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FACULTY OF MEDICINE AND BIOMEDICAL SCIENCES

DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY

POST RHEUMATIC VALVULAR HEART DISEASE IN PREGNANCY: CASE REPORT AND LITERATURE REVIEW

Dissertation submitted in partial fulfilment of the requirements for the award of a University Diploma

in

Management of High-risk Pregnancies by;

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2023-2024 Academic year

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DEDICATION

This work is dedicated to my parents

Mr. SULE GARBA DANLADI

And

Mrs. SODA IDIRISU ZENABU

ACKNOWLEDGEMENTS

TO ALMIGHTY GOD: THANK YOU, ALLAH, FOR EVERYTHING.

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LIST OF LECTURERS FOR THE UNIVERSITY DIPLOMA PROGRAMME IN MANAGING HIGH-RISK PREGNANCIES

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Dr KUABAN Alain

Dr ZAMBO Huguette

PROGRAMME OVERVIEW

The University Diploma in Management of High-Risk Pregnancies is the fruit of a long struggle and unprecedented hard work by our lecturers at the Faculty of Medicine and Biomedical Sciences of the University of Yaoundé I, Professor MEKA Née NGO UM Esther, Associate Professor of Obstetrics and Gynaecology, assisted by Professors NOA NDOUA Claude Cyrille and ESSIBEN Félix, both Associate Professor of Obstetrics and Gynaecology. The aim of the Diploma in Management of High-Risk Pregnancies is to provide health professionals such as obstetricians, midwives, nurses and other allied health professionals with the basic and advanced knowledge necessary to provide effective prenatal and perinatal care.

The University Diploma in Management of High-Risk Pregnancies is a crucial step in addressing the complexities associated with high-risk pregnancies. By equipping healthcare professionals with the knowledge, skills and practical experience required for effective management, this programme not only enhances individual career prospects, but also contributes to improved maternal and fetal health outcomes. As the demand for specialist care continues to grow, the importance of such training initiatives cannot be overstated, ultimately leading to healthier pregnancies and brighter futures for mothers and their children.

Our expectations during this DU were high, based mainly on the quality of the training and the acquisition of all possible skills to be a good perinatolologist, aware of the obligations of scientific competence and technical efficiency normally expected, in order to make my modest contribution to reducing maternal and perinatal mortality. In this report, we invite you to take a look at our training and the clinical case we have reported.

PRESENTATION OF UNIVERSITY DIPLOMA IN MANAGEMENT OF HIGH-RISK PREGNANCIES

The University Diploma in Management of High-Risk Pregnancies is an educational diploma organised by the Faculty of Medicine and Biomedical Sciences (FMBS) of the University of Yaoundé I. The course is specifically designed for health care providers who wish to enhance their expertise in maternal-fetal medicine.

To achieve these objectives, our training programme was structured as follows:

- Four lecture seminars over four months, i.e. one week of lectures per month in the new pedagogical block of the Faculty of Medicine and Biomedical Sciences of the University of Yaoundé I,
- Timetable: 08:00-17:00 Monday to Friday with one hour break
- Practical sessions
- culminating in a final examination consisting of multiple-choice questions, open questions and clinical cases.

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LIST OF ABBREVIATIONS

AHA: American Heart Association

AIDS: acquired immunodeficiency syndrome

aPTT: activated partial thromboplastin time

AR: aortic regurgitation

CARPREG: Cardiac Disease in Pregnancy

CHD: congenital heart disease

CMR: cardiac magnetic resonance

ECG: electrocardiogram

EROA: effective regurgitant orifice area

INR: international normalized ratio

LMWH: low molecular weight heparin

LVEDD: left ventricular end-diastolic diameter

LVEF: left ventricular ejection fraction

LVESD: left ventricular end systolic diameter

MR: mitral regurgitation

NYHA: New York Heart Association

PAP: pulmonary artery pressure

PET: positron emission tomography

PHT: pressure half time

RVol: regurgitant volume

TEE: transoesophageal echocardiography

TTE: transthoracic echocardiography

UFH: unfractionated heparin

VHD: valvular heart disease

ZAHARA: Zwangerschap bij Aangeboren HARtAfwijkingen I

ABSTRACT

Valvular heart disease in pregnancy is rare (less than 1%), but its presence increases the risk of adverse maternal, fetal and neonatal outcomes. valvular heart disease is almost exclusively the consequence of childhood rheumatic fever in developing countries.

This case report describes the management of a 20-year-old patient with a history of rheumatic heart disease who presented in labour with decompensated mitral-aortic regurgitation. Despite an initially uneventful pregnancy, she developed significant cardiac symptoms including palpitations, dyspnoea and arrhythmias at 36 weeks' gestation. Comprehensive investigations, including echocardiography, confirmed severe cardiac complications characterised by mitral and aortic regurgitation, left ventricular dysfunction and pulmonary hypertension. A multidisciplinary team decided to perform an emergency caesarean section due to the mother's deteriorating cardiac condition. Post-operative management focused on controlling cardiac symptoms and preventing complications, which included fluid management, diuretics and antiarrhythmic therapy. The patient's recovery was complicated by acute pulmonary oedema, which required further interventions. Although her condition improved over five days, she presented with persistent tachycardia and signed for discharge against medical advice.

This case highlights the importance of pre-pregnancy counselling, risk stratification and tailored management plans for pregnant women with valvular heart disease, as well as the impact of pregnancy on cardiac health and vice versa. We conclude that although pregnancy carries additional risks for women with valvular heart disease, appropriate care can reduce adverse outcomes for both mother and child.

Keywords: valvular heart disease, pregnancy, rheumatic heart disease, mitral-aortic regurgitation, materno-fetal outcome

RESUME

Les cardiopathies valvulaires sont rares pendant la grossesse (moins de 1 %), mais leur présence augmente le risque de complications pour la mère, le fœtus et le nouveau-né. Elle est presque exclusivement la conséquence du rhumatisme articulaire aigu de l'enfant dans les pays en voie de développement.

Ce rapport de cas décrit la prise en charge d'une patiente de 20 ans ayant des antécédents de cardiopathie rhumatismale et qui s'est présentée en travail avec une régurgitation mitrale-aortique décompensée. Malgré une grossesse initialement sans incident, elle a développé des symptômes cardiaques importants, notamment des palpitations, une dyspnée et des arythmies, à la 36e semaine d'aménorrhée. Des examens complets, y compris une échocardiographie, ont confirmé des complications cardiaques graves caractérisées par une insuffisance mitrale et aortique, un dysfonctionnement du ventricule gauche et une hypertension pulmonaire. Une équipe multidisciplinaire a décidé de pratiquer une césarienne d'urgence en raison de la détérioration de l'état cardiaque de la mère. La prise en charge postopératoire a consisté à contrôler les symptômes cardiaques et à prévenir les complications, y compris la gestion des fluides, des diurétiques et d'une thérapie antiarythmique. Le rétablissement de la patiente a été compliqué par un œdème pulmonaire aigu, qui a nécessité d'autres interventions. Bien que son état se soit amélioré en cinq jours, elle a présenté une tachycardie persistante et a signé pour sortir de l'hôpital contre avis médical.

Ce cas souligne l'importance du conseil avant la grossesse, de la stratification des risques et des plans de gestion personnalisés pour les femmes enceintes atteintes d'une cardiopathie valvulaire, ainsi que l'impact de la grossesse sur la santé cardiaque et vice-versa. Nous concluons que, bien que la grossesse comporte des risques supplémentaires pour les femmes souffrant de cardiopathie valvulaire, des soins appropriés peuvent réduire les conséquences négatives pour la mère et l'enfant.

Mots clés: cardiopathie valvulaire, grossesse, cardiopathie rhumatismale, insuffisance mitrale-aortique, issue materno-fœtale

BACKGROUND

Cardiovascular disease complicates ≈1% to 3% of all pregnancies and is responsible for 10% to 15% of maternal mortality. Valvular heart disease accounts for about a quarter of all cardiac conditions that complicate pregnancy. As more women with congenital or acquired heart disease reach childbearing age and desire children due to improved medical and surgical care, the incidence of cardiovascular disease in pregnancy is increasing. In developing countries, heart valve disease is almost exclusively the result of rheumatic fever in children. Valvular heart disease may be a pre-existing complication of pregnancy or may be diagnosed for the first time during pregnancy. Pregnancy is associated with significant haemodynamic changes that may exacerbate valvular heart disease and may be associated with significant adverse events for both mother and foetus. Accurate diagnosis, tailored therapy and an understanding of the physiology and pathophysiology of pregnancy are necessary components of management, best achieved through a multidisciplinary approach. We present a case of post-rheumatic mitral aortic regurgitation in a 20-year-old nulliparous patient with the aim of discussing the course of the pregnancy and the particularities of maternal and foetal care.

CASE HISTORY

20 years old, student, G1P0, GA: 37wks+2days with a past medical history of rheumatic heart disease since 6 years old on captopril but non-compliant to treatment (for about 4 years), no surgical history, presented to our facility (Yaoundé Central Hospital) in labour.

She attended 5 Antenatal consultations at CASS Nkoldongo. First contact at 18 weeks, baseline blood pressure (BP) was 127/77mmHg. She received 2 doses of anti-tetanus vaccine, 4 doses of IPTp, iron and calcium supplementation during her pregnancy. She is Blood Group O+, Hb Electrophoresis AA, RDT for malaria negative, FBS = 0.79 g/dl, TPHA/VDRL negative, HBsAg negative, Hepatitis C antibody negative, HIV negative, Toxoplasmosis/Rubella immunized, FBC normal (WBC 7000, HB 12.7g, PLT 175,000). Two obstetric ultrasounds were performed at the 2nd and 3rd trimesters, which were uneventful.

The course of the pregnancy was uneventful until 36 weeks, when the patient developed persistent palpitations and fatigue, motivating a cardiology consultation. BP: 109/74, pulse: 126bpm, B1 and B2 heart sounds present but irregular, systolic murmur (MR: 3/6, AR: 2/6) and bilateral pitting oedema. The ECG showed supraventricular tachycardia at 175 bpm and left ventricular hypertrophy (Figure 1). Cardiac ultrasound revealed post-rheumatic polyvalvulopathy with associated severe Carpentier type III mitral regurgitation (EROA:

200mm2, RVol: 187ml) and moderate aortic regurgitation (PHT: 288ms). Repercussions: Left ventricular dilatation (LVEDD: 70mm, LVESD: 52mm), alteration in left ventricular systolic function (LVEF at 46%). Severe bi-atrial dilatation with ectasia of the left atrium (OGi volume 470ml/m2); moderate pulmonary hypertension (PAPs 51 mmHg).

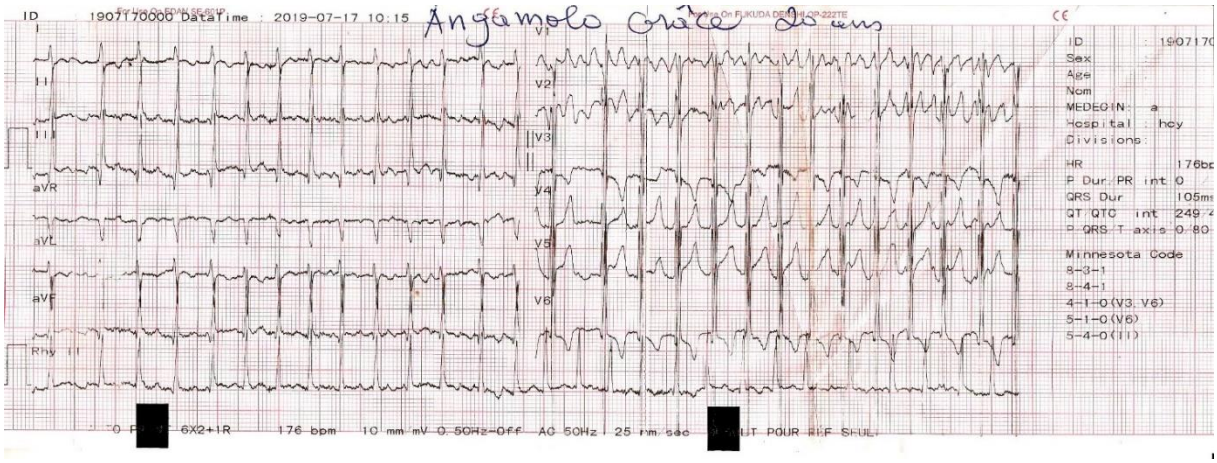

Figure 1: ECG showing supraventricular tachycardia at 175bpm, and left ventricular hypertrophy

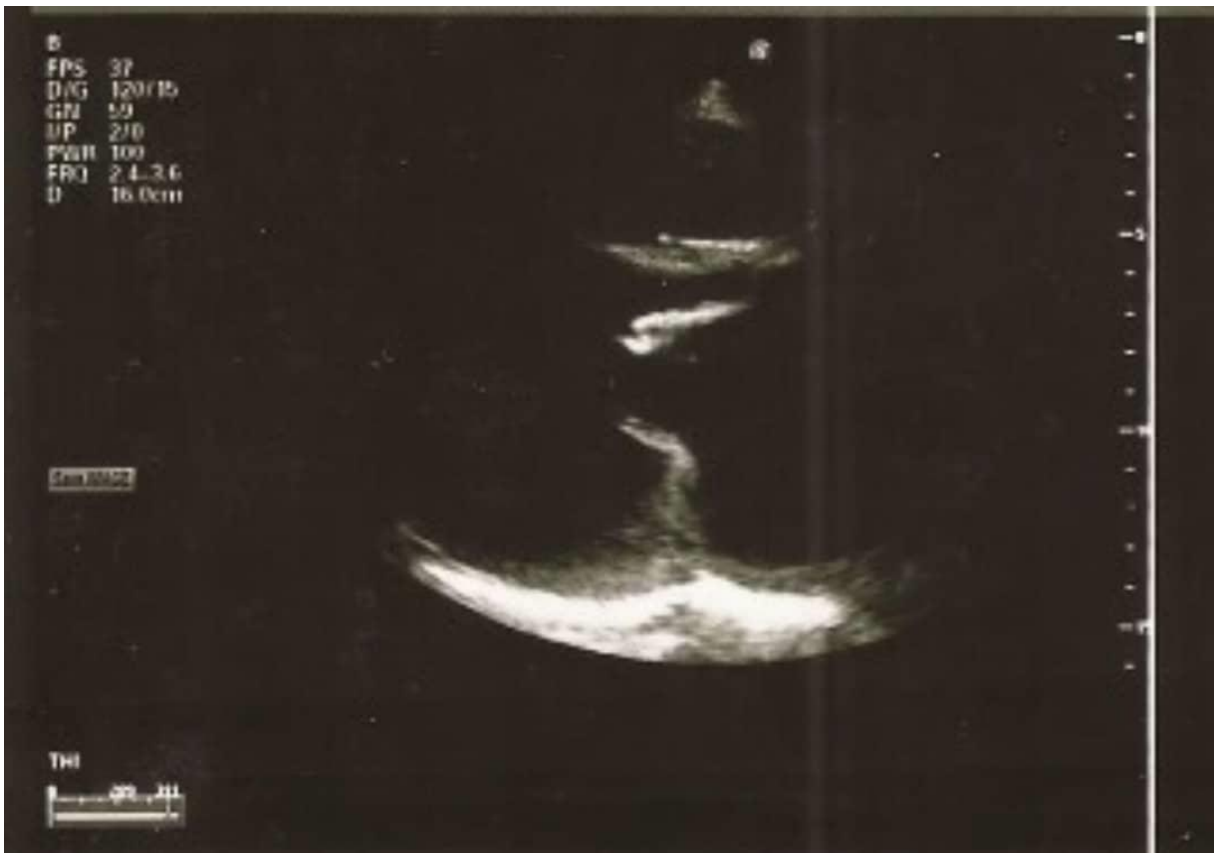

Figure 2: Image showing dilated left ventricle, calcified and remodelled incompetent mitral valve

Figure 3: Depicts severe mitral regurgitation with regurgitant volume over 100ml

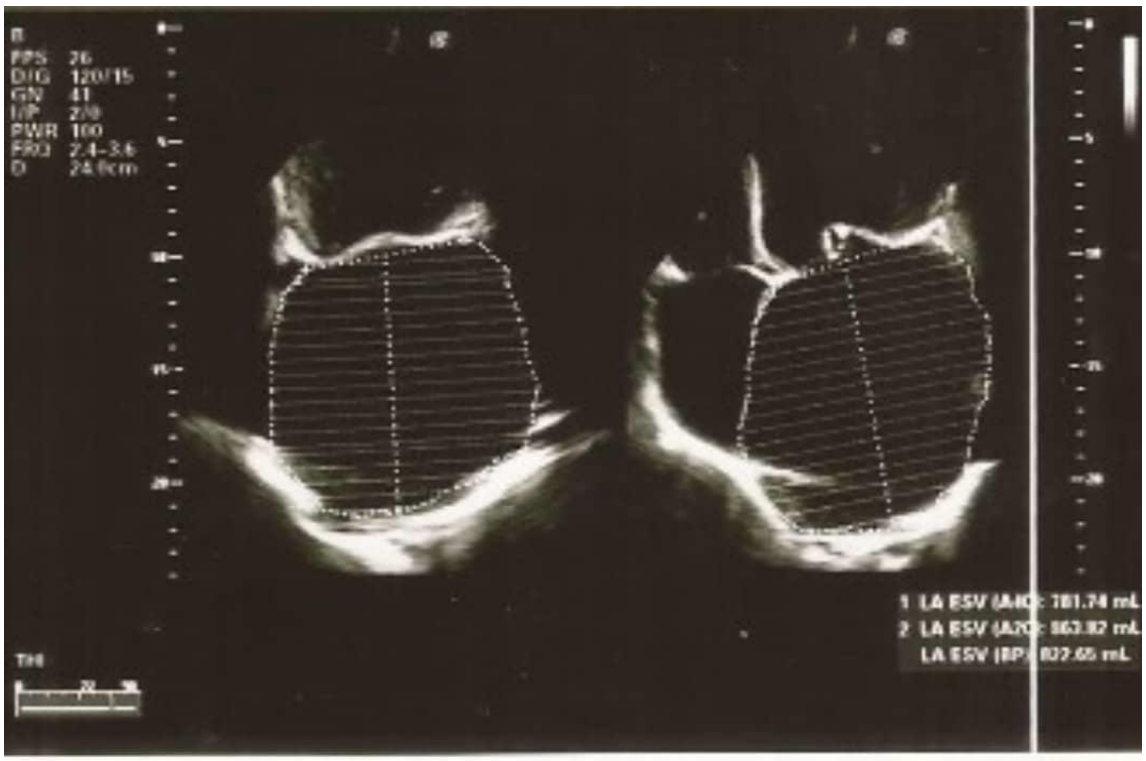

Figure 4 : Image shows markedly dilated left atrium

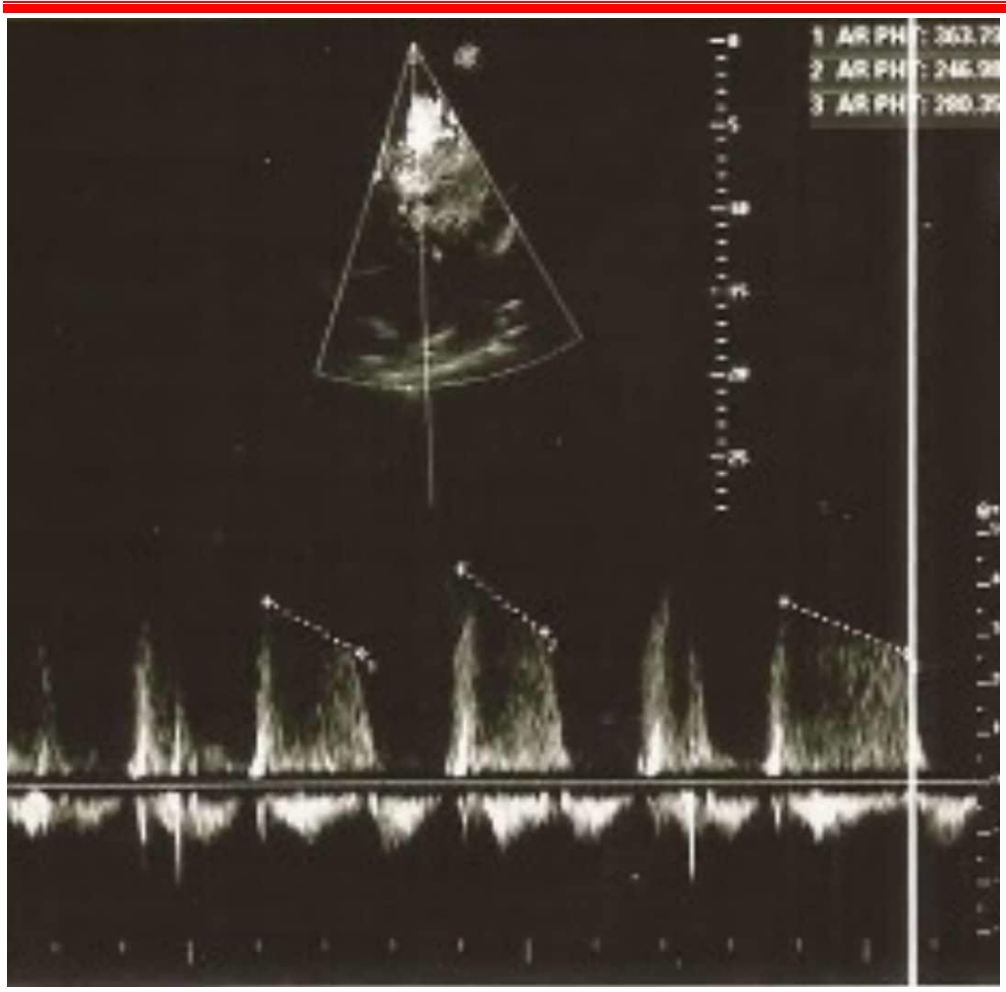

Figure 5: Image shows moderate aortic regurgitation

Review of systems revealed stage IV dyspnoea (NYHA), polypnea, fatigue.

Physical examination revealed altered general condition, BP 103/89mmHg, pulse 165bpm, temp 36.8oC, RR 28bpm, SPO2 98% (AA), systolic murmur (MR:4/6, AR:2/6), FH 30cm, cephalic presentation, FHT 138-141bpm, 2 uterine contractions in 10min. Cervix was dilated to 3cm, 80% effaced, station -1 with intact membranes, clinically adequate pelvis. Bilateral pitting oedema. The rest of the physical examination was unremarkable.

We concluded on a latent phase of labour in the context of a decompensated mitral-aortic regurgitation by a left heart failure (stage IV dyspnoea, moderate pulmonary hypertension and arrhythmias).

A multidisciplinary meeting (obstetrician, cardiologist and anaesthetist) decided on emergency caesarean section (C/S) as the mode of delivery.

The pre anaesthetic work up was uneventful and spinal anaesthesia was chosen with a pre anaesthetic fluid administration of 1500cc to avoid fluid overload.

C/S resulted in the delivery of a male baby weighing 2530g, APGAR 9, 10. EBL 400CC, urine output 0.83ml/kg/h. The operation was well tolerated.

The aim of post operative management was to improve symptoms notably by reducing dyspnoea and increasing tolerance to exertion. Also, we set out to prevent complications either related to surgery or valvular heart disease. Fluids 30ml/kg/day, ATB prophylaxis, analgesics, LMWH (enoxaparin), compression stockings and stress ulcer prophylaxis with proton pump inhibitor.

Two hours post op, patient complained of exacerbation of dyspnoea and orthopnoea. She also had cough with frothy expectorations. Physical examination showed a hypoxemic patient in respiratory distress with SP02 at 83% on ambient air and use of accessory respiratory muscles. A diagnosis of acute pulmonary oedema following decompensated mitral aortic regurgitation was made. A chest x-ray, FBC, serum electrolytes, urea/creatinine, ASAT/ALAT and urinalysis were ordered but not performed due to financial difficulties.

The patient was placed in semi-recumbent position (45^0), oxygen therapy, fluids 1000cc/24h, salt restriction, diuretics (furosemide), antiarrhythmic (amiodarone), factor Xa inhibitor (apixaban).

Days 1 to 5 post-op were marked by progressive resolution of dyspnoea, but persistent irregular tachycardia with systolic murmurs.

Amiodarone was discontinued and replaced with bisoprolol, a selective beta-1 receptor blocker. The surgical and obstetrical course were uneventful.

The patient was transferred to the cardiology unit for further evaluation of the valvulopathy but signed for discharge against medical advice.

LITERATURE REVIEW AND DISCUSSION

PREVALENCE OF VALVULAR DISEASE IN PREGNANCY

Valvular heart disease (VHD) is any cardiovascular disease process (structural or functional) involving one or more of the four valves of the heart. The prevalence and distribution of VHD varies according to where the patient comes from. In developed countries, advances in the medical and surgical management of patients with complex congenital heart disease (CHD) have resulted in an increase in the number of patients reaching adulthood and childbearing age. CHD currently accounts for ≈30% to 50% of all cardiac disease during pregnancy [1]. In

developing countries, rheumatic heart disease (RHD) accounts for ≈90% of all heart disease in women of childbearing age [2], with an estimated global incidence of 282 000 new cases per year [5].

Although accurate statistics are lacking, the estimated incidence of rheumatic fever in sub-Saharan Africa is ≈13 cases per 100 000 per year based on clinical screening [3], while estimates ranging from 21.5 to 30.4 per 1000 have been reported in Cambodia and Mozambique using echocardiographic screening [4].

In the European Registry on Pregnancy and Heart Disease [5], mitral stenosis and/or regurgitation were the most common types of valvular disease (63%), followed by aortic valve disease (23%).

Other causes of valvular disease in younger women include myxomatous mitral valve disease (mitral valve prolapse), previous endocarditis, valvular disease associated with systemic disease (Marfan syndrome, systemic lupus erythematosus, inflammatory vascular disease) and radiation-induced valvular disease [6].

In our case, the aetiology of our patient's valvopathy was post-rheumatic heart disease when she was 6 years old. This is consistent with the literature where rheumatic heart disease is the major cause in developing countries.

Table I : Aetiology of valvular heart disease Boudoulas et al. 2013 [6].

Heritable	Congenital	• Bicuspid aortic valve • Endocardial cushion defects
	Connective tissue	• Marfan's • Ehlers–Danlos
Inflammatory/infectious	Rheumatic fever	
	Syphilis	
	AIDS	
	Kawasaki	
Endocardial disorders	Infective	
	Noninfective	
Myocardial disease	Ischemic	
	Nonischaemic	
	Hypertrophic	
Neoplastic		
Degenerative		
Iatrogenic		
Drugs		
Radiation		
Trauma		
Infiltrative		

CLASSIFICATION

VHD can be classified according to the anatomical heart valve involved, the number of heart valves involved (mono vs polyvalvopathy), the functional abnormality (stenosis vs regurgitation) and the onset (acute vs chronic). Stenosis and insufficiency/regurgitation are the dominant functional and anatomical consequences associated with valvular heart disease [7]. Irrespective of the disease process, valvular changes occur that result in one or a combination of these conditions.

Insufficiency and regurgitation are synonymous terms that describe the inability of the valve to prevent blood from flowing backwards because the valve leaflets do not meet properly [3]. Stenosis is characterised by a narrowing of the valve opening that prevents adequate blood flow.

Stenosis can also lead to insufficiency if thickening of the annulus or leaflets results in inadequate leaflet closure [3].

Table II : Classification of valvular heart disease

Valve involved	Stenotic disease	Insufficiency/regurgitation disease
Aortic valve	Aortic valve stenosis	Aortic insufficiency/regurgitation
Mitral valve	Mitral valve stenosis	Mitral insufficiency/regurgitation
Tricuspid valve	Tricuspid valve stenosis	Tricuspid insufficiency/regurgitation
Pulmonary valve	Pulmonary valve stenosis	Pulmonary insufficiency/regurgitation

In our case, the patient had a mixed valvular lesion with mitral and aortic regurgitation.

HAEMODYNAMIC CHANGES IN PREGNANCY

Pregnancy causes significant haemodynamic changes that progress through pregnancy and further change during labour and delivery and in the postpartum period [6]. During pregnancy, cardiac demands increase due to the placental circulation and hormonal effects, with a 30–50% increase in cardiac output, a 10–20 bpm increase in heart rate and a 30–50% increase in blood volume. Systemic vascular resistance decreases so that blood pressure remains low, despite the increase in cardiac output. Owing to the increase in blood volume with an unchanged red cell mass, there is a fall in haematocrit [7-9] During the third trimester, preload reduction may occur due to compression of the inferior vena cava by the gravid uterus. Most of these haemodynamic changes begin early in the first trimester, peak during the second trimester and reach a plateau phase in the third trimester. During the third trimester, preload reduction may occur due to compression of the inferior vena cava by the gravid uterus.

During labour and delivery, there is an increase in cardiac output, heart rate, blood pressure and systemic vascular resistance, all of which are accentuated with each contraction [7,10,11]. Pain, and anxiety contribute to the increase in heart rate and blood pressures, such that pain control and anxiolytics help reduce the acuity of the haemodynamic changes. Delivery of the placenta increases afterload by removing the low- resistance vascular bed, and rapidly increases preload with venous return of blood to the maternal circulation. Blood loss may result in further decrease in the haematocrit. These changes pose a substantial demand on cardiac function in a patient with valvular disease, sometimes necessitating invasive haemodynamic monitoring and aggressive medical treatment in the peripartum period. In a delivery complicated by excessive

blood loss, infection, arrhythmia or complex obstetric issues, these demands are further amplified.

Table III: Hemodynamic changes in pregnancy, labour and postpartum.

	First Trimester	Second Trimester	Third Trimester	Stage 1 Labor	Stage 2 Labor	Early Postpartum	3–6 months Postpartum
Cardiac output	↑5–10%	↑↑35–45%		↑30%	↑↑50%	↑↑↑60–80% immediately, then rapidly decreases within the first hour	Return to prepregnancy values
Heart rate	↑3–5%	↑10–15%	↑15–20%	During uterine contractions: ↑40–50%		↓5–10% within 24 hours; continues to decrease throughout the first 6 weeks	Return to prepregnancy values
Blood pressure	↓10%	↓5%	↑5%	During uterine contractions: ↑SBP 15–25% ↑DBP 10–15%		↓SBP 5–10% within 48 hours; may increase again between days 3–6 due to fluid shifts	Return to prepregnancy values
Plasma volume	↑	↑↑40–50%		↑	↑↑	↑↑↑500 mL due to autotransfusion	Return to prepregnancy values

Normal pregnancy is accompanied by changes in the coagulation and fibrinolytic systems. These include increases in a number of clotting factors (I, II, VII, VIII, IX, X and XII), a decrease in protein S levels and inhibition of fibrinolysis. As gestation progresses, there is also a significant fall in the activity of activated protein C, an important anticoagulant. While these physiological changes may be important for minimizing intrapartum blood loss, they entail an increased risk of thromboembolism during pregnancy and the post-partum period [4].

EFFECT OF PREGNANCY ON VALVULAR HEART DISEASE

Each of these hemodynamic alterations may have effects that can be beneficial or detrimental depending on the characteristics of the existing valvular abnormality. In general, stenotic valvular lesions carry a higher pregnancy risk than regurgitant lesions [12]. In valvular stenosis, the increased cardiac output associated with the pregnancy increases the transvalvular gradient and, therefore, upstream pressure [13]. In addition, the fall in the peripheral vascular resistance will provoke fluid retention and volume expansion. Further, the increased heart rate may be poorly tolerated, as the left-ventricular filling depends on an adequate diastolic filling time.

Conversely, the effects of mitral or aortic regurgitation are usually ameliorated in early pregnancy by the dominant physiological change, peripheral vasodilatation. The increased plasma volume is offset by the reduction in systemic vascular resistance and consequently, the extent of the regurgitation diminishes. The plasma volume, however, peaks in the middle of the third trimester and that, together with a rise in vascular resistance, may lead to worsening

regurgitation and the onset of symptoms and signs consistent with fluid overload or pulmonary oedema [14].

The most common maternal complications of valvular heart disease during pregnancy are heart failure, arrhythmias, and thromboembolic complications. Postpartum haemorrhage can be a common complication for women on anticoagulation [15].

This pathophysiology is related to our case, in which our patient was asymptomatic until 36 weeks' gestation, when she began to present with complications of pulmonary oedema and arrythmias, which could be related to the peak in plasma volume and increase in peripheral resistance observed in the middle of the third trimester.

Table IV: Hemodynamic effect of pregnancy on VHD [13].

Cardiac lesion	Hemodynamic effect of VHD	Effects of pregnancy
Mitral/aortic stenosis	↓ LV filling ↑ PVR Eventual pulmonary HTN	Fixed CO; tachy- or bradycardia will ↓ LV filling and ↓ CO Left atrial dilation leading to pulmonary congestion Arrhythmias leading to thrombus formation
Mitral valve regurgitation	Component of regurgitation LV hypertrophy Eventual LV failure Eventual pulmonary HTN	Complications occur late in life; generally asymptomatic during pregnancy The ↓ SVR of pregnancy improves forward flow ↑ SVR during labor increases regurgitation
Aortic regurgitation	LV volume overload Left heart failure Pulmonary congestion	The ↓ SVR and ↑ HR of pregnancy reduce regurgitant flow During labor, ↑ intravascular volume, ↑ SVR, and stress of labor can lead to LV dysfunction
Prosthetic valve	Component of regurgitation	Risk of embolization Valvular dysfunction Endocarditis

EFFECT OF VALVULAR HEART DISEASE ON PREGNANCY

This depends on the type and number of valvular lesions involved, severity, ventricular decompensation, effects on pulmonary or systemic circulation, arrhythmias and medications taken.

In mitral and aortic stenosis, the risk of prematurity (20-30%), intrauterine growth retardation (5-20%), stillbirth (1-3%) and low birth weight in up to 25% [13]. Conversely, the risk of fetal complications has been reported to be low with regurgitation lesions, as in our case where we had no adverse fetal outcome [14].

When valvular heart disease is of congenital origin, there is a risk of fetal congenital heart disease. In addition, certain drugs used in the treatment of valvular heart disease have teratogenic effects.

Table V: Stages of progression of VHD

Stage	Definition	Description
A	At risk	Risk factors for developing VHD
B	Progressive	Progressive VHD (mild to moderate severity, asymptomatic)
C	Asymptomatic severe	Asymptomatic patients with severe VHD
C1		Left and right ventricle remain compensated
C2		Decompensation of left or right ventricle
D	Symptomatic severe	Symptoms resulting from VHD

SIGNS AND SYMPTOMS

Many of the normal symptoms of pregnancy are also symptoms of cardiac decompensation. However, while dyspnoea on exertion, orthopnoea, ankle oedema and palpitations are expected, symptoms of angina, resting dyspnoea, paroxysmal nocturnal dyspnoea or a sustained arrhythmia are not normal, even in pregnancy.

The normal physical exam in pregnancy can often mimic disease [2,4]. Increased plasma volume may result in a systolic flow murmur, which can be heard in most normal pregnant patients. This murmur is usually systolic and soft (usually ≤ grade II/VI). A venous hum can also be heard, which is usually related to increased mammary blood flow. In normal pregnant patients, echocardiographic changes in the form of mild dilation of the left ventricle and left atrium are commonly seen. Trivial valvular regurgitation is commonly seen in the mitral, tricuspid and pulmonic valves. This may be related to the physiologic hemodynamic changes of pregnancy. In addition, mild tricuspid and mitral annular dilatation may be present, which can contribute to mild valvular regurgitation [6].

Patients with mitral and aortic regurgitation may present with heart failure symptoms, such as dyspnoea on exertion, orthopnoea and paroxysmal nocturnal dyspnoea, palpitations, pulmonary oedema and angina pectoris [15]. In acute cases patients may experience cyanosis and circulatory shock [1]. We could find similar signs and symptoms which were present in our patient.

DIAGNOSIS AND WORK- UP

Valvular heart disease can present in myriad ways ranging from a decrease in functional capacity to florid pulmonary oedema. Cardiac disease should be considered for any pregnant woman who presents with dyspnoea, chest pain, palpitations, limitation of physical activity, or arrhythmias [16]. History and physical exam establishes symptom severity, comorbidities, valve disease presence and severity, and presence of heart failure.

The standard work- up for patients with known VHD and/or potential cardiac symptoms includes measurement of heart rate and blood pressure, pulse oximetry, electrocardiography and echocardiography. Exercise testing: this should be considered in women with known VHD planning pregnancy or to 80% of the predicted maximum if already pregnant. Magnetic resonance imaging (MRI): if other imaging modalities are not sufficient, MRI may be an alternative, but gadolinium- containing contrast is contraindicated in pregnancy. Invasive hemodynamic monitoring is strictly reserved for symptomatic patients in whom the clinical and non-invasive evaluations are discordant.

Table VI: Evaluation of patients with known or suspected VHD [17]

Reason	Test	Indication
Initial evaluation: All patients with known or suspected valve disease	TTE*	Establishes chamber size and function, valve morphology and severity, and effect on pulmonary and systemic circulation
	ECG	Establishes rhythm, LV function, and presence or absence of hypertrophy
Further diagnostic testing: Information required for equivocal symptom status, discrepancy between examination and echocardiogram, further definition of valve disease, or assessing response of the ventricles and pulmonary circulation to load and to exercise	Chest x-ray	Important for the symptomatic patient; establishes heart size and presence or absence of pulmonary vascular congestion, intrinsic lung disease, and calcification of aorta and pericardium
	TEE	Provides high-quality assessment of mitral and prosthetic valve, including definition of intracardiac masses and possible associated abnormalities (e.g., intracardiac abscess, LA thrombus)
	CMR	Provides assessment of LV volumes and function, valve severity, and aortic disease
	PET CT	Aids in determination of active infection or inflammation
	Stress testing	Gives an objective measure of exercise capacity
	Catheterization	Provides measurement of intracardiac and pulmonary pressures, valve severity, and hemodynamic response to exercise and drugs
Further risk stratification: Information on future risk of the valve disease, which is important for determination of timing of intervention	Biomarkers	Provide indirect assessment of filling pressures and myocardial damage
	TTE strain	Helps assess intrinsic myocardial performance
Preprocedural testing: Testing required before valve intervention	CT coronary angiogram or invasive coronary angiogram	Gives an assessment of coronary anatomy

*TTE is the standard initial diagnostic test in the initial evaluation of patients with known or suspected VHD. CMR indicates cardiac magnetic resonance; CT, computed tomography; ECG, electrocardiogram; HF, heart failure; LV, left ventricular; PET, positron emission tomography; STS, Society of Thoracic Surgeons; TAVI, transcatheter aortic valve implantation; TEE, transoesophageal echocardiography; TTE, transthoracic echocardiography; and VHD, valvular heart disease.

The chronic nature of the polyvalvopathy in our case was reflected by left ventricular hypertrophy on ECG and left atrio-ventricular dilatation on transthoracic echocardiography, which was due to rheumatic heart disease when she was 6 years old. The severe mitral and moderate aortic regurgitation decompensated at 36 weeks' gestation due to a peak in cardiac output and an increase in peripheral vascular resistance in the middle of the third trimester. This led to an increase in left ventricular end-diastolic pressure and left atrial pressure. As a result, pulmonary hypertension, congestion and oedema were seen on transthoracic echocardiography. This explains the patient's signs and symptoms of left heart failure.

MATERNAL RISK STRATIFICATION

There are three well-known models to assist with maternal cardiac risk stratification. The Cardiac Disease in Pregnancy (CARPREG) risk score was derived from a prospective multicentre study of pregnant women with congenital and acquired heart disease. This risk score stratifies women based on four predictors, one of which includes left-sided heart obstruction [18] The Zwangerschap bij Aangeboren HARtAfwijkingen I (ZAHARA) risk score was derived from a study of pregnant woman with CHD. In contrast to the CARPREG score, valvular heart and mechanical prosthesis are used in the risk prediction model [19]. The World Health Organization (WHO) classification divides women with congenital and acquired heart disease into four classes, ranging from low to high risk. Women who fall into WHO Class IV are at the highest risk, and thus pregnancy is contraindicated due to the risk of mortality [20]. A recent prospective validated study compared the ZAHARA score, CARPREG score, and WHO classification in pregnant women with CHD. Although none of these are ideal, the WHO risk assessment model performed the best in estimating cardiovascular risk in pregnant women with CHD [21].

European Society of Cardiology recommend performing a risk assessment in all women with known cardiac diseases of childbearing age before conception using the modified WHO (mWHO) classification of maternal cardiovascular risk in order to plan the appropriate management. VHD in pregnant women is heterogeneous and its management varies from simple surveillance (mWHO Class I) to contraindication or termination of pregnancy (mWHO Class IV) [11].

Our patient did her first cardiac evaluation at 36 weeks of pregnancy when she started manifesting signs and symptoms of left heart failure. Had a systemic ventricular ejection fraction of 46%, systemic ventricular dysfunction NYHA class III and moderate pulmonary hypertension. She was mWHO class IV.

Table VII: CARPREG risk score [11]

Risk Factor	Score and Risk of Cardiac Complications
• Prior cardiac event or arrhythmia • New York Heart Association (NYHA) Class >II or cyanosis • Left heart obstruction • Systemic ventricular dysfunction (ejection fraction <40%)	0: 5% risk 1: 27% risk >2: 75% risk

Table VIII: ZAHARA risk score [11]

Risk Factor and Weight	Points	Score and Risk of Cardiac Complications
		0-0.5: 2.9% risk
History of arrhythmia	1.5	0.51-1.5: 7.5% risk
Cardiac medication prior to pregnancy	1.5	1.51-2.5: 17.5% risk
NYHA Class ≥II	0.75	2.51-3.5: 43.1% risk
Left heart obstruction	2.5	>3.51: 70% risk
Systemic atrioventricular valve regurgitation (moderate or severe)	0.75	
Pulmonic atrioventricular valve regurgitation (moderate or severe)	0.75	
Mechanical valve prosthesis	4.25	
Cyanotic heart disease (corrected or uncorrected)	1.0	

Table IX: mWHO classification for pregnancy [11]

Risk Classification	Cardiac Lesion
I: No detectable increased risk of maternal mortality and no/minimal increase in maternal morbidity	• Uncomplicated, small or mild pulmonary stenosis; ventricular septal defect; patent ductus arteriosus; mitral valve prolapse with no more than trivial mitral regurgitation • Successfully repaired simple lesions (atrial or ventricular septal defect, patent ductus arteriosus, anomalous pulmonary venous drainage) • Isolated ventricular extrasystoles and atrial ectopic beats
II: Small increased risk of maternal mortality or moderate increase in morbidity	Unoperated atrial or ventricular septal defect, repaired tetralogy of Fallot, most arrhythmias
II-III: Depending on patient	Mild ventricular impairment, heart transplantation, hypertrophic cardiomyopathy, native or tissue valvular heart disease not considered WHO I or IV, repaired coarctation, Marfan syndrome without aortic dilatation, bicuspid valve with aorta <45 mm
III: Significantly increased risk of maternal mortality or severe morbidity; expert cardiac and obstetric pre-pregnancy, antenatal, and postnatal care are required	Mechanical valve, systemic right ventricle, Fontan circulation, unrepaired cyanotic heart disease, other complex CHD, Marfan syndrome with aorta 40–45 mm, bicuspid aortic valve with aorta 45–50 mm
IV: Pregnancy is contraindicated	• Pulmonary hypertension/Eisenmenger syndrome, systemic ventricular ejection fraction <30% or systemic ventricular dysfunction with NYHA class III–IV • Severe MS, severe symptomatic AS, Marfan syndrome with aorta >45 mm, • bicuspid aortic valve with aorta >50 mm, native severe coarctation • Prior peripartum cardiomyopathy with any residual impairment of ventricular function

GENERAL MANAGEMENT OF PREGNANT WOMEN WITH VALVULAR DISEASE

Women with valvular disease may not tolerate the haemodynamic changes of pregnancy or labour and delivery, even when asymptomatic before pregnancy. In addition, some patients with only mild valve dysfunction before pregnancy develop acute valvular dysfunction for example, acute mitral regurgitation owing to chordal rupture of acute stenosis of a thrombosed mechanical prosthesis.

The first step in management of women with valvular disease is to establish the level of maternal and fetal risk, based on functional status, severity and type of valvular disease, left ventricular function and pulmonary pressures.

In addition to the medical issues of pregnancy, the inheritability of the maternal condition is addressed. Low- risk patients can be reassured that pregnancy is not contraindicated and that management by the primary physician is appropriate. Women at moderate risk should be evaluated at specialised centres, with management coordinated between the primary physician and specialty centre. Women at high risk of maternal and fetal complications are best cared for at centres with an experienced multidisciplinary team.

PRECONCEPTION COUNSELLING AND EVALUATION

Reproductive age women with valvular heart disease should undergo counselling before conception by a collaborative Pregnancy Heart Team consisting of a maternal fetal medicine (MFM) specialist and cardiologist with experience in caring for pregnant women with heart disease [10]. The goal of preconception counselling is to review and individualize the maternal and fetal risk of pregnancy.

Baseline cardiac function should be assessed with an electrocardiogram and echocardiogram to start. Exercise stress testing can be an important tool to assess exercise capacity, development of arrhythmias and symptomatic response, which may guide risk stratification and treatment before conception. Additional imaging modalities such as cardiac MRI or computed tomography may be used to further assess valvular function, anatomy of structures not well seen by echocardiogram, and associated acropathies. Medications should be reviewed for safety during pregnancy. Our patient did no prior periconceptional evaluation.

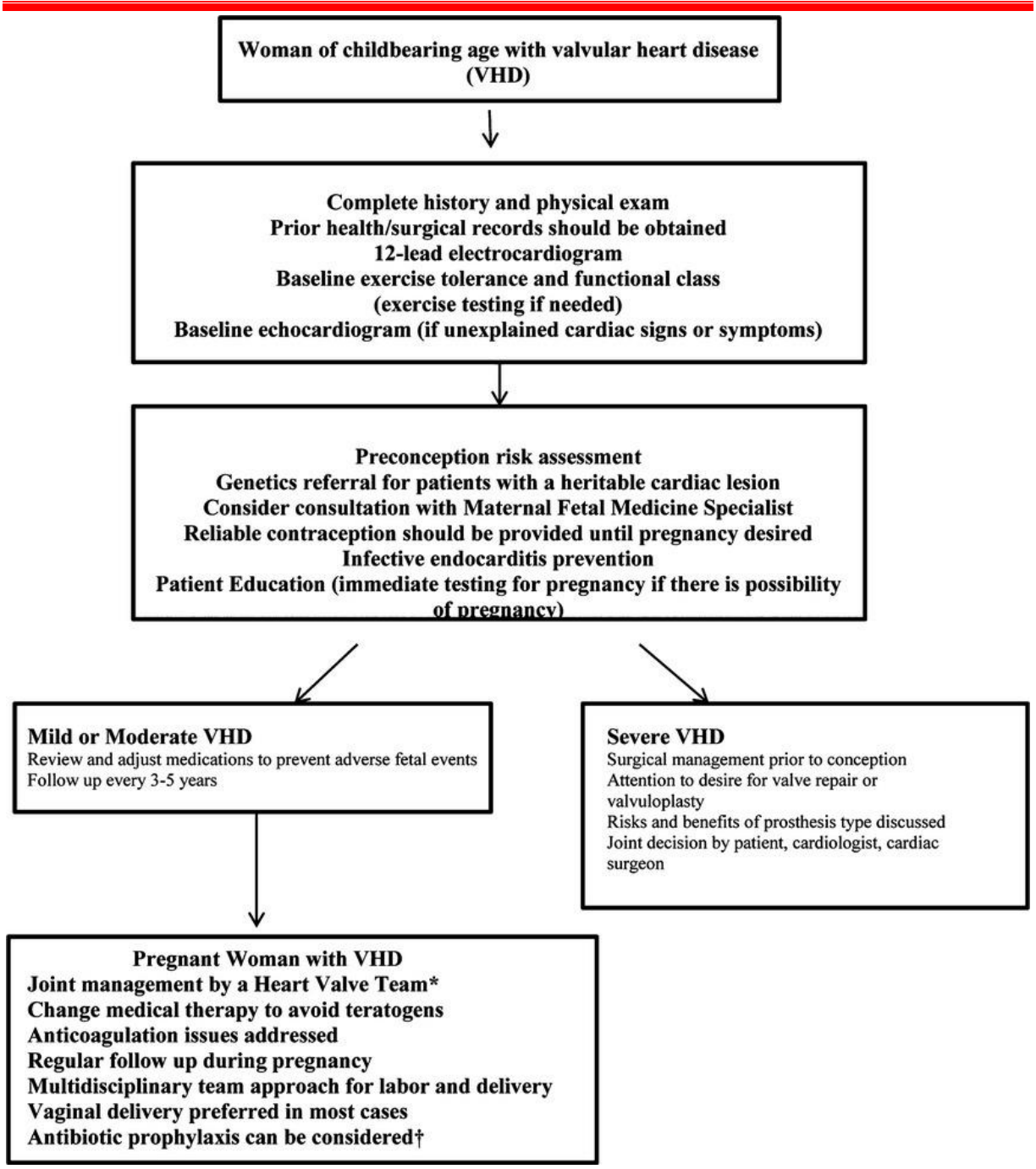

Figure 6: Algorithm of preconception counselling and evaluation [10].

ANTENATAL AND OBSTETRIC CARE

Antenatal care plans should be dependent on the risk stratification [21]. Women in WHO class I have a very low risk and cardiology follow up during pregnancy can be limited to one or two visits. Women in WHO class II are deemed to be low or moderate risk and follow up once during each trimester of pregnancy is recommended. Women in WHO class III are high risk with an increased risk of complications. These women should be followed up at least monthly, then increasing to twice a month during the latter stages of pregnancy. Women in WHO class

IV are extremely high risk. In these women, pregnancy is considered contra-indicated but if they do fall pregnant and decline termination of pregnancy, these women should have close cardiology follow up monthly if not twice monthly.

Key principles in the antepartum management of VHD focus on minimizing cardiac work while optimizing perfusion of the tissues, including the utero placental circulation. Factors that may contribute to cardiac decompensation, such as anaemia, infections, arrhythmias, hypertension and hyperthyroidism should be actively sought so they may be avoided or corrected [22,23]. The patient's functional status should be closely monitored as pregnancy progresses. Any diminution in cardiac function or worsening of maternal functional class should prompt further evaluation and consideration for hospitalization.

Women who develop heart failure symptoms or left ventricular dysfunction can be treated with dietary interventions aimed at lowering sodium intake, diuretics and vasodilators, such as hydralazine or nitrates, with care to avoid hypotension which can lead to placental hypoperfusion. Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers are contraindicated during pregnancy. Patients who are functional class III or IV will need to significantly limit their physical activity and have specified daily rest periods.

The foetal baseline scan, 13-week nuchal translucency scan and 20-week foetal anomaly scan should all be done routinely, with an extra emphasis given to excluding cardiac disease in the foetus. Growth scans should be performed as obstetrically indicated but in addition, those women with severe cardiac disease, cyanotic congenital heart disease or on medications known to cause growth restriction should have serial growth scans to detect foetal growth restriction.

The combined cardiac and antenatal clinic visits are the time when decisions regarding timing and mode of delivery, and the analgesic and anaesthesia options available are discussed and preferences are documented. Cardiac monitoring, antibiotic prophylaxis and thromboprophylaxis will need to be individualised. The entire team, including the patient, should be involved in the decision-making process.

Timing of delivery is controversial and should be individualized based on the patient's cardiac lesion, functional status, and any other maternal or fetal complications of the pregnancy. Generally, induction of labour should be considered at or after 39 weeks. In patients without cardiac disease, scheduling induction of labour at this time reduces the risk of emergency caesarean delivery by 12% and the risk of stillbirth by 50%. The benefits for patients with VHD may be greater still. From a practical perspective, when possible, delivery timing should be optimized to have the adult CHD team available. However, planned delivery prior to 39 weeks

of gestation must weigh potential maternal benefit with the increased risk of fetal lung immaturity.

Our patient had five antenatal contacts, she was recommended specialized follow up of her pregnancy from the initial antenatal contact which was not done until onset of decompensation at around 36 weeks of pregnancy.

USE OF DRUGS

Avoiding all drugs is not always possible in pregnant women with valvular disease, particularly when heart failure, significant arrhythmias or a prosthetic valve is present. Although no drugs are truly safe in pregnant women, treatment may be essential to maintain cardiac stability. Medications that are contraindicated during pregnancy include ACE inhibitors, angiotensin receptor blockers, amiodarone and nitroprusside, so a transition to alternate treatment before pregnancy is desirable. Cardiac drugs that are commonly used during pregnancy include β-blockers, hydralazine, diuretics and digoxin [13,14].

ANTICOAGULATION [18]

Pregnancy is a hypercoagulable state, and pregnant women with mechanical heart valves or cardiac failure are at especially high risk of thromboembolism. The American College of Cardiology and American Heart Association (AHA) recommend that all women with mechanical heart valves undergo therapeutic anticoagulation during pregnancy (Nishimura et al. 2014). However, anticoagulation management is challenging as no therapy is devoid of maternal and fetal risk, therefore an individualized treatment plan should be formulated with the patient using a joint decision- making model.

• **Warfarin:** although warfarin is the most effective therapy in preventing thrombosis associated with mechanical valves, it readily crosses the placenta and has adverse fetal effects throughout pregnancy. If used in the first trimester, it increases the risk of early pregnancy loss or may result in warfarin embryopathy, which includes abnormal cartilage formation and a hypoplastic midface. If used in the second or third trimesters, warfarin increases the risk of pregnancy loss, growth restriction, and abnormalities caused by vascular disruption like cerebral bleeding or limb reduction defects. There are some data suggesting that these complications are less likely if the daily dose is 5 mg or less per day, therefore the AHA recommends continuation of warfarin therapy for these patients. For these patients, the goal should be to maintain an international normalized ratio (INR) of 2.5–3.5. Many women will elect to discontinue warfarin when attempting pregnancy or immediately after conception and receive unfractionated heparin (UFH) or low molecular weight heparin (LMWH) until 12

weeks' gestation. The various forms of heparin do not cross the placenta and are therefore safe for the foetus but are not completely effective in preventing thrombosis. However, reports indicate a 12–30% incidence of thromboembolic complications and a 4–15% incidence of mortality for pregnant women with mechanical valves taking heparin.

• **LMWH:** patients electing for LMWH often require greater than weight-based dosing twice daily to achieve a target anti- Xa level of 0.8–1.2 U/mL measured four hours after administration of the third dose, and a trough level prior to the next dose of 0.6–0.8 U/mL. Subcutaneous UFH is not recommended due to the difficulty of attaining stable therapeutic levels. However, if the patient chooses UFH, it should be given as a continuous infusion in pregnancy. In addition to anticoagulation, all women with mechanical valves should receive antiplatelet therapy in the form of low- dose aspirin (70–100 mg/day). Patients on anticoagulation need a coordinated delivery plan.

Patients on warfarin should be switched to UFH infusion in hospital at least one week prior to delivery to avoid persistent neonatal warfarin effect, which includes increased risk of intracranial haemorrhage during vaginal delivery. The timing of delivery should be individualized. Ideally, patients are switched to therapeutic UFH at 35–36 weeks. As UFH has a short half- life (1.5 hours), its effects can be rapidly reversed with protamine sulphate, and an activated partial thromboplastin time (aPTT) can rapidly confirm that its effects have resolved. Patients should be instructed to withhold UFH at the onset of labour or 8–12 hours prior to a planned induction of labour or caesarean, primarily so they can receive neuraxial anaesthesia. Should urgent delivery occur while the patient is still fully anticoagulated, those on UFH or LMWH should be reversed with protamine sulphate. Of note, the half- life of LMWH is longer than UFH, so repeated dosing or an infusion may be required. Patients on warfarin should receive four- factor prothrombin complex concentrate with a goal INR of ≤1.5 as it is more effective than fresh frozen plasma. The dose of four- factor prothrombin complex is weight and pretreatment INR based. Vitamin K may also be administered orally or intravenously but may take 8–12 hours to achieve efficacy. The dose varies from 1 mg to 10 mg based on the level of INR and severity of bleeding. The effect of vitamin K persists and can make anticoagulation after delivery more of a challenge. Of note, the neonate may also remain anticoagulated for 8–10 days and may also require fresh frozen plasma as well as vitamin K.

Postpartum, therapeutic heparin (either UFH or LMWH) and warfarin should be restarted 4–6 hours after vaginal delivery or 6–12 hours after caesarean delivery, as long as the patient has no significant bleeding. The timing of warfarin initiation is variable, but 48 hours is reasonable.

It is crucial to maintain therapeutic doses of UFH or LMWH until the INR is in the therapeutic range for two successive days, which is usually at least five days

Table X: Recommended approach for anticoagulation prophylaxis

Higher Risk	Lower Risk
First generation PHV (e.g. Starr-Edwards, Bjork Shiley) in the mitral position, atrial fibrillation, history of TE on anticoagulation	Second generation PHV (e.g, St. Jude Medical, Medtronic-Hall) and any mechanical PHV in the aortic postion
Warfarin (INR 2.5–3.5) for 35 weeks, followed by UFH (mid-interval aPTT >2.5) or LMWH (pre-dose anti-Xa ~0.7) + ASA 80–100 mg q.d.	SC UFH (mid-interval aPTT 2.0–3.0) or LMWH (pre-dose anti-Xa ~0.6) for 12 weeks, followed by warfarin (INR 2.5–3.0) for 35 weeks, then SC UFH (mid-interval aPTT 2.0–3.0) or LMWH (pre-dose anti-Xa level ~0.6)
OR	OR
UFH (aPTT 2.5–3.5) or LMWH (pre-dose anti-Xa ~0.7) for 12 weeks, followed by warfarin (INR 2.5–3.5) to 35th week, then UFH (aPTT >2.5) or LMWH (pre-dose anti-Xa~0.7) + ASA 80–100 mg q.d.	SC UFH (mid-interval aPTT 2.0–3.0) or LMWH (pre-dose anti-Xa ~0.6) throughout pregnancy

aPTT – activated partial thromboplastin time; ASA – acetylsalicylic acid; INR – international normalized ratio; LMWH – low molecular weight heparin; PHV – prosthetic heart valve; SC – subcutaneous; TE – thromboembolism; UFH – unfractionated heparin.

In our case, there was a lack of periconceptional evaluation and cardiac evaluation during pregnancy. Therefore, the initial risk of thromboembolism was not assessed. However, the decompensation of her VHD at 36 weeks with signs of left heart failure (stage IV dyspnoea, pulmonary oedema and arrhythmias) placed her at high risk of thromboembolism. Prevention was achieved with LMWH (enoxaparin) and a Factor Xa inhibitor (apixaban).

LABOUR AND DELIVERY [19]

Unless indicated for obstetric indications, a vaginal delivery is preferred for most women with valvular heart disease [1]. Vaginal delivery is associated with less blood loss, more rapid recovery, and less thrombogenic and infectious risk. Patients at elevated risk of complications should discuss a delivery plan in consultation with a multidisciplinary team consisting of a MFM specialist, cardiologist, and obstetric anaesthesiologist. Women with stable cardiac disease can undergo full-term delivery at 39 weeks of gestation [1]. Good pain control with regional anaesthesia during vaginal delivery can minimize the catecholamine release associated with sudden increases in heart rate and stroke volume. Epidural is preferred over spinal anaesthesia due to lower rates of hypotension. Women with moderate to severe left-sided obstructive lesions may benefit from an assisted second stage of labour using forceps or vacuum, which shortens the time to delivery and minimizes the frequency and intensity of maternal effort with Valsalva manoeuvre, which transiently drops cardiac output.

Caesarean delivery results in increased blood loss (on average twice that associated with vaginal delivery), greater hemodynamic fluctuations, as well as increased risks of infection, thromboembolism, and postoperative complications. Generally, elective caesarean delivery confers no maternal benefit and results in earlier delivery and lower birthweight. Caesarean delivery should be considered in women with severe heart failure (New York Heart Association [NYHA] class III-IV), high-risk aortic disease, severe forms of pulmonary hypertension and those who require valve replacement immediately after delivery [6]. For women requiring delivery while fully anticoagulated on warfarin, Caesarean delivery should be considered to minimize the risk of fetal intracranial haemorrhage. Telemetry is recommended during labour and delivery and up to 24 hours after delivery in women at risk for developing arrhythmias. Women with severely stenotic or symptomatic valvular disease may require monitoring in a cardiac care or telemetry unit for at least 24 hours after delivery, with close monitoring of hemodynamic and volume status.

In a woman with valvular disease, a short and pain- free labour and delivery helps to minimise haemodynamic fluctuation. Particularly with severe left- sided valve stenosis, the rapid changes in heart rate, cardiac output, venous return and vascular resistance are difficult to manage, often requiring haemodynamic monitoring, including continuous monitoring of oxygen saturation, ECG, arterial pressure, pulmonary artery and wedge pressures, and cardiac output. Fetal monitoring is another means of assessing the adequacy of cardiac treatment because fetal distress is an indicator of impaired cardiac output.

The immediate postpartum period is critical for the patient with VHD. Blood loss must be minimized and blood pressure maintained, on one hand, and attention must be paid to avoid congestive failure from fluid overload, on the other. Postpartum, high- risk patients may require further monitoring in the cardiac intensive care unit. Lactation should be considered to optimize medications for breastfeeding as some medications like amiodarone can pass into breast milk with hazardous effect on the newborn.

In our case a caesarean delivery was indicated due to a systemic ventricular ejection fraction of 46%, systemic ventricular dysfunction NYHA class IV, moderate pulmonary hypertension and arrythmia. Post op was complicated by pulmonary oedema, which was control by fluid and salt restriction, diuretics (furosemide), antiarrhythmic (amiodarone), and a beta blocker (bisoprolol).

ANAESTHESIA [24]

Conduction anaesthesia is the preferred method of intrapartum pain control for patients with VHD. However, it is important to avoid hypotension when establishing regional anaesthesia. Careful administration of intravenous crystalloid before placement of the catheter, close monitoring of fluid status, and slow administration of the anaesthetic agent help to prevent this complication. Ephedrine is generally the agent of choice for the treatment of hypotension associated with regional anaesthesia because it does not constrict the placental vessels. However, as ephedrine increases the maternal heart rate, phenylephrine may be more appropriate for patients in whom tachycardia and increased myocardial work must be avoided (e.g., those with mitral and aortic stenosis). A single- dose spinal technique is relatively contraindicated in patients with significant cardiac disease because hypotension frequently occurs during establishment of the spinal block. A narcotic epidural is an excellent alternative method and may be particularly effective for patients in whom systemic hypotension must be avoided (e.g., those with pulmonary hypertension, etc.).

Anaesthesia for patients on anticoagulation therapy can be a challenge. Patients should stop anticoagulation at the onset of labour. Ideally, patients are transitioned from warfarin at least one week prior to planned procedures and LMWH should be stopped 36 hours before. Those patients on UFH should hold it 8–12 hours prior to a planned induction of labour or caesarean, primarily so they can receive neuraxial anaesthesia. Regional anaesthesia should only be administered during labour after clotting studies return to normal to avoid spinal or epidural hematomas. Epidural anaesthesia is generally considered safe for patients with a normal aPTT and platelet count. However, anaesthesia guidelines often recommend stopping aspirin 7–10 days prior to neuraxial anaesthesia for patients on joint anticoagulation therapy; both the risks and benefits must be weighed for pregnant patients with mechanical valves. Therefore, it may be reasonable to continue aspirin therapy, but the choice of anaesthesia should be decided in conjunction with an anaesthesiologist.

FOLLOW- UP [23]

Most of the cardiovascular changes of pregnancy will have resolved approximately 4–6 weeks after delivery and so the patient should be reevaluated by a cardiologist. However, the hemodynamic changes of pregnancy may not fully resolve for six months. Therefore, additional follow- up visits at 4–6 months may be warranted to further evaluate and adjust medications. Based on the outcome of the pregnancy and the results of the cardiac re-evaluation, the patient should be counselled regarding the risks for subsequent pregnancy and provided with appropriate contraception if desired.

CARDIAC SURGERY IN PREGNANCY

For patients with an appropriate indication, surgery prior to pregnancy can reduce pregnancy risk. Surgical intervention not only improves the patient's ability to tolerate pregnancy, but also increases fertility and decreases fetal risks for patients with cyanotic heart disease. These women should discuss surgical and transcatheter interventions for VHD, including risks and benefits of mechanical prostheses, bio prostheses, and valve repair. Particular attention should be paid to the risks of anticoagulation during pregnancy with mechanical valves compared to the limited life span of bio prostheses and technical feasibility of valve repair.

If an intervention is required during pregnancy, cardiac surgery should be avoided if possible due to the significant risk of cardiopulmonary bypass for the foetus, related to non-pulsatile blood flow and reduced uteroplacental flow [12,13] Coronary bypass surgery or valvular surgery may be considered during pregnancy only when conservative and medical therapy has failed, and in situations that threaten the mother's life or that are not amenable to percutaneous treatment, which remains the first choice (Class IIb, LOE C) [11]. To date, an urgent or emergency structural intervention during pregnancy, could be required in the following clinical scenarios:

• Patients with high-risk VHD (mWHO Class IV) that was unknown at the time of conception (the incidence of this phenomenon is increasing due to migration flows) and when pregnancy interruption is refused.

• Patients with lower risk VHD, haemodynamic instability and refractory symptoms despite optimal medical therapy.

• Patients with acute onset of severe VHD during pregnancy (i.e. acute severe mitral regurgitation due to chordal rupture).

Valve surgery with cardiopulmonary bypass performed during pregnancy is associated with rates of fetal death up to 30%, especially when surgery is emergent and/or performed at early gestational age [11,12]. If surgery is needed, however, the second trimester is the preferred time frame with use of high flow on cardiopulmonary bypass to provide adequate placental perfusion [13].

CONTRACEPTION

Contraception should be discussed with all women with valvular heart disease, but highly effective contraception should be particularly recommended for women at high risk of pregnancy complications. Estrogen-containing contraception increases the risk of venous and arterial thrombosis and hypertension and should be avoided in women with cardiac disease,

especially those at increased thrombotic risk. In such patients, Barrier methods and long-acting progesterone-only methods are recommended, such as an intrauterine device or subdermal implants [16].

COMPLICATIONS OF VALVULAR HEART DISEASE IN PREGNANCY

The most common maternal complications of valvular heart disease in pregnancy are heart failure, arrhythmias and thromboembolic complications. Postpartum haemorrhage can be a common complication in women on anticoagulation [12].

Fetal complications include miscarriage, prematurity, intrauterine growth retardation, stillbirth and low birth weight [13]. stenotic lesions are associated with these complications as compared with regurgitant lesions.

CONCLUSION

The haemodynamic changes of pregnancy may precipitate cardiac symptoms in previously stable women or worsen symptoms in those with pre-pregnancy symptoms. Regurgitant lesions are generally better tolerated than stenotic lesions. The mWHO model is the most widely used to assess the risk of cardiovascular events during pregnancy, and preconception planning is often required to replace teratogenic pre-pregnancy medications with non-teratogenic ones. In women with severe symptoms despite maximal medical therapy, percutaneous and surgical valvular interventions can be considered, although these should generally be considered as last resort therapies because of the high risk of fetal mortality. Spontaneous vaginal delivery is generally safe in the majority of patients, but planned caesarean section with epidural anaesthesia is appropriate for higher-risk patients. Anticoagulation strategies require advance planning and close monitoring. The process of delivering obstetric care provides an opportunity to ensure continuity of postpartum care, with an emphasis on preventive therapy and contraception.

REFERENCES

1. Sliwa K, Johnson MR, Zilla P, Roos-Hesselink JW. Management of valvular disease in pregnancy: a global perspective. Eur Heart J. 2015 May 7;36(18):1078–89.

2. Anthony J, Osman, A, Sani MU. Valvular heart disease in pregnancy. Cardiovasc J Afr. 2016;27(2):111–8.

3. Fraccaro C, Tence N, Masiero G, Karam N. Management of Valvular Disease During Pregnancy: Evolving Role of Percutaneous Treatment. Interv Cardiol Rev. 2020 Jul 29;15: e10.

4. Nanna M, Stergiopoulos K. Pregnancy Complicated by Valvular Heart Disease: An Update. J Am Heart Assoc. 2014 May 22;3(3): e000712.

5. Bortnick AE, Levine LD. Valvular Heart Disease in Pregnancy. Clin Obstet Gynecol. 2020 Dec;63(4):910–22.

6. Lewey J, Andrade L, Levine LD. Valvular Heart Disease in Pregnancy. Cardiol Clin. 2021 Feb;39(1):151–61.

7. Shapiro H, Alshawabkeh L. Valvular Heart Disease in Pregnancy. Methodist DeBakey Cardiovasc J. 2024 Mar 14;20(2):13.

8. Zwerling B, Hameed AB. Valvular Heart Disease in Pregnancy. In: Queenan JT, Spong CY, Lockwood CJ, editors. Protocols for High- Risk Pregnancies [Internet]. 1st ed. Wiley; 2020 [cited 2024 Jul 25]. p. 141–64. Available from: https://onlinelibrary.wiley.com/doi/10.1002/9781119635307.ch17

9. Sanghavi M, Rutherford JD. Cardiovascular physiology of pregnancy. Circulation. 2014 Sep 16;130(12):1003–8.

10. Bremme KA. Haemostatic changes in pregnancy. Best Pract Res Clin Haematol. 2003 Jun;16(2):153–68.

11. Stout KK, Otto CM. Pregnancy in women with valvular heart disease. Heart Br Card Soc. 2007 May;93(5):552–8.

12. Reimold SC, Rutherford JD. Valvular Heart Disease in Pregnancy. N Engl J Med. 2003

13. ACOG practice bulletin No. 212: pregnancy and heart disease. Obstet Gynecol 2019;133: e320–56. [PubMed: 31022123]

14. Petersen EE, Davis NL, Goodman D, et al. Vital Signs: pregnancy-related deaths, United States, 2011-2015, and strategies for prevention, 13 states, 2013-2017. MMWR Morb Mortal Wkly Rep 2019;68: 423–9. [PubMed: 31071074]

15. Roos-Hesselink J, Baris L, Johnson M, et al. Pregnancy outcomes in women with cardiovascular disease: evolving trends over 10 years in the ESC Registry of Pregnancy and Cardiac disease (ROPAC). Eur Heart J 2019; 40:3848–55. [PubMed: 30907409]

16. Siu SC, Sermer M, Colman JM, et al., Investigators on behalf of the CD in P (CARPREG). Prospective multicenter study of pregnancy outcomes in women with heart disease. Circulation 2001. Available at: 10.1161/hc3001.093437.

17. Nanna M, Stergiopoulos K. Pregnancy complicated by valvular heart disease: an update. J Am Heart Assoc 2014;3: e000712. [PubMed: 24904015]

18. Regitz-Zagrosek V, Roos-Hesselink JW, Bauersachs J, et al. 2018 ESC Guidelines for the management of cardiovascular diseases during pregnancy. Eur Heart J 2018; 39:3165–241. [PubMed: 30165544]

19. Sanghavi M, Rutherford JD. Cardiovascular physiology of pregnancy. Circulation 2014; 130:1003–8. [PubMed: 25223771]

20. Robson SC, Hunter S, Boys RJ, et al. Serial study of factors influencing changes in cardiac output during human pregnancy. Am J Physiol 1989;256:H1060–5. [PubMed: 2705548]

21. Kamel H, Navi BB, Sriram N, et al. Risk of a thrombotic event after the 6-week postpartum period. N Engl J Med 2014; 370:1307–15. [PubMed: 24524551]

22. Mehta LS, Warnes CA, Bradley E, et al. American Heart Association Council on Clinical Cardiology; Council on Arteriosclerosis, Thrombosis and Vascular Biology; Council on Cardiovascular and Stroke Nursing; and Stroke Council. Cardiovascular Considerations in caring for pregnant patients: A Scientific Statement from the American Heart Association. Circulation 2020;141: e884–903. [PubMed: 32362133] Jul 3;349(1):52–9.

23. Silversides CK, Grewal J, Mason J, et al. Pregnancy outcomes in women with heart disease: the CARPREG II study. J Am Coll Cardiol 2018; 71:2419–30. [PubMed: 29793631] ‘

24. Drenthen W, Boersma E, Balci A, et al. Predictors of pregnancy complications in women with congenital heart disease. Eur Heart J 2010;31: 2124–32. [PubMed: 20584777]

25. Steinberg ZL, Dominguez-Islas CP, Otto CM, et al. Maternal and fetal outcomes of anticoagulation in pregnant women with mechanical heart valves. J Am Coll Cardiol 2017; 69:2681–91. [PubMed: 28571631]