

Maternal Cardiopulmonary Failure in Rheumatic Heart Disease: Gaps in Cardio-Obstetric Care in Resource-Limited Settings

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Received Date: 02 Apr 2026

Accepted Date: 21 Apr 2026

Published Date: 27 Apr 2026

Citation:

Temiloluwa D. Owolabi. Maternal Cardiopulmonary Failure in Rheumatic Heart Disease: Gaps in Cardio-Obstetric Care in Resource-Limited Settings. International Journal Clinical and Medical Case Reports, 2026; 7: 1-7

1. Abstract

Rheumatic heart disease (RHD) continues to be a major cause of maternal death in low- and middle-income countries (LMICs), where access to preconception counseling and coordinated cardio-obstetric care is often limited. We describe the case of a 35-year-old woman with a known history of RHD who stopped cardiac follow-up after becoming pregnant. She presented in her second trimester with worsening breathlessness and was found to have advanced valvular disease and pulmonary hypertension. Despite multidisciplinary support and medical management, her condition deteriorated. She experienced intrauterine fetal demise and later died from progressive cardiopulmonary failure. This case highlights the critical role of early risk assessment, structured care, and system-wide support in improving outcomes for high-risk pregnancies in resource-limited settings.

2. Keywords: Rheumatic heart disease, Pregnancy, Low- and Middle-Income Countries, Cardio-Obstetric, Preconception counseling

3. Abbreviations: RHD - Rheumatic Heart Disease; LMICs - Low- and middle-income countries Bpm - Beats per minute; VDRL - Venereal Disease Research Laboratory Hb - Hemoglobin; Hct - Hematocrit; RBC - Red blood cells WBC - White blood cells; CT - Computerized Tomography; IUGR - Intrauterine Growth Restriction PO - per os; CPAP - Continuous Positive Airway Pressure SpO₂ - Peripheral oxygen saturation; HIV - Human Immuno Deficiency

Virus HCV - Hepatitis C virus; HbsAg - Hepatitis B surface antigen BP - Blood Pressure; HR - Heart Rate; AST - Aspartate aminotransferase ALT - Alanine aminotransferase LDH - Lactate dehydrogenase; CARPREG II - Cardiac Disease in Pregnancy Study ZAHARA - Zwangerschap bij Aangeboren HARTafwijking WHO - World Health Organization; ICU - Intensive Care Unit

4. Introduction

Rheumatic heart disease (RHD) has become rare in high-income countries but remains a common and serious condition in many low- and middle-income countries (LMICs), where access to healthcare and cardiac specialists is limited. In these settings, RHD is one of the leading causes of maternal and fetal complications. Many women with pre-existing RHD only seek care when they become pregnant or when complications arise, which increases the risks for both mother and baby [1]. Pregnancy in women with RHD can lead to serious problems like heart failure, arrhythmias, pulmonary hypertension, and even maternal death [2]. On the fetal side, outcomes like preterm birth, low birth weight, and fetal death are not uncommon [2,3].

Even though we know that early detection, proper risk assessment, preconception counseling, and multidisciplinary care can make a big difference, these approaches are still not widely used in LMICs [1,2,4]. Specialized care isn't always available, and many women miss the chance to receive proper follow-up or planning before pregnancy. As a result, many return to care only when complications start to appear. There's also a lack of clear guidelines or protocols for risk stratification, antenatal surveillance, and delivery planning for high-risk pregnancies in resource-limited settings [5].

In reviewing this case, we also look at the current literature around preconception counseling, multidisciplinary team-based management, and the challenges specific to LMICs. Although the disease burden is highest in these regions, most of the available research and guidelines come from high-income countries, which often doesn't reflect the real-world realities in low-resource settings. Available research focuses more on newly diagnosed cardiac cases which presents a critical gap in continuity of care in high-risk pregnancies. There is also not much data on how effective multidisciplinary cardio-obstetric teams are in these areas, or how to implement such models of care in a sustainable way. All of this shows the need for more practical, locally relevant strategies to reduce the risks that women with RHD face during pregnancy.

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Hence, we report the case of a pregnant woman with a pre-existing RHD who stopped attending cardiac follow-up after becoming pregnant, thereby missing the opportunity for structured cardio-obstetric care and counseling. This case highlights how the lack of preconception planning and integrated cardio-obstetric care influenced the clinical course and complications in pregnant women with pre-existing rheumatic heart disease in low- and middle-income countries (LMICs).

5. Case Presentation

A 35-year-old Dominican woman, gravida 4 para 2 (G4P2A1), with a known history of childhood rheumatic fever, presented at 24.6 weeks of gestation with signs of decompensated heart failure. She was initially evaluated at her prenatal clinic on July 11, 2024, where she was unable to lie supine due to severe dyspnea. Due to progressive respiratory distress, she was referred to cardiology, then immediately sent to the emergency obstetric unit at a regional hospital in Dominican Republic. She had no known toxic habits or prior surgical history.

Her past medical history was significant for rheumatic fever in childhood, with a known diagnosis of severe mitral stenosis, left atrial dilatation, pulmonary hypertension and left ventricular diastolic dysfunction. She had been on bisoprolol 5 mg daily for four years and was being followed at a specialist hospital in preparation for cardiac surgery, which she discontinued upon confirmation of pregnancy. No preconception counseling or risk assessment had been performed.

On arrival at the emergency unit, the patient was in acute respiratory distress. Vital signs showed tachycardia (130 bpm), tachypnea (35/min), hypotension (110/70 mmHg), and oxygen saturation of 93% on room air. Physical examination revealed jugular venous distension, a hyperdynamic precordium with costal and subcostal retractions, and bilateral basal crackles on lung auscultation. Cardiac auscultation detected irregular heart sounds and a loud diastolic murmur. Abdominal examination showed generalized edema with a uterine height of 27 cm; Leopold's maneuvers were inconclusive due to distension and tenderness, and 4+ peripheral edema extended to the abdominal wall. These findings-pulmonary congestion, jugular venous distension, murmur, and marked volume overload-were consistent with acute cardiopulmonary decompensation in the context of known rheumatic valve disease. Obstetric ultrasound on admission dated the pregnancy to 23 weeks and revealed moderate polyhydramnios.

Initial laboratory evaluation revealed normocytic anemia (Hb 10.9 g/dL; reference 12-16 g/dL), leukocytosis (WBC $10.3 \times 10^9/L$; reference $4-10 \times 10^9/L$), hypoalbuminemia (2.9 g/dL; reference 3.5-4.8 g/dL), and proteinuria with leukocyturia, as documented in Table 2 and Table 3. Serologic test-ing with Venereal Disease Research

Laboratory (VDRL) tests performed in May and July confirmed syphilis as seen in Table 1. An electrocardiogram performed on July 12, 2024 confirmed inactive rheumatic heart disease with a double mitral lesion, severe mitral stenosis and mild insufficiency. Electrocardiography findings are shown in Figure 2. Additional findings included moderate aortic regurgitation, septal shift, severe tricuspid regurgitation, pulmonary hypertension with estimated pulmonary artery pressure of 65.3 mmHg (severe; normal <35 mmHg) and reduced right ventricular ejection fraction of 31% (moderately reduced; normal $>45\%$).

According to patient records, a CT chest scan demonstrated pulmonary congestion with interstitial edema and patchy alveolar opacities, raising concern for hospital-acquired pneumonia, which prompted antibiotic escalation. Between July 13 and 25, she exhibited progressive leukocytosis (peaking at $38.3 \times 10^9/L$; reference $4-10 \times 10^9/L$), rising creatinine (2.5 mg/dL; reference 0.72-1.16 mg/dL), and elevated LDH (769 U/L; reference 230-460 U/L), traced in the hematology panel and chemistry charts (Table 2 and Table 3, respectively), consistent with multiorgan stress. Arterial blood gases revealed hypoxemia, respiratory acidosis, and metabolic derangement. On July 21, intrauterine growth restriction (IUGR) was diagnosed, with an estimated fetal weight of 613 g (below the 3rd percentile).

The patient was started on oxygen therapy for respiratory distress. She received intravenous furosemide (20 mg every 6 hours) for volume overload and bisoprolol (5 mg PO daily) to control heart rate.

Supportive therapies included omeprazole, ceftriaxone for suspected infection, enoxaparin for thromboprophylaxis, IV albumin, and later, broad-spectrum antibiotics and corticosteroids. Given her instability, a multidisciplinary care plan was initiated with consultations from cardiology, pulmonology, internal medicine, perinatology, nephrology, and infectious disease teams. A Foley catheter was inserted and fluid balance, fetal viability, renal function, and respiratory status were closely monitored. On July 15, a drop in fetal heart rate prompted intrauterine resuscitation through lateral positioning. Fluids were withheld due to maternal cardiac risk. Fetal heart rate returned to baseline after 25 minutes. On July 19, a blood transfusion was indicated following hematological evaluation for worsening anemia and hemodynamic changes. As respiratory and infectious parameters worsened, further imaging was ordered and her antibiotic regimen was escalated.

On July 22, 2024, at approximately 2:15 a.m., the patient experienced spontaneous rupture of membranes and delivered a nonviable fetus weighing 490 grams. On the evening of July 25, 2024, she developed acute pulmonary edema with hemoptysis, severe hypoxia (SpO_2 44%), hypotension (BP 80/60 mmHg), and bradycardia (HR 61 bpm). Continuous Positive Airway Pressure (CPAP) support was initiated, and vasopressors (dopamine and

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norepinephrine) were started. She was intubated due to respiratory failure, during which over 400 cc of bloody secretions were aspirated. Despite initial return of spontaneous circulation after the first cardiac arrest, the patient suffered a second arrest shortly after. Extensive resuscitative efforts, including multiple doses of epinephrine and CPR, were unsuccessful. She was pronounced dead at 8:35 p.m.; marking the terminal event of a progressive clinical decline summarized in Figure 1.

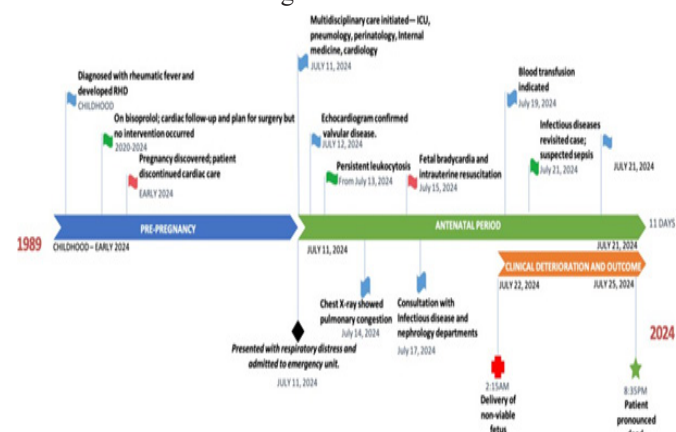


Figure 1: Clinical Progression Roadmap

Timeline of the patient's clinical course highlighting a childhood diagnosis of rheumatic heart disease (RHD), chronic medication use, key investigations and interventions throughout care, multi-disciplinary consultations, fetal diagnosis of intrauterine growth restriction (IUGR), subsequent fetal and maternal death.

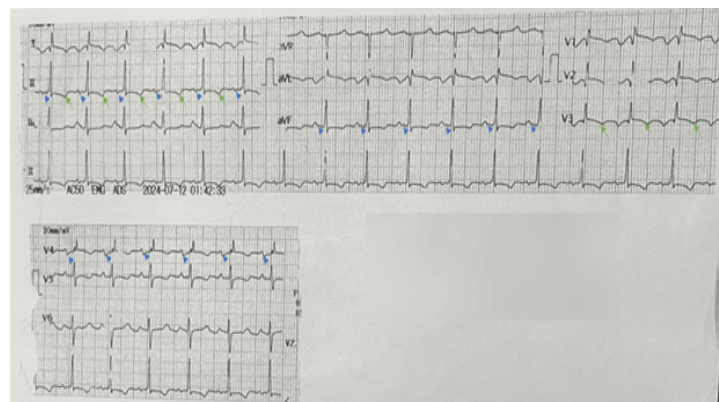


Figure 2: Electrocardiogram

Electrocardiogram done on the 12th of July showing biphasic P waves in leads DII, aVF, and V4 (blue arrowheads) and ST-segment inversion in representative leads DII and V3 (green arrows), also present in DIII, aVF, and V2–V4. These findings are consistent with biatrial enlargement and re-polarization changes likely related to rheumatic mitral valve disease.

DATE	28.05.2024	16.07.2024
HIV	NEGATIVE	NEGATIVE
HCV	-	NEGATIVE
Hbs Ag	-	NEGATIVE

VDRL	REACTIVE*	REACTIVE*
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Table 1: Serologic Screening for Infectious Diseases.

The patient tested negative for HIV, HCV, and Hepatitis B surface antigen (HbsAg). VDRL test was reactive (marked by an asterisk *) in both columns. In the absence of clinical symptoms, these results were interpreted as latent syphilis.

DATE	" R B C s (Reference range= 4.2 - 5.4 x 10 ¹² /L)"	Hemoglobin (Reference range= 12–16 g/dL)	Hematocrit (Reference range= 36- 46%)	Platelets (Reference range = 150 - 450 x10 ⁹ /L)	Leukocytes/ W B C (Reference range= 4-10 xx10 ⁹ /L)
11-07-2024	3.22*	10.9*	31.8*	213	10.3*
12-07-2024	3.31*	12.1	32.9*	233	10.3*
13-07-2024	3.49*	11.4*	34.2*	181	22.0*
13-07-2024	3.46*	10.4*	32.5*	225	23.8*
14-07-2024	3.37*	10.5*	33.0*	247	20.9*
15-07-2024	3.31*	10.5*	32.7*	179	23.9*
16-07-2024	2.94*	9.6*	30.9*	181	18.4*
17-07-2024	3.17*	10.1*	31.0*	148*	25.5*
18-07-2024	3.17*	10.2*	32.5*	138*	38.3*
19-07-2024	2.75*	8.9*	28.3*	117*	23.3*
20-07-2024	2.49*	8.0*	25.8*	105*	17.6*
21-07-2024	2.70*	8.6*	26.9*	96*	16.3*
22-07-2024	3.22*	12	33.9*	147*	22.9*
23-07-2024	2.89*	9.6*	29.8*	112*	24.3*
24-07-2024	3.18*	10.3*	33.5*	117*	25.7*
25-07-2024	2.90*	9.3*	29.6*	85*	21.0*

Table 2: Trend of Hematologic Parameters During Admission.

Values of hemoglobin (Hb), hematocrit (Hct), red blood cells (RBC), leukocytes or white blood cells (WBC), and platelets. Abnormal values are marked with an asterisk (*).

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Date	25.07	24.07	23.07	22.07	21.07	20.07	19.07	18.07	17.07	16	15	14	Value of reference
	0.2024	0.2024	0.2024	0.2024	0.2024	0.2024	0.2024	0.2024	0.2024	7.2	7.2	7.2	
										24	24	24	
Albumin	3.8	3.9	3.3	3.5	3.3	3.3	3.2	3.4	3.7	3.3	3.5	3.1	3.5 -
	g/dL	g/dL	g/dL	g/dL	g/dL	g/dL	g/dL	g/dL	g/dL	g/dL	g/dL	g/dL	4.8
			*		*	*	*	*		*		*	g/dL
Direct bilirubin	0.6	0.4	1.2	1	0.5	0.4	0.4	0.7	0.5	0.3	0.3	0.3	0 - 0.3
	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/ dL	mg/ dL	mg/ dL	mg/dL
Indirect bilirubin	1.1	1.4	1.1	1.6	0.5	0.7	0.8	1.6	1.3	0.7	0.4	0.6	0 – 1
	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/d L	mg/d L	mg/d L	mg/d L*	mg/d L*	mg/ dL	mg/ dL	mg/ dL	mg/dL
Total bilirubin	1.7	1.8	2.3	2.6	1	1.2	1.2	2.4	1.9	0.9	0.7	0.9	0 – 1
	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/d L	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/ dl	mg/ dL	mg/ dL	mg/dL
Creatinine	1.94	2.09	2.5	1.78	1.95	2.06	2.11	1.43	0.6	0.84	0.88	0.74	0.72 –
	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/ dL	mg/ dL	mg/ dL	1.16
													mg/dL
Glucose	133	120	218	63	112	81	94	95	63	65	88	90	70 –
	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/d L	mg/d L	mg/d L	mg/d L*	mg/ dL*	mg/ dL	mg/ dL	110
													mg/dL
AST	27	26	23	29	17	18	23	30	35	27	22	20	0 – 38
	U/L	U/L	U/L	U/L	U/L	U/L	U/L	U/L	U/L	U/L	U/L	U/L	U/L
ALT	20	21	20	19	17	18	23	28	26	23	28	26	0 – 41
	U/L	U/L	U/L	U/L	U/L	U/L	U/L	U/L	U/L	U/L	U/L	U/L	U/L
LDH	610	769	579	731	536	494	477	581	718	548	503	499	230 –
	U/L*	U/L*	U/L*	U/L*	U/L*	U/L*	U/L*	U/L*	U/L*	U/L	U/L	U/L	460
										*	*	*	U/L
Total protein	5.7	5.8	5.2	5.7	5.3	5.1	4.6	4.8	5.8	5.5	5.9	5.5	6.1 –
	g/dL	g/dL	g/dL	g/dL	g/dL	g/dL	g/dL	g/dL	g/dL	g/dL	g/dL	g/dL	7.9g/dL
	*	*	*	*	*	*	*	*	*	*	*	*	
Urea	91	105	122	88	62	63	59	39	34	39	34	27	10 – 50
	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/d L	mg/d L	mg/ dL	mg/ dL	mg/ dL	mg/dL
Uric acid	10.7	11	11.8	10.3	9	8.5	7.9	7.2	6.2	6.8	6.7	5.7	2.5 – 6
	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/d L*	mg/ dL*	mg/ dL*	mg/ dL	mg/dL
Amylase	-	-	-	-	-	161	-	-	-	-	-	-	0 – 125
						U/L*							U/L
Lipase	-	-	-	-	-	82	-	-	-	-	-	-	13 -60
						U/L*							U/L

Table 3: Trend of Blood Chemistry Parameters.

Serial blood chemistry results obtained between July 14 and July 25, 2024; showing albumin, bili-rubin (direct, indirect, and total), creatinine, glucose, liver enzymes [AST: aspartate aminotransfer-ase, ALT: alanine aminotransferase], lactate dehydrogenase (LDH), total protein, urea, uric acid, amylase, and lipase levels. Abnormal values (both above and below the reference range) are marked with an asterisk (*).

6. Discussion

This case underscores the urgent need for systemic interventions to reduce maternal mortality related to pre-existing cardiac disease in pregnancy, particularly in low- and middle-income countries

(LMICs) where rheumatic heart disease (RHD) remains prevalent. The following recommendations are proposed to address the gaps identified in this patient's care and improve outcomes for women with similar risk profiles:

6.1. Strengthen Preconception Care And Education

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Preconception care includes any interventions and programs aimed at optimizing health before pregnancy to improve maternal and child health outcomes. It addresses risks associated with bio-medical, behavioral, and social factors, including modifiable elements such as nutrition, physical activity, substance use, and the management of existing conditions like infectious and chronic diseases [6].

Women with known cardiac diseases should receive targeted reproductive planning and individualized management plans for pregnancy, labor and delivery [7]. This includes accurate maternal and fetal risk stratification, optimizing cardiac lesions, discussing possible pre-pregnancy interventions and reviewing the safety of medications in pregnancy [1].

6.2. Implement Standardized Risk Stratification

Pregnancy is associated with profound hemodynamic changes that can significantly increase the risk of complications in women with pre-existing or newly diagnosed cardiac disease [7]. To support clinical decision-making and pregnancy planning, several risk stratification tools are available, including CARPREG II (Cardiac Disease in Pregnancy Study), ZAHARA (Zwangerschap bij Aangeboren HARTafwijking), and the modified World Health Organization (WHO) classification system [3,7].

Although no tool is perfect [5], they provide a solid foundation for estimating risk and can be further refined using lesion-specific and patient-centered information. These tools are especially useful in preconception counseling, determining the frequency of antenatal monitoring, guiding delivery planning and postpartum care planning [7].

For women at excessively high risk of severe morbidity or mortality, alternative reproductive options such as adoption or the use of a gestational carrier should be considered [7]. It is also important to discuss the emotional and ethical implications of continuing a pregnancy in the event that cardiac decompensation occurs early or mid-pregnancy, when termination may be the only medically viable option. Although most women with cardiac disease become symptomatic later in pregnancy, these sensitive conversations should ideally take place before conception, with full awareness of local laws and access to medically indicated interventions [7].

6.3. Establish And Adapt Multidisciplinary Cardio-Obstetric Teams

Establishing and adapting multidisciplinary cardio-obstetric teams is crucial for optimizing the care of pregnant women with cardiac disease, particularly in the context of rheumatic heart disease and other complex cardiac conditions. These teams should be composed of maternal-fetal medicine specialists, cardiologists with expertise in pregnancy, cardiac anesthesiologists, neonatologists, and specialized nursing coordinators, working collaboratively to

manage high-risk pregnancies from preconception counseling through postpartum care [8,9]. Standardizing care pathways within these teams enables proactive management of severe cardiac lesions by facilitating timely surveillance, medication safety reviews, and informed decisions regarding the timing and mode of delivery.

Importantly, the multidisciplinary team model must be tailored to local resources and realities. In resource-limited settings, this may involve leveraging regional referral networks, virtual consultation platforms, or targeted training for available healthcare personnel to recognize early signs of decompensation and escalate care promptly [10,11]. Investment in these team-based structures has been associated with improved maternal and fetal outcomes, reduced delays in care, and more efficient resource utilization across diverse clinical environments [9-11]. Adapting the cardio-obstetric team approach to fit local contexts ensures that even in settings with limited resources, high-risk women receive coordinated, evidence-based care throughout the continuum of pregnancy.

6.4. Develop Referral And Escalation Protocols

Timely identification and referral of high-risk patients remain key components of effective maternal care [4]. Structured escalation pathways should be implemented to guide frontline providers in transferring patients to higher levels of care when specific warning signs are detected. This includes protocols for red flags such as exertional syncope, persistent tachycardia, new or worsening dyspnea, orthopnea, or signs of heart failure during pregnancy.

Clear, evidence-based thresholds for ICU admission, advanced cardiac imaging, or interventions (e.g., balloon valvotomy) should be documented and disseminated across maternity units and emergency departments [12]. Algorithms tailored for primary, secondary, and tertiary care settings can bridge the gap between resource-rich and resource-limited environments, improving triage and coordination.

6.5. Strengthen Health Systems And Policy Support

Improving outcomes for pregnant women with cardiac disease requires comprehensive health system reforms substantiated by targeted policy initiatives. Central to this is the allocation of dedicated funding to ensure the availability of accessible diagnostics and essential medications at all levels of care [13,14]. Governments and health institutions should invest in specialized training programs for healthcare providers, focusing on the risk assessment and management of maternal cardiac disease. Such efforts will empower frontline providers to recognize and respond to cardiac complications early, reducing delays in care [13,15].

Public health campaigns are equally important to raise awareness

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among women of childbearing age, especially those with known heart disease, about the risks associated with pregnancy and the importance of preconception counseling and regular follow-ups [14,15]. Finally, integrating cardi-ovascular specialists into national maternal mortality review committees can help identify system-level gaps and inform the design of context-specific solutions tailored to the regional burden of disease [14,16]. These coordinated policy and health system actions are essential to address the unique challenges faced by pregnant women with cardiac disease, particularly in low- and middle-income countries where rheumatic heart disease remains a significant contributor to maternal morbidity and mortality [16].

Evidence from multiple studies in low- and middle-income countries (LMICs) has shown that early preconception counseling, structured risk stratification using tools like the modified WHO classification, and the implementation of multidisciplinary cardio-obstetric care significantly reduce maternal morbidity and mortality in women with rheumatic heart disease (RHD) [7,17]. Interventions such as early echocardiographic assessment, optimized medical therapy, and timely surgical procedures like balloon valvotomy have been associated with improved maternal and fetal outcomes. In contrast, the patient in our case despite having a known diagnosis of RHD since childhood did not receive preconception counseling or early specialist referral. She presented late in pregnancy with advanced cardiopulmonary decompensation and multiorgan dysfunction, resulting in spontaneous delivery of a nonviable fetus and maternal death. This outcome underscores the urgent need to implement and scale proven interventions, particularly in resource-limited settings where structural and systemic barriers persist.

7. Conclusion

In conclusion, delayed intervention and fragmented cardio-obstetric care can lead to preventable maternal and perinatal mortality in high-risk cardiac pregnancies. While evidence-based strategies such as preconception counseling, structured risk stratification, early specialist referral, and coordinated cardio-obstetric management have demonstrated clear benefits in improving outcomes, their implementation in low- and middle-income countries (LMICs) remains inconsistent and often inaccessible. The fatal outcome in this case results from systemic gaps in reproductive and cardiovascular care. There is an urgent need to adapt and scale these interventions within low- and middle-income countries (LMICs) through health system strengthening, community-based screening, policy development, and global partnerships. Ensuring that high-risk women receive timely, appropriate, and coordinated care is not only feasible but essential to reducing maternal deaths from cardiac disease.

8. Acknowledgements

We acknowledge the healthcare providers involved in the patient's

care in the Dominican Republic for providing access to medical records that formed the basis of this report. We extend our sincere gratitude to Dr. Sorivel Sosa for her supervision, guidance, and clinical insight throughout the preparation of this manuscript. No radiologic chest images (X-ray or CT) are included because the case details were abstracted from patient records, and original radiology files were unavailable for publication.

Echocardiographic images were available and included. We also acknowledge the use of "Elicit" for literature organization. The authors maintained full control of the manuscript content and approved the final version.

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