TYPES OF CARDIAC DISEASES IN WOMEN PRESENTING WITH FEATURES SUGGESTIVE OF CARDIAC DISEASE IN PERIPARTUM PERIOD AND THEIR PREGNANCY OUTCOMES IN MUHIMBILI NATIONAL HOSPITAL, DAR ES SALAAM.

By

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A Dissertation Submitted in Partial Fulfilment of the Requirements for the Degree of

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Muhimbili University of Health and Allied sciences

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CERTIFICATION

The undersigned certify that they have read and hereby recommend for the examination of a dissertation entitled *Types Of Cardiac Diseases In Women Presenting With Features Suggestive Of Cardiac Disease In Peripartum Period And Their Pregnancy Outcomes In Muhimbili National Hospital, Dar Es Salaam,* in fulfilment of the requirements for the degree of Master of Medicine (Internal Medicine) of the Muhimbili University of Health and Allied Sciences

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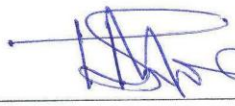
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ABSTRACT

Background: Cardiac diseases continue to be a risk factor for maternal and neonatal morbidity and mortality.

Objective: The aim of this study was to identify the types of cardiac conditions among women presenting with features suggestive of cardiac disease in the peripartum period and the maternal and perinatal outcomes at a tertiary care centre, Muhimbili National Hospital, Dar es Salaam, Tanzania.

Methods: A cross section study was done from April 2006 to January 2007. During the ten months period, a total of 97 women with features suggestive of cardiac disease in their third trimester of pregnancy and in puerperal period were evaluated and Echocardiography was done to identify the types of cardiac conditions, maternal and foetal outcomes were recorded and analyzed.

Results: The majority (46.4%) had echocardiographic changes consistent with chronic hypertension (30.9% of them had LVH due to Hypertension and 15.5% had Hypertensive dilated cardiomyopathy). The second most frequent echocardiographic diagnosis was peripartum cardiomyopathy in 27.8% subjects, followed by Rheumatic heart disease in 16.5%subjects. Other diagnoses included, mitral valve prolapse in 3.1% patients, Atrial septal defects in 2.0% patients and pericarditis in 1(1.0%) patients. Majority (54.6%) of the patients were in NYHAIII/IV. There was no case of maternal mortality. Three women developed life-threatening complications, cerebrovascular accidents. Intrauterine foetal complications were found in 17.5% of patients in total, IUGR accounting for 8.2% and IUFD, 9.3%

There were 9.3% still births and perinatally, there were10.3% reported deaths.

Babies born from mothers with NYHA III/IV had statistically significant low Apgar score than babies born from mothers with NYHA I/II.

There were more perinatal deaths from women who were in NYHA III/IV than in those who were in NYHA class I/II. This was statistically significant.

Birth weight of babies born to mothers with functional classes III and IV were lower than those of functional classes I—II, though not statistically significant

Conclusions: Hypertensive cardiac complications and Peripartum cardiomyopathy were the most frequent Echocardiographic diagnoses followed by Rheumatic heart disease in MNH, Dar es Salaam.

Maternal morbidity and perinatal mortality increased with worsening maternal cardiac functional class.

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DEFINITION OF TERMS:

1. Anaemia - reduction in the oxygen-carrying capacity of the blood as a result of fewer Circulating erythrocytes than is normal or a decrease in the concentration of haemoglobin (Hb).

Haemoglobin concentrations below which anaemia is likely to be present at sea level are usually defined in pregnant females as 11 g/dL

Mild anaemia between is 9-11g/dL

Moderate anaemia is 7-9g/dL

Severe less than 7g/dL anaemia in pregnancy has been defined as <7g/dL (WHO 1989).

2. Hypertension during pregnancy is a systolic pressure $\geq$ 140 mm Hg and/or a diastolic pressure $\geq$ 90 mm Hg.

3. Peripartum period- is a period beginning from the last month of gestation or the first few (5) months after delivery, with reference to the mother.

ABBREVIATIONS:

AHD	Acquired Heart Disease
CHD	Congenital Heart Disease
ECHO	Echocardiogram
ECG	Elecrocardiogram
NYHA	New York Heart Association
LVEF	Left Ventricular Ejection Fraction
ASD	Atrial Septal Defect
VSD	Ventricular Septal Defect
PDA	Patent Ductus Arteriosus
(cTGA)	Corrected Transposition of the Great Arteries.
LVH	Left Ventricular Hypertrophy
HT	Hypertension
RHD	Rheumatic Heart Disease
MR	Mitral Regurgitation
MS	Mitral Stenosis
GUCHD	Grown up Congenital Heart Disease
PPCM	Peripartum Cardiomyopathy
MNH	Muhimbili National Hospital
MUHAS	Muhimbili University Of Health and Allied Sciences
PMR	Perinatal Mortality Rate
SVD	Spontaneous Vertex Delivery
CS	Caesarean Section.

1 INTRODUCTION AND LITERATURE REVIEW

1.1 CARDIAC DISEASES IN PREGNANCY

As more women with congenital or acquired cardiac diseases survive into the childbearing years, physicians must be aware of the special difficulties that these women face during pregnancy. Cardiac diseases complicate 1-4% of pregnancies in women without preexisting cardiac abnormalities[1]. A working knowledge of the normal physiology of pregnancy is often helpful in the management of patients with heart diseases[1]

1.2 NORMAL PHYSIOLOGICAL CHANGES IN PREGNANCY

Profound haemodynamic alterations occur during pregnancy, labor and delivery, and in the post-partum period. These changes begin during the first five to eight weeks of pregnancy and reach their peak late in the second trimester.Haemodynamic deterioration may become clinically manifest in the diseased heart at this time

Knowledge of these cardiovascular adaptations is required to correctly interpret haemodynamic and cardiovascular tests in the gravida, to predict the effects of pregnancy on the woman with underlying cardiac disease, and to understand how the fetus will be affected by maternal cardiac disorders[1]

Blood volume increases 40 to 50 percent during normal pregnancy, in part due to estrogen-mediated activation of the renin-aldosterone axis. The rise in blood

volume is greater than the increase in red blood cell mass, contributing to the fall in hemoglobin concentration (i.e. the "anemia of pregnancy").

Cardiac output rises 30 to 50 percent above baseline. It peaks by the end of the second trimester, after which it reaches a plateau until delivery. The change in cardiac output is mediated by:

1. Increased preload due to the rise in blood volume,

2. Reduced after load due to a fall in systemic vascular resistance,

3. A rise in the maternal *heart rate* by 10 to 15 beats per minute.

Stroke volume increases during the first and second trimesters, but declines in the third trimester due to caval compression by the gravid uterus.

The direct effect of pregnancy on cardiac contractility is controversial.

Blood pressure typically falls, usually reaching a nadir of 10 mm Hg below baseline by the end of the second trimester. The decline in blood pressure is mediated by a fall in *systemic vascular resistance* induced by hormonal changes and by the addition of a low-resistance circuit through the utero placental bed.

During *labor and delivery* Haemodynamic fluctuations can be profound. Each uterine contraction results in the displacement of 300 to 500 ml of blood into the general circulation. Stroke volume increases, with a resultant rise in cardiac output

by an additional 50 percent with each contraction. Mean systemic pressure also rises, in part due to maternal pain and anxiety

Blood loss during delivery (300 to 400 ml for a vaginal delivery and 500 to 800 ml for a caesarian section) can further compromise the Haemodynamic state. The haemodynamic changes during the *post-partum* state are mainly due to relief of vena caval compression after delivery. The resultant increase in venous return augments cardiac output and causes a brisk diuresis. The haemodynamic changes return to the pre-pregnant baseline within 3 to 4 weeks following delivery. The marked haemodynamic changes associated with pregnancy explain the characteristic *signs and symptoms* that occur in the pregnant patient. Normal pregnancy is often associated with fatigue, dyspnea, and decreased exercise capacity.[1]

1.2.1 Cardiovascular Symptoms and Signs

Many of the normal symptoms of pregnancy, such as fatigue, dyspnea, orthopnea, and palpitations, can mimic symptoms of cardiac disease.[2] Careful examination for abnormal cardiovascular signs and symptoms is an important part of prenatal care because cardiac disease may be unmasked by physiologic changes of pregnancy.[3]

Normal	Abnormal
Fatigue	Syncope
Dyspnea	Paroxysmal nocturnal Dyspnoea
Occasional palpitations	SustainedArrhythmia
Systolic murmur (1–2/6)	Tachycardia>120beats/min
Pulsation of neck veins	Summation Gallop
Dependent edema (lower extremities)	Systolic murmur (4–6/6)
Distension of Neck veins	Diastolic murmur
Loud, wide split S1	Chest pain
	Haemoptysis
	Cyanosis
	Shortness of breath at rest

1.2.2 Physical examination:

Pregnant women usually have peripheral edema in the last trimester and jugular venous distension. Most pregnant women have audible a "new" physiologic systolic ejection murmur best heard over the mid or lower left sternal border, created by augmented blood flow.

A physiologic third heart sound (S3), reflecting the volume-overloaded state, a

prominent left and right ventricular apical impulse, basilar rales, exaggerated heart sounds, and Preexisting murmurs will be louder. One of the challenges for the clinician is how to distinguish between these normal symptoms and signs of pregnancy and similar symptoms that may indicate underlying heart disease in the mother. During normal pregnancy, *noninvasive testing* of the heart may include an electrocardiogram, a chest radiograph, and an echocardiogram.[1]

1.2.3 Electrocardiographic changes:

Shortening of the PR and QT intervals may accompany the increase in heart rate. Frontal lead axis changes are rare despite significant elevation of the diaphragm; when seen, slight rightward shifts are more common than leftward deviations.[4]

Nonspecific abnormalities of the ST segments and T waves appear in 4 to 14 percent of pregnancies.[5]

These changes predominate in left precordial leads and resolve in the majority of patients after delivery, but may recur during subsequent pregnancies.

Echocardiography is an invaluable tool for diagnosis and evaluation of suspected cardiac disease in the pregnant patient. Normal changes attributable to pregnancy include increased left ventricular mass and dimensions.

1.3 ASSESMENT OF RISK IN PATIENTS WITH PRE-EXISTING CARDIAC LESIONS.

1.3.1 Maternal and Fetal outcome

Whenever possible, women with preexisting cardiac lesions should receive.Pre-conception counseling, including a discussion of contraception, maternal and fetal risks during pregnancy, and potential long-term maternal morbidity and mortality. The *New York Heart Association functional class* is often used as a predictor of outcome.[1]

New York Heart Association (NYHA) Classification:

Class I: patients with no limitation of activities; they suffer no symptoms from ordinary activities.

Class II: patients with slight, mild limitation of activity; they are comfortable with rest or with mild exertion.

Class III: patients with marked limitation of activity; they are comfortable only at rest.

Class IV: patients who should be at complete rest, confined to bed or chair; any physical activity brings on discomfort and symptoms occur at rest.

Women with NYHA class III and IV face a mortality rate upwards of 7% and a morbidity rate of over 30%. These women should be strongly cautioned against pregnancy.[1]

In a study of 252 completed pregnancies in women with cardiac disease, five factors were found to be predictive of maternal cardiac complications.[1]

An updated risk index composed of the following four risk factors was shown to accurately predict a woman's chance of having adverse cardiac or neonatal complications.[1]

1.3.2 Predictors of Maternal Risk for Cardiac Complications:

(1) A prior cardiac event (heart failure, transient ischemic attack, stroke prior to pregnancy),

(2) Cyanosis or poor functional class, NYHA functional class >II,

(3) Left heart obstruction (Valvular and outflow tract obstruction (aortic valve area < 1.5 cm^2, mitral valve area < 2 cm^2, or left ventricular outflow tract peak gradient > 30 mm Hg), and

(4) Systemic ventricular dysfunction. Myocardial dysfunction (LVEF < 40% or restrictive cardiomyopathy or hypertrophy cardiomyopathy.[6]

An additional risk factor is,

5) Prior arrhythmia (symptomatic sustained tachyarrhythmia or bradyarrhythmia requiring treatment.

1.3.3 Effects of Heart Lesions in pregnancy (Fetal outcome):

1. Intrauterine growth retardation of baby

2. Pre term delivery (delivery before time)

3. Intrauterine deaths

4. Low birth weight babies

5. Babies with low Apgar score

Pregnant woman with cardiac problem may have a fatal out come at 28-32 weeks of pregnancy, during labour, immediately following delivery or at the end of 1st week after delivery.[1]

The fetus/neonate is exposed not only to the risks associated with maternal cardiac disease in pregnancy, but also to the increased risk of genetic transmission of a cardiac defect. In the general population, the risk of congenital heart disease is less than 1%. When either parent is affected with congenital heart disease, the risk to the fetus increases 10-fold. Therefore, genetic counseling and fetal echocardiography are recommended in these cases.[7] Babies born to pregnant women who have cardiac disease are at risk for neonatal and cardiac complications.[8]

In one study, pregnant women who had heart disease were followed during 302 pregnancies, and the frequency of maternal cardiovascular and neonatal complications was compared with a group of women who did not have heart disease and who experienced a total of 572 pregnancies.[8]

Neonatal complications, such as intraventricular hemorrhage, preterm delivery at less than 34 weeks' gestation, and fetal/neonatal death, occurred in 6% of the group

who had heart disease compared with 2% in the control group, a statistically significant difference.

The relationship between maternal heart disease and neonatal complications is believed to be caused by maternal cyanosis, low maternal oxygen saturation, and uteroplacental insufficiency. Uteroplacental insufficiency can be linked to poor maternal NYHA functional classification and left heart obstruction.[6,8]

In the setting of maternal cardiac disease, especially functional class III and IV, intensive fetal surveillance with ultrasonography and antepartum testing is mandatory.

A retrospective analysis of perinatal mortality at Muhimbili National Hospital (MNH), Dar es Salaam, Tanzania was conducted between 1999-2003 in order to categorise/classify perinatal deaths as well as to identify key factors in perinatal care that could be improved.

Data were retrieved from the MNH obstetric database and causes of early neonatal deaths were traced from the neonatal ward register. The study includes all foetuses weighing =500g. A modified Nordic-Baltic classification was used for classification of perinatal deaths.

Over a 5-year period there were 77,815 babies born with a perinatal mortality rate

of 124 per 1000 births, 78% of which was labour related stillbirth. The PMR was 913/1000 for singleton births and 723/1000 for multiple births for babies weighing less than 1500 grams and 65/1000 for singleton births and 116/1000 for multiple births for babies weighing 2500 grams or more. Babies weighing less than 1500 grams contributed 26% of PMR, whereas 41% occurred in babies weighing 2500 grams or more. The majority (79%) of neonatal deaths had Apgar score <7 at 5 minutes and the most common causes of neonatal mortality were birth asphyxia (37%) and prematurity (29%). Labour related deaths were more common in multiple pregnancies.There was no mention on the specific causes of perinatal deaths, in particular the contribution of cardiac disease in pregnancy.[9]

Another study was done by Kidanto et al, using hospital data looked at Maternal mortality at Muhimbili National Hospital, Tanzania, 1999 – 2005: levels, causes and characteristics and they found a maternal mortality ratio of 512 per 100,000 live births (95%CI, 465/100,000 – 559/100,000 live births) during the study period.

The top three direct obstetric causes of maternal deaths included eclampsia, 108 (23.5%), postpartum hemorrhage, 107 (23.3%) and anaemia in pregnancy, 52 (11.3%).

Anaemia in pregnancy has effect to both the mother and the foetus; the effects of

anaemia are made even worse in anaemic pregnant women with cardiac disease.

Major causes of anaemia in pregnancy in tropical Africa are malaria, iron deficiency, folate deficiency and haemoglobinopathies: now there is added also the acquired immune deficiency syndrome (AIDS).

Anaemia progresses through 3 stages: compensation, with breathlessness on exertion only; decompensation, with breathlessness at rest and haemoglobin (Hb) below about 7g/deci litre; cardiac failure, with Hb below about 4g/deci litre. Without treatment, over half of the women with haematocrit less than 0.13 and heart failure die.

Maternal anaemia, malaria and deficiencies of iron and folate cause intrauterine growth retardation, premature delivery and, when severe, perinatal mortality. Surviving infants have low birth weights, immune deficiency and poor reserves of iron and folate. They have entered already the vicious cycle of infection, malnutrition and impaired immunity.[98]

1.4 SPECIFIC CONGENITAL OR ACQUIRED CARDIAC LESIONS:

Specific congenital or acquired cardiac lesions can be classified as low, intermediate, or high risk.[10]

1.4.1 Low risk lesion

Young women with uncomplicated secundum-type *atria septal defect* (ASD) or

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isolated ventricular septal defect (VSD) usually tolerate pregnancy well. *Patent ductus arteriosus* (PDA) is not associated with additional maternal risk for cardiac complications if the shunt is small to moderate and if pulmonary artery pressures are normal. Antibiotic prophylaxis prior to labor and delivery is recommended for VSD and PDA (except in patients who are $\geq$ 6 months after repair); it is not required for ASD.[10]

Mitral regurgitation:

Chronic mitral regurgitation most commonly is the result of rheumatic heart disease or myxomatous degeneration and usually is well tolerated during pregnancy. However, new onset atrial fibrillation or severe hypertension can precipitate hemodynamic deterioration. Acute mitral regurgitation (e.g., from rupture of chordae tendineae) may produce flash pulmonary edema and life-threatening cardiac decompensation. Women with severe mitral regurgitation prior to pregnancy are advised to undergo operative repair prior to conception. *Mitral valve prolapse* in isolation rarely causes any difficulties during pregnancy.[10]

Aortic regurgitation:

Aortic regurgitation may be encountered in women with rheumatic heart disease, a congenitally bicuspid or deformed aortic valve, infective endocarditis, or

connective tissue disease. Aortic regurgitation generally is well tolerated during pregnancy. Ideally, women with severe aortic regurgitation should undergo operative repair prior to conception. Women with bicuspid aortic valves, with or without aortic regurgitation, are at increased risk for aortic dissection and should be followed carefully for signs and symptoms of this complication.[10] Congestive heart failure from either mitral or aortic regurgitation can be treated with digoxin, diuretics, and vasodilators such as hydralazine. Angiotensin converting enzyme inhibitors are teratogenic and therefore contraindicated. Beta blockers are generally safe during pregnancy, although fetal bradycardia and growth retardation has been reported.Antibiotic prophylaxis is recommended for both AR and MR during labor and delivery.[11]

1.4.2 Moderate risk lesions:

Mitral stenosis

Mitral stenosis in women of childbearing age is most often rheumatic in origin. Patients with moderate to severe mitral stenosis often experience hemodynamic deterioration during the third trimester and/or during labor and delivery. The physiologic increase in blood volume and rise in heart rate lead to an elevation of left atrial pressure, resulting in pulmonary edema. Additional displacement of blood

volume into the systemic circulation during contractions makes labor particularly hazardous.[10]

Aortic Stenosis

The most common etiology of aortic stenosis in women of childbearing age is a congenitally bicuspid valve. Mild to moderate aortic stenosis with preserved left ventricular function usually is well tolerated during pregnancy. Severe aortic stenosis (aortic valve area $\leq$ 0.7 cm^2, mean gradient > 50 mm Hg), in contrast, markedly increases the risk of pregnancy. Symptoms such as dyspnea, angina pectoris, or syncope usually become apparent late in the second trimester or early in the third trimester.[10]

1.4.3 High-risk lesion :

The high-risk conditions are associated with increased maternal and fetal mortality. Pregnancy is not advised. If pregnancy should occur, therapeutic abortion is advised. These patients are best managed with the assistance of a cardiologist.[10] Mechanical prosthetic heart valves. Mechanical heart valves are a major source of concern during pregnancy. The risk to the fetus of warfarin (risk of embryopathy in the first trimester, hemorrhage, and fetal loss throughout pregnancy) needs to be balanced against the risk of heparin to the mother. Heparin does not cross the placenta, but is a less effective anticoagulant; even if meticulously monitored the

mother is at risk of potentially fatal valve thrombosis.[10]

The fetal risk of warfarin is significantly reduced if the woman is adequately anticoagulated on ≤5 mg warfarin.

Coarctation of the aorta:

It is usually women with repaired coarctation who present for pregnancy. Magnetic resonance imaging should be used to assess the coarctation site before pregnancy, but can be safely performed during pregnancy if there is concern about recoarctation or aneurysm formation. The only caveats are to perform the scan in the left lateral position to avoid syncope caused by inferior vena cava compression by the gravid uterus, and the possibility that the third trimester patient may not fit in the scanner. Rarely a pregnant woman presents with a native coarctation. The major maternal risk is of aortic dissection, but mortality from pregnancy appears to be low whether or not there is adequate repair of the coarctation.[12]

Marfan syndrome and aortic dissection:

The main maternal risk in Marfan syndrome is type A aortic dissection, repair of which carries a 22% maternal mortality. Even without aortic root dilation the risk of dissection for pregnant women with Marfan syndrome is about 1%. Patients with poor family history, cardiac involvement, and aortic root > 4 cm diameter or a

rapidly dilating aorta are at high risk of dissection and should be advised accordingly. Those who elect to proceed with pregnancy should be treated with ß blockers and undergo elective caesarean section. Patients should also be aware of the 50% recurrence risk. Aortic dissection can occur without pre-existing disease in pregnancy, probably because of the hormonal changes and increased cardiovascular stress of pregnancy. Bicuspid aortic valve with dilated aortic root may also be a risk factor for aortic dissection in pregnancy, with similar histological findings to that of Marfan syndrome.[13]

2 EPIDEMIOLOGY:

There is wide regional variation (0.3—3.5%) in incidence of maternal heart disease during pregnancy with a global figure of around 1%.6.Heart disease in pregnant women is most commonly due to rheumatic heart disease, congenital abnormalities and less commonly due to ischaemic heart disease or cardiomyopathy.[14]

The Magnitude and the types of cardiac diseases in Africa are not well documented. The Data available for South Africa show that 0.65% of all pregnant women have heart diseases, and there is an unacceptably high morbidity and mortality rate (9.5%).[15] In developing countries, rheumatic heart disease is still predominant and continues to be a major cause of maternal mortality.[16] The likelihood of an adverse

maternal and fetal outcome is related to the type and severity of maternal valvular disease and the resulting abnormalities of functional capacity.[17]

In Egypt, heart disease is the most common indirect cause of maternal mortality.[18, 19].Rheumatic mitral stenosis is the most common valvular abnormality encountered in pregnant women and may be associated with pulmonary oedema, and atrial arrhythmias during pregnancy or soon after delivery. Mortality among pregnant women with minimal symptoms is less than 1%.[37, 38] Patients with severe symptoms, class III or IV according to New York heart association classification (NYHA) or tight mitral stenosis, carry a significant risk (5—15%) of maternal mortality 39.Patients who undergo valvuloplasty or valve surgery before pregnancy appear to have fewer complications than women who are treated medically.[20]

Fetal mortality increases with deteriorating maternal functional capacity and is around 30% when the mother is in NYHA class IV disease.[21] In the U.K, heart diseases in pregnancy are mainly Grown up congenital heart disease (GUCHD) in origin and are the major cause of morbidity and mortality not due to obstetric problems in 0.4-4% of pregnant women. In one study, the prevalence of heart diseases in pregnancy in a west London population revealed that 3%had significant cardiac lesions de novo. The spectrum of cardiac diseases complicating pregnancy

was illustrated in a large series from Brazil that described 1000 patients cared for at a single referral center. The type and frequency of cardiac diseases included rheumatic heart disease (56 percent), congenital heart disease (19 percent), Chagas' disease (9 percent), cardiac arrhythmias (5 percent), cardiomyopathies (4 percent), and others (7 percent).[15] Three-quarters of the women did not experience a cardiovascular complication during pregnancy; the reminder had the following: congestive heart failure (12 percent), cardiac arrhythmias (6 percent), thromboembolism (2 percent), angina (1 percent), hypoxemia (1 percent), infective endocarditis (less than 1 percent), and other complications (less than 1 percent). Twenty percent of patients required interventional procedures.[15] In the USA, the incidence of heart disease during pregnancy has not changed in 25 years but is uncommon, mainly because of the marked decline in rheumatic heart disease. Cardiac disorders during pregnancy are predominantly congenital diseases, which often have been previously corrected surgically. Although cardiac disease complicates only 1 to 4 percent of all pregnancies in the United States; maternal cardiac disease is a major cause of non-obstetric maternal morbidity and mortality. [22,23] Knowing that the patient has a history of heart disease is helpful because diagnosis of cardiac lesions during pregnancy is complicated by frequent systolic

functional murmurs, venous distention, tachycardia, and chest x-ray distortions that are related to pregnancy and not to disease. However, the unexpected finding of a diastolic or presystolic murmur during pregnancy requires investigation. In about 25% of women with mitral stenosis, symptoms first appear during pregnancy. In women with preexisting heart disease, maternal mortality is about 1%, accounting for about 10% of all maternal mortality. Successful pregnancy does not shorten life or permanently impair the functional capacity of women with heart disease.[0] Heart disease is the joint most common cause of maternal death in the UK. The most common cardiac causes of death involve cardiomyopathy and pulmonary hypertension. Congenital heart disease (CHD) is infrequently associated with maternal death, but forms the greatest workload in a cardiac antenatal clinic and may be associated with significant maternal morbidity.[25] Congenital heart disease in pregnancy is increasingly common because of the advances in surgery and medical therapy, which have taken place over the last 30 years, which means that more affected women are surviving into the reproductive age. Antenatal counseling needs to be tailored to the specific lesion, with pulmonary hypertension and cyanotic disease presenting a risk of maternal mortality of up to 50%. The use of anticoagulants in women with artificial valves presents a particular challenge,

heparin being safer for the baby and warfarin for the mother. Peripartum cardiomyopathy and Marfan's syndrome may be less dangerous than once thought. The risk of congenital heart disease in the fetus is increased, from twice to 20-fold, depending on the nature of the mother's lesion. Care throughout pregnancy and in the puerperium should be multidisciplinary and include cardiologists, obstetricians and midwives with experience of such cases, preferably in a tertiary centre.[26]

3 SPECIFIC HEART DISEASES

3.1 ACQUIRED HEART DISEASES

3.1.1 Ischaemic heart disease

Acute myocardial infarction is rare in pregnant women, occurring in 1:10 000 pregnancies, but the incidence is increasing, perhaps reflecting the trend towards older maternal age. Mortality is 37–50%.[11] and the risk of death is greatest if the infarct occurs late in pregnancy, in women under 35 years old, or if delivery is within two weeks of the infarct. Acute myocardial infarction during pregnancy is rare, occurring in 0.01 percent of pregnancies. Most myocardial infarctions occur during the third trimester in women over age 33 that have had multiple prior pregnancies. Coronary spasm, in situ coronary thrombosis, and coronary dissection occur more frequently than classic obstructive atherosclerosis. The diagnosis and management of acute myocardial infarction in the pregnant patient should follow

the guidelines established for the general population.Medical therapy for acute myocardial infarction must be modified in the pregnant patient. Thrombolytic agents increase the risk of maternal hemorrhage substantially to 8%. Low dose aspirin and nitrates are considered safe. Beta-blockers generally are safe. Short-term heparin administration has not been associated with increased maternal or fetal adverse effects. Angiotensin-converting enzyme (ACE) inhibitors and statins are contraindicated during pregnancy. Hydralazine and nitrates may be used as substitutes for ACE inhibitors.

3.1.2 Peripartum cardiomyopathy

Peripartum cardiomyopathy (PPCM) is a disorder of unknown cause in which initial left ventricular systolic dysfunction and symptoms of heart failure occur between the last month of pregnancy and the first 5 months postpartum.[28, 29] An essential element in diagnosis of PPCM is the demonstration of left ventricular systolic dysfunction. Because echocardiography was not available at the time of Demakis' original description of the condition[27] specific echocardiographic measures of left ventricular systolic dysfunction have been proposed as additional criteria.[29] These measures include an ejection fraction of less than 45%, fractional shortening of less than 30%, or both, and end-diastolic dimension of greater than 2·7 cm/m2 body surface-area.

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The need for echocardiographic confirmation of left ventricular systolic dysfunction often presents a difficulty in developing countries where the necessary technology is not available. However, even in settings of poverty, echocardiographic strategies have been shown to be possible and practical.[30, 31] PPCM remains a diagnosis of exclusion. No additional specific criteria have been identified to allow distinction between a peripartum patient with new onset heart failure and left ventricular systolic dysfunction as PPCM and another form of dilated cardiomyopathy. Therefore, all other causes of dilated cardiomyopathy with heart failure must be systematically excluded before accepting the designation of PPCM.

Peripartum cardiomyopathy is therefore defined as cardiac failure with left ventricular ejection fraction < 45%, occurring in the last month of pregnancy or within five months of delivery, in the absence of any identifiable cause of heart failure.[28].It is a rare condition, occurring in 1 in 3000–15 000 pregnancies. The pathogenesis is poorly understood, but some form of myocarditis, possible viral, has been postulated. Fetal outcome is good, as expected in a condition, which is not symptomatic until the end of the third trimester. However, there is an increased incidence of prematurity and low birth weight suggesting that the underlying disease process begins before the onset of maternal symptoms.[14]

Maternal mortality is up to 20%. Adverse risk factors include age > 30 years, multiparity, twin pregnancy, and the 50% of women in whom left ventricular dilatation and dysfunction persist.[14]

Treatment is the same as for any form of cardiomyopathy with impaired systolic ventricular function. The major challenge for the cardiologist is the assessment of recurrence risk in future pregnancies. If left ventricular function is persistently abnormal a year following delivery, the risk of death in a subsequent pregnancy is around 20%. If left ventricular function is completely normal, the risk of death in a subsequent pregnancy is probably small; however, there is a substantial risk of recurrence of symptomatic heart failure and permanent impairment of left ventricular function in any subsequent pregnancy

Pregnancy is poorly tolerated in women with dilated cardiomyopathy. The risk of maternal death is approximately 7% if the patient is in New York Heart Association (NYHA) functional class III or IV. Other adverse risks factors include ejection fraction < 20%, mitral regurgitation, right ventricular failure, atrial fibrillation, and systemic hypotension. Such patients should be counseled against pregnancy: as well as the risk of maternal death, there is a high risk of overt heart failure occurring during pregnancy, irreversible deterioration in ventricular function, and fetal loss.

Recent observations from Haiti[14]suggest that a latent form of PPCM without clinical symptoms might exist. The investigators identified four clinically normal postpartum women with asymptomatic systolic dysfunction on echocardiography, which subsequently either developed clinically detectable dilated cardiomyopathy, or improved and completely recovered heart function.

Although it seems likely that women of reproductive age all over the world have some risk of developing PPCM, good data about incidence are unavailable because so few population-based registries exist. Recent reports suggest an estimated incidence of one case per 299 live births in Haiti [34]one case per 1000 livebirths in South Africa [35]and one case per 2289 live births [35,36]to one case per 4000 livebirths in the USA.[28] These more recent data from the USA suggest a higher incidence than reported in earlier studies [32, 37]

The reasons for this variation in incidence between countries remain unknown. Cases of PPCM that adhere to the stated diagnostic criteria probably represent a similar disease, despite geographical variation in contributing factors. The remarkable propensity for recovery of left ventricular function observed in the diverse locations provides further evidence for a similar disease process. It should not be surprising that varying genetic pools and diverse environmental factors play

parts of varying importance in different areas.

However, cultural practices in some areas such as heavy intake of lake salt and body heating in clay beds might precipitate pregnancy-associated heart failure that does not always fit the diagnostic criteria for PPCM.[38-42] Although not clearly delineated, suggested risk factors associated with PPCM have included age, gravidity or parity, African origin, toxaemia or hypertension of pregnancy, use of tocolytics, and twin pregnancy.

The incidence of these presumed risk factors reported in the three largest series of patients with PPCM.[31, 34, 43, 44] and in Demakis and colleague's.[27, 45]

Comparison of potential risk factors and mortality rates in PPCM

Although PPCM is thought to be more prevalent in the upper and lower extremes of childbearing age, and in older women of high parity[35, 46], it is important to note that 24–37% of cases may occur in young primigravida patients.[27, 28, 31, 35, 44, 45]

The largest prospectively identified case series reports, from Haiti and South Africa[44]did not show a disproportionate role for older age, multiparity, and long-term use of tocolytic agents in the development of PPCM. The association with race might be confounded by the observed increased frequency of PPCM in women of lower socioeconomic status.

Aetiology

The cause and mechanism of pathogenesis of PPCM remains unknown,

Early suggestions that nutritional disorders, such as deficiencies in selenium and other micronutrients, might contribute could not be confirmed in studies of Haitian patients with PPCM [47]although unidentified nutritional factors might exist.

Proposed factors investigated contributing to the pathogenesis for PPCM[47-52]protective signalling pathway, viral antigen persistence, stress-activated cytokines, autoimmunity, micronutrients, microchimerism, myocyte apoptosis, and excessive prolaction production. Because of immune-system changes related to pregnancy, associations with autoimmune mechanisms and inflammation have been studied since the 1970s. Melvin and colleagues[53]proposed myocarditis as the cause for PPCM and reported a dense lymphocyte infiltrate with variable amounts of myocyte oedema, necrosis, and fibrosis in right ventricular biopsy specimens. Treatment with prednisone and azathioprine resulted in clinical improvement and loss of inflammatory infiltrate on repeated biopsies in the three patients studied. Sanderson and colleague [54] and subsequently Midei and colleagues[55]emphasised the association between myocarditis and development of PPCM

Although their findings were intriguing, they failed to establish a causal link.

Additionally, Rizeq and colleagues found an inflammatory component in less than 10% of biopsy samples from patients with PPCM, a proportion similar to that found in age-and-sex-matched patients with idiopathic dilated cardiomyopathy.

A decade later, Felker and colleagues [56]confirmed that the absence or presence of inflammation on endomyocardial biopsy tissue did not predict outcome in patients with PPCM.[49]

A viral trigger for the development of PPCM has been postulated and investigated in several studies. Bultmann and colleagues[57] identified viral genomic material in endomyocardial biopsy tissue from patients with PPCM; however, the same incidence and types of viral positivity were noted in controls. The findings of Kuhl and colleagues[58,59]in idiopathic dilated cardiomyopathy are more convincing evidence for the role of virus as a trigger for cardiomyopathy.

The investigators demonstrated the presence of viral genomic material in endomyocardial biopsy tissue, including enterovirus (coxsackievirus), parvovirus B19, adenovirus, and herpes virus. They also showed that clinical improvement of left ventricular systolic function was associated with viral clearing. If future results show that viruses play a part in the development of PPCM (and some forms of idiopathic dilated cardiomyopathy), endomyocardial biopsy could become

increasingly important in PPCM and unexplained cardiomyopathy, especially in patients who do not improve with conventional treatment in the early weeks after diagnosis. [58, 59]

Ansari and colleagues[57]investigated the role of fetal microchimerism (fetal cells in maternal blood during and after pregnancy) in patients with PPCM. In a small sample of patients, the amount of male chromosomal DNA in maternal plasma was significantly greater in patients with PPCM than in control mothers without PPCM during the third trimester of pregnancy, at term, and in the first week postpartum. The lower concentrations of this foreign protein could possibly contribute to tolerance of the fetus, permitting successful completion of pregnancy; increased levels could theoretically lead to the initiation of autoimmune disease, including an autoimmune myocarditis.

Findings from South Africa [31, 44]and Haiti lend [31, 41] support to the hypothesis that immune activation contributes to the pathogenesis of PPCM. Sliwa and colleagues identified increased [31,44]concentrations in plasma of the inflammatory cytokine tumour necrosis factor α (TNFα), C-reactive protein, and a plasma marker of apoptosis, Fas/Apo-1, in a large population of newly diagnosed patients with PPCM. Baseline concentrations of C-reactive protein correlated positively with baseline left

ventricular end-diastolic and end-systolic diameters and inversely with left ventricular ejection fraction. Concentrations of Fas/Apo-1 in plasma were significantly higher in patients with PPCM than in healthy controls, and were a predictor of mortality. Concentrations of C-reactive protein in plasma can vary substantially between different ethnic groups, and as much as 40% of this variation is genetically determined.[61] Hypothetically, an increase in the intensity of an inflammatory response could be one of the many factors contributing to development of PPCM. The importance of raised high-sensitivity C-reactive protein in plasma of patients with new and evolving PPCM [62, 63] merits additional evaluation.

A high incidence of postpartum mortality in mice with a cardiac-tissue-specific signal transducer and activator of transcription 3 (STAT 3) knockout of has been reported[48]before death, female mutant mice presented with symptoms of heart failure, reduced cardiac function, and apoptosis.

Data from this study suggested that the cardiac-myocyte-specific STAT3 pathway is necessary for protection of the heart from postpartum stress, and that prolactin cleavage is crucially involved in the pathogenesis of PPCM. Additionally, data from another mouse model of PPCM suggest that apoptosis of cardiomyocytes has a

causal role in PPCM, and that pharmacological inhibition of apoptosis by a caspase inhibitor might offer novel therapeutic strategies.[51] The finding of raised Fas/Apo-1 in women with PPCM [50]provides additional evidence for a role of apoptosis.

Warraich and colleagues [60] investigated the effect and clinical relevance of PPCM on humoral immunity and evaluated the immunoglobulins (class G and subclasses G1, G2, G3) against cardiac myosin in 47 patients with PPCM from South Africa, Mozambique, and Haiti. The immunoglobulin profiles were similar in the three regions and were markedly and non-selectively present.

Comparison of immunoglobulin profiles in patients with PPCM from South Africa, Haiti, and Mozambique versus 15 healthy mothers from South Africa. Although various causes for PPCM have been proposed, including abnormal hormonal regulation, the role of innate and adaptive immune systems, and the participation of autoantibodies, progenitor dendritic cells, T and B lymphocytes, cytokines, and chemokines[52-72] so far no cause has been clearly identified. It is likely that the aetiology is multifactorial, and that the search for a cause is hampered by the rarity of this condition in the developed countries, where more research funds are available, by the lack of adherence to diagnostic guidelines, and by the heterogeneity of the populations studied.

Clinical presentation

The most common presentation of PPCM is with symptoms and signs of systolic heart failure[27,30,31,35]Clinical examination of 97 patients seen in South Africa12 over 4 years revealed a displaced hypodynamic apical impulse in 72% and gallop rhythm in 92% of the patients. Functional mitral regurgitation was present in 43%. Left ventricular hypertrophy by electrocardiographic voltage criteria was present in 66% and ST-T wave abnormalities in 96% of the patients. Additional symptoms and signs include dependent oedema, dyspnoea on exertion, orthopnoea, paroxysmal nocturnal dyspnoea, persistent cough, abdominal discomfort secondary to passive congestion of the liver and other organs, precordial pain, and palpitations. In the later stages postural hypotension may be prominent, reflecting low cardiac output and low blood pressure.

Early signs and symptoms of heart failure can be obscured by pregnancy, because often the patient considers them to be a normal part of pregnancy. New York Heart Association (NYHA) cardiac functional classification varies from NYHA I to IV, although most frequently initial presentation is with NYHA III and IV. Sudden cardiac arrest might occur in a situation where cardiopulmonary resuscitation is neither available nor successful.[72] Delayed diagnosis can be associated with

increased morbidity and mortality. Clinicians should think of PPCM in any peripartum patient with unexplained disease. A broad index of suspicion is important, since unusual and non-heart failure presentations are also possible, as illustrated by the following evidence. Left ventricular thrombus is common in PPCM patients with a left ventricular ejection fraction of less than 35%.[61] With progression of disease, four-chamber dilation may be seen, with thrombus formation in the left atrium and right ventricle. Peripheral embolisation then becomes possible to any part of the body, including arterial occlusion of the lower extremities with compromised circulation [30,73,74] cerebral embolism with hemiplegia, aphasia, and in coordination[30,75] mesenteric artery occlusion with an acute abdomen syndrome secondary to bowel infarction[30] and acute myocardial infarction secondary to coronary artery embolism.[35,36]Haemoptysis may be the presenting feature of co-existing pulmonary embolus, for which the patients also have an increased risk.[35]

A recent report[76]identified a 5-weeks postpartum 35-year-old mother who progressively deteriorated with acute hepatic failure and was being considered for liver transplant. An echocardiogram identified dilated cardiomyopathy as the reason for heart failure with subsequent passive congestion of the liver and severe hepatic

failure. Appropriate treatment led to survival and recovery from liver failure as well as complete recovery of left ventricular systolic function.

Management and prognosis

The medical management of patients with PPCM is similar to that for other forms of heart failure.[77] Treatment aims to reduce after load and preload, and to increase contractility.Angiotensin-converting enzyme (ACE) inhibitors are usually used to reduce afterload by vasodilation if PPCM occurs after pregnancy. Because of potential toxic effects on the fetus, hydralazine (with or without nitrates) replaces ACE inhibitors during pregnancy. β blockers are used, since high heart rate, arrhythmias, and sudden death often occur in patients with PPCM. Digitalis, an inotropic agent, is also safe during pregnancy and may help to maximise contractility and rate control, but has to be closely monitored since excessive digoxin concentrations in serum have been associated with worse outcomes in women.[78, 79] Women in general and pregnant women in particular, may be more sensitive than men to its effect. Diuretics are also safe and are used to reduce preload and relieve symptoms. Because of the high incidence of thromboembolism in these patients, the use of heparin is also considered necessary, followed by warfarin in those with left ventricular ejection fractions of less than about 35%.

Although PPCM shares many features with other forms of non-ischaemic cardiomyopathy, an important distinction is that women with PPCM have a higher rate of spontaneous recovery of ventricular function. However, in a single centre prospective study of 100 South African patients with the condition, 15% died, and only 23% recovered normal left ventricular function after 6 months of treatment, despite optimal medical therapy with ACE inhibitors and β blockers.[44] In a longer follow-up study of prospectively identified patients in Haiti over 5 years, the mortality rate was also 15%, and 29 of 92 (31·5 %) recovered normal left ventricular function.[34] Continuing improvement was observed in the second and third years after diagnosis, confirming that the recovery phase is not limited to the first 6–12 months. Subsequent monitoring depends on response to treatment, and includes a follow-up echocardiogram in the first several weeks to confirm improvement of left ventricular systolic function. After that, an echocardiogram is indicated about every 6 months until recovery is confirmed or a plateau is reached. The best time to discontinue ACE-inhibitors or β blockers is unknown; however, one or both of these medications should be continued for at least 1 year. Where resources exist, left ventricular assist devices and heart transplantation may be used if necessary[80, 81]

Limited studies have been done with the use of immunosuppressive drugs such as azathioprine and steroids, and have shown mixed results. Use of these agents should be reserved pending further assessments, and perhaps should be restricted to patients with biopsy-proven lymphocytic myocarditis in the absence of viral particles. Recent studies in some patients with idiopathic dilated cardiomyopathy suggest a prominent role for cardiotrophic viruses.[49, 59] As noted previously, only one investigation to date has identified viral genomic material in endomyocardial tissue from patients with PPCM. Hence, PCR testing for a range of cardiomyotropic viruses could become important in the investigation of patients who do not improve in the early weeks following diagnosis.Aside from their haemodynamic benefits, the angiotensin-converting enzyme inhibitors, β blockers, and angiotensin-receptor blockers may have an additional benefit to dampen an over-active immune system that plays a role in the basic pathophysiology of PPCM.[82-84] Given the potential inflammatory nature of PPCM there may also be a role for other immunomodulatory therapy. A prospective study of 59 consecutive women with PPCM reported a significant reduction in the inflammatory marker TNFα and improved outcome in patients receiving the immunomodulating agent pentoxifylline in addition to conventional therapy [31]

Long-term prognosis

Very few studies have been done to investigate the long-term survival and recovery outcomes in patients with PPCM. Felkner and colleagues [56]assessed the survival of patients with initially unexplained cardiomyopathy referred for endomyocardial biopsy Johns Hopkins hospital. Patients with PPCM seemed to have a better prognosis than those with other forms of cardiomyopathy. However, patients who died soon after diagnosis were probably not included in that study. Fett and co-workers [34]with over 5 years of echocardiographic observations noted continuing improvement in cardiac function well beyond the initial 6–12 months after diagnosis.

One of the greatest concerns of PPCM patients is the safety of additional pregnancies. Since many patients with PPCM develop the disease during or shortly after their first pregnancy, it is especially important to be able to provide them with information about relapse. In a retrospective study, Elkayam and colleague [85] found that subsequent pregnancy in women with PPCM was associated with significant decrease in left ventricular function that resulted in clinical deterioration and even death. Symptoms of heart failure occurred in 21% of those who entered the subsequent pregnancy with normal left ventricular systolic function and 44% of

those who entered the subsequent pregnancy with abnormal left ventricular systolic function. However, all deaths (three of 44, 7%) occurred in the group who entered the subsequent pregnancy with abnormal systolic function. Eventual recovery of left ventricular systolic function occurred more frequently in women who had an ejection fraction of greater than 30% at original diagnosis of PPCM. Serial studies of left ventricular function and TNFα in prospectively-studied African patients with subsequent pregnancy after PPCM showed that deterioration of left ventricular function occurred postpartum to the subsequent pregnancy in five of six patients and was accompanied by raised concentrations of TNFα in plasma.[62] In a study [62] of 15 prospectively-identified Haitian patients with a subsequent pregnancy after PPCM, seven patients tolerated the subsequent pregnancy without worsening heart failure whereas eight had a relapse. Of those who relapsed, only one regained normal left ventricular systolic function during the 2-year follow-up after the subsequent pregnancy. All those who did not relapse went on to recover normal left ventricular systolic function. The longer follow-up allowed identification of both late recovery and late deterioration, again emphasizing that recovery of heart function is not limited to the first 6–12 months after diagnosis or relapse. Family-planning counseling is an important aspect of the care of patients after a diagnosis

of PPCM. Subsequent pregnancy after a diagnosis of PPCM carries higher risk of relapse if left ventricular systolic function is not fully recovered first; and even with full recovery some additional risk of relapse remains [85]

3.1.3 Hypertrophic cardiomyopathy

Asymptomatic patients with hypertrophic cardiomyopathy (HCM) usually tolerate pregnancy well and few develop symptoms for the first time during pregnancy [86] Maternal death is uncommon and there is no evidence to suggest the risk of sudden death is increased by pregnancy. There is little evidence that echocardiographic findings correlate with maternal morbidity, although massive hypertrophy may be an adverse risk factor. In addition, patients with heart failure and severe symptomatic restrictive physiology are unlikely to tolerate the haemodynamic changes of pregnancy and they should be advised against pregnancy.[87]

3.1.4 Hypertension in Pregnancy

Hypertension during pregnancy is a systolic pressure $\geq$ 140 mm Hg and/or a diastolic pressure $\geq$ 90 mm Hg. Hypertension during pregnancy can be classified into three main categories: chronic hypertension, gestational hypertension, and preeclampsia, with or without preexisting hypertension. In general, hypertensive disorders can complicate 12 to 22 percent of pregnancies and are a major cause of

maternal morbidity and mortality.[88]

Chronic hypertension is a blood pressure ≥ 140/90 mm Hg present prior to pregnancy, before the 20th week of gestation, or persisting beyond the 42nd post-partum day. Drug therapy for diastolic blood pressure ≥ 110 mm Hg has been shown to reduce the risk of stroke and cardiovascular complications.[88]

Gestational hypertension is hypertension that develops in the latter part of pregnancy, is not associated with proteinuria or other features of preeclampsia, and resolves by 12 weeks post-partum.This condition may portend the development of chronic hypertension in the future, but otherwise is associated with good maternal and fetal outcomes. [88]

Preeclampsia occurs in 3 to 8 percent of pregnancies in the United States. Primagravid women under the age of 20 years are at the highest risk for development of this disorder. The classic clinical triad involves gradual onset of hypertension, proteinuria (>300 mg/24 hours), and edema. Symptoms usually begin in the third trimester and resolve with delivery. The etiology of preeclampsia is still unclear. *Eclampsia* is the development of grand mal seizures in a woman with preeclampsia.

Untreated preeclampsia is a risk to both the fetus and the mother. When pre eclampsia is accompanied by risk factors including seizures, severe hypertension, HELLP syndrome (hemolysis, elevated liver enzymes, and low platelets), placental abruption, cerebral hemorrhage, pulmonary edema, renal failure, or liver failure, the fetus must be delivered immediately. Antihypertensive agents are usually initiated after the diastolic blood pressure exceeds 105 mm Hg and the systolic pressure is greater than 160 to 180 mm Hg.[88] Hydralazine, labetolol, and methyldopa are first line agents in the treatment of hypertension in pregnant women. Calcium channel blockers, such as amlodipine or diltiazem, are also used. Hypertension due to preeclampsia typically improves within a few days of delivery and should return to baseline by 12 weeks following delivery.[88]

3.1.5 Tachyarrhythmia

Many women with a normal heart experience palpitation during pregnancy and treatment is rarely necessary. For those with structural abnormalities, the increased sympathetic drive of pregnancy often worsens arrhythmia; if possible they should be established on an anti arrhythmic without fetal effects pre-pregnancy. In practice this is often difficult and some patients with refractory arrhythmia need to continue potentially toxic drugs such as amiodarone, balancing the risk to the fetus from the

drug against the risk to both the mother and the fetus of an arrhythmia with haemodynamic compromise. Adenosine, verapamil, and DC cardio version are safe for both mother and fetus. The population of pregnant women with heart disease has changed in recent decades, with rheumatic disease becoming uncommon and complex CHD increasing. Cardiomyopathy and pulmonary hypertension continue to account for most maternal deaths. As the number and complexity of adults survivors of CHD continue to grow, cardiologists and obstetricians need to be able to manage safely the majority of women who can tolerate a pregnancy and be able to recognise those few in whom a pregnancy is very high risk. Collaboration between cardiologist and obstetrician should result in a fall in maternal deaths from conditions already diagnosed before pregnancy and ensure that CHD does not feature more prominently in the next UK analysis of maternal deaths

Premature atrial and/or ventricular complexes are not associated with adverse maternal or fetal outcomes and do not require antiarrhythmic therapy. *Atrial fibrillation* and *atrial flutter* are rare during pregnancy. Rate control can be safely achieved with digoxin and beta-blockers. Electrical cardioversion can be performed safely during any stage of pregnancy. Other arrhythmias should be managed with the assistance of a cardiologist.

3.2 CONGENITAL HEART DISEASES

3.2.1 Unoperated secundum atrial septal defect:

This common lesion frequently remains undiagnosed until pregnancy when the pulmonary flow murmur is accentuated. There is a small risk of developing atrial arrhythmias in older women with longstanding right heart volume overload. The only other risk is of paradoxical embolism, so if the patient is immobilized they should receive heparin thrombosis prophylaxis and support stockings.Endocarditis prophylaxis is unnecessary. [89]

3.2.2 Repaired tetralogy of Fallot:

Pregnancy is well tolerated in women who have had radical repair of Tetralogy Of Fallot, as long as ventricular function is good and there is no significant right ventricular outflow tract obstruction. Many have significant pulmonary regurgitation and may become more tired and breathless as pregnancy progresses, occasionally needing diuretic treatment or admission for bed rest. [89]

3.2.3 Congenitally corrected transposition of the great arteries (cTGA).

Pregnancy is likely to be well tolerated in asymptomatic patients with (cTGA). However, systemic right ventricular function may deteriorate during pregnancy,

presenting with heart failure. Systemic tricuspid atrioventricular (AV) valve regurgitation may also worsen during pregnancy but is usually well tolerated. Management is as for any patient with heart failure and AV valve regurgitation.The AV node is absent in cTGA, complete heart block occurring at any stage in life; pregnancy is better-tolerated post-pacing.[89]

3.2.4 Post-Mustard or Senning repair of simple transposition of the great arteries:

Following atrial re-routing, the tricuspid valve and right ventricle support the systemic circulation, with the same implications for pregnancy as in cTGA. AV node function is usually normal, but patients are at risk of atrial arrhythmia and sinus node disease as a result of extensive atrial surgery. The pulmonary venous pathway must be demonstrated to be unobstructed pre-pregnancy, by transoesophageal echo or magnetic resonance imaging if transthoracic imaging is inadequate. A narrowed pulmonary venous pathway may only become symptomatic once pregnancy occurs, when the fixed obstruction acts as the physiological equivalent of severe mitral stenosis. A further caveat is that some patients develop unexplained pulmonary vascular disease in which case pregnancy is very high risk.

With these exceptions, pregnancy is usually well tolerated in NYHA class I–II patients' post-Mustard [90]

3.2.5 Post-Fontan type operation

A Fontan type operation is the definitive palliative procedure for cyanotic patients with a unctionally single ventricle in whom a biventricular repair is not possible. The ventricular mass is used to support the systemic circulation; there is no "pump" to support the pulmonary circulation so systemic venous blood reaches the pulmonary artery without pulsatile flow limited ability to increase cardiac output, dependent on high systemic venous filling pressures tendency to poorly tolerated atrial arrhythmia prothrombotic circulation. As a consequence, these patients are often advised against pregnancy. However, the maternal risk of pregnancy is not prohibitively high in NYHA class I–II patients provided ventricular function is good.[91] Ventricular failure and arrhythmia are the main causes of maternal morbidity. Care must be taken to maintain filling pressures during labour and delivery, avoiding dehydration and vasodilation. The risks to the fetus include: High (30%) risk of fetal loss.Warfarin should be converted to low molecular weight heparin antiarrhythmic drugs such as amiodarone may need to be continued; the risk of uncontrolled arrhythmia may outweigh the fetal risks of amiodarone. [91]

3.2.6 Cyanotic heart disease in the absence of pulmonary hypertension

Women with complex cyanotic heart disease and low or normal pulmonary artery pressures do not carry the prohibitively high risk of death of the Eisenmenger syndrome patient with pulmonary hypertension. Maternal risk for such patients depends largely on ventricular function. Other risks are: Hemorrhage: impaired clotting factors and platelet function Paradoxical embolism: all cyanotic patients shunt right to left. Heart failure: precipitated by the additional volume load of pregnancy on an already volume loaded ventricle Increasing cyanosis: caused by the vasodilation of pregnancy.The major risks to the fetus are fetal loss, low birth weight, and prematurity related to maternal cyanosis and maternal haemodynamic decompensation. If pre-pregnancy maternal oxygen saturation is < 85%, the chance of a live birth is only 12%, compared with 92% if the oxygen saturation is > 90%.[92] Prolonged admission for bed rest is an effective method of maintaining maternal oxygen saturation and hence fetal circulation. Although spontaneous normal vaginal delivery is safest for the mother, in practice fetal problems often necessitate an elective section. Care must be taken to achieve meticulous haemostasis, maintain good hydration, and to avoid vasodilation (for example, with high dose epidural) and air emboli through venous lines.

3.2.7 Pulmonary hypertension and the Eisenmenger syndrome

Pulmonary hypertension of any cause carries a high risk of maternal death even if pulmonary artery pressures are 50% systemic. The risk of death for a patient with Eisenmenger syndrome is 40–50%, unchanged in the last four decades.[93]

Patients should be advised against pregnancy. Laparoscopic sterilization may be considered, but carries a significant risk. The progesterone sub dermal implant (Implanon) carries no cardiovascular risk and is at least as effective as sterilisation.

In the event of pregnancy, therapeutic termination should be offered. For those who elect to continue, admission for rest and treatment of right heart failure may be necessary. Treatment may include heparin, oxygen, prostacyclin, and nitric oxide, but there is no evidence that any intervention reduces mortality. The high risk of death continues for at least two weeks postpartum and may be sudden or with irreversible hypoxia.

4 RATIONALE

Women with Cardiac diseases in NYHA class III and IV face a mortality rate upwards of 7% and a morbidity rate of over 30%.[1] High morbidity caused by heart diseases account for high costs of treatment due to prolonged duration of Hospitalization, high costs of medication and loss of working hours, all these lead to high burden to the families of these patients and a nation at large. An estimated maternal mortality rate appears to be small, but this applies only to the developed countries, this figure might be higher in the developing countries where generally there is suboptimal obstetric care. Fetal outcomes are also severely affected by the maternal health condition; sick mothers give birth to stillborns, premature babies and low Apgar score babies, which increase the perinatal mortality. There are no published data on the types of cardiac diseases among peripartum women in MNH. This study, therefore aims at describing types of cardiac diseases among women presenting with features suggestive of cardiac lesions in peripartum period at Muhimbili National Hospital and the outcomes of such pregnancies.

5 OBJECTIVES

5.1 Broad Objectives

To identify types of cardiac diseases in women presenting with features suggestive of heart disease in their peripartum period and the maternal and foetal outcomes at MNH, Dar es Salaam.

5.2 Specific Objectives

1. To describe demographic characteristics of women presenting with features suggestive of cardiac disease in their peripartum period.
2. To describe common presenting clinical features in women suspected to have cardiac disease in their peripartum period.
3. To identify echocardiographic diagnosis of types of cardiac diseases in women presenting with features suggestive of cardiac disease in their peripartum period.
4. To assess the effect of NYHA functional class of peripartum women with cardiac disease on maternal and foetal outcome.

6 METHODOLOGY

6.1 Study Design: Descriptive Cross-sectional study.

6.2 Study setting: Obstetric wards, Antenatal clinics, postnatal clinics, female Medical Wards and cardiac clinic at Muhimbili National Hospital, Dar es Salaam.

Dar es Salaam is the largest city (population 3,500,000 estimated 2003), economic centre and former capital of Tanzania. It has three districts, each with a municipal hospital. Muhimbili National Hospital (MNH) is a tertiary referral and teaching hospital, situated in the middle of the City of Dar es Salaam. It also serves as a regional hospital for Ilala,Temeke and Kinondoni municipality hospitals. It serves about 1000 outpatients per day and admission bed occupancy of about 1500 patients a week.The hospital runs, among other departments, the department of Internal Medicine, and obstetrics and gynaecology for both in and outpatients. It also runs postnatal clinic where women and their babies are followed up to 42 days post partum.

6.3 Study Subjects:

Pregnant women from 28 weeks of pregnancy to six weeks postpartum attending MNH were screened for cardiac symptoms and signs using a structured questionnaire.

Inclusion Criteria:

1. Women in their peripartum period presenting with features suggestive of cardiac disease.

2. Women with hypertension in their periparum period presenting with features suggestive of cardiac disease.

3. Women in their peripartum period with previously confirmed heart disease.

Exclusion criteria:

Women who did not give consent to participate in the study.

Women who had history of asthma

Enrolment of subjects:

Consecutive enrolment of all eligible study subjects during the study period.

In this study, 97 subjects were recruited from 1st April, 2006 to 31stJanuary, 2007, making total study duration to be ten months.

6.4 Sample size:

All eligible study subjects were included till the end of the study.

6.5 Study Procedures:

Study subjects were identified by the attending doctors at clinics and wards using a checklist of symptoms and signs listed for them. The investigator was then informed and re-evaluated these patients for suitability into the study. Patients were screened using a checklist of features given to all the attending doctors, also posted on the note boards in both outpatient obstetric clinics and wards

The following were the screening features for cardiac disease:

1. Shortness of breath on exertion
2. Awareness of heart beats
3. Fainting attacks
4. Precordial pain
5. Easy fatigability
6. Swelling of lower extremities
7. High Blood pressure
8. Raised JVP
9. Murmur.
10. Known cardiac disease.

Those with at least 3 out of the above listed features with or without elevated blood

pressure were recruited. Thorough history was taken, physical examination done and Hemoglobin (Hb) level estimated, and Echocardiogram (ECHO) done for all eligible patients. The investigator supervised by a senior cardiologist at Muhimbili National Hospital ECHO room, performed ECHOS on all recruited participants. Echocardiogram was done using a PHILIPS, HP 7550 Echocardiography machine with a 2.5.0MHz transducer. ECHO images of all patients were printed in papers and recorded in Video tapes. All the information on the demographic information was collected using structured questionnaire. HIV serostatus of the participants was checked from their antenatal cards as part of routine antenatal screening. For those who were found to have Peripartum cardiomyopathy and their HIV serostatus was unknown, they were pre test counseled, and tested to know their serostatus. Pregnancy outcome was determined based on both maternal as well as fetal outcome through a follow up made by the author at clinics and wards.

Maternal outcome was determined on the basis of:

-maternal mortality

-mode of delivery, whether Vaginal or caesarean section

-Duration of hospitalization

Foetal outcome was based on:

-perinatal mortality

-Birth weight

-APGAR score at 5minutes.

-Intrauterine growth retardation as assessed by obstetric ultrasound.

-Intra uterine Foetal death.

6.6 Data entry, cleaning and analysis

Information obtained was collected using questionnaires. All questionnaires were daily checked for completeness and consistencies. Then data were entered into computer using SPSS version 10 for further cleaning, categorizing of continuous variables and eventually analyses. Analyses included comparison of different categories by the use of χ^2 test especially when comparing different proportions. The cut-off point for rejecting the null hypotheses was when p-values were less than 5%.

6.7 ETHICAL CONSIDERATION:

The study received ethical clearance from the Research and Publication committee of MUHAS and the Ethics department of MNH. Patients were included after

informed verbal and written consent. All patients who were found to have Cardiac disease were reviewed by the author, examined and given treatment in the respective area i.e. clinics or wards and later followed in cardiac clinic. In case more expertise was needed on treatment of these patients, a senior cardiologist was consulted. The report on the well being of the mother and baby at home was assesed through follow clinics and in cases of failure to attend clinic,the information was obtained through phone numbers given to the investigator by the study subjects. Patients therefore had the benefit of having best medical care, however, there was no financial gain obtained by participating in this study.

7 RESULTS:

7.1 Baseline maternal characteristics at the beginning of the study:

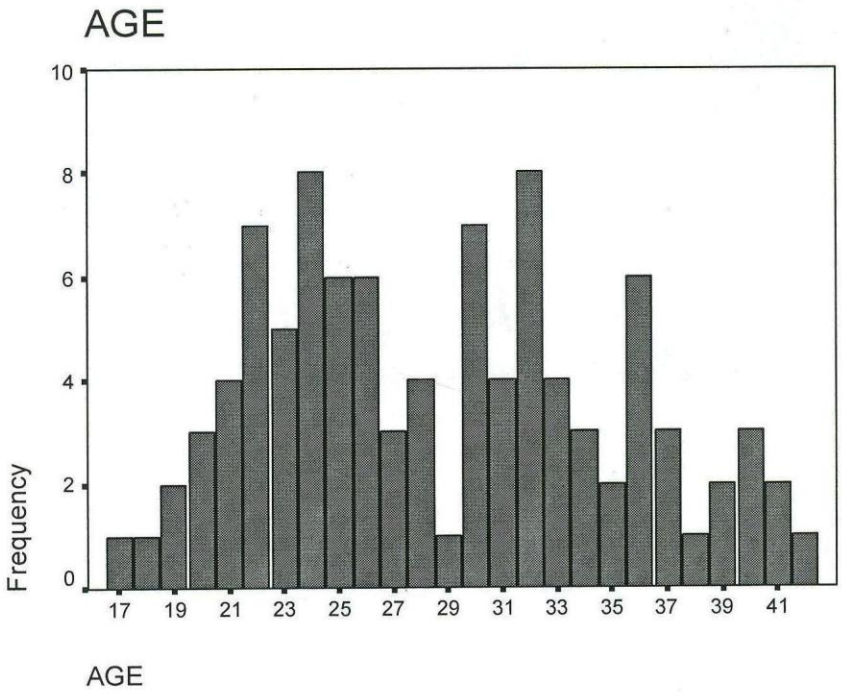
A cross section study was done between 1st April 2006 to 31st of January2007 at a tertiary national referral and teaching hospital to identify types of cardiac lesions in women presenting with features suggestive of cardiac disease in their peripartum period. During the ten months duration, there was a total of 13,171women who attended MNH for obstetric services, 7,941(6,623 ANC and 1,318 postnatal clinics) were from outpatient clinics and 5230 were admitted in the obstetric wards. Out of these, 97 were suspected to have cardiac disease and 94(97%) were confirmed to have different types of cardiac disease. Eight subjects (8.2%) had history of heart disease diagnosed before recruitment into this study, their diagnoses were, atrial septal defects in 2 patients, 3 patients had mitral leaflet prolapse and the remaining 3 had rheumatic heart disease. Out of 8 patients with known cardiac disease, 5 of them (5.2%) were on treatment. Of the patients with Rheumatic heart disease, one had undergone valvular surgery and was on heparin.

Table1; Baseline maternal characteristics at recruitment=97

Maternal characteristics	Numbers (%)
Age of the patients (years)	
Range	17-43
Mean (±SD)	28.74±6.26
Obstetric History	
Pregnant	11(11.3%)
Postpartum	86(88.7%)
Disease status	
Known to have cardiac disease	8(8.2%)
Undiagnosed	89(91.8%)
Known Hypertensives	26(26.8%)
Newly detected hypertensives	17(17.5%)
NYHA (Functional class)	
I	17(17.5%)
II	26(26.8%)
III	39(40.2%)
IV	15(15.5%)
Anaemia	
Mild	22(22.7%)
Moderate	31(32.0%)
Severe	5(5.2%)
HIV Positive	1(1.0%)

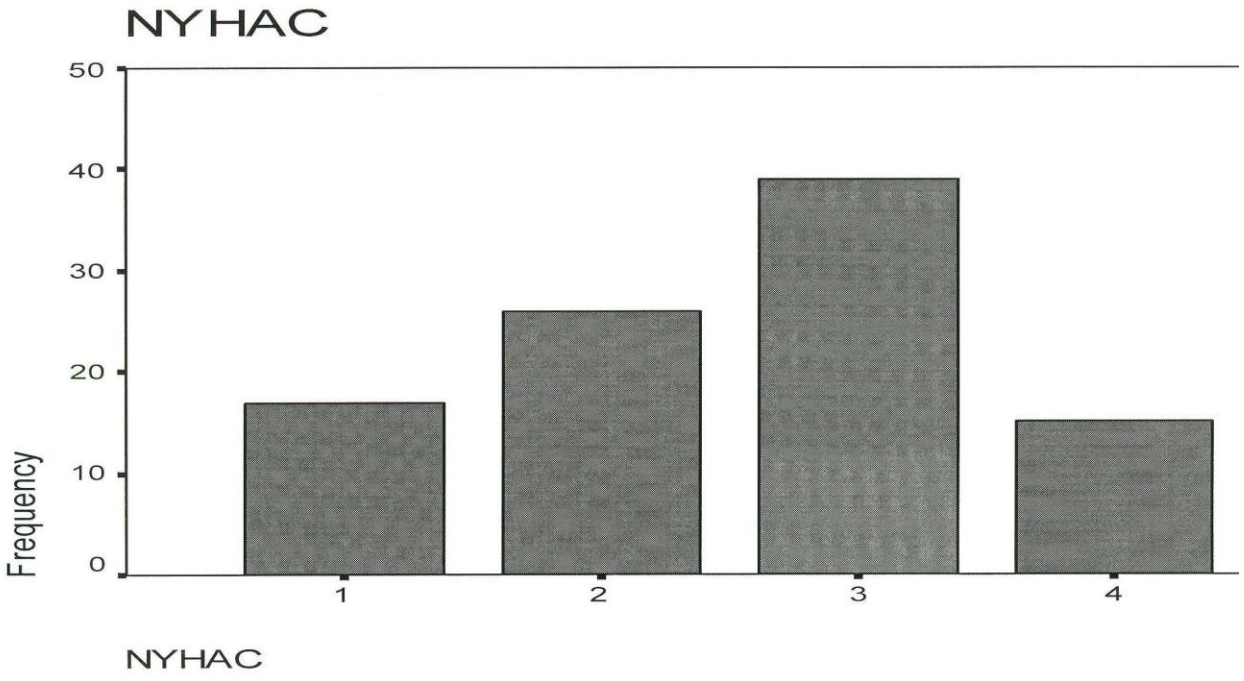
A few (5.2%) of patients had severe anaemia. Only 1(1.0%) of patients had tested positive for HIV, 6(6.1%) of patients had their serostatus for HIV unknown, the rest were seronegative

Fig1. Bar chart showing age distribution of study participants



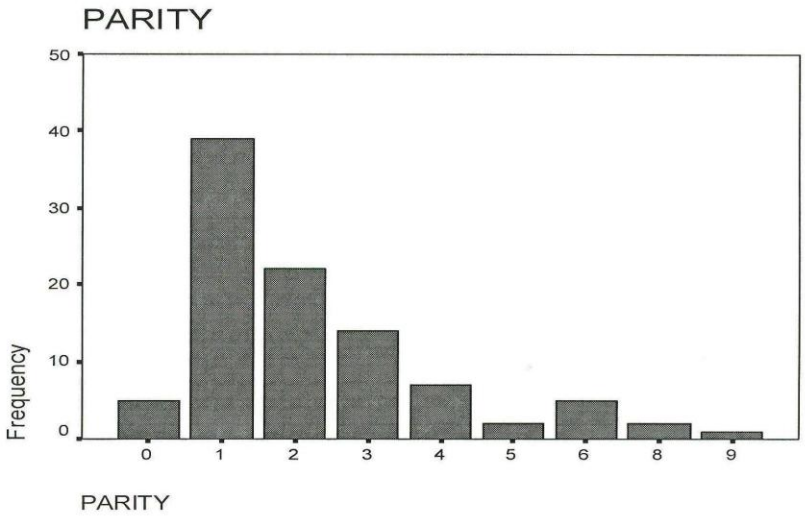
There were two peak ages, at around 24years and at around 32 years.

Fig 2.Distribution of Study participants according to their NYHA



The majority (40.2%) were in NYHA clinical functional class III, the rest were in NYHA class I/II and IV

Fig 3.Distibution of Study participants according to their parity



Majority (41.2%) of women were Primiparous

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Clinical presentation of patients at enrolment is summarized as in table 2

Table2: Clinical symptoms and signs of study patients:

Clinical Feature	Frequency (%)
Palpitations	93(95.9%)
Exertional dyspnoea	91(93.8%)
Easy fatigue	90(92.8%)
Oedema of lower limbs	76(78.4%)
Tachycardia	36(37.1%)
Elevated JVP	36(37.1%)
Murmur	31(32.0%)
Precordial pain	28(28.9%)
Syncope	13(13.4%)

The most frequent presenting symptom was palpitations in (95.9%) patients and syncope was the least common presenting symptom (13.4%)

7.2 Echocardiographic diagnosis of symptomatic patients

Table3: Echocardiographic diagnosis of symptomatic patients.

Echo diagnosis	Frequency (%)
LVH due to HT	30(30.9%)
Ht Dilated CM	15(15.5%)
Peripartum Cardiomyopathy	27(27.8%)
Rheumatic Heart Disease	16(16.5%)
Mitral regurgitation	16(100%)
Mitral regurgitation+stenosis	5(31.2%)
Multivalvular lesions*	7(43.7%)
MVP	3(3.1%)
Normal Echo	3(3.1%)
CHD	2(2.1%)
Pericarditis	1(1.0%)

*Combination of MR, MS, with TR or AR

Hypertensive cardiac disease was the most frequent echocardiographic diagnosis followed by Peripartum Cardiomyopathy, Rheumatic Heart Disease and others were: ASD, MVP and Pericarditis.

7.3 Maternal and foetal outcomes of symptomatic patients.

Table 4:Maternal and Foetal outcomes in women presenting with features suggestive of Cardiac disease.

Outcome	Numbers (%)	95% CI
Maternal outcomes		
Maternal survival	97(100%)	
Mode of Delivery		
SVD	85(87.6%)	81-94
C/S	12(12.4%)	5.8-18.9
Duration of hospitalization (days)		
Range	1-84	
Mean	11.59±12.85	
Foetal outcomes		
Intrauterine fetal complications		
Intrauterine Growth Restriction	8(8.0%)	
Intrauterine Foetal Death	9(9.0%)	
Twin Pregnancy	3(3.1%)	
Neonatal outcomes		
Liveborns	81(81.0%)	73.3-88.7
Stillbirths	9(9%)	11.3-26.7*
Postnatal deaths	10(10%)	
Mean birth weights (Kg)		
≥2.5	64(64.0%)	54.6-73.4
<2.5	36(36.0%)	26.6-45.4
APGAR score at 5 minutes		
Less than	17 (17.5%)	
Prematurity	19(19.6%)	

*Confidence interval for Stillbirths and postnatal (perinatal) deaths

None of the mothers died during the course of study. Two mothers with Peripartum

cardiomyopathy suffered cerebrovascular accidents, one had thrombus in left ventricular cavity and the other had no echocardigraphic evidence of intracardiac thrombus. The other mother with cerebrovascular accident had LVH due to Hypertension on Echo. Majority of the subjects, 85(87.6%) delivered by spontaneous Vaginal delivery, the rest 12(12.4%) by caesarean section

In this study, there were a total of 100 babies born, from 97 mothers, three women had twin deliveries. Intrauterine foetal complications were found in17 (17.5%) of patients, IUGR (diagnosed by obstetric ultrasound) accounting for 8(8.2%) and IUFD in 9(9.3%).

Out of 100 babies delivered, 81(81.0%) were live borns, others 9 (9.0%) were still births and 10 (10.0%) neonatal deaths. Causes of neonatal deaths were low birth weights, birth complications or maternal poor cardiac functional status. About one third of babies, 33 (33.0%) had low birth weights, 17 (17.5%) of neonates had Apgar score of less than 5 at 5minutes. Prematurity was found in 19(19.6%) of patients, Mean gestational age was 35.6±3.03 weeks.

7.4 Effect of cardiac functional class on Maternal and fetal outcomes:

Table5.Effect of cardiac functional class on Maternal and fetal outcomes:

Outcome	NYHA I /II	NYHA III/IV	p-value
Maternal outcome			
Maternal Survival	43(100%)	54(100%)	
Mode of Delivery			
SVD	34(36.1%)	48(51.0%)	
C/S	6(6.2%)	6(6.2%)	0.673
Duration of hospitalization			
More than 7days	17	42	
Foetal Outcome			
Live	40(41.2%)	38(39.2%)	
Died	3(3.1%)	16(16.5%)	0.005
Birth Weight (kg)			
≥2.5	30(30.9%)	34(35%)	
<2.5	13(13.4%)	20(20.6%)	0.482
Prematurity	10(10.3%)	9(9.27%)	0.417
IUGR	4(4.1%)	4(4.1%)	0.736
IUFD	3(3.1%)	6(6.2%)	0.486
APGAR score<5			
At 5min	3 (17.6%)	14/17(82.3%)	0.015

Till the end of the study, there was no case of maternal death reported in subjects presenting in all four cardiac functional classes. Out of the 94 who had cardiac disease, 82 (87.6%) delivered by SVD, Majority of them, 48(56.5%) were in NYHA class III/IV the rest in class I/II.

There were more perinatal deaths from women who were in NYHA III/IV than

those who were in NYHA class I/II. This was statistically significant with p-value of 0.005.

Babies who were born with low birth weight (33babies), majority20 (60.6%) their mothers were in NYHA III/IV. This shows that, NYHA class of mothers affected birth weights of babies.

8 DISCUSSION:

Cardiac diseases continue to be a risk factor for maternal and neonatal morbidity and mortality.[1] This study reflects on the types of cardiac diseases in women presenting with features suggestive of cardiac disease during peripartum period and maternal and foetal outcomes at a tertiary care center in Tanzania. In developed countries, the diagnosis and management of cardiac disease starts early. In developing countries, the sub optimal health network and poor treatment seeking behavior of patients results in late diagnosis and even diagnosis in pregnancy when haemodynamic compromise has already begun.

In this study, 97 women with features suggestive of cardiac disease in their peripartum period were evaluated; thorough history, physical examination and echocardiography were done to identify the types of cardiac diseases in these women. Of the 97 women involved in this study, 8 had history of being diagnosed to have cardiac disease before joining this study(Two had Atrial septal defect, Three had Rheumatic heart disease and the rest three had mitral valve leaflet prolapse)

It was found on Echocardiography that; 94(96.9%) had cardiac diseases, the rest, 3(3.1%) had normal echocardiographic findings. Eighty six out of 89(96.6%) patients therefore were diagnosed to have cardiac disease for the first time.

In a recent study by Desai et al including 128 patients with Mitral Stenosis, 42% of them were diagnosed for the first time during pregnancy.[94] This reflects that, routine examination of the cardiovascular system in the antenatal clinic using features suggestive of cardiac disease is therefore essential for early diagnosis and appropriate management.

This study revealed that, the majority 45(47.8%) had echocardiographic features consistent with chronic hypertension, 30(31.9%) of them having Left Ventricular Hypertrophy due to Hypertension and 15(15.9%) having Hypertensive dilated cardiomyopathy. The second most frequent echocardiographic diagnosis was peripartum cardiomyopathy in 27(27.8%) women, followed by Rheumatic heart disease in 16(16.5%) subjects. Other less frequent diagnoses included, mitral valve prolapse in 3(3.2%) patients, atrial septal defects in 2(2.12%) patients and pericarditis in 1 (1.0%) patients.

These findings are different from the prospective study done by Abdel -Hady et al [6] in Egypt in 2005 involving 86 pregnant women with cardiac disease where he found almost 90%of patients had Rheumatic heart disease. In another retrospective study done by Bhatla el al in 2002 in India involving 207 pregnant women with

cardiac disease, the most frequent Diagnosis was Rheumatic Heart disease in 88.0% of patients.[2]

These different findings could be attributed to the fact that; in this study, Symptomatic hypertensive patients, who made a large proportion out of all studied patients, were included using screening symptoms. In contrary, in both the studies mentioned above, only selected group of patients already diagnosed to have cardiac disease (excluding hypertensive patients) were included and followed up over time. A small sample size of this study could also contribute to these findings.

In this study, the most common presenting symptom was Palpitations in 93(95.9%) patients, Exertional dyspnoea, 91(93.8%) and easy fatigue, 90(92.8%) patients. The rest presented with oedema of Lower limbs in 76(78.4%) patients, precordial pain in 28 (28.8%) patients and lastly, syncope was the presenting symptom in the minority, 13(13.4%) of the patients.

Fever was present in only 2 (2.1%) of patients; one had malaria and was treated and got improvement. The other had chest infection which responded well to antibiotics. About one third, 36(37.1%) of patients had tachycardia, and the rest, 62(62.9%) patients had normal heart rate. None of the patients had bradycardia. Jugular Venous pressure was found to be elevated in 36(37.1%) of the patients, and

31(32.0%) of patients had murmurs at auscultation. In a study done by Desai et al [35] in South Africa in1995 involving 97 patients with Peripartum Cardiomyopathy over 4 years revealed, displaced hypodynamic apical impulse in 72% and gallop rhythm in 92% of the patients. Functional mitral regurgitation was present in 43%. Left ventricular hypertrophy by electrocardiographic voltage criteria was present in 66% and ST-T wave abnormalities in 96% of the patients. Additional symptoms and signs included dependent oedema, dyspnoea on exertion, orthopnoea, paroxysmal nocturnal dyspnoea, persistent cough, abdominal discomfort secondary to passive congestion of the liver and other organs, precordial pain, and palpitations. In the later stages postural hypotension may be prominent, reflecting low cardiac output and low blood pressure. The Study by Desai involved only a selected group of patients who were confirmed to have Peripartum Cardiomyopathy before joining the study, while in my study, I used screening features to include patients into the study before their diagnoses were known.

There was no reported maternal death in all patients involved in a study presenting in different classes of NYHA.There were 3(3.1%) cases of life threatening cardiac complications in this study, these were cerebrovascular accidents which occurred in three patients, two of them had peripartum cardiomyopathy, one had severe left

ventricular hypertrophy due to hypertension. All these patients were in NYHA class IV.

In a study done by Abdel-Hady et al in a tertiary care centre in Egypt in 2005 involving 86 women with cardiac disease it was found that there was one case of maternal mortality(1.16%), and 10 (11.6%) cases of life threatening cardiac complications . All complications occurred in rheumatic patients and the majority occurred in patients with functional classes III and IV. Poor Maternal outcome in terms of Maternal Mortality and Morbidity is influenced by Maternal NYHA cardiac functional Class. Sawhney et al [95]reported 10 maternal deaths out of 486 (2.0%) pregnant Indian patients with rheumatic heart disease, of which eight patients were in NYHA III and IV. There were eight maternal deaths in a series of 1093 Mexican women (0.73%)[96] ,compared with no maternal mortality in the Saudi report [97] this may be due to differential severity of cardiac disease and/or different level of health care.

Both these studies had big sample size, which gives the likelihood of having more maternal mortality contrally to my study which had a small sample size of only 97 subjects.

Another reason for no mortality in my study could be due to the fact that, majority of women with severe congenital and acquired heart disease do not survive to child bearing age, and therefore only those with mild to moderate heart disease they become pregnant and hence succumb to less mortality but high maternal morbidity and foetal complications.

Overall duration of hospitalization in patients with feature suggestive of heart disease was from 1-84days; mean duration of stay was (11.59±12) days. This average duration was longer compared to standard duration of hospitalization set for this hospital which is six hours for those who have delivered by uncomplicated vaginal delivery and three days for those who had caesarean section.

Eight patients were re-admitted for the same illness, their duration of admission ranged between 5-25 days, mean 15±7.33.

In a study done by Abdel-Hady et al in a tertiary care centre in Egypt in 2005 involving 86 women with cardiac disease they compared duration of hospitalization in patients with cardiac disease with those with low risk caesarean section and they found that the average duration of hospital admission for cardiac patients was (22±6) days, compared with (5±1) days for a low risk elective caesarean delivery. The average duration of hospital admission for classes III and IV was (28±7) days.

This duration of hospital stay for cardiac patients at MNH was about four times longer compared to the standard duration set by the Hospital,this is due to long term morbidity of these patients, which, accounts for high costs of care to both the hospital and the patient.

Of all patients, 82 (90.4%) delivered by SVD, Majority of them, 48(56.5%) were in NYHA class III/IV the rest in class I/II

Equal numbers of patients in NYHAI/II and III/IV delivered by Caesarean section. In a study done by Abdel-Hady et al in Egypt, it revealed that, the incidence of Caesarean delivery increased steadily from 49% in functional class I to 100% in class IV.In another retrospectively study done by Bhatla in India it showed that patients in NYHA class III/IV required cesarean section more often than those in NYHA Class I/II (46.34% vs. 13.86%).In both these studies, the mode of delivery was decided by a team of Obstetrician, Cardiologist and a senior obstetric anaesthetist. This was not the case in Muhimbili National Hospital where the decision was given by only attending Obtetrician and the decision on the mode of delivery was based on obstetric indications and not NYHA functional. Subjecting Sick patients in NYHA ClassIII/IV to deliver by SVD increases the risk of both maternal morbidity and mortality and perinatal deaths.

Of 19 babies who died, majority of their mothers 16/19(84.2%) vs 3/1(15.8%) were in NYHA III/IV and I/II respectively. Therefore accounting for more perinatal deaths from women who were in NYHA III/IV than those who were in NYHA class I/II.This was statistically significant (p-value of 0.005).The severe form of heart failure could be due to Anaemia which worsens the cardiac failure in women who have pre existing cardiac disease as was shown in this study that 58% of women had Anaemia, and 5% of them had severe anaemia.

These are no literature which gives the specific causes of perinatal deaths at Muhimbili National Hospital. The study done by Kidanto et al [9] at Muhimbili National Hospital (MNH), Dar es Salaam, between1999-2003, retrospective analyzed perinatal mortality in order to categorise/classify perinatal deaths as well as to identify key factors in perinatal deaths, it was found that, out of 77,815 babies born, perinatal mortality rate was124 per 1000 births, 78% of which was labour related stillbirths. Although there was no mention on the specific causes of perinatal mortality, the overall mortality rate of 124 per 1000 births (12.4%) is still lower than the mortality rate of 19 per 97 births (19.6%) from mothers with cardiac diseases found in my study.

This study shows that, there were more low birth weight babies from mothers who

were in NYHAIII/IV 20(20.6%) than in mothers in NYHAI/II (13.4%); however this did not show statistical significance(p-value 0.482). Bhatla etal also in his study showed that, there were more low birth weight newborns (56.41% vs. 33.33%) in patients in NYHA III/IV

Ebdel-Hady in his case-control study involving 86 mothers with cardiac diseases revealed that, birth weight of babies born to mothers with functional classes I and II was (3260±1.08) g and was not statistically significant from the control group (p =0.4). The birth weight for those with functional classes III and IV was (2980±190) g and this was significantly different from both the control group (p =0.001) and those with functional classes I and II (p =0.02).This shows that, NYHA class of mothers had clinically significant effect on birth weight of babies despite lack of statistical significance in my study.

There was no difference in the prematurity in the two groups of patient according to their NYHA class 10(34.5%) in NYHAI/II and 9(31.0) in NYHA III/IV.

There was no difference in IUGR in the two groups of patients (4) in NYHAI/II and (4) in NYHA III/IV.

There were more IUFD in mothers in NYHA III/IV (6patients) than in NYHAI/II (3patients).There were more babies14/17 born with low Apgar score of less than 5

at 5minutesfrom mothers in NYHAIII/IV than those3/17 born from mothers in NYHAI/II.This was statistically significant(p-value 0.015).

Bhatla et al in their study showed that (4.32%) neonates had an Apgar score of 5 at 10minutes. But none of the required mechanical ventilation.There was no perinatal mortality

9 CONCLUSIONS:

1. Using a set of screening features used in this study, 94peripartum women were identified to have cardiac diseases out of 97peripartum women who presented with features suggestive of cardiac disease

2. Hypertensive cardiac complications were the most common echocardiographic findings in women presenting with features suggestive of cardiac disease in their peripartum period, followed by peripatum cardiomyopathy and Rheumatic Valvular Disease.

3. Cardiac diseases accounted for greater maternal morbidity and hence contributed for prolonged duration of hospitalization.

4. There were more foetal deaths and low Apgar score in Foetuses of women with NYHAII/IV than in those in NYHAI/II.

10 RECOMMENDATIONS:

1. This is the first study to be done in Tanzania, it involves only a small sample size .I therefore recommend a study with larger sample size should be conducted to increase the power of this study.

2. Obstetricians should have a very high index of suspicion to suspect, investigate, and treat as early as possible women in peripartum period who present with features of cardiac disease.

3. Patients should be managed by Multi disciplinary team of obstetrician, cardiologist and anaesthetist to appropriately decide on the Mode of delivery of women in severe Heart failure

11. LIMITATIONS OF THE STUDY

1. The sample size was small; therefore, the power of the study was also small to give concrete conclusion on the findings in this study.

2. This was a hospital based study, so it was possible to recruit only very sick women who were attending a tertiary care centre.

3. Inclusion of women with hypertensive cardiac disease, who formed large part of the study subjects, would be the reason for different results I got on the types of cardiac disease in women as compared to other studies which did not include hypertensive subjects.

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Appendix I

QUESTIONNAIRE

TYPES OF CARDIAC DISEASES IN WOMEN PRESENTING WITH FEATURES SUGGESTIVE OF CARDIAC DISEASE IN PERIPARTUM PERIOD AND THEIR OUTCOMES AT MNH, DAR ES SALAAM.

1. Demographic Characteristics:

Date of interview.....

Serial Number.....

File No.....

Name.....

Date of birth..... (Age.....yrs)

Ward Admitted/Clinic attending.....

Residency.....

Tel.....

Gravida.....Para.....

Gestational age.....

Date of delivery.....

2. History

1. Have you been previously diagnosed to have heart disease or operation?

1. YES..... 2. NO.....

2. If yes, are you on any treatment for heart disease or operation?

1. YES..... 2. NO .....

3. Have you experienced shortness of breath on exertion for the past one month?

1. YES.... 2.NO.....

4. Have you ever-experienced Syncope (sudden loss of consciousness)

1. YES..... 2. NO.....

5. Have you experienced awareness of your heartbeats in the past 4 weeks?

1. YES..... 2. NO.....

6. Have you experienced precordial pain in the past 4 weeks?

1. YES..... 2. NO.....

7. Have you experienced easy fatigability in the past 4 weeks?

1. YES..... 2. NO.....

8. Have you noted any swelling of your lower extremities in the past 4 weeks?

1. YES..... 2. NO.....

9. Have you been diagnosed to have high blood pressure?

1. YES…… 2. NO……

3. PhysicalExamination

10. Body Temperature (degree centigrade)

1. <37…… (Normal) 2. >37…… (Fever)

11. Body weight (Kg)……

12. Height (cm)……

13. Clinical Anemia 1.YES…… 2.NO……

14.Radial pulse

1.Normal….. 2. Bradycardia …… 3. Tachycardia……

15. Jugular Venous Pressure 1.Normal…. 2. Raised…..

16. Blood Pressure (mmHg

1. Normal… 2. Low….. 3. High…..

17. Murmur 1. YES….. 2. NO…..

18. If there is a murmur, Grade

I……. II…… III…… IV……. V…… VI……

19. Functional state of Heart failure (NYHA class)

I …… II…… III …….... IV……..

4. Investigations:

20. Haemoglobin Level (g/dl)-Anemia

1. Normal…… 2. Mild …….. 3. Moderate…….....4. Severe……

21. HIVSerostatus

1. Negative…. 2. Positive….

22. ECHOCARDIOGRAPHY

1. Rheumatic Heart Disease…… 2. LVH due to HT……..

3.Ht Dilated cardiomyopathy 4. Dilated Cardiomyopathy

5. Hypertrophic Cardiomyopathy... 6. Restrictive Cardiomyopathy…

7. Congenital Heart Disease…….. 8. Mitral Valve prolapse…

9. Others……..

5. Outcomes

23. Mode of Delivery

1. SVD…… 2. C/S……

24. Maternal outcome

1. Live……
2. Dead……

25. Fetal outcome

1. Live……
2. Dead……

26. Birth Weight

1. Normal……
2. Low birth weight……

27. Prematurity

1. YES….
2. NO…….

28. Intra Uterine Growth retardation

1. YES……
2. NO……

29. Intrauterine Fetal death

1. YES……
2. NO…….

30. APGAR SCORE

1. At 1minute…
2. At 5minutes…

Appendix II

IDHINI YA KUSHIRIKI KATIKA UTAFITI

Nambari ya Usaili.....

Ndugu, hujambo?

Mimi naitwa..... mwanafunzi wa udhamili Chuo Kikuu cha Tiba Muhimbili. Nafanya utafiti kuhusu " **AINA ZA MAGONJWA YA MOYO KWA WANAWAKE WAJAWAZITO NA WALIOJIFUNGUA WENYE DALILI ZA MAGONJWA YA MOYO NA ATHARI ZA MAGONJWA HAYO KWA MAMA NA MTOTO, HOSPITALI YA TAIFA MUHIMBILI"**

Madhumuni ya Utafiti

Katika utafiti huu, lengo ni kusajiri wagonjwa watakaoonekana kuwa na dalili za magonjwa ya moyo na kuwafanyia kipimo cha moyo kiitwacho ECHOCARDIOGRAPHY ili kujua ni aina gani ya ugonjwa wa moyo alionao mgonjwa na unaathiri vipi afya ya mama na mtoto.

Ushiriki wa wagonjwa

Kama unakubali kushiriki katika utafiti huu utaombwa kujibu maswali kadhaa na pia kufanyiwa kipimo cha moyo.Kipimo kitafanyika bure bila wewe kuchangia pesa.

Utunzaji wa siri

Taarifa zote zitatunzwa kwa siri na mtafiti.

Madhara na athari

Hakuna madhara yoyote yanayotegemewa kutokana na ushiriki kwenye utafiti huu.Kipimo hiki ni salama kwa afya ya mama na mtoto na wala hakitoi mionzi inayohatarisha afya..

Uhuru wa kushiriki

Ni hiari ya mgonjwa kushiriki kwenye utafiti huu na pia anaweza kujitoa wakati wowote. Hata hivyo kutoshiriki au kujitoa kwenye utafiti hakumnyimi mgonjwa haki yake ya kupata huduma za matibabu. Mgonjwa aliyejitoa kushiriki utafiti akiamua kurudi atapokelewa na kuendelea kupata huduma zote kwa mujibu wa utaratibu uliopo.

Faida za utafiti

Kipimo hiki cha gharama kubwa utafanyiwa bure.Mgonjwa atafaidi kwa kuhudumiwa na mtafiti pindi mgonjwa anapohitaji utaalamu maalum wa magonjwa ya moyo.

Pia afya ya mshiriki itafuatiliwa kwa ukaribu na mtafiti kwenye kliniki maalum ya magonjwa ya moyo katika hospitali ya Taifa Muhimbili

Ni matumaini kwamba taarifa za utafiti huu zitasaidia kuboresha tiba kwa wagonjwa wengine siku za baadaye.Kuna kamati ya kusimamia udhibiti wa utafiti Kamati hii hutoa taarifa za matokeo ya utafiti ambayo hutoa elimu kubwa kuhusu tatizo hili.

Taarifa

Endapo utahitaji kupata maelezo kuhusu haki zako au kutoa taarifa ya madhara ambayo unahisi yametokana na kipimo hiki cha moyo, wasiliana na Prof. A.Y. Massele, mwenyekiti wa kamati ya utafiti,S.L.P 655001, Dar es Salaam. Namba: 2150302-6.

Je unakubali kushiriki kwenye utafiti? (weka alama) Ndiyo.....

Hapana.....

Mimi……..nimeelezwa/nimesoma maelezo haya.Maswali yangu yamejibiwa.

Nimekubali kushiriki kwenye utafiti huu

Sahihi ya mgonjwa……

Sahihi ya ndugu/shahidi……..

Sahihi ya Mtafiti……...

Tarehe……..