

# The Effect of Valvular Heart Disease on Maternal and Fetal Outcome of Pregnancy in Young Pregnant Females

ASIA ALTAF<sup>1</sup>, SAIRA FAIZ<sup>2</sup>, SAMSON S. BADALYAN<sup>3</sup>, RAMSHA KHAN<sup>4</sup>, SHEHERBANO YAHYA<sup>5</sup>

<sup>1,2</sup>MBBS, Mohi ud Din Isamic Medical College, AJK Mirpur, Pakistan.

<sup>3</sup>First Moscow State Medical University, Moscow, Russian Federation.

<sup>4</sup>Demonstrator, Ganju Khan Medical College, Swabi.

<sup>5</sup>Senior Lecturer, Hazrat Bari Imam Srkar Medical and Dental College, Islamabad.

Correspondence to: Asia Altaf

## ABSTRACT

**Objective:** The objective of this study is to determine the effect of valvular heart disease on maternal and fetal outcomes of pregnancy in young pregnant females

**Study design:** Retrospective study

**Methodology:** A retrospective study was conducted to get an analysis of 321 healthy women and 312 women with valvular heart disease who received care at a tertiary care hospital during the same time period and compared the results of their pregnancies. The chi (2)-test was used for statistical analysis, with a significance set at 0.05.

**Results:** In comparison to women in the control group, women with valvular heart disease had significantly higher rates of congestive heart failure (5.1 percent vs. 0 percent, P0.001), mortality (0.64% [two women] vs. 0 percent), and surgical interventions during pregnancy (13.4 percent (balloon mitral valvotomy) vs. 0.6 percent [ovarian cystectomy]). Additionally, the perinatal outcome was worse in the valvular heart disease group than in the control group, with higher preterm delivery rates (48.3 percent vs. 20.5 percent), lower birth weights (2434±599 g vs. 2653±542 g; P0.001), and a higher incidence of APGAR scores below 8 (8.3 percent vs. 4 percent; P0.01). Additionally, the rate of instrumental delivery increased (9.9 percent vs. 3.4 percent). The proportion of cesarean deliveries, however, was comparable across the two groups.

**Conclusion:** In conclusion, there is a substantial link between valvular heart disease and maternal and neonatal mortality. Close fetal and maternal monitoring are required, and valvular stenosis should be repaired before conception. To avoid difficulties for both the mother and the fetus, balloon valvuloplasty should be taken into consideration in cases of severe stenosis during the second trimester of pregnancy. The hospitals that deal with heart illness and pregnancy should create practical recommendations.

**Keywords:** Valvular heart disease (VHD), Pregnancy outcome; maternal outcome; Fetal outcome, young pregnant.

## INTRODUCTION

Young women with valvular heart disease are more likely to have rheumatic heart disease, congenital anomalies, or prior endocarditis; this can raise the risks to the mother and fetus from pregnancy. (Desai et al., 2000) Pregnancy is a common time for valvular heart disease. During pregnancy, the physiology of the mother undergoes major changes that result in significant increases in cardiac output and blood volume, which may reveal or exacerbate cardiac illness. Rheumatic fever is the most common cause of acquired valvular lesions, particularly in people who have emigrated from impoverished countries.

The type and severity of maternal valvular illness and the ensuing anomalies of functional capacity left ventricular function, and pulmonary pressure is related to the chance of a poor outcome. (Coetzee et al., 2020) Clinical recommendations for individuals with valvular heart disease during pregnancy are based on sparse information from case reports and observational research, or inferences drawn from information for patients in other patient groups. A normal pregnancy is associated with an increase in cardiac output of 30 to 50% and a 30 to 50% increase in blood volume. (Fessehaye et al., 2021) These gains start in the first trimester, reach their peak between 20 and 24 weeks of pregnancy, and either continue until term or start to decline. 1 The stroke volume increases, the systemic vasomotor resistance significantly lowers, the heart rate increases by 10 to 20 beats per minute, and blood pressure decreases all at the same time. Cardiovascular output increases during childbirth, and blood pressure rises in tandem with uterine contractions. Due to vena cava decompression and uterine blood returning to the systemic circulation right after birth, the heart filling pressure may rise substantially. After birth, the cardiovascular adaptations related to pregnancy fade by about six weeks.

The prevalence and distribution of VHD (1). Depending on the patient's origin country, The number of people with complicated congenital heart disease (CHD) who are becoming adults and having children has increased in the developed world as a result of advancements in the medical and surgical management of these

patients. (Avila et al., 1992) Currently, CHD makes up about 30% to 50% of all heart conditions that affect pregnant women. In non-industrialized areas, 90% of all heart disorders in women of childbearing age have rheumatic origins. Rheumatic heart disease (RHD), once the most common cause of valvular disease in the developed world, is still a common disease.

Although clinically significant maternal cardiac disease is uncommon during pregnancy (likely less than 1%<sup>4</sup>), it raises the risk of unfavorable maternal, fetal, and neonatal outcomes. 5 Based on the type of valvular anomaly and the functional class established by the New York Heart Association (NYHA), the American Heart Association and the American College of Cardiology have categorized maternal and fetal risk during pregnancy. Additional clinical considerations also affect the absolute danger that pregnancy poses to a particular woman.

About 40% of the female participants had main valve disease, 13 percent of completed pregnancies resulted in adverse maternal cardiac events, including pulmonary edema, persistent Brady arrhythmias or tachyarrhythmia requiring therapy, stroke, cardiac arrest, or death. These events were significantly more likely in women with reduced left ventricular systolic function (an ejection fraction below 40 percent), left heart obstruction (aortic stenosis with a valve area of less than 1.5 cm<sup>2</sup> or mitral 4 percent of the women who had none of these risk factors, 27 percent of the women who had one risk factor, and 62 percent of the women who had two or more risk factors experienced these results. The three dead ladies had two or more risk factors in common. Fetal mortality was 4% among pregnancies carried by women who had one or more of these risk factors, compared to 2% among those who had none.

Negative fetal outcomes were likewise at low risk. Before conception, comprehensive clinical evaluation and echocardiography are necessary for individuals with valve disease like the woman in the vignette to measure functional capacity and find any left ventricular dysfunction or valvular dysfunction. Women with valvular heart disease have a restricted capacity of their cardiovascular system to meet the demands of pregnancy. (Salazar et al., 1999) During labor and delivery, when various

changes to the circulatory system may cause hemodynamic decompensation, these restrictions are particularly obvious. 15 The suddenness of these changes can be difficult, especially for women who have a less cardiovascular reserve. Maternal hemodynamics during labor and delivery is affected by uterine contractions, discomfort, anxiety, maternal position, blood loss, the Valsalva maneuver, and analgesia if utilized.

In patients with VHD, ongoing risk assessment and a customized care strategy during pregnancy are crucial, depending on the severity of the underlying condition.

The American Heart Association/American College of Cardiology Valvular Heart Disease recommendations stress a pre-pregnancy assessment and conversation. It is still challenging to determine the risk of unfavorable maternal and fetal outcomes during pregnancy complicated by valvular heart disease. (Gupta et al., 1998)

The patient should be advised about the risk of negative cardiac outcomes if she has an aberrant cardiac function, left ventricular dysfunction, valve obstruction, or a history of heart failure or embolic events. Pregnancy may not be detectable in people with more than one of these risk factors.

**Literature Review:** This study took place in 2014 on valvular cardiovascular disease that causes 1 to 3 percent of pregnancies to become complicated and accounts for 10 to 15 percent of maternal deaths. Heart disease is the most common cause of maternal death, despite having a low prevalence rate among pregnant women. The prevalence of cardiovascular disease in pregnancy is rising as more women with congenital or acquired heart disease reach childbearing age and want to have children as a result of better medical and surgical care. Patients with severe congenital or acquired valvular heart disease (VHD) have care concerns when they become pregnant.

Before becoming pregnant, many individuals with substantial VHD are unaware of their condition, and the diagnosis is only discovered when the hemodynamic demands of pregnancy cause clinical symptoms. (Wada et al., 1996). A 5-year, multicenter prospective research including 60 pregnant individuals with valvular heart disease was conducted between 2005 and 2009. 38 patients (64%) had only one affected valve, with mitral stenosis being the most common lesion (50 percent). Ten patients (16.7%) had interventional and surgical corrections made before becoming pregnant. An NYHA class III-IV patient was detected in 14 (24%) patients. This study's objective was to assess the effects of pregnancy on the mother and the fetus in cases of valvular heart disease (VHD). Preterm delivery and newborns that were undersized for gestational age occurred at rates of 11.69 percent and 13.36 percent, respectively. (Horstkotte et al., 2003)

The main goal of this review is to, assess the risk assessment, and management concerns about the care of women of reproductive age with VHD who are pregnant or may become pregnant. Before becoming pregnant, many individuals with substantial VHD are unaware of their condition, and the diagnosis is only discovered when the hemodynamic demands of pregnancy cause clinical symptoms. Despite substantial improvements in diagnosis, medicinal treatment, and surgical treatment for VHD, the path for many of these patients during and after pregnancy can be wrought with serious ill effects for both the mother and fetus. The prevalence of cardiovascular disease in pregnancy is rising as more women with congenital or acquired heart disease reach childbearing age and want to have children as a result of better medical and surgical care. Patients with severe congenital or acquired valvular heart disease (VHD) have care concerns when they become pregnant. As it concluded that cardiovascular disease causes 1 to 3 percent of pregnancies to become complicated and accounts for 10 to 15 percent of maternal deaths. (Rajput and Zeltser, 2022)

The purpose of the study was to compare how pregnant women with valvular heart disorders fared. We saw 259 pregnant women with cardiac conditions, ranging in age from 18 to 42. 158 patients in Group I have mitral valve disease, including 51 patients

with mitral valve prolapse, 44 patients with mitral regurgitation, and 33 patients with mixed mitral valve disease.

Group III: 47 patients following valve replacement; Group II: 54 patients with aortic valve disease, including 32 patients with aortic stenosis and 22 patients with aortic regurgitation (36 mechanical; 11 homograft valves). During each of the pregnancy's three trimesters, as well as after birth, medical history and physical examination, an evaluation of the patient's NYHA class, an ECG, and an echocardiogram were conducted. (Fennira et al., 2008)

## METHODOLOGY

A retrospective study was conducted to get an analysis of 321 healthy women and 312 women with valvular heart disease who received care at a tertiary care hospital during the same time period and compared the results of their pregnancies. The chi (2)-test was used for statistical analysis, with a significance set at 0.05.

**Study Design:** Randomized Clinical Trial.

**Setting:** Different hospitals in Lahore.

**Duration of Study:** 12 months after approval of synopsis.

**Sample Size:** The sample size of 321 cases of healthy women and 312 with valvular heart disease cases; were calculated with a confidence level of 0.5, by using the following formula.

Where  $n$  is the sample size required in each group ( $i=1,2$ ),  $Z$  is the value from the standard normal distribution reflecting the confidence level that will be used (e.g.,  $Z = 1.96$  for 95%), and  $E$  is the desired margin of error.  $p_1$  and  $p_2$  are the proportions of successes in each comparison group. To generate a 95% confidence interval for the difference in unknown proportions, the formula to estimate the sample sizes needed requires  $p_1$  and  $p_2$ . To estimate the sample size, we need approximate values of  $p_1$  and  $p_2$ . The values of  $p_1$   $p_2$  that maximize the sample size are  $p_1=p_2=0.5$ . Thus, if there is no information available to approximate  $p_1$  and  $p_2$ , then 0.5 can be used to generate the most conservative, or largest, sample sizes (Boston University, 2016).

**Sampling technique:** Purposive Nonprobability consecutive sampling

**Statistical Analysis:** Continuous variables were expressed as mean values and standard deviation for normally distributed data, and as the median and interquartile range for non-normally distributed data. Categorical variables were expressed as frequencies and percentages. Paired t-tests or Chi-square tests were used for group comparison. For all steps, a p-value of  $<0.05$  was regarded as statistically significant. All analyses were performed with commercially available software (SPSS software version 24.0, SPSS Inc., Chicago, IL). 5 mg/day to 10 mg mg/d

## RESULTS

Results showed that 38 patients—25 women from Group I, 6 from Group II, and 7 from Group III—had clinical deterioration. 250 healthy newborns (ten premature and twelve with intrauterine growth retardation), six abortions, two stillbirths, and one neonatal death. 200 deliveries were vaginal and 53 were cesarean sections.

A high-risk category of potentially fatal consequences affects pregnant women with severe mitral valve stenosis, researchers have concluded (2) In women with severe aortic stenosis, pregnancy may cause a precipitous decline in clinical status. (3) Patients with enlarged left ventricles and diminished function should be prepared for cardiac problems. (L4) 312 pregnant women with valvular heart disease made up the study group (group 1), while 321 pregnant women without heart disease made up the control group. Table 1 displays the traits of the women in the two groups. Age, parity, ethnicity (religion), and prenatal care were closely matched between the two groups. However, group 1 had a higher incidence of previous abortions (22.1% vs. 5.2%;  $P=0.01$ ), stillbirths (6.4% vs. 0.62%), and preterm births (0.96% vs. 0;  $P=0.05$ ). The quantity and proportion of booked and unbooked cases in the two groups did not statistically differ from one another (Table 1).

According to Table 1, the majority of women received their first antenatal appointment in the second (49 percent vs. 48.6 percent; P) 0.05) or third trimester (42.9 percent vs. 42.7 percent; P) 0.05). Table 2 displays the different valvular lesions the women investigated had. The majority of women (52.6 percent) either had mitral stenosis alone or in conjunction with other valve diseases (33.3 percent). Aortic stenosis was the least prevalent condition, with only 58 (12.5 percent) women experiencing mitral regurgitation (1.6 percent). 175 (56.1%) of the women in the study group had New York Heart Association (NYHA) Class I illness at their initial visit, whereas 96 (30.8%), 26 (8.3%), and 15 (4.8%) had Class II, III, and IV disease, respectively.

Table 3 provides information on the prenatal history, including surgical intervention and medical problems. All problems were observed in patients with heart disease at much higher rates than in controls. 42 (13.4%) women with severe mitral stenosis underwent balloon mitral valvotomies. In the control group, two women (0.6%) underwent ovarian cystectomies when they were pregnant. Significantly more cases of maternal problems were observed in the study group (18.6 percent vs. 1.2 percent; P-0.001). 16 (5.1%) of the women had congestive heart failure, while 23 (7.3%) of the women had cardiac arrhythmias, the bulk of which were atrial fibrillation (0.9%) and ventricular ectopic beats (6.4%). One woman in the control group had ventricular ectopic beats, and one lady suffered paroxysmal supraventricular ventricular tachycardia, whereas one woman also experienced sinus tachycardia. Two maternal fatalities occurred in the trial group, and both patients had congestive heart failure and multivalvular heart disease. Table 4 displays the major obstetric issues and fetal outcomes. Incidences of twin pregnancies, hypertension, hematemesis and gestational diabetes mellitus were not significantly different between the two groups.

However, a higher percentage of women with heart disease (8.65 percent vs. 4.36 percent; P-0.05) had severe anemia. A worse fetal outcome was linked to valvular heart disease. The study group had a significantly greater rate of premature births and other obstetric problems (48.3 percent vs. 20.5 percent; P-0.001). Additionally, the study group saw higher instances of intrauterine growth retardation than the control group did (35.37"5.49 weeks vs. 37.7"2.68 weeks; P0.001). The study group's mean birth weight was substantially lower (2434"599 g vs. 2653"542 g; P-0.001) than the control groups. The study group contained a considerably larger proportion of newborns with low birth weight and an Apgar score below 8. The incidence of congenital defects and stillbirths between the two groups, however, did not significantly differ from one another. The delivery methods are given in Table 5.

Table 1: Characteristics of study

Characteristics	Group1	Group2	P-value	Significance
Number of women	312	321	>0.05	NS
Marriage mean"S.D., years	25.2±3.7	24.4"2.9	>0.05	NS
Mean parity, mean"S.D.	0.95"1.03	0.76"0.82		NS
Religion, N (%)				
Muslim	230 (73.7)	249 (77.6)	>0.05	NS
Hindu	72 (26.3)	26.3 (82)	>0.05	NS
Obstetric history, N (%)				
Abortion	69 (22.1)	17 (5.2)	<0.001	NS
Stillbirth	20 (6.4)	2 (0.62)	<0.001	NS
Preterm delivery	3 (0.96)	0	>0.05	NS
Present preference, N (%)				
Booked	109 (11.5)	115 (35.8)	>0.05	NS
Unbooked	96 (30.8)	98 (30.5)	>0.05	NS
Registered	107 (34.2)	108 (33.6)	>0.05	NS
First visit, N (%)				
First trimester	25 (8)	28 (8.7)	>0.05	NS
Second trimester	153 (49.0)	156 (48.6)	>0.05	NS
Third trimester	134 (42.9)	137 (42.7)	>0.05	NS

Table 2: Valvular lesions investigation

Type of lesion	No. (%)
Mitral stenosis (MS)	164 (52.6)

Mitral regurgitation (MR)	56 (12.5)
aortic regurgitation (AR)	7 (2.2)
tricuspid regurgitation (TR)	
pulmonary artery	3 (0.8)
Hypertension	
NYHA Class	
Class I	175 (56.1)
Class II	96 (30.8)
Class III	26 (8.3)
Class IV	15 (4.8)

Table 3: Parental History

Event	Study group	Control group	P-value	Significance
Operative intervention				
Balloon mitral valvotomy	42 (13.4)	0	-0.001	HS
Ovarian cystectomy	0	2 (0.6)	>0.05	NS
Antenatal complication				
No complications	254 (81.4)	318 (98.8)	<0.001	HS
Complications	56 (18.6)	3 (1.2)	<0.001	HS
Congestive heart failure	16 (5.1)	0	<0.001	HS
Cardiac arrhythmias	23 (7.3)	3 (1.2)	<0.001	HS
Ventricular ectopic beats	20 (6.4)	0	<0.001	HS
Atrial fibrillation	3 (0.9)	3 (0.93)	>0.05	HS
Paroxysmal sinus ventricular tachycardia	0	1 (0.31)	>0.05	HS
Sinus tachycardia	0	1 (0.31)	>0.05	NS
Maternal mortality	2 (0.64)	-0.01	<0.001	HS

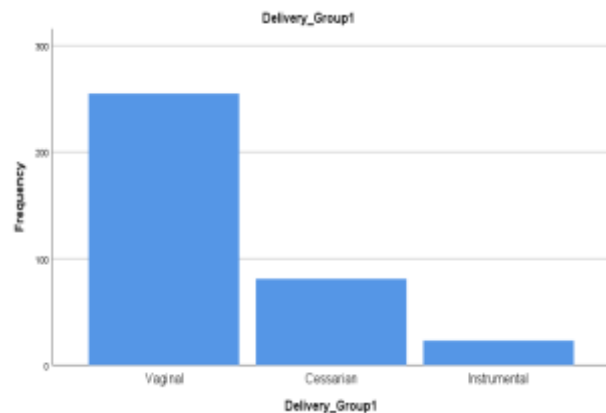
Table 4: The major obstetric issues and fetal outcomes

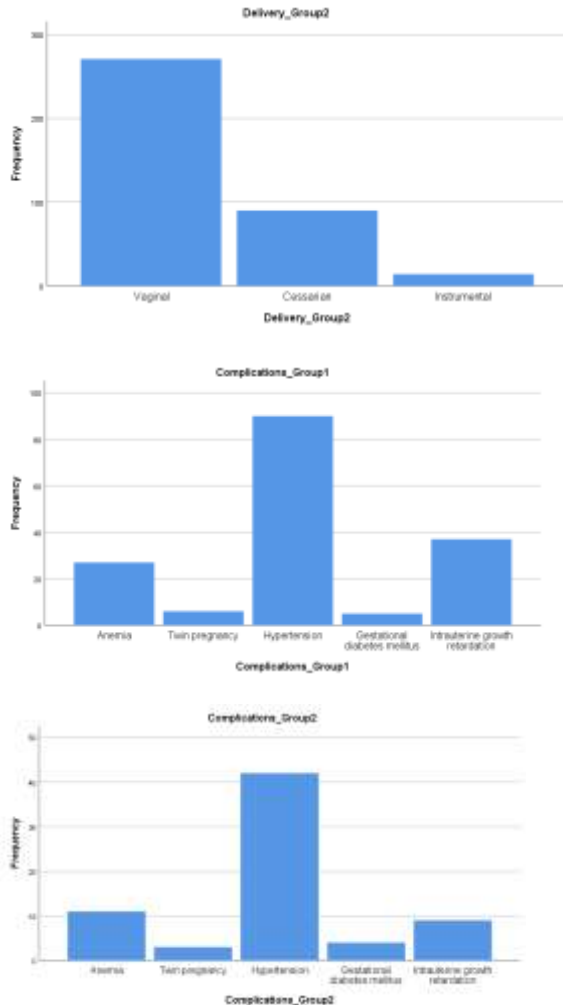
Complication	Group 1	Group 2	P-value	Significance
Severe anemia	27 (8.65)	14 (4.36)	<0.05	S
Twin pregnancy	6 (1.9)	3 (0.9)	>0.05	NS
Hypertension				
Essential hypertension	2 (0.6)	2 (0.6)	>0.05	NS
Pregnancy-induced	28 (8.7)	28 (8.7)	>0.05	NS
Eclampsia	4 (1.2)	4 (1.2)	>0.05	NS
Hematemesis	0	0	>0.05	NS
Gestational diabetes mellitus	2 (0.6)	2 (0.6)	>0.05	NS
Intrauterine growth retardation	5 (1.5)	5 (1.5)	<0.001	S
Preterm delivery	66 (20.5)	66 (20.5)	<0.001	HS
Gestational age, mean"S.D., weeks	37.7"2.7	37.7"2.7	<0.001	HS
Congenital anomalies	0	0	>0.05	NS
Birth weight, mean"S.D., g	2653"542	2653"542	<0.001	HS
Low birth weight	109 (33.9)	109 (33.9)	<0.01	S
Newborn with Apgar score	13 (4)	13 (4)	<0.01	S
Stillbirth	9 (2.8)	9 (2.8)	>0.05	NS

Table 5: The delivery methods

Mode	Group 1(N %)	Group 1(N %)	P-value	Significance
Vaginal	255 (81.7)	271 (89.4)	>0.05	NS
Cesarean	26 (8.9)	39 (21.1)	>0.05	NS
Instrumental	29 (9.9)	11 (3.4)	<0.001	HS

Abbreviations: HS, highly significant; NS, not significant.





## DISCUSSION

Cardiac illness during pregnancy is still a serious health issue, especially in developing nations, and it is linked to high rates of maternal and neonatal death and morbidity w1, 2x. The most prevalent and hazardous condition that significantly increases maternal mortality in pregnancy is mitral stenosis. (González Maqueda et al., 2000)

For severe mitral stenosis in the second trimester of pregnancy, surgical procedures such as balloon mitral valvotomy and commissurotomy have been used, with pronounced clinical improvement and excellent maternal and fetal outcomes (Wiederhorn et al., 1992)

Although quick diagnosis and treatment are necessary, most hospitals may not have access to effective surgical facilities. Additionally, performing surgery while pregnant is frowned upon. Angina and left ventricular failure can be brought on by left ventricular outflow tract obstruction during pregnancy. (Shotan et al., 1997)

There is an ongoing debate over the best course of action for pregnant women who have artificial valves, particularly in regards to whether to continue taking warfarin or switch to heparin. The usage of a bio prosthesis in young women planning future pregnancies is decreasing as more research shows that these prostheses degrade more quickly during pregnancy. However, a recent extensive investigation from a significant cardiac facility in Mexico found no evidence of a speeding up of the deteriorating process for bovine pericardial bio

prostheses. A recent large study from a major cardiac center in Mexico did not find any acceleration in the rate of deterioration of bovine pericardial bio prostheses.

In this present study, the most prevalent lesion was mitral regurgitation alone, which was followed by various valvular diseases (33.3 percent) (12.5 percent). Women with valvular heart disease experienced pregnancy problems at much higher rates than women in the general population (18.6 percent vs. 1.2 percent;  $P < 0.001$ ). There were considerably more instances of cardiac arrhythmias (ventricular ectopic beats and atrial fibrillation) in the study group than in the control group (7.3 percent vs. 0.93 percent;  $P < 0.001$ ). 5.1 percent of patients had congestive heart failure, compared to none in the control group. Two maternal fatalities occurred in the research group. Our findings are consistent with those from other investigations. In their investigation of valvular heart disease during pregnancy, Hameed et al. w2x revealed that 38 percent of congestive heart failures, 15 percent of arrhythmias, and 2 percent of maternal deaths occurred. In their examination of pregnant women with mitral stenosis, Desai et al. w1x found that 38% of the patients had congestive heart failure. Numerous other studies from India, Japan, and Mexico have found higher maternal complications. Arrhythmias may also be attributed to the prenatal hormonal influence and a probably increased susceptibility to catecholamine during pregnancy, and they may also be explained by the new beginning or aggravation of existing arrhythmias due to the increased hemodynamic burden. (Brodsky et al., 1992)

In comparison to the control group, there were more unfavorable fetal outcomes in the group with valvular heart disease. Lower birth weight newborns (43.9 percent vs. 33.9 percent;  $P < 0.01$ ), preterm birth rate (48.3 percent vs. 20.5 percent;  $P < 0.001$ ), intrauterine growth retardation (5.7 percent vs. 1.5 percent); and neonates with Apgar scores less than 8 were all considerably more common (98.3 percent vs. 4 percent;  $P < 0.01$ ). In valvular disease patients, the mean gestation period was significantly shorter (35.37"5.49 weeks vs. 37.7"2.68 weeks;  $P < 0.001$ ), and the mean birth weight was likewise significantly lower

Fortunately, only two cases of maternal mortality were observed in this study as a result of the team approach to the management of pregnant and laboring women with cardiac disease in collaboration with an advanced cardiac center. Therefore, our data support the observation made by other authors that mortality in medically treated pregnant women with valvular heart disease can be rare with early diagnosis and close monitoring.

## CONCLUSION

In conclusion, there is a substantial link between valvular heart disease and maternal and neonatal mortality. Close fetal and maternal monitoring are required, and valvular stenosis should be repaired before conception.

To avoid difficulties for both the mother and the fetus, balloon valvuloplasty should be taken into consideration in cases of severe stenosis during the second trimester of pregnancy. The hospitals that deal with heart illness and pregnancy should create practical recommendations,

## REFERENCES

1. AVILA, W. S., GRINBERG, M., DÉCOURT, L. V., BELLOTTI, G. & PILEGGI, F. 1992. [Clinical course of women with mitral valve stenosis during pregnancy and puerperium]. *Arq Bras Cardiol*, 58, 359-64.
2. BRODSKY, M., DORIA, R., ALLEN, B., SATO, D., THOMAS, G. & SADA, M. 1992. New-onset ventricular tachycardia during pregnancy. *Am Heart J*, 123, 933-41.
3. COETZEE, A., SADHAI, N., MASON, D., HALL, D. R. & CONRADIE, M. 2020. Evidence to support the classification of hyperglycemia first detected in pregnancy to predict diabetes 6-12 weeks postpartum: A single center cohort study. *Diabetes Res Clin Pract*, 169, 108421.
4. DESAI, D. K., ADANLAWO, M., NAIDOO, D. P., MODLEY, J. & KLEINSCHMIDT, I. 2000. Mitral stenosis in pregnancy: a four-year

- experience at King Edward VIII Hospital, Durban, South Africa. *Bjog*, 107, 953-8.
5. FENNIRA, S., REJEB, M. A., ELLOUZE, Y., KHALDI, H., OUERTANI, W., TELLILI, S., BATTIKH, K., LONGO, S., KRAIEM, S. & SLIMENE, M. L. 2008. [Heart diseases in pregnant women]. *Tunis Med*, 86, 584-90.
  6. FESSEHAYE, A., TAFERE, Y. T. & ABATE, D. D. 2021. Postpartum maternal collapse-a first-time presentation of severe mitral stenosis: a case report. *Journal of medical case reports*, 15, 225-225.
  7. GONZÁLEZ MAQUEDA, I., ARMADA ROMERO, E., DÍAZ RECASENS, J., GARCÍA DE VINUESA, P. G., GARCÍA MOLL, M., GONZÁLEZ GARCÍA, A., FERNÁNDEZ BURGOS, C., IÑIGUEZ ROMO, A. & RAYO LLERENA, I. 2000. [Practice Guidelines of the Spanish Society of Cardiology for the management of cardiac disease in pregnancy]. *Rev Esp Cardiol*, 53, 1474-95.
  8. GUPTA, A., LOKHANDWALA, Y. Y., SATOSKAR, P. R. & SALVI, V. S. 1998. Balloon mitral valvotomy in pregnancy: maternal and fetal outcomes. *J Am Coll Surg*, 187, 409-15.
  9. HORSTKOTTE, D., FASSBENDER, D. & PIPER, C. 2003. [Congenital heart disease and acquired valvular lesions in pregnancy]. *Herz*, 28, 227-39.
  10. RAJPUT, F. A. & ZELTSER, R. 2022. *Aortic Valve Replacement*. StatPearls. Treasure Island (FL): StatPearls Publishing
  11. Copyright © 2022, StatPearls Publishing LLC.
  12. SALAZAR, E., ESPINOLA, N., ROMÁN, L. & CASANOVA, J. M. 1999. Effect of pregnancy on the duration of bovine pericardial bioprostheses. *Am Heart J*, 137, 714-20.
  13. SHOTAN, A., OSTRZEGA, E., MEHRA, A., JOHNSON, J. V. & ELKAYAM, U. 1997. Incidence of arrhythmias in normal pregnancy and relation to palpitations, dizziness, and syncope. *Am J Cardiol*, 79, 1061-4.
  14. WADA, H., CHIBA, Y., MURAKAMI, M., KAWAGUCHI, H., KOBAYASHI, H. & KANZAKI, T. 1996. [Analysis of maternal and fetal risk in 594 pregnancies with heart disease]. *Nihon Sanka Fujinka Gakkai Zasshi*, 48, 255-62.
  15. WIDERHORN, J., WIDERHORN, A. L., RAHIMTOOLA, S. H. & ELKAYAM, U. 1992. WPW syndrome during pregnancy: increased incidence of supraventricular arrhythmias. *Am Heart J*, 123, 796-8.