Outcome of Heart Disease in Pregnant Women: a case series in a Tertiary Care Hospital in West Bengal

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ABSTRACT

Cardiovascular diseases are a significant cause of maternal morbidity and are increasingly common due to factors such as obesity, hypertension, and diabetes. In our case series study, we have observed various types of heart disease in pregnant women, with some cases diagnosed during pregnancy. A multidisciplinary approach adhering to guidelines and conducting cardiovascular investigations before and during pregnancy is recommended to prevent and treat maternal and fetal morbidity and mortality.

KEYWORDS

Cardiac disease, Pregnancy, Multidisciplinary approach

Cardiovascular diseases are responsible for significant maternal morbidity and common reason for intensive care admission¹. It is also responsible for one fourth of all pregnancy related moralities ^{2,3}. It is becoming more prevalent due to higher rates of obesity, hypertension and diabetes⁴. Also more number of women with congenital heart diseases are becoming pregnant. American college of obstetricians and gynaecologists created task force on pregnancy and heart disease.

Risk factors for cardiovascular disease related morbidity and mortality:

- 1. RACE: Non Hispanic black women have a 3.4 times higher risk of cardiovascular disease related pregnancy complications compared with non Hispanic white women².
- 2. AGE: More than 40 years of age have 30 times more risk of cardiovascular disease related death than at age of 20 years ^{5,6}

- 3. HYPERTENSION: Hypertensive disorders in pregnancy lead to increased cardiovascular compromise in pregnancy ^{7,8,9}.
- 4. OBESITY: Pre-pregnancy obesity is important risk factor for cardiovascular disease related morbidity¹⁰.

Cardiovascular Changes during Pregnancy:

Pregnancy is a stress test for cardiovascular system. There is continuous increase in plasma volume and cardiac output and decrease in maternal systemic vascular resistance¹¹. After initial decrease blood pressure increases in third trimester^{12,13}. There is postural hypo-tension syndrome in second and third trimester due to mechanical compression of uterus¹⁴. In first week of puerperium increased maternal Atrial Natriuretic Peptide causes postpartum diuresis¹⁵. There are dramatic changes in cardiac output, heart rate, blood pressure, and plasma volume during labor and after delivery^{16,17}. Heart rate and blood pressure normally decrease within 48 hours postpartum, but blood pressure may increase between day 3 to 6 due to fluid shifts¹⁸. During this period, patient should be monitored for hypertensive complications, pulmonary edema and those related to fluid overload¹⁹. Maternal hemodynamics usually returns to pre-pregnancy level 3-6 months after delivery. The heart ventricles adapt to the increased plasma volume during pregnancy. Left ventricle EDD increases by 10%²⁰ and left and right ventricular mass increase by approximately 50% and 40% respectively²¹. Ejection fraction in some cases show no changes²⁰, although others decrease^{22,23}. Approximately 20% of women have diastolic dysfunction, may be associated with dyspnea on exertion ^{22,24}. Structural changes of the maternal heart returns to baseline after 1 year postpartum.

Hematologic, coagulation and metabolic changes are important contributors to cardiovascular risk. There increased red cell mass by 20-

30% which is proportionally less than increase in plasma volume, resulting in anemia from hemodilution. Hemoglobin or hematocrit level should be checked in each trimester as severe anemia may lead to heart failure and myocardial ischemia. There is increased risk of thromboembolism including hypercoagulability, venous stasis, decreased venous outflow, compression of inferior vena cava and pelvic veins by enlarging uterus and decreased mobility²⁵. Pregnancy also alters the levels of coagulation factors responsible for hemostasis amplify thrombogenic state and certain disorders such as antiphospholipid antibody syndrome, smoking further increase risk of thrombosis and embolism during pregnancy. As pregnancy is catabolic state that leads to insulin resistance and an atherogenic lipid profile with elevated serum fatty acids.

Case Series:

Sl No	Age (Year)	Parity & POG	Known H/O Cardiac disease	Cardiac disease	Indication for admission	Labour at admissi on	Echo data	NYHA class	Sponta neous labour	Mode of delivery
1	27	P1+0 38weeks	No	Subtle Septal Hypokinesia	Pain abdomen	Yes	Subtle Septal Hypokinesia,L VEF- 55%	NYHA Class-II	No	Emergency LSCS
2	26	P1+1 40weeks	Yes	Congenital complete heart block	Persistent abnormal bradycardia	No	Complete Heart Block in ECG	NYHA Class-I	No	Emergency LSCS
3	30	P0+0 39Weeks	No	Mild global hypokinesia, MR, Mild prolapsed AML tip	Safe confinement	No	Mild global hypokinesia , Grade II MR, AML tip mild prolapsed	NYHA Class-II	No	Elective LSCS
4	28	P0+0 32 Weeks	Yes	Fallot's physiology with BT shunt	Safe confinement	Yes	Tetralogy of Fallot's Physioloy, Situs Solitus, levocardia	NYHA Class-II	Yes	Vaginal Delivery

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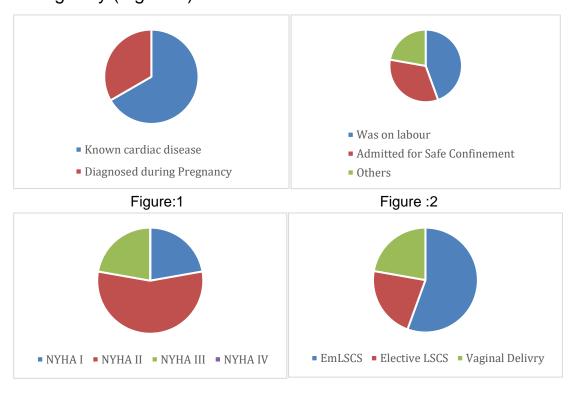
5	20	P0+0	Yes	Rheumatic Heart	Pain abdomen	Yes	RHD, AML/	NYHA	Yes	Vegine!
3	20		res		Pain abdomen	res	, and the second		res	Vaginal
		37Weeks		Disease, moderate MS,			PML	Class-II		Delivery
				mild PAH, Moderate TR			thickened-			
							calcified			
							mildly,			
							MVA0.9cm2,			
							mild MR&AR			
6	27	P0+0	Yes	Eisenmenger Syndrome	Safe	No	Large	NYHA	No	Elective LSCS
	27	34Weeks	103	Disemienger byndrome	Confinement	110	Perimembrano	Class-II	110	Licetive Eses
		34 WCCKS			Commement		us	Class II		
							VSD(17mm)			
							with severe			
							PAH(34mm of			
							`			
							Hg), TR,			
							bidirectional			
							shunt, LVEF-			
							62%			
7	20	P0+0	Yes	Dextrocardia, large	Bleeding PV	Yes	Dextrocardia,	NYHA	Yes	Emergency
		39Weeks		ASD, severe TR			Large OS-	Class-I		LSCS
							ASD(21mm),S			
							evere TR,			
							dilated RA,			
							RV, mild PAH			
							.,			
]						

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8	27	P0+2	No	Peripartum	Respiratory	No	LV Global	NYHA	Yes	Emergency
		36Weeks		Cardiomyopathy	distress		Hypokinesia,	Class-III		LSCS
							LVEF-37%,			
							mild			
							MR,AR,PAH,			
							moderate TR			
9	46	P0+1	Yes	Rheumatic Heart	Safe	No	AV- sclerosed	NYHA	No	Emergency
		33Weeks,		disease with progressive	confinement		and calcified,	Class-III		LSCS
		Twin		MS			AVmax-			
							2.5m/sec, mild			
							MR, AML,			
							PML-			
							thickened			

DISCUSSION:

In this work of case series we have observed 9 pregnant women among whom 6(66.6%) women had known cardiac disease and 3(33.3%) women diagnosed with Cardiac Disease during Pregnancy (Figure:1). We have seen various type of heart disease in our case series. We have seen 1(11.1%) case of Syndrome, 1(11.1%) Eisenmenger case of Peripartum Cardiomyopathy, 2(22.2%) cases of Rhematic Heart Disease, 1(11.1%) case of Fallot's Tetralogy, 1(11.1%) case of Congenital Complete Heart Block, 1(11.1%) case of Dextrocardia. In our case series 3(33.3%) women had septal defect. During admission 4(44.4%) women was on labour, 3(33.3%) admitted for Safe Confinement (Figure:2). 2 cases was NYHA Class I, 5 cases was NYHA Class II and 2 cases was NYHA Class IIII (Figure:3). Among the patients EmLSCS done in 5(55.5%) cases, Elective LSCS done in 2(22.2%) cases and 2(22.2%) women delivered Vaginally (Figure:4).



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Figure:3

Figure:4

CONCLUSION:

Pregnancy can exacerbate heart diseases which are most common

non-obstetric cause of maternal and fetal morbidity and mortality.

So, during pre-conception and early antenatal period couple should

be counseled regarding it's outcome and all cardio-logical

investigation to be done. A multidisciplinary approach to be taken to

prevent cardiac decompensation and feto-maternal loss. We may

conclude that with multidisciplinary approach and adhering to

guidelines we can prevent feto-maternal morbidity and mortality.

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