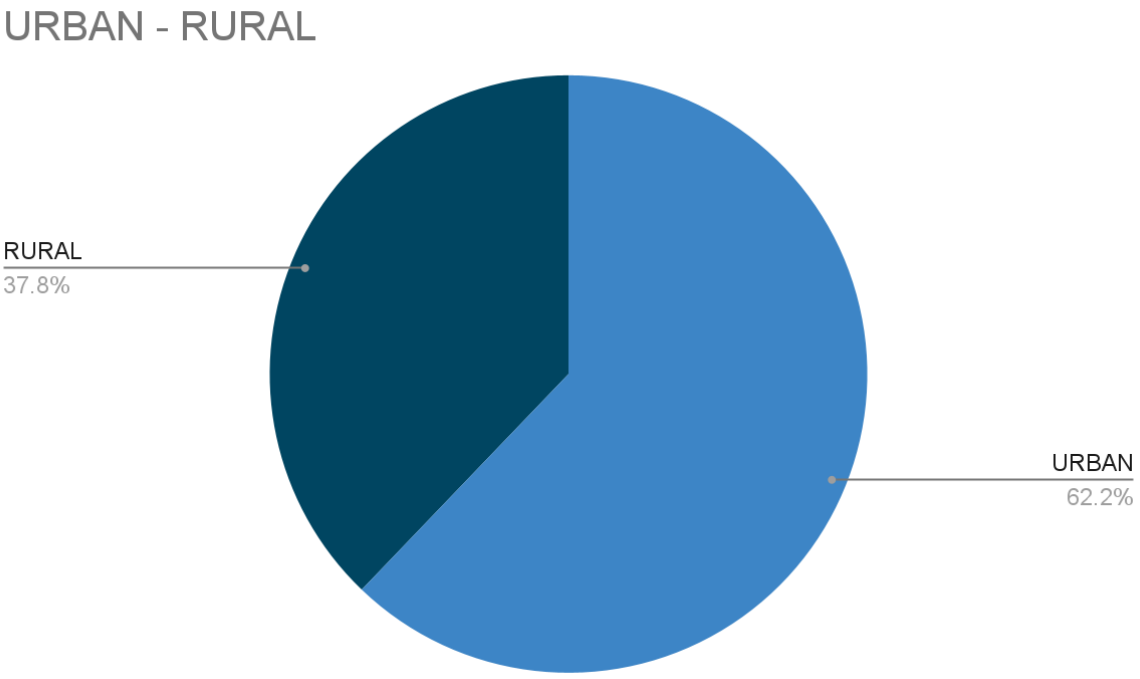


CHART 2: URBAN - RURAL DISTRIBUTION

SOCIO ECONOMIC STATUS DISTRIBUTION

TABLE 6

SOCIOECONOMIC CLASS	NO. OF CASES	PERCENTAGE
I	0	0
II	1	0.67%
III	17	11.48%
IV	76	51.35%
V	54	36.48%

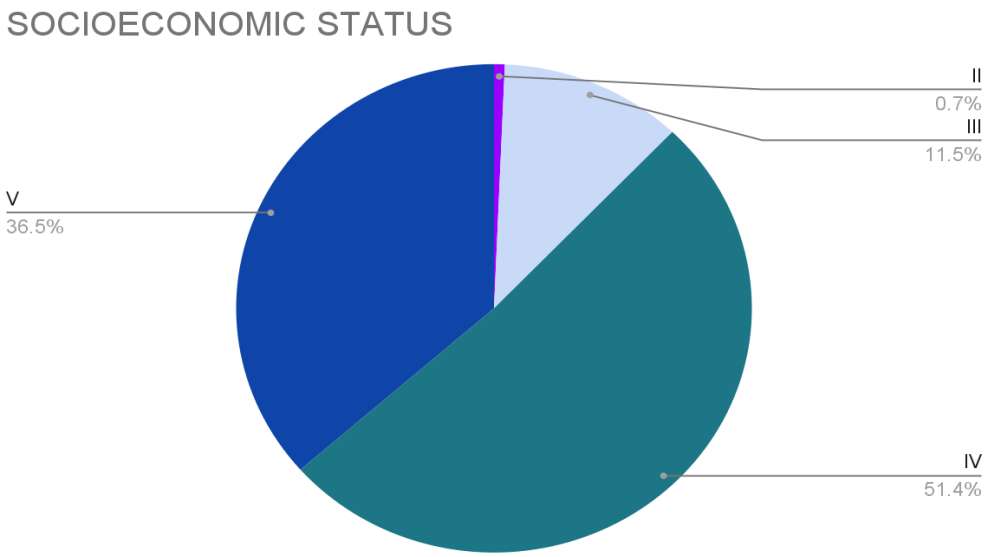


CHART 3: SOCIO ECONOMIC STATUS DISTRIBUTION

BOOKED STATUS

138 patients were admitted for safe confinement. Out of the total 138 cases, 134 (97.10%) cases were booked cases and 4 cases (2.90%) were unbooked cases. All the unbooked cases were multigravida and they were diagnosed to have heart disease only in this pregnancy

One neonate was admitted in neonatal intensive care unit for respiratory distress syndrome. There was no maternal or perinatal mortality among the unbooked cases .

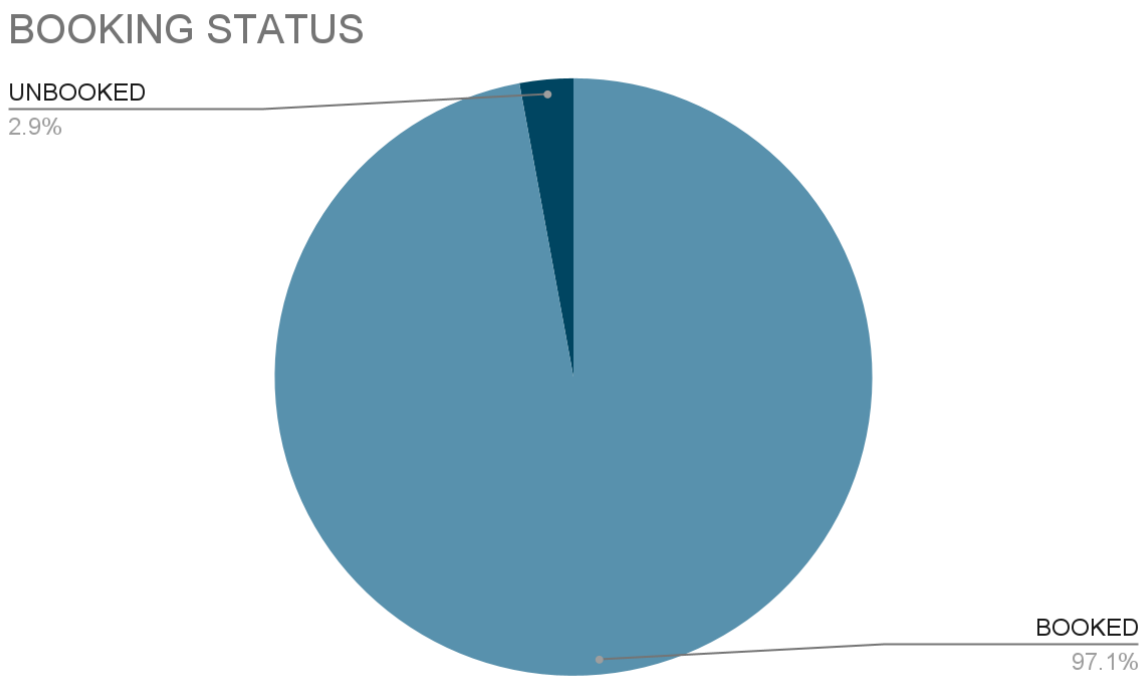


CHART 4: BOOKING STATUS

AGE DISTRIBUTION:

TABLE 7:

AGE	NO. OF CASES	PERCENTAGE
<19	14	9.45%
20-24	66	44.59%
25-29	50	33.78%
30-34	12	8.10%
>35	6	4.05%

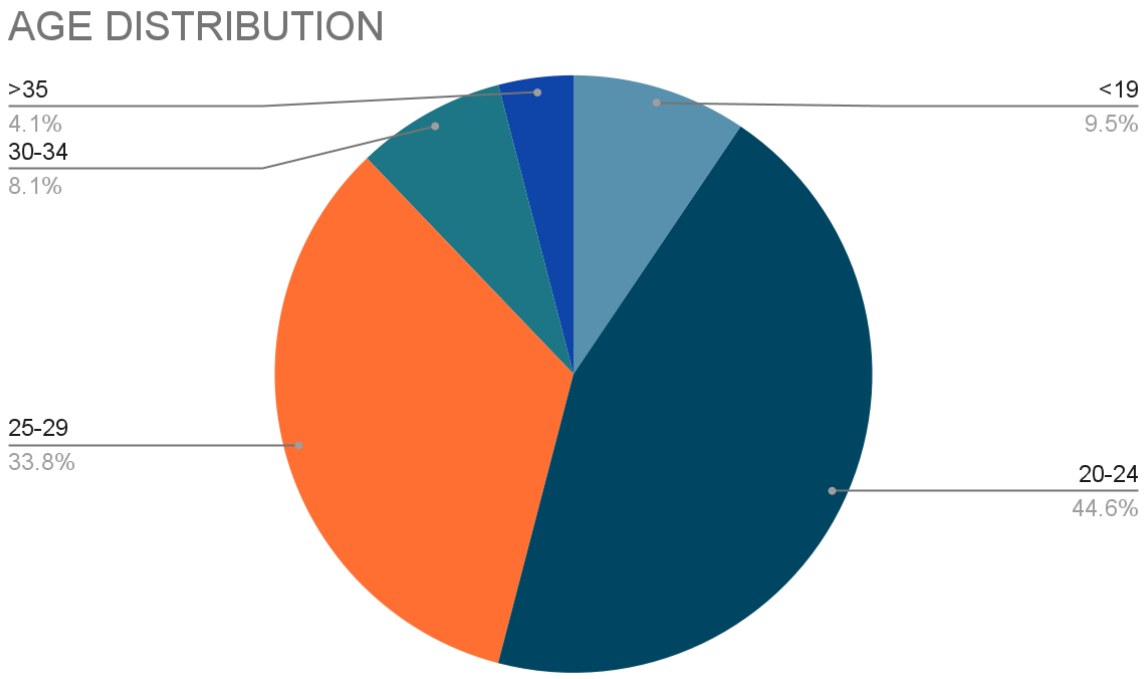


CHART 4: AGE DISTRIBUTION

PARITY DISTRIBUTION:

TABLE 8:

GRAVIDA	NO. OF CASES	PERCENTAGE
PRIMI	76	51.35%
2nd GRAVIDA	44	29.72%
3rd GRAVIDA	24	16.24%
G4 AND ABOVE	4	2.70%

Among the patients, 56% of the heart diseases were diagnosed for the first time in current pregnancy and 44% of the patients were known cases of heart diseases before the present pregnancy. Out of the 56% of the patients diagnosed in this pregnancy 40% were diagnosed only they were evaluated for cardiac symptoms and 60% were diagnosed during the routine echocardiogram done in the antenatal patients.

Routine evaluation of cardiac status in antenatal patients is being brought into practice nowadays, thus helping in diagnosis of new heart diseases and evaluation of known cases.

DISTRIBUTION OF TYPE OF HEART DISEASE:

TABLE 9:

TYPE OF HEART DISEASE	NO. OF CASES	PERCENTAGE
RHEUMATIC	62	41.89%
CONGENITAL	40	27.02%
MITRAL VALVE PROLAPSE SYNDROME	32	21.62%
PRIMARY PULMONARY HYPERTENSION	4	2.70%
CARDIOMYOPATHY	6	4.05%
HEART BLOCK	4	2.70%

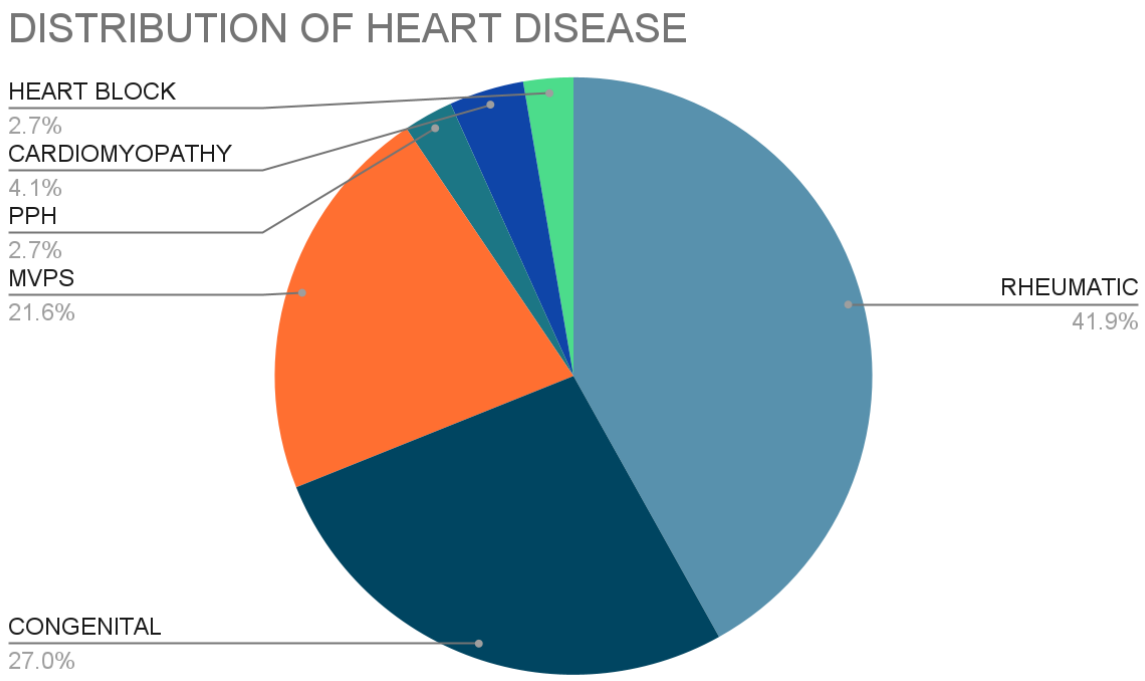


CHART 5: DISTRIBUTION OF HEART DISEASE

DISTRIBUTION OF RHEUMATIC HEART DISEASE:

The distribution of the valvular lesions in patients with rheumatic heart disease was studied. 27 patients had isolated mitral regurgitation. 14 patients had isolated mitral stenosis with or without pulmonary hypertension. 8 patients had mitral stenosis with mitral regurgitation. 6 patients had combined lesions ie.,stenotic and/ or regurgitation of more than one heart valve ie., mitral, aortic, tricuspid and pulmonary.

TABLE 10:

TYPE OF LESION	NO. OF CASES	PERCENTAGE
MR	27	33.87%
MS	14	22.88%
MS WITH MR	8	12.90%
MULTIVALVULAR LESIONS	6	9.67%
AS	2	3.22%
AR	2	3.22%
AS WITH AR	3	4.83%

DISTRIBUTION OF CONGENITAL HEART DISEASE:

The distribution of patients with congenital heart disease was studied and tabulated

A total of 40 cases with 11 different congenital heart diseases were identified.

TABLE 11:

TYPE OF HEART DISEASE	NO. OF CASES	PERCENTAGE
ASD	16	40%
VSD	7	17.5%
PDA	4	10%
PS	3	7.5%
BICUSPID AORTIC VALVE	1	2.5%
EBSTEIN ANOMALY	1	2.5%
DEXTROCARDIA	1	2.5%
EISENMENGER SYNDROME	1	2.5%
WPW SYNDROME	2	5%
TETRALOGY OF FALLOT	2	5%
IAS ANEURYSM	2	5%

DISTRIBUTION OF MITRAL VALVE PROLAPSE SYNDROME:

Distribution of Mitral valve prolapse syndrome in the patients were studied:

TABLE 12:

TYPE	NO. OF CASES	PERCENTAGE
NO FUNCTIONAL CHANGE	15	46.87%
MR	11	34.37%
MR/TR	4	12.5%
TR	1	3.125%
PR	1	3.125%

DISTRIBUTION ACCORDING TO NYHA STATUS:

The functional class of the patients according to the NYHA classification is shown in the following table. The classification of the functional status is done at the first time of presentation in our institution.

TABLE 13:

NYHA STATUS	NO. OF CASES	PERCENTAGE
I	92	62.16%
II	36	24.32%
III	12	8.10%
IV	8	5.40%

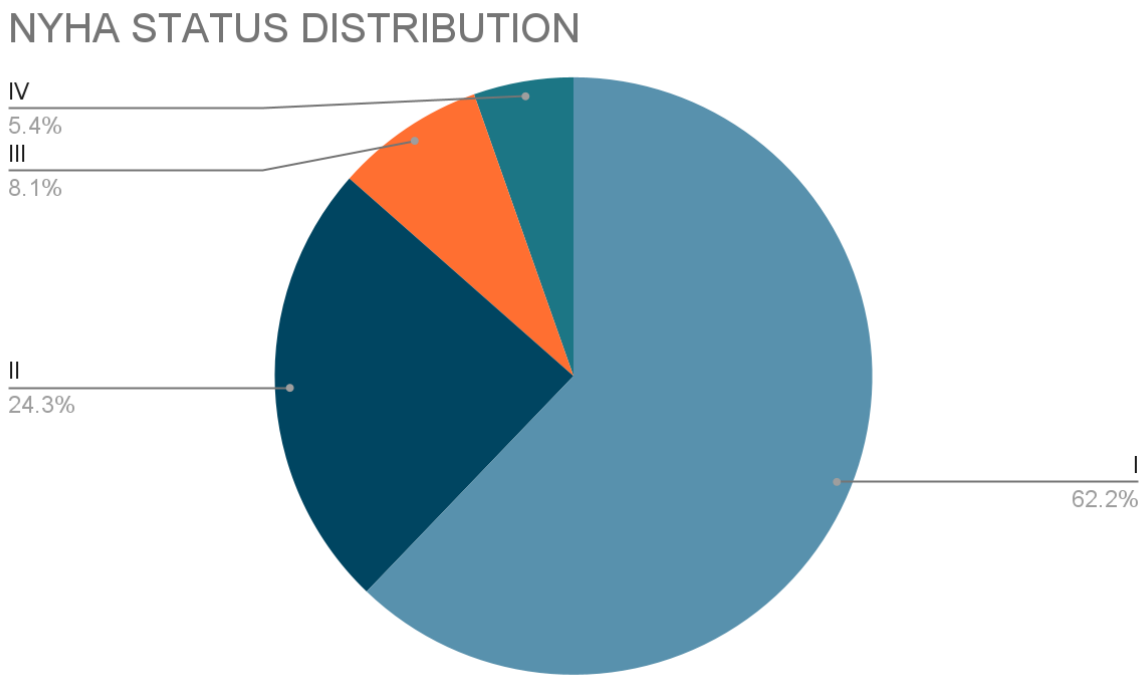


CHART 6: NYHA STATUS DISTRIBUTION

PREGNANCY OUTCOME IN RARE HEART DISEASE:

We had 2 cases of WPW syndrome admitted for safe confinement. One patient was multigravida and delivered by outlet forceps and the other was G2A1 and delivered by emergencyLSCS. There was no maternal or perinatal morbidity or mortality in these patients. There were 5 cases of cardiomyopathy in the study group. 1 case of dilated cardiomyopathy, and 4 peripartum cardiomyopathy cases were in the group. 3 cases of peripartum cardiomyopathy were delivered by vacuum assisted delivery and one case was taken up for LSCS. There was no maternal or foetal complication in dilated cardiomyopathy. Out of the 4 cases of peripartum cardiomyopathy, there was one maternal death and there was no perinatal mortality.

We had 4 cases of primary pulmonary hypertension identified. All cases were booked cases. All the patients were diagnosed to have primary pulmonary hypertension only after conception. All were primigravida. One case of primary pulmonary hypertension was delivered by LSCS in view of maternal cardiac status and IUGR baby. The mother died in post op period due to recurrent flare ups and pulmonary edema. No other morbidity or mortality was there in these patients.

There were 2 cases of 1st degree heart block and 1 case of 2nd degree heart block and 1 case of 3rd degree heart block. These patients were subjected to continuous

ECG monitoring and had no maternal or perinatal mortality during pregnancy and delivery.

COMPLICATIONS OF HEART DISEASE:

The various complications that developed in pregnancy complicated by heart disease were studied. 6 patients developed congestive cardiac failure, 11 patients had acute pulmonary edema, 1 patient developed atrial fibrillation, 2 patients had embolic manifestation, 2 patients developed supraventricular tachycardia, 1 patient had severe Right ventricular obstruction.

TABLE 14:

COMPLICATION	NO. OF CASES	PERCENTAGE
Acute pulmonary edema	11	47.82%
Congestive cardiac failure	6	26.08%
Atrial fibrillation	1	4.34%
Embolic manifestation	2	8.69%
Supraventricular tachycardia	2	8.69%
Severe Right ventricular obstruction	1	4.34%

MEDICAL DISORDERS ASSOCIATED IN THE STUDY GROUP:

The table below shows the various pregnancy related complications. 17 patients had anemia, 8 had gestational hypertension, 3 patients had pre-eclampsia, 3 patients had gestational diabetes, 12 patients had hypothyroid, 1 case was hyperthyroid, 1 patient had epilepsy, 9 patients had lower respiratory tract infection,1 patient had bronchial asthma, 3 patients were Rh- Negative, 1 had urinary tract infection. 3 of the patients had fever evaluated for infective endocarditis in the study group, treated with prophylactic antibiotics and turned out to be culture negative. Some of the patients had more than one diseases.

TABLE 15:

MEDICAL DISEASE	NO. OF CASES	PERCENTAGE
ANEMIA	17	28.81%
GHT	8	13.55%
PRE ECLAMPSIA	3	5.08%
GDM	3	5.08%
HYPOTHYROID	12	20.33%
HYPERTHYROID	1	1.69%
EPILEPSY	1	1.69%
LRTI	9	15.25%
BRONCHIAL ASTHMA	1	1.69%
Rh NEGATIVE	3	5.08%
UTI	1	1.69%

DISTRIBUTION OF SURGICALLY CORRECTED HEART DISEASES:

The patients who had heart diseases surgically corrected were evaluated. 21 patients were studied. 5 of the patients underwent closed mitral valve commissurotomy (CMC), 1 underwent balloon valvuloplasty, ASD closure was done in 8 patients, PDA ligation was done in 2 patients, VSD closure was done in 3 patients, mitral valve replacement was done in 2 patients.

Among the patients with Rheumatic heart disease, mitral valve surgery was the commonest surgery done. CMC was the commonest among the procedures followed by MVR.

Most of the patients who underwent mitral valve surgery were in NYHA class I or II. Surgical correction of congenital heart disease was the most common procedure in the study group especially ASD closure was the commonest procedure among the study group. These patients had no perinatal or maternal mortality or morbidity during the study period.

The maternal and foetal outcome was favourable in all the surgically treated patients. Except for one preterm death, there was no other maternal or foetal death. All these patients were hemodynamically stable during pregnancy, labour, delivery and puerperium.

OUTCOME OF PREGNANCY IN THE STUDY GROUP:

TABLE 16:

MODE OF DELIVERY	NO. OF CASES	PERCENTAGE
VAGINAL DELIVERY	86	62%
CAESAREAN SECTION	52	38%

Among the 138 patients, 86 patients delivered by vaginal route and 52 patients delivered by caesarean section.

DISTRIBUTION OF VAGINAL DELIVERY:

This table shows the split up of deliveries that occurred by vaginal route. 61 patients had normal vaginal delivery. 16 patients had vacuum extraction with episiotomy, 12 delivered by outlet forceps with episiotomy, 1 case of assisted breech delivery and 2 spontaneous expulsion of dead foetus with placenta and membranes in toto.

TABLE 17:

TYPE OF DELIVERY	NO. OF CASES	PERCENTAGE
NORMAL DELIVERY	61	66.30%
VACUUM EXTRACTION	16	17.39%
OUTLET FORCEPS	12	13.04%
ASSISTED BREECH	1	1.08%
SPONTANEOUS EXPULSION	2	2.17%

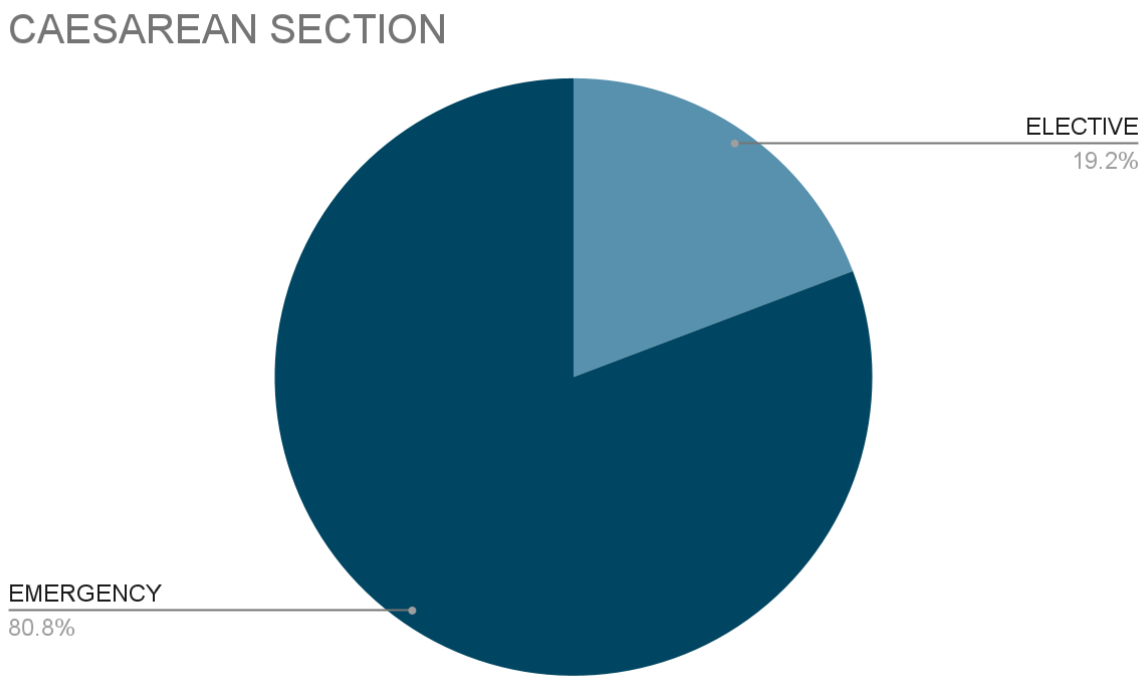
DISTRIBUTION OF CAESAREAN SECTION:

Out of the 52 caesarean sections done, the distribution of cases is studied. All caesarean sections were done for obstetric indication out of which 42 were Emergency caesarean sections and 10 were Elective caesarean sections.

TABLE 18:

CAESAREAN SECTION	CASES	PERCENTAGE
ELECTIVE	10	19.23%
EMERGENCY	42	80.76%

CHART 7:

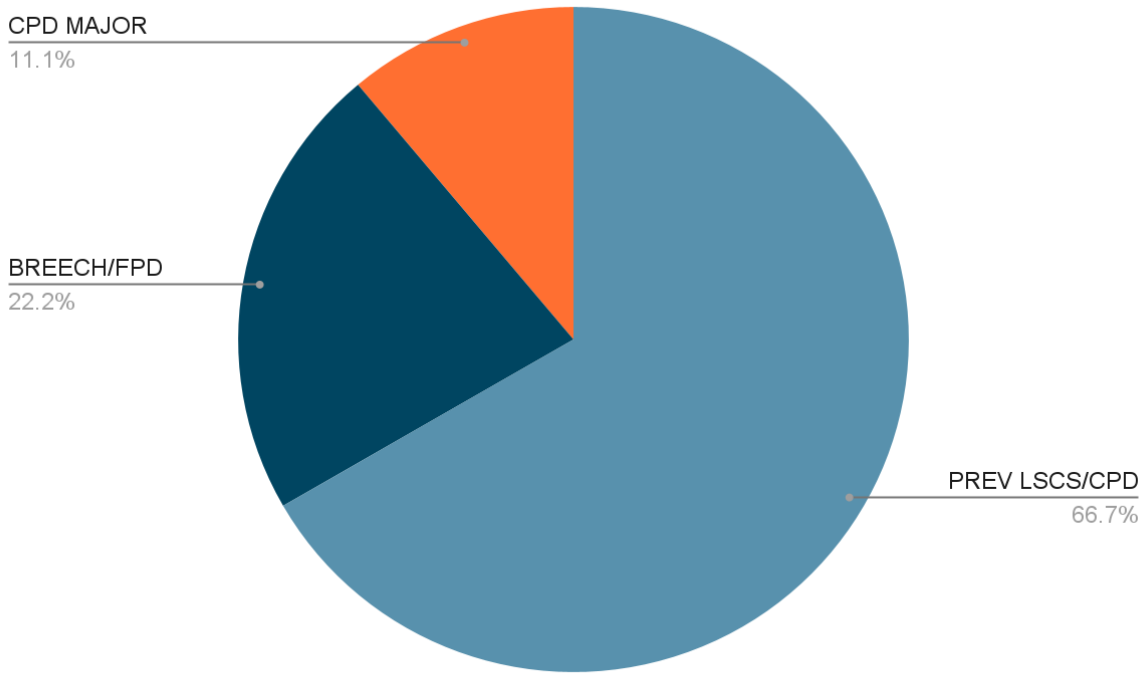


INDICATIONS FOR CAESAREAN SECTION:

The various indications for termination of pregnancy in heart disease patients by caesarean section were studied both in emergency and elective cases and were tabulated.

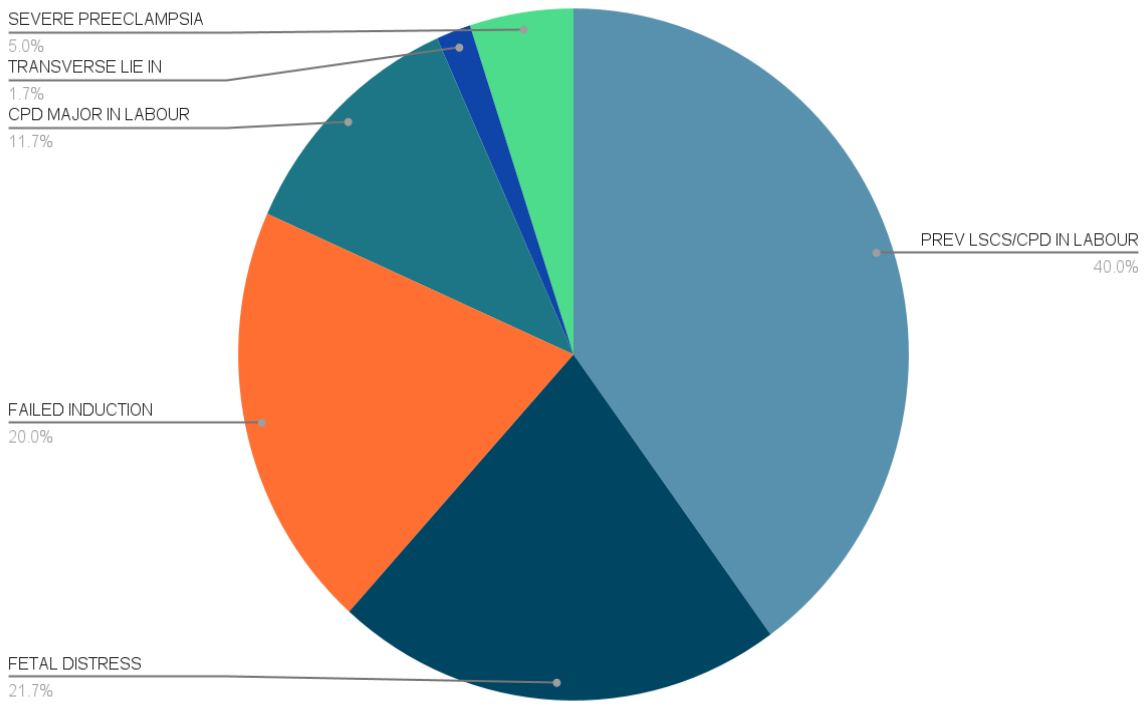
INDICATIONS FOR ELECTIVE CAESAREAN SECTION:

CHART 8:



INDICATIONS FOR EMERGENCY CAESAREAN SECTION:

CHART 9:



DISTRIBUTION OF BIRTH WEIGHT:

The following table shows the distribution of birth weight of the babies delivered. 140 babies were delivered, of which 2 were twin deliveries. Birth weight of 6 babies were ≤1.5 kg, weight of 12 babies were between 1.51 – 2 kg, 40 babies were between 2.01 – 2.5 kg, weight of 50 babies was between 2.51 – 3 kg, 30 babies were between 3.01 – 3.5 kg and 2 babies were above 3.51 kg.

TABLE 19:

BIRTH WEIGHT	NO. OF BABIES	PERCENTAGE
≤1.5 kg	6	4.28%
1.51 – 2 kg	12	8.57%
2.01 – 2.5 kg	40	28.57%
2.51 – 3 kilos	50	35.71%
3.01 – 3.5 kg	30	21.42%
>3.5 kg	2	1.42%

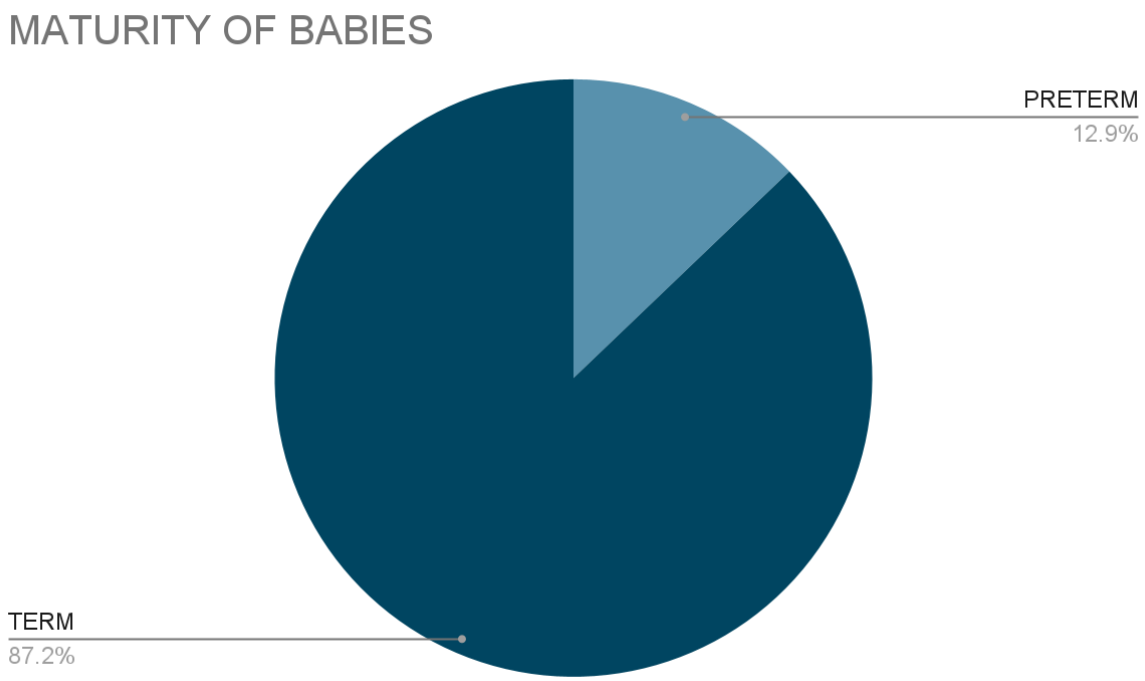
DISTRIBUTION ACCORDING TO MATURITY OF BABIES:

Out of the 140 babies delivered, 18 babies were preterm and 122 babies were term babies. There was no evidence of congenital heart disease in the babies born to the mothers with congenital heart disease. Out of the 18 preterm babies 2 were extreme preterm and delivered as spontaneous expulsion with placenta and membranes in toto.

TABLE 20:

MATURITY	NO. OF BABIES	PERCENTAGE
PRETERM	18	12.85%
TERM	122	87.15%

CHART 10:



DISTRIBUTION OF PERINATAL MORBIDITY:

Out of 138 live born babies, 32 babies required admission into the neonatal intensive care unit. 20 admissions were due to respiratory distress, 5 admissions were due to SGA, 4 were due to IUGR, 3 were due to meconium stained liquor.

TABLE 21:

PURPOSE OF ADMISSION	NO. OF BABIES	PERCENTAGE
RESPIRATORY DISTRESS	20	62.5%
SGA	5	15.62%
IUGR	4	12.5%
MECONIUM STAINED	3	9.37%

DISTRIBUTION OF PERINATAL DEATH:

Reasons for perinatal mortality was respiratory distress syndrome in 2 preterm babies, prematurity in 2 babies, and severe IUGR and birth asphyxia in one term baby. Perinatal mortality is 3% in the study.

CONTRACEPTION:

The following table shows the different methods of contraception- both temporary and permanent among the 148 patients included in the study. The methods of contraception used in the patients were either temporary or permanent. No method of contraception was used in 34 patients due to some maternal or foetal complications and were advised to adopt barrier contraceptive measures.

The distribution of the contraceptive measures is discussed: Out of the patients 62 Copper-T insertions were done which included both postplacental and interval copper- T insertions, 38 had concurrent sterilisation with caesarean section, 10 patients underwent puerperal sterilisation, 4 patients had concurrent sterilisation with MTP.

Out of the 62 copper –T insertions, 56 were following deliveries and 6 were following MTP.

TABLE 22:

DISTRIBUTION OF METHODS OF CONTRACEPTION:

METHOD	NO. OF CASES	PERCENTAGE
COPPER T	62	54.38%
CONCURRENT STERILISATION	38	33.33%
PUERPERAL STERILISATION	10	8.77%
MTP WITH STERILISATION	4	3.50%

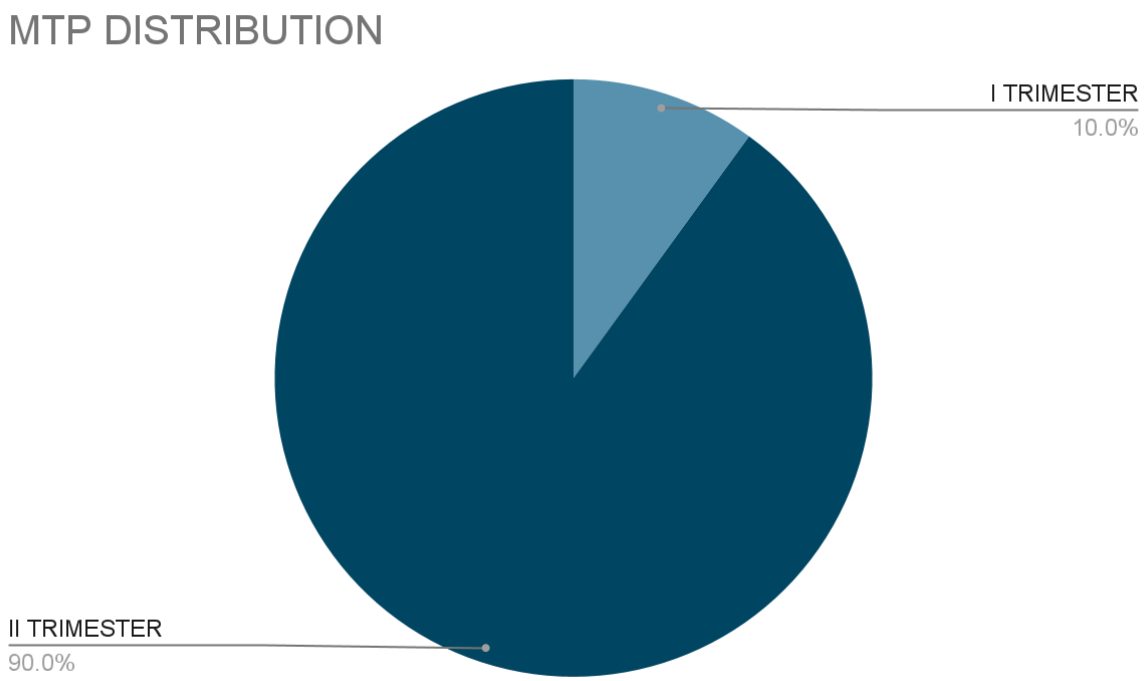
In the study group, 38 patients underwent concurrent sterilisation with LSCS,10 patients underwent puerperal sterilisation following vaginal deliveries. Out of the 148 patients, 35.10% of the patients adopted a permanent method of sterilisation.

DISTRIBUTION OF MTP:

TABLE 23:

TRIMESTER	NO. OF CASES	PERCENTAGE
I	9	90%
II	1	10%

CHART 11:



Only one MTP was done in the II trimester and 9 were done in the I trimester. MTP was done in one primigravida because of Eisenmenger's syndrome.

DISTRIBUTION OF USE OF CONTRACEPTION FOLLOWING MTP:

TABLE 24:

MODE OF CONTRACEPTION	NO. OF CASES	PERCENTAGE
COPPER T	6	60%
STERILISATION	4	40%

Out of the 10 patients admitted for MTP, 9 patients underwent MTP as they had completed their family, 1 patient underwent MTP as it was indicated because of their heart disease. As mentioned in the table, 4 patients underwent tubectomy, 6 patients had copper-T insertion following MTP. All patients were stable during the procedure and after the procedure and discharged without any complication. The first trimester MTPs were done by manual vacuum aspiration using an MVA syringe.

ANALYSIS OF DEATH IN PREGNANT PATIENTS WITH HEART DISEASE:

There were 4 deaths among the total 148 patients studied. Cause of death was cardio respiratory arrest in 2 cases, pulmonary oedema in 1 case and pulmonary embolism in 1 patient. All cases had been booked and they were referred to tertiary care centre for further management of cardiac disease.

Functional status of 3 patients was class IV at the time of admission and class II for 1 patient. 1 of the patients had pre- eclampsia and anaemia as coexisting medical disorders and the others had no comorbidities.

1 patient was a known case of heart disease before pregnancy and the other 3 were newly diagnosed. 1 of the patients developed myocarditis and 2 patients developed peripartum cardiomyopathy in the present pregnancy. 2 of the cases were started on anti-failure drugs and despite treatment the condition deteriorated.

One patient with critical mitral stenosis acquired ventilator associated pneumonia infection which proved to be fatal in her on the 4th postnatal day, the patient had severe to critical mitral stenosis.

1 patient had primary pulmonary hypertension, two patients had peripartum cardiomyopathy, and one had myocarditis. No patient had undergone any surgical correction of heart disease before pregnancy. All the patients were less than 30 years of age.

DISCUSSION

INCIDENCE

In our study, the incidence of heart disease among the pregnant patients was 1.43%. It is comparable and is similar to the incidence reported by various other authors in their study

AUTHOR	YEAR	INCIDENCE
MUDALIAR- MENON	1972	0.97%
CHIA	1998	0.7%
DeSWIET& FIDDLE	1999	0.5%
WILLIAMS	2001	1%
IOG STUDY	2011	1.41%

SOCIO ECONOMIC STATUS:

The outcome of the patients with heart disease is proved to be influenced by their socio economic status. 87.83% of the patients included in our study were class IV and V. Comorbid conditions like anaemia, underweight and other infections are more common in the lower socioeconomic status and these conditions in turn complicate the pregnancy affecting the maternal and foetal outcome in a significant manner. Nafeesa and associates published a report in 1985 stating that 92% in their

study group belonged to class IV and V. About 65.8% of the study group patients were from overcrowded urban areas.

ANTENATAL CARE:

In the study group, 2.7% patients were unbooked and 97.3% were booked. Majority of the study population was booked in coimbatore and surroundings. Most of the patients who presented with class IV NYHA status were booked elsewhere and were referred to tertiary care centre at a later stage for institutional management. All the unbooked cases were multigravida and they were diagnosed to have heart disease only in this pregnancy. There was no maternal or perinatal mortality among the unbooked.

56% of the heart disease patients in the study group were diagnosed to have heart disease only during this pregnancy and 44% were known cases of heart disease before this pregnancy. Patients who had regular routine antenatal check-up and tertiary care follow up had a good maternal and perinatal outcome.

AGE AND PARITY:

Mudaliar and Menon in their study stated that in patients with Rheumatic Heart Disease the cardiac condition worsens with time preferably due to the progressive nature of the organic lesion and rather than the increasing parity of the patient.

Among the study group, 87.82% of the cardiac patients were below 30 years of age. Though there is a consensus that, the younger the age the better is the prognosis of the patient, it also depends on the functional status of the patient.

In a report published by Dr.LalithaSubramaniam, 20.5% patients with heart disease were grand multigravida whereas in the present study, the grand multigravida accounted for 1.8% of the study group and this illustrates the betterment in the knowledge regarding the risk of pregnancy among the patients with heart disease and increase in the awareness about various sterilisation methods for limiting the family size.

TYPE OF HEART DISEASE:

In the various studies conducted by Mudaliar and Menon (1972) Kamala Sidkar(1980), the incidence of rheumatic heart disease among 69 the pregnant cardiac patients is 90 – 96%. The studies published in western countries by Tan and De Swiet in 1998 stated that the incidence of rheumatic heart disease is 12%. Chia in 1998 gave a report that the incidence of rheumatic heart disease is 61.6% and the incidence of congenital heart disease is 38.4% In our study, 62% of the patients had heart disease of rheumatic origin. 40% patients were diagnosed to have congenital heart disease. 32% of the patients had Mitral valve prolapse. 4% patients were

diagnosed to have primary pulmonary hypertension, 6% had cardiomyopathy. Heart block in 4 patients, and viral myocarditis in one case.

RHEUMATIC HEART DISEASE

According to Szekely and Snaith, the distribution is as follows: dominant mitral stenosis lesion is seen in 90%, mitral regurgitation in 6.6%, aortic stenosis in 1% and aortic regurgitation is seen in 2.5%.

In contrast to this report, in our study, among the rheumatic heart disease patients, isolated mitral regurgitation was seen in 33.87%,isolated mitral stenosis is seen in 22.88%, aortic stenosis was seen in 3.22%, 3.22% patients had aortic regurgitation, mitral stenosis with regurgitation was seen in 12.90% and aortic stenosis with regurgitation in 4.83% and 9.67% had multivalvular lesions. The diagnosis of these lesions is very important in management of these patients during labour and delivery. The use of intravenous fluids mitral stenosis has to be restricted whereas in case of aortic stenosis, the patient is managed on the wet side.

CONGENITAL HEART DISEASE

The incidence of congenital heart disease is comparable with the incidence of congenital heart disease in the general population. In our study, 40% had atrial septal defects. 17.5% patients had ventricular septal defect, 10% patients had patent

ductus arteriosus, 7.5% had pulmonic stenosis, 5% had tetralogy of fallot and 5% had interatrial septal aneurysm. Incidence of Wolff Parkinson White syndrome was 5%. There was one case each of ASD with MVPS, VSD with MVPS, Dextrocardia without situs inversus, Eisenmenger syndrome.

The maternal and perinatal outcome in these patients was influenced by the functional status of these patients according to the NYHA classification. The outcome was generally good in most of these patients especially in those who had undergone surgical correction of the defects before pregnancy. In one case of Eisenmenger syndrome, pregnancy was terminated at 11 weeks due to poor maternal outcome in these patients. The case of Tetralogy of Fallot was of acyanotic type. The inter atrial septal aneurysm was congenital in origin and is associated with atrial septal defect in 2 patients, There was no thromboembolic event among these patients. One case of Ebstein's anomaly had a successful pregnancy outcome without any maternal or fetal complications. In 7 patients Copper-T was inserted and all others adopted the permanent methods of sterilisation.

MITRAL VALVE PROLAPSE;

Mitral valve prolapse syndrome is most often seen in relation to pregnancy as its frequency of occurrence is common in young women (15%). In the present study, 21.62% of the pregnant patients had mitral valve prolapse syndrome. In most of the cases, mitral valve prolapse was diagnosed during the routine cardiology workup

done in the antenatal patients and the patients were totally asymptomatic. Although it is an incidental finding, patients with this syndrome might develop complications like arrhythmias, subacute bacterial endocarditis or any thromboembolic complication. In our study, 46.87% of patients diagnosed to mitral valve prolapse had no functional derangement like regurgitation. 34.37% patients had mitral regurgitation, 3.125% had tricuspid regurgitation,12.5% patients had both Mitral and tricuspid regurgitation and 3.125% had pulmonary regurgitation. Mitral valve prolapse associated with regurgitant lesions has been found to be a significant risk factor for bacterial endocarditis.

RARE CARDIAC DISEASES IN PREGNANCY:

We had 2 cases of WPW syndrome admitted for safe confinement. One patient was multigravida and delivered by outlet forceps and the other was G2A1 and delivered by emergency LSCS. There was no maternal or perinatal morbidity or mortality in these patients. There were 4 cases of cardiomyopathy in the study group. 1 case of dilated cardiomyopathy and 4 peripartum cardiomyopathy cases were in the group. There was no maternal or fetal complication in dilated cardiomyopathy. Out of the 4 cases of peripartum cardiomyopathy, there were two maternal deaths and one neonatal death. We had 4 cases of primary pulmonary hypertension during the study period. All cases were booked elsewhere and where referred to IOG in II and III

trimesters for further management. All the patients were diagnosed to have primary pulmonary hypertension only after conception. All were primigravida. There was 1 maternal death in patients with pulmonary hypertension. Thus primary pulmonary hypertension had 25% mortality in our institute. No other morbidity or mortality was there in these patients.

There was a case of dextrocardia evaluated for situs inversus, a case of Ebsteins anomaly diagnosed before pregnancy and 2 cases of tetralogy of Fallot all discharged without any mortality and morbidity.

COMPLICATIONS OF HEART DISEASE IN PREGNANCY:

Among the patients who developed cardiac complications, rheumatic heart disease was the most common underlying heart disease. Various complications seen in the patients with heart disease were acute pulmonary edema, congestive cardiac failure, supraventricular tachycardia, pulmonary embolism and severe right ventricular outlet obstruction.

CARDIAC FAILURE:

In the study group,4 patients were in class IV NYHA status and had cardiac failure at the time of admission. 4 out of the 6 patients with failure improved with anti-failure measures and supportive care in the intensive care unit. These patients

were managed with expert opinion from the cardiologists and anaesthesiologists. Development of decompensation in the heart disease patients depends on the functional status of the patient before pregnancy and the hemodynamic burden imposed on the diseased heart by pregnancy

ACUTE PULMONARY EDEMA:

Most common heart disease which developed Pulmonaryedema as a complication was rheumatic mitral stenosis. In our study group, 11 patients (47,82%) developed pulmonary edema. 0ut of these 11 patients, 5 patients had severe mitral stenosis, 1 had critical mitral stenosis and 1 patient had mitral valve prolapse with preeclampsia. In one patient with severe mitral stenosis acute pulmonary edema was precipitated by Lower respiratory tract infection. Pulmonary edema has been the single most important cause of death in patients with heart disease. 2 out of 11 patients with pulmonary edema could not be revived and they succumbed to the disease. The risk of developing pulmonary edema coincided with period of pregnancy in which there is increased volume expansion and increased hemodynamic overload on the heart. In one patient pulmonary edema occurred in 32 weeks, in 2 patients it occurred near term and in 8 patients it developed after delivery. All patients were connected to ventilator with anti-failure measures and inotropic agents as per cardiologist’s advice. 2 out of 11 patietns could not be

revived and they expired. In cases of severe mitral stenosis, the patients were already on anti-failure drugs and despite these measures they developed pulmonary edema. This emphasises the importance of pre-conceptional counselling and early termination of pregnancy if required when the patient is diagnosed to have a heart disease with poor cardiac reserve, so that death can be prevented in these patients.

SUPRAVENTRICULAR TACHYCARDIA:

Supraventricular tachycardia complicated 2 pregnancies with atrial septal defect. Both patients were at term. 1 case developed the complication in the antenatal period and the other patient developed it 12 hours after delivery. Because of the intensive treatment and the prompt use of drugs, the patients were saved.

PULMONARY EMBOLISM:

One patient with a primary pulmonary hypertension developed pulmonary embolism on the 2nd postnatal day and expired. The baby was a preterm and died due to respiratory distress. One patient with Ventricular septal defect developed pulmonary embolism and was managed with anticoagulants.

SEVERE RIGHT VENTRICULAR OUTLET OBSTRUCTION:

One case of acyanotic tetralogy of fallot had severe right ventricular outlet obstruction. The patient was under critical care and intensive monitoring and did not develop any complications and was discharged in a stable condition.

ATRIAL FIBRILLATION:

Szekely and Snaith in 1989 reported that atrial fibrillation increases the risk of thromboembolism and heart failure. Pulmonary edema can develop in pregnancy when atrial fibrillation occurs in patients with mitral stenosis leading to the increase in maternal and 79 perinatal morbidity and mortality. The incidence of atrial fibrillation reported by Szekely and Snaith(1974) was 6.5%. None of the patients in our study developed atrial fibrillation.

COMORBID CONDITIONS:

ANAEMIA:

In our study 28.81% patients had co-existing anaemia. 10 patients had mild anaemia, 6 patients had moderate anaemia and 1 patient had severe anaemia. Improved antenatal care has decreased the incidence of anaemia in the study group. The patient with severe anaemia is an unbooked case. Patients with anaemia in our study belonged to low socio-economic status. Anaemia was linked to various

complications like preterm delivery, IUGR, heart failure etc. 2 patients had preterm delivery, 1 patient had IUGR. There was no maternal or perinatal mortality.

GESTATIONAL HYPERTENSION AND PREECLAMPISA:

In our study, 8 patients with heart disease had gestational hypertension and 3 patients had pre-eclampsia. The association of preeclampsia with heart disease had a poor perinatal outcome due to further compromise in blood flow. 2 patients with pre-eclampsia had IUD and one patient with gestational hypertension developed grade –II abruption and IUD. 2 more babies had respiratory distress but recovered.

RESPIRATORY COMPLICATIONS:

4 patients had lower respiratory infection and one had bronchial asthma. One patient with mitral stenosis acute pulmonary edema was triggered by lower respiratory tract infection. Cardiorespiratory disorder in a heart disease patient further reduced the cardiopulmonary reserve and fetal complications are more due to increased hypoxic stress.

OTHER CONDITIONS

3 of the patients had gestational diabetes, 12 patients had hypothyroidism, 1 had hyperthyroidism,and 1 had epilepsy. 37 cases had oligohydramnios and 2 cases had polyhydramnios, 1 had placenta praevia, 2 had PPROM and 8 had PROM and 1 had UTI.

INFECTIVE ENDOCARDITIS:

2 patients in the study group had fever but no case of infective endocarditis was noted in our study group. All patients received antibiotics during parturition and infective endocarditis prophylaxis was given for all patients with valvular lesions with more pressure gradient across the valves.

SURGICALLY CORRECTED CONGENITAL HEART DISEASE:

8 patients had undergone ASD closure in the childhood and adolescence period. 3 patients with VSD had undergone closure and in 4 patients PDA ligation was done. All these patients had a favourable pregnancy outcome and they were hemodynamically stable during pregnancy, labour, delivery and puerperium. There was no maternal mortality in this study group.

PURPOSE OF ADMISSION:

In our study, 93.3% patients with heart disease were admitted for safe confinement and 6.7% patients were admitted for medical termination of pregnancy. 1 patient was admitted in the II trimester for MTP. 10 patients were admitted for anemia evaluation and correction and was diagnosed to be a case of heart disease. 1 out of the 6 patients in heart failure was an unbooked case/ previous LSCS and she was admitted for severe anaemia in labour. 3 patient were admitted for pre-eclampsia. 1 patient was admitted in second trimester was admitted for PPROM.

MODE OF DELIVERY:

In the study group, among the 138 patients admitted for safe confinement 62% had vaginal delivery, 38% delivered by caesarean section. Caesarean section was done for obstetric reasons. In a study published by Dr.K.Sidkar, 92% of the study group delivered vaginally and caesarean section was done in 1.7% and 3.5% patient died undelivered. In the present study the rate of caesarean sections done had increased but they were done mainly for obstetric indications.36.14% were primary section and 63.86% were repeat caesarean section. In our study, 62% delivered vaginally and 30.43% were prophylactic outlet forceps and vacuum extraction delivery. 16 cases of vacuum extraction,12 cases of outlet forceps delivery, 1 assisted breech delivery and 2 spontaneous expulsion of dead fetus were under the study.

TWIN PREGNANCY OUTCOME IN HEART DISEASE:

Twin pregnancy in heart disease predisposes the mother to greater risk of decompensation due to much higher hemodynamic overload in these patients. There were 2 twin pregnancies among the patients studied. 1 out of the 2 pregnancies delivered preterm and one delivered at term. All 2 underwent emergency caesarean section, 1 for fetal distress and for previous LSCS with 1st twin in breech presentation. The preterm babies were admitted in neonatal care unit due to respiratory distress. Weights of these babies were between 1.1kg and 2.6 kg. There was no maternal or perinatal mortality among these patients. This indicates that if the patient has good pre pregnancy cardiac reserve, the pregnancy will have a favourable maternal and perinatal outcome but the incidence of preterm labour is increased in the study group.

FETAL OUTCOME:

Surgne and associates in 1981 proposed that the perinatal outcome in rheumatic heart disease in pregnancy is generally good and comparable to the patients without heart disease.

MATURITY AND BIRTH WEIGHT:

In our study 12.85% of the babies were preterm babies and 87.15% were term babies. 5 babies were SGA and 4 were IUGR babies. 41.42% of the babies had birth

weight below 2.5 kg. 57.13% had birth weight between 2.51 – 3.5 kg and 1.42% babies were above 3.51 kg. One baby was 4.1 kg.

There were no congenital anomalies or congenital heart disease in the babies born to mothers with congenital heart disease. Incidence of congenital anomalies among babies born to mother with congenital heart disease in various studies is shown in the following table:

AUTHOR	INCIDENCE
Whitemore et al 1982	10%
Shime et al 1987	13%
Perloff 1997	5-10%

PERINATAL MORTALITY RATE:

There were 2 intra uterine deaths and 4 perinatal deaths out of the 140 babies delivered. Perinatal mortality was seen in 3 preterm babies and 1 term baby accounting for 2.8% of the babies in the study.

Reasons for perinatal mortality was respiratory distress syndrome in 2 preterm babies, prematurity in 1 baby, and severe IUGR and birth asphyxia in one term baby. There is a decline in the perinatal mortality and morbidity with improvement in the antenatal care, prompt diagnosis and treatment of the comorbid conditions

and treatment of infection if present, institutional delivery of the patients with heart disease and advancements in neonatal care.

CONTRACEPTION:

According to Cheslay in 1978, the severity of rheumatic heart disease increases with age as there is progressive endomyocardial changes in the diseased heart. Hence the patients with rheumatic heart disease are generally advised to complete their family at a younger age with an inter pregnancy interval of at least 2 to 3 years. This emphasises the counselling of the patient regarding the temporary and permanent methods of contraception. The use of improper contraceptives can be even life threatening in heart disease patients according to Brenner in 1975. Somerville in 1998 stated that patients with heart disease can be treated as any normal pregnant patient with regard to the use of contraceptives. In our study out of the total 148 patients, 114 patients adopted some form of contraception. Out of the 114 patients, 45.60% patients had undergone permanent sterilisation. All the patients had at least one live child. Out of the 45.60% patients, 8.77% underwent puerperal sterilisation, 33.33% patients underwent concurrent sterilisation with LSCS and 3.50% underwent tubectomy following MTP.

MEDICAL TERMINATION OF PREGNANCY:

In 90% of cases, MTP was done in the I trimester by manual vacuum aspiration. 1 patient underwent II trimester MTP. II trimester MTP was performed using oral mifepristone and vaginal misoprostol without any complication. Out of the 10 patients admitted for MTP, all patients underwent MTP as they had completed their family and MTP was done as per cardiologist's advice. Copper- T was inserted in 60% patients following MTP. And 40% underwent tubectomy. All patients were stable during the procedure and after the procedure and discharged without any complication.

MATERNAL MORTALITY:

Maternal mortality in relation to heart disease in pregnancy has declined over the past 2 decades. Yet heart disease continues to be an important cause of maternal mortality in developing countries. Case fatality rate of heart disease in the present study is 2.7%.

The statistics show that heart disease still contributes significantly to maternal mortality even in the most developed countries like the United States. In the U.S. heart disease continues to be the cause for 10% of maternal death and this rate has remained relatively constant over the past 5 decades according to Eastby (1998). Case fatality rate of heart disease in pregnancy in developing countries continues to

be high ranging from 3 -6% mainly because of anaemia, lack of prenatal care and emergency admissions.

SUMMARY

- In the Department of obstetrics and gynecology, Coimbatore medical college and hospital, Coimbatore during the study period from July 2020 to June 2021.
- 148 cases of heart disease complicating pregnancy were studied.
- The incidence of heart disease in pregnant patients is 1.43%.
- 65.8% were admitted from overcrowded areas.
- 87.83% of the patients with heart disease belonged to class IV and V socioeconomic status.
- 97.3% patients in the study group were booked cases.
- 56% of the patients were diagnosed to have heart disease during pregnancy and 44% were diagnosed before pregnancy.
- 87.82% of the patients were young and were less than 30 years of age.
- Only 1.8% of the study group were grand multigravida.
- 62% had rheumatic heart disease, 40% had congenital heart disease, 32% had mitral valve prolapse syndrome, 4% had primary pulmonary hypertension and 6% had cardiomyopathy.
- Isolated cases of mitral stenosis contributed to 14% of the cases and mitral stenosis was seen in combination with other valvular lesions in 20.01% of the patients.

- ASD is the most common congenital heart disease seen in the study group with an incidence of 40%.
- 21.62% patients had MVPS in the study group.
- 114 patients adopted some form of sterilisation out of which 45.60% underwent permanent sterilisation. Copper – T was inserted in 54.38% of the patients.
- The perinatal mortality in the study group was 2.85%. Maternal mortality was 2.7% in our study group.
- Among the rare heart diseases, 1. Primary Pulmonary Hypertension patients had 25% mortality. 2. Peripartum cardiomyopathy had 50% mortality.

CONCLUSION

Heart disease in pregnancy continues to be the major cause of maternal mortality, preterm birth and perinatal mortality. Favourable outcome in pregnancies complicated by heart disease depends on the following factors:

1. Age, socioeconomic status
2. Functional capacity of the heart and hemodynamic status of the patient
3. Early Booking and comprehensive antenatal care
4. Comorbid conditions
5. Quality of medical care

Early termination of pregnancy and prompt use of permanent sterilisation methods improve the survival of women with high risk cardiac disease. Once the pregnant patient seeks medical care, risk stratification is achieved and pregnancy can be continued in low risk group and the patients in high risk group are counselled for termination if necessary.

Multidisciplinary approach with a team of obstetricians, cardiologist, anaesthetist, neonatologist combined with patient education provides the best opportunity to continue pregnancy with a good maternal and perinatal outcome. Maternal mortality in heart disease patients can be brought down significantly by effective preconceptional counselling, and improvements in medical, surgical, antenatal, intrapartum, and postnatal care and effective adaptation of contraception.

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PROFORMA

NAME: IP.NO: LMP: DOA:

AGE: UNIT: EDD: DOD:

SEX: GPLA: DODIS:

ADDRESS: BOOKING STATUS:

IMMUNISATION:

1.G.A. AT PRESENTATION :

ASSOCIATED PREGNANCY :

COMPLICATION

TYPE OF HEART DISEASE : CHD/ RHD/ OTHERS

ANATOMICAL LESION :

NYHA CLASS :

ASSOCIATED FACTORS : OBESITY/ANAEMIA/FEVER/

INFECTIONS ETC.

PAST OBSTETRIC OUTCOME: EVENTFUL/ UNEVENTFUL

WHAT AND WHEN

PAST MEDICAL HISTORY : Diagnosed/Undiagnosed

When

COURSE :

QUALITY OF CARE :

SURGERIES IF ANY :

IUGR :

CONGENITAL ANOMALIES :

ADMISSION TO NICU :

PUERPERIUM : EVENTFUL/UNEVENTFUL

CONTRACEPTIVE ADVICE :

STERILISATION : TYPE :

ANAESTHESIA :

WHEN DONE :

POST OPERATIVE PERIOD : EVENTFUL/UNEVENTFUL

MATERNAL MORTALITY :

PERINATAL MORTALITY :

INVESTIGATIONS :

PRESENT PREGNANCY

1. Effect of Pregnancy on Heart Disease :

Heart failure/ Arrhythmias / pulmonary

Edema

Management :

2. Effect of Heart Disease on Pregnancy :

Preterm Labour/ IUGR :

Management :

3. Labour Stages

First stage:

Duration :

Second stage: Duration :

Prophylactic forceps

/ vacuum :

Third stage : Complications :

Management :

4. Caesarean Section : Elective/ Emergency

Indication :

Type of Anaesthesia :

Complications :

Management :

5. Baby – Male/ Female :

Birth weight :

APGAR :

Preterm / Term / Postdated :

CONSENT FORM

STUDY TITLE:

STUDY OF **"MATERNAL AND FETAL OUTCOME IN HEART DISEASE**

COMPLICATING PREGNANCY"

STUDY CENTRE:

COIMBATORE MEDICAL COLLEGE AND HOSPITAL

PARTICIPANT NAME: AGE: SEX: LD NO:

I confirm that I have understood the above study. I have the opportunity to ask. The question and all my questions and doubts have been answered to my satisfaction.

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason. I understand the investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I understand that my identity will not be revealed in any information released to third parties of published, unless as required under the law. I agree not to restrict the use of any results that arise from the study.

I hereby consent to consent to participate in this study titled **"MATERNAL AND FETAL**

OUTCOME IN HEART DISEASE COMPLICATING PREGNANCY"

Signature of investigator: Place:

Study investigator's name: Date:

Signature / thumb impression of patient:

ஒப்புதல் படிவம்

பெயர் :

பாலினம் :

முகவரி :

வயது :

கோவை அரசு மருத்துவக் கல்லூரியில் kfg;ngW kw;Wk; mWitrpfpr;ir பட்ட மேற்படிப்பு பயிலும் khztd; jpnd#;.vk;.Mh;. மேற்கொள்ளும் **"MATERNAL AND FETAL OUTCOME IN HEART DISEASE COMPLICATING PREGNANCY"**" குறித்த அனைத்து விவரங்களையும் கேட்டுக் கொண்டதுடன் எனது அனைத்து சந்தேகங்களையும் தெளிவுபடுத்திக் கொண்டேன் என்பதை தெரிவித்துக் கொள்கிறேன் நான் இந்த ஆய்வில் முழு சம்மதத்துடனும் சுய சிந்தனையுடனும் கலந்துகொள்ள சம்மதிக்கிறேன் இந்த ஆய்வில் என்னுடைய அனைத்து விவரங்களும் பாதுகாக்கப்படுவதுடன் இதன் முடிவுகள் ஆய்விதழில் வெளியிடுவதில் எனக்கு எந்த ஆட்சேபனையும் இல்லை என்பதை தெரிவித்துக் கொள்கிறேன் . எந்த நேரத்திலும் இந்த ஆய்விலிருந்து விலகிக் கொள்ள எனக்கு உரிமை உண்டு என்பதை அறிவேன் .

இடம் பாதுகாவலர்/ பெற்றோர்

தேதி கையொப்பம் ரேகை

MASTER CHART

S.No	NAME	AGE	PARITY	SOCIO ECONOMIC STATUS	BOOKING STATUS	HEART DISEASE TYPE	PRESENTATION	NYHA	OUTCOME OF PREGNANCY	SEX OF BABY	B.WT (in KG)	MATURITY	COMPLICATIONS	MATERNAL COMPLICATION	FETAL COMPLICATION	CONTRACEPTION
1	KUMARI	23	PRIMI	IV	B	Mild MS/MR	SC	II	OUTLET FORCEPS	M	2.7	T	NO	-	-	Cu-T
2	SHOBANA	21	PRIMI	IV	B	MVPS	SC	I	EMER.LSCS -FAILED INDUCTION	M	3	T	NO	-	-	-
3	KAVITHA	20	PRIMI	IV	B	MVPS/MILD MR	SC	II	LABOUR NATURALE	M	3.1	T	NO	-	-	-
4	HEMALATHA	23	G2P1L1	IV	B	ASD	SC	I	LABOUR NATURALE	F	2.75	T	NO	-	-	-
5	RAJATHI	22	G2P1L0	IV	B	MR/ trivial TR	SC	I	EMER.LSCS-FETAL DISTRESS	F	2.75	T	Yes	-	MECONIUM STAINED	Cu-T
6	MAHESWARI	23	PRIMI	IV	B	moderate MS	SC	II	EMER.LSCS-FETAL DISTRESS	M	3	T	NO	-	-	Cu-T
7	SUMATHI	20	PRIMI	IV	B	mild MS	SC	II	LABOUR NATURALE	M	2.5	T	YES	-	RDS	-
8	BHAVANI	24	G3P1L1A1	IV	B	severe MS/ moderate MR/ TR/ PHT	SC	III	OUTLET FORCEPS	M	2.7	T	Yes	pulmonary edema	-	Cu-T
9	RENUKA	20	PRIMI	IV	B	MS/ MR/ TR/ PHT	SC	II	LABOUR NATURALE	M	3.05	T	NO	-	-	Cu-T
10	MANIMEGALAI	26	G3P2L1	II	B	mild MS	SC	II	VACUUM DELIVERY	F	2.8	T	NO	-	-	-
11	SHANTHI	24	G2P1L1	III	B	PDA OPERATED	SC	I	LABOUR NATURALE	F	2.75	T	NO	-	RDS	PS
12	NAMBEESWARI	24	Primi	V	B	2nd DEGREE HEART BLOCK	SC	II	OUTLET FORCEPS	F	2.75	T	NO	-	-	Cu-T
13	INDRA	24	G2P1L1	IV	B	ASD CLOSURE DONE	SC	I	LABOUR NATURALE	F	2.8	T	NO	-	-	PS
14	SEETHALAKSHMI	24	G2P1L1	IV	B	trivial MR	SC	I	LABOUR NATURALE	F	2.25	T	NO	-	-	PS
15	JAYANTHI	24	G2P1L1	IV	B	VSD	SC	I	LABOUR NATURALE	M	2.6	T	NO	-	-	-
16	KOMALAVALLI	29	G2P1L1	IV	B	ASD	SC	II	LABOUR NATURALE	F	2.5	T	YES	-	RDS	-
17	NITHYA	24	G2P1L1	V	B	MS/MVR done	SC	I	LABOUR NATURALE	M	2.4	T	NO	-	-	Cu-T
18	YOGESWARI	20	PRIMI	V	B	mild MR	SC	I	LABOUR NATURALE	M	3.15	T	YES	-	IUD	-
19	RAMA	26	PRIMI	III	B	mild MR/ mild MS/ trivial TR	SC	II	LABOUR NATURALE	F	2.5	T	NO	-	-	-
20	GOVINDAMMAL	28	G2P1L1	V	B	ASD	SC	II	ELECTIVE RPT LSCS-PREV LSCS/ BREECH	M	2.95	T	NO	-	-	LSCS-ST

S.No	NAME	AGE	PARITY	SOCIO ECONOMIC STATUS	BOOKING STATUS	HEART DISEASE TYPE	PRESENTATION	NYHA	OUTCOME OF PREGNANCY	SEX OF BABY	B.WT (in KG)	MATURITY	COMPLICATIONS	MATERNAL COMPLICATION	FETAL COMPLICATION	CONTRACEPTION
21	REVATHI	22	G2P1L1	V	B	PRIMARY PULMONARY HYPERTENSION	SC	III	EMER.LSCS-FETAL DISTRESS	M	2.5	T	Yes	pulmonary edema	-	LSCS-ST
22	ANJALI	21	PRIMI	III	B	ASD CLOSURE DONE	SC	I	LABOUR NATURALE	M	2.45	T	YES	-	RDS	Cu-T
23	LAKSHMI	28	G2P1L1	IV	B	MS/MR/MVR done	SC	I	VACCUM EXTRACTION	M	2.6	T	NO	-	-	Cu-T
24	NIRANJANA	23	G2P1L1	IV	B	mild MR	SC	I	LABOUR NATURALE	F	2.5	T	NO	-	-	PS
25	NITHYA	21	PRIMI	IV	B	PRIMARY PULMONARY HYPERTENSION	SC	II	VACCUM EXTRACTION	F	2.9	T	YES	PULMONARY EMBOLISM	-	Cu-T
26	DHANAKODI	21	PRIMI	IV	B	mild TR	SC	II	LABOUR NATURALE	F	2.2	T	YES	-	SGA	Cu-T
27	LAKSHMI	28	G2P1L1	IV	B	moderate MS	SC	II	EMER.RPT.LSCS-PREV. LSCS/1ST TWIN BREECH	M	1.5,1.4	PT,PT	YES	-	RDS	LSCS-ST
28	PAPPATHI	28	PRIMI	IV	B	PRIMARY PULMONARY HYPERTENSION	SC	IV	OUTLET FORCEPS	F	2.75	T	NO	-	-	Cu-T
29	KALPANA	24	G2P1L1	III	B	Mild MR	SC	I	EMER.LSCS-FAILED INDUCTION	M	2.5	T	YES	-	RDS	LSCS-ST
30	DEVI	27	G4P2L1	III	B	moderate MS/ mild MR	SC	II	ELECTIVE RPT LSCS- PREV LSCS/CPD	M	2.6	T	yes	-	MECONIUM STAINED	LSCS-ST
31	ILAKKIYA	19	PRIMI	V	B	moderate MS/ severe MR	SC	III	OUTLET FORCEPS	M	2.6	T	NO	-	-	Cu-T
32	REKHA	22	G2P1L1	V	B	severe MR/ MVR DONE	SC	II	EMER.LSCS-FETAL DISTRESS	F	2.775	T	Yes	pulmonary edema	-	LSCS-ST
33	REVATHI	24	PRIMI	V	B	ASD CLOSURE DONE	SC	I	LABOUR NATURALE	F	2.3	PT	NO	-	-	-
34	BHARATHI	28	PRIMI	IV	B	moderate MR	SC	I	OUTLET FORCEPS	M	2.6	T	NO	-	-	Cu-T

S.NO.	NAME	AGE	PARITY			HEART DISEASE TYPE	PRESENTATION	NYHA	OUTCOME OF PREGNANCY		B.WT (in KG)	MATURITY	COMPLICATIONS	MATERNAL COMPLICATION	FETAL COMPLICATION	CONTRACEPTION
35	LAKSHMI	28	G3P1L1A1	V	UB	PERIPARTUM CARDIOMYOPATHY	SC	IV	VACUUM DELIVERY	M	2.5	T	YES	CCF	-	Cu-T
36	MENAKA	22	G2A1	III	B	severe AS/ severe AR	SC	III	OUTLET FORCEPS	M	1.5	PT	YES	-	RDS	Cu-T
37	VENDALAVANYA	23	G2P1L1	IV	B	severe MS	SC	III	EMER.RPT.LSCS-PREVLSCS/CPD	F	3.1	T	Yes	pulmonary edema	-	LSCS-ST
38	NITHYA	27	G2P1L1	IV	B	mild MR/ MVPS	SC	I	EMER.RPT.LSCS-PREVLSCS/CPD	F	2.9	T	NO	-	-	LSCS-ST
39	NIRMALA	26	G2P1L1	V	B	mild MS	SC	I	ELECTIVE RPT LSCS- PREV LSCS/CPD	M	3.2	T	NO	-	-	LSCS-ST
40	RAMYA	20	PRIMI	V	B	mild MR	SC	II	LABOUR NATURALE	F	2.75	T	NO	-	-	-
41	PARAMESWARI	22	G2P1L1	V	B	mild MS	SC	I	LABOUR NATURALE	M	2.7	T	NO	-	-	Cu-T
42	MOHANALAKSHMI	28	PRIMI	V	B	VSD	SC	I	LABOUR NATURALE	F	2.6	T	NO	-	-	-
43	DHANALAKSHMI	21	G2P1L1	V	B	trivial MR	SC	I	LABOUR NATURALE	M	2.25	T	NO	-	-	PS
44	VASUKI	24	G3P1LOA 1	V	B	MS/ MR/ AR	SC	II	VACUUM DELIVERY	F	2	PT	Yes	pulmonary edema	-	-
45	BRINDHA	25	G3P2L1	V	B	mild TR	SC	I	EMER.LSCS-FAILED INDUCTION	M	3.25	T	NO	-	-	LSCS-ST
46	SRIDEVI	30	PRIMI	V	B	ASD CLOSURE DONE	SC	I	EMER.LSCS-FAILED INDUCTION	M	2.25	T	NO	-	-	-
47	SHANMUGALAKSHMI	27	G2P1L1	V	B	Mild MS/ CMC DONE	SC	II	EMER.RPT.LSCS-PREVLSCS/CPD	M	2.4	T	NO	-	-	LSCS-ST
48	SAKIYA	24	G2P1L1	V	B	trivial MR	SC	I	ELECTIVE RPT LSCS- PREV LSCS/ CPD	M	3	T	NO	-	-	LSCS-ST
49	PARAMESWARI	20	PRIMI	III	B	VSD	SC	I	LABOUR NATURALE	F	2.5	T	NO	-		-
50	SARANYA	22	G2P1L1	IV	B	mild MR	SC	I	EMER LSCS-FETAL DISTRESS	F	1.7	T	YES	-	IUGR/RDS	LSCS-ST
51	ESTHERKANI	25	G3P2L1	IV	B	MR/TR/ MVPS	SC	II	ASSISTED BREECH DELIVERY	F	3.1	T	NO	-	-	-
52	AMSA	28	G3P1L1A1	IV	B	MS/ MR	SC	I	LABOUR NATURALE	F	2.25	T	NO	-	-	-
53	LAKSHMI	23	G2P1L1	III	B	mild to moderate MR/ MVPS	SC	I	ELECTIVE RPT LSCS-PREVLSCS/CPD	F	3.25	T	NO	-	-	LSCS-ST
54	RUKMANI	20	PRIMI	V	B	moderate AS/ mild AR	SC	I	EMER.LSCS-CPD/FAILED ACCELERATION	F	2.6	T	NO	-	-	Cu-T
55	SULOCHANA	35	G3P2L2	V	UB	Mild MR	SC	I	LABOUR NATURALE	M	2.5	T	NO	-	-	PS

S.N0.	NAME	AGE	PARITY			HEART DISEASE TYPE	PRESENTATION	NYHA	OUTCOME OF PREGNANCY		B.WT (in KG)	MATURITY	COMPLICATIONS OF HD	MATERNAL COMPLICATION	FETAL COMPLICATION	CONTRACEPTION
55	ABIRAMI	22	PRIMI	V	B	mild MR	SC	I	LABOUR NATURALE	M	3	T	YES	-	IIUD	-
56	RAMA	26	PRIMI	V	B	Mild MS/ CMC DONE	SC	II	LABOUR NATURALE	M	2	PT	YES	-	IUGR/RDS	Cu-T
57	SATHYAVANI	22	PRIMI	IV	B	ASD	SC	I	EMER.LSCS-CPD/FAILED ACCELERATION	M	2.75	T	NO	-	-	-
58	SEETHA	21	PRIMI	IV	B	MVPS OF AML	SC	I	EMER.LSCS-PRIMI/BREECH	M	1.75	PT	YES	-	RDS	-
59	JAYANTHI	25	PRIMI	IV	B	VSD	SC	I	LABOUR NATURALE	F	2.7	T	YES	PULMONARY EMBOLISM	-	-
60	AMUL	23	G2P1L1	IV	B	VSD OPERATED	SC	I	EMER.LSCS-BREECH	M	3.5	T	NO	-	-	LSCS-ST
61	KAVERI	23	G2P1L1	V	B	ASD	SC	I	LABOUR NATURALE	F	2.5	T	NO	-	-	PS
62	USHARANI	25	PRIMI	V	B	ASD CLOSURE DONE	SC	I	EMER.LSCS-SEVERE PREECLAMPSIA	F	2.15	T	NO	-	-	Cu-T
63	JEEVA	36	G5P1L1A3	V	B	MVPS/trivial mr	SC	I	ELECTIVE RPT LSCS-PREVLSCS/CPD	F	2.75	T	NO	-	-	LSCS-ST
64	HEMALATHA	23	PRIMI	V	B	mild TR	SC	I	VACCUM EXTRACTION	M	3	T	NO	-	-	Cu-T
65	IRFANA	23	G2P1L1	V	B	severe MS/ trivial MR/ moderate PHT	SC	III	OUTLET FORCEPS	M	2.5	T	Yes	pulmonary edema	-	Cu-T
66	CHITHRA	23	G2P1L1	V	B	DILATED CARDIO MYOPATHY	SC	III	OUTLET FORCEPS	F	2.4	T	YES	-	RDS	-
67	SUGANYA	18	PRIMI	V	B	mild MR	SC	II	Spontaneous expulsion of dead fetus	F	0.5	PT	NO	-	-	-
68	ADHILAKSHMI	20	G2P1L1	III	B	AS / CONGENITAL BICUSPID VALVE	SC	II	VACUUM DELIVERY	M	2.75	T	NO	-	-	Cu-T
69	SIRIYAPUSHPAM	24	PRIMI	IV	B	severe MS	SC	II	LABOUR NATURALE	M	3	T	Yes	CCF	-	Cu-T
70	KALAISELVI	28	G2P1L1	IV	B	moderate MR	SC	II	ELECTIVE RPT LSCS-PREVLSCS/FLEXED BREECH	F	2.9	T	yes	Yes	-	LSCS-ST
71	AMUDHA	26	G2P1L1	IV	B	MS/ MVR	SC	I	LABOUR NATURALE	F	1.8	PT	YES	-	IUGR/RDS	-
72	KRISHNAVENI	25	G2P1L1	IV	B	mild MR	SC	I	LABOUR NATURALE	F	2.5	T	NO	-	-	PS
73	NAGAMANI	19	PRIMI	IV	B	mod MS/ MR/ mild PHT	SC	I	LABOUR NATURALE	M	2.1	T	YES	CCF	-	Cu-T
74	POONGODI	32	G4P2L2A1	IV	UB	MVPS	SC	I	LABOUR NATURALE	M	3.5	T	NO	-	-	-
75	AMBIKA	22	G3P1L1A1	IV	B	ASD	SC	I	ELECTIVE RPT LSCS-PREV.LSCS/CPD	F	2.65	T	NO	-	-	LSCS-ST
76	LOGESWARI	18	PRIMI	IV	B	ASD CLOSURE DONE	SC	I	LABOUR NATURALE	M	2.7	T	NO	-	-	-
77	RAZIA	23	PRIMI	III	B	ASD	SC	I	LABOUR NATURALE	F	2.75	T	NO	-	-	Cu-T

S.NO.	NAME	AGE	PARITY			HEART DISEASE TYPE	PRESENTATION	NYHA	OUTCOME OF PREGNANCY		B.WT (in KG)	MATURITY	COMPLICATIONS OF HD	MATERNAL COMPLICATION	FETAL COMPLICATION	CONTRACEPTION
78	RENUKA	26	G2P1L2	V	B	MVPS of AML	SC	I	LABOUR NATURALE	F	3.15	T	NO	-	-	PS
79	PAVITHRA	18	PRIMI	V	B	ASD/ mild TR/ mild PHT	SC	II	OUTLET FORCEPS	M	2.5	T	Yes	pulmonary edema	RDS	Cu-T
80	SHOBANA	23	PRIMI	IV	B	Mild MR	SC	II	LABOUR NATURALE	M	2	PT	YES	-	IUGR	-
81	PADMA	30	G3P1L1A1	IV	B	PDA OPERATED	SC	I	EMER.RPT LSCS-PREV.LSCS/CPD	F	2.75	T	NO	-	-	LSCS-ST
82	SHAMEEM	25	G2P1L0	V	B	Mild MR	SC	II	EMER.RPT LSCS-PREV.LSCS/THREATENE D RUPTURE	F	2.9	T	NO	-	-	LSCS-ST
83	SARIDHA BAI	21	PRIMI	IV	B	ASD	SC	I	LABOUR NATURALE	M	2.5	T	**YES**	**SUPRAVENTRICULAR TACHYCARDIA**	-	-
84	NAJIMUNISHA	35	G3P1L1A1	IV	B	ASD CLOSURE DONE	SC	I	LABOUR NATURALE	M	2.25	PT	NO	-	-	-
85	SRIMATHI	19	PRIMI	IV	B	mild to moderate MR/ MVPS	SC	I	LABOUR NATURALE	F	3.2	T	NO	-	-	Cu-T
86	AMUDHA	25	G2P1L1	IV	B	MVPS	SC	I	ELECTIVE RPT LSCS-PREV.LSCS/CPD	F	3.45	T	NO	-	-	LSCS-ST
87	VALARMATHI	23	PRIMI	IV	B	acyanoticTETRALOGY OF FALLOT	SC	II	OUTLET FORCEPS	M	2.5	T	YES	SEVERE RIGHT VENTRICULAR OBSTRUCTION	-	Cu-T
88	ANITHA	24	G2P1L1	IV	B	ASD CLOSURE DONE	SC	I	EMER.LSCS-FETAL DISTRESS	F	3.75	T	NO	-	-	LSCS-ST
89	SATHYA	24	G2P1L0	IV	B	Mild MS	SC	II	VACUUM DELIVERY	M	3.45	T	NO	-	-	Cu-T
90	REKHA	22	G3P2L1	IV	B	mild MR	SC	I	ELECTIVE RPT LSCS/ PREV.LSCS/ CPD	M	2.9	T	NO	-	-	LSCS-ST
91	AMUL	25	G2P1L1	III	B	mild MS/ MR/ TR	SC	I	LABOUR NATURALE	F	2.5	T	YES	-	RDS	Cu-T
92	NASIMA	22	G4P1L1A2	III	B	Mild AR	SC	I	EMER.RPT.LSCCS-PREV.LSCS/ CPD	M	3.8	T	NO	-	-	LSCS-ST

S.NO.	NAME	AGE	PARITY			HEART DISEASE TYPE	PRESENTATION	NYHA	OUTCOME OF PREGNANCY		B.WT (in KG)	MATURITY	COMPLICATIONS OF HD	MATERNAL COMPLICATION	FETAL COMPLICATION	CONTRACEPTION
93	VANITHA	25	G2P1L1	IV	B	PDA CLOSURE DONE	SC	I	EMER.LSCS-FETAL DISTRESS	F	2.5	T	YES	-	MECONIUM STAINED	LSCS-ST
94	VISALAM	26	PRIMI	IV	B	Mod AR	SC	I	EMER.LSCS-FETAL DISTRESS	M	2.6	T	YES	-	RDS	Cu-T
95	VANI	37	G3P1L1A1	IV	B	TR/ MVPS	SC	I	EMER.LSCS-FETAL DISTRESS	M	2.75	T	NO	-	-	LSCS-ST
96	ZENATH	24	G2P1L1	IV	B	mild TR	SC	I	EMER.LSCS-FETAL DISTRESS	M	3	T	NO	-	-	LSCS-ST
97	KUMUDHA	20	G2P1L1		B	MVPS	SC	I	EMER.LSCS-FETAL DISTRESS	F	3.1	T	NO	-	-	LSCS-ST
98	KALPANA	19	G2P1L1	IV	B	VSD OPERATED	SC	I	EMER.LSCS-CPD MAJOR	F	3.8	T	NO	-	-	LSCS-ST
99	AMMU	27	G2P1L1	IV	B	mild AR	SC	II	EMER.RPT.LSCS- PREV.LSCS/ CPD	M	2.8	T	NO	-	-	LSCS-ST
100	GOMATHI	28	G3P2L2	IV	B	severe MS	SC	IV	OUTLET FORCEPS	M	2.6	T	**YES**	**MYOCARDITIS** -	-	Cu-T
101	MANJULA	25	G2P1L1	IV	B	MVPS AML	SC	I	ELECTIVE RPT LSCS-PREV.LSCS/CPD	M	2.8	T	NO	-	-	LSCS-ST
102	REVATHI	22	G2P1L1	IV	B	mild TR	SC	I	LABOUR NATURALE	F	2	PT	NO	-	-	PS
103	BANU	21	G2P1L1	III	B	mild MS/ CMC DONE	SC	I	LABOUR NATURALE	M	2.6	T	NO	-	-	-
104	GEETHA	25	G2P1L1	V	B	Moderate AS	SC	III	VACUUM DELIVERY	F	3.45	T	Yes	-pulmonary edema	-	LSCS-ST
105	DEVI	27	G2P1L1	V	B	ISOLATED DEXTROCARDIA	SC	II	EMER.LSCS-FETAL DISTRESS	M	2.8	T	NO	-	-	LSCS-ST
106	KALAIVANI	29	PRIMI	IV	B	Eisenmerger syndrome	SC	IV	VACUUM DELIVERY	F	2.3	T	YES	-	SGA	Cu-T
107	GOWRAMMAL	21	G2A1	V	B	WPW SYNDROME	SC	II	VACUUM DELIVERY	M	3.3	T	NO	-	-	Cu-T
108	CHITHRA	35	G3P2L2	V	B	moderate MR	SC	II	LABOUR NATURALE	M	2	PT	YES	-	RDS	Cu-T
109	SEETHA	24	G3P1L1A1	IV	B	mild TR	SC	I	LABOUR NATURALE	M	2.75	T	NO	-	-	Cu-T
110	SANGEETHA	28	PRIMI	III	B	IAS aneurysm	SC	I	ELECTIVE LSCS-CPD MAJOR	F	2.9	T	NO	-	-	Cu-T

S.NO.	NAME	AGE	PARITY			HEART DISEASE TYPE	PRESENTATION	NYHA	OUTCOME OF PREGNANCY		B.WT. (in KG)	MATURITY	COMPLICATIONS OF HD	MATERNAL COMPLICATION	FETAL COMPLICATION	CONTRACEPTION
111	DEEPA	27	G2P1L1	V	B	PERIPARTUM CARDIOMYOPATHY	SC	IV	VACUUM DELIVERY	M	3.5	T	**YES**	CCF	-	LSCS-ST
112	PANDIAMMAL	23	PRIMI	IV	B	PDA CLOSURE DONE	SC	I	Spontaneous expulsion of dead fetus	F	0.4	PT	NO	-	-	-
113	KALA	21	PRIMI	III	B	Mild PS	SC	III	OUTLET FORCEPS	M	2.4	T	Yes	pulmonary edema	-	Cu-T
114	PARAMESWARI	28	PRIMI	V	B	IAS aneurysm	SC	I	ELECTIVE LSCS- CPD MAJOR	M	2.25	T	NO	-	-	Cu-T
115	JAYACHITHRA	26	G3P2L2	IV	B	MVPS	SC	I	LABOUR NATURALE	F	1.85	PT	NO	-	RDS	-
116	SHEELA	18	PRIMI	IV	B	Mild MR	SC	I	LABOUR NATURALE	M	3	T	NO	-	-	Cu-T
117	JAYAPRADHA	22	PRIMI	III	B	Moderate AS	SC	II	VACUUM DELIVERY	F	1.6	PT	NO	-	RDS/SGA	Cu-T
118	MEERA	20	G2P1L1	V	B	MVPS/ trivial TR/ trivial MR	SC	I	EMER.RPT.LSCS-THREATENED RUPTURE	M	2.5	T	NO	-	RDS	LSCS-ST
119	ASHA	24	G2P1L1	IV	B	2nd degree heart block	SC	II	LABOUR NATURALE	F	3.25	T	NO	-	-	Cu-T
120	LATHA	25	G2P1L1	IV	B	PERIPARTUM CARDIOMYOPATHY	SC	IV	ELECTIVE RPT LSCS-PREV.LSCS/ CPD	F	3.75	T	**YES**	CCF	-	LSCS-ST
121	DHANALAKSHMI	25	G2P1L1	V	B	HYPERTROPHIC NON OBSTRUCTIVE CARDIO MYOPATHY	SC	III	EMER.LSCS-TRANSVERSE LIE	M	3.25	T	NO	-	-	LSCS-ST
122	JOTHILAKSHMI	23	PRIMI	IV	B	MVPS	SC	I	VACCUM EXTRACTION	M	2.4	PT	NO	-	SGA	-
123	SURYA	22	PRIMI	V	B	MVPS	SC	I	EMER.LSCS- CPD MAJOR	F	2.5	T	YES	-	RDS	Cu-T
124	MANJU	21	PRIMI	IV	B	MVPS	SC	I	EMER.LSCS- CPD/ FAILED ACCELERATION	M	3.25	T	NO	-	-	-
125	SUMATHI	25	PRIMI	III	B	Mild PS	SC	II	VACUUM DELIVERY	M	2.9	T	NO	-	-	Cu-T

S.NO.	NAME	AGE	PARITY			HEART DISEASE TYPE	PRESENTATION	NYHA	OUTCOME OF PREGNANCY		B.WT. (in KG)	MATURITY	COMPLICATIONS OF HD	MATERNAL COMPLICATION	FETAL COMPLICATION	CONTRACEPTION
126	ASHA	22	G2P1L1	IV	B	MVPS	SC	I	LABOUR NATURALE	M	2.3	T	NO	-	-	-
127	REVATHI	21	PRIMI	IV	B	Mild PS	SC	I	EMER.LSCS- FETAL DISTRESS	F	2.85	T	NO	-	-	Cu-T
128	PAVUN	23	PRIMI	IV	B	EBSTEIN'S ANOMALY	SC	III	EMER.LSCS- FETAL DISTRESS	F	2	PT	NO	-	RDS/SGA	Cu-T
129	SUBHA	21	PRIMI	IV	B	Mild MR	SC	I	EMER.LSCS- CPD/ FAILED INDUCTION	M	2.8	T	NO	-	-	Cu-T
130	SARALA	22	G2P1L1	IV	B	MVPS	SC	II	LABOUR NATURALE	M	2.8	T	NO	-	-	PS
131	DEEPA	21	PRIMI	IV	B	MVPS/ mild PR	SC	I	EMER.LSCS- FETAL DISTRESS	M	2.2	T	NO	-	-	-
132	GEETHA	25	G2P1L0	IV	B	MVPS/mild MR	SC	I	LABOUR NATURALE	F	2.25	T	NO	-	-	-
133	SATHYA	21	G2P1L1	IV	B	ASD	SC	I	LABOUR NATURALE	M	2.3	T	**YES**	**SUPRAVENTRICULAR TACHYCARDIA**	-	-

S.NO.	NAME	AGE	PARITY			HEART DISEASE TYPE	PRESENTATION	NYHA	OUTCOME OF PREGNANCY		B.WT. (in KG)	MATURITY	COMPLICATIONS OF HD	MATERNAL COMPLICATION	FETAL COMPLICATION	CONTRACEPTION
134	VASUKI	32	G3P1L1A1	V	B	MVPS	SC	I	LABOUR NATURALE	M	3	T	NO	-	-	Cu-T
135	LUBINAMARY	23	Primi	V	B	3rd degree heart block	SC	I	ELECTIVE RPT LSCS-PREV.LSCS / CPD	F	3.75	T	NO	-	-	LSCS-ST
136	UMA	24	PRIMI	V	B	MVPS/mod MR	SC	I	LABOUR NATURALE	M	2.6	T	NO	-	-	-
137	AMUDHA	20	PRIMI	IV	B	MVPS/ MR/TR	SC	I	LABOUR NATURALE	F	2.5	T	NO	-	-	Cu-T
138	VALARMATHI	25	G2P1L1	IV	B	MVPS/mild MR	SC	I	LABOUR NATURALE	F	3	T	NO	-	-	
139	SOWDAMANI	27	G2P1L1	IV	B	MVPS/mildMR/TR	SC	I	LABOUR NATURALE	F	3.2	T	NO	-	-	Cu-T
140	PAPPUTHAI	25	G3P1L1A1	V	B	WPW SYNDROME	SC	I	LABOUR NATURALE	M	2	PT	NO	-	-	Cu-T
141	MUNIYAMMAL	39	G8P5L5A2	V	UB	MVPS	SC	I	LABOUR NATURALE	F	3.1	T	NO	-	-	-
142	GAYATHRI	27	PRIMI	V	B	Tetrology of fallot	SC	IV	VACUUM DELIVERY	M	2.8	T	NO	-	-	Cu-T
143	DHANABAKKIYAM	26	G2P1L1	V	B	1st degree heart block	SC	I	LABOUR NATURALE	F	2.7	T	NO	-	-	Cu-T
144	SEETHA	21	G2P1L1	V	B	Primary pulmonary hypertension	SC	III	VACUUM DELIVERY	M	3.5	T	YES	CCF	-	Cu-T
145	SELVI	22	PRIMI	V	B	moderate AS/ mild MR	SC	I	LABOUR NATURALE	F	2.9	T	NO	-	-	Cu-T
146	ANITHA	23	PRIMI	V	B	MVPS/mild MR	SC	I	LABOUR NATURALE	M	2.5	T	NO	-	-	-
147	SUDHA	22	PRIMI	V	B	MVPS/mild MR	SC	I	LABOUR NATURALE	F	2.7	T	NO	-	-	Cu-T
148	KARPAGAM	24	PRIMI	V	B	Peripartum cardiomyopathy	SC	IV	VACUUM DELIVERY	M	2.7	T	YES (PULMONARY EDEMA)	PULMONARY EDEMA	-	-