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Decompensated Rheumatic Mitral Stenosis in the Context of Pregnancy and Atrial Fibrillation: A Case Report

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Abstract

We report a case of a 36-year-old Indian origin woman who presented with heart failure (HF) and atrial fibrillation (AF). She was found to be pregnant on admission, which she was unaware of, and further biochemical testing confirmed that she had a miscarriage. Transthoracic echocardiography (TTE) revealed severe rheumatic mitral stenosis (MS), and transoesophageal echocardiography (TOE) demonstrated a left atrial appendage (LAA) thrombus. This case highlights the importance of echocardiography in diagnosing rheumatic heart disease (RHD) and in identifying complications. Furthermore, it demonstrates the challenges of managing decompensated MS in the context of pregnancy and AF.

Categories: Obstetrics/Gynecology, Cardiology, Internal Medicine
Keywords: atrial fibrillation, case report, echocardiography, mitral stenosis, pregnancy, rheumatic valvular heart disease

Introduction

Rheumatic valvular heart disease remains the leading cause of acquired mitral stenosis (MS) even in the Western world, aided by global migration [1]. Severe MS in pregnancy carries significant risk of complications, which is further exacerbated by atrial fibrillation (AF) and can be complex to manage [2]. There is a 50% increase in transvalvular gradient during pregnancy with MS, especially from the first to second trimester, which increases the risk of maternal and foetal complications such as maternal death, intrauterine growth retardation, prematurity and foetal loss [2]. AF further intensifies the hemodynamic burden on the heart and is the most common arrhythmia associated with rheumatic heart disease (RHD) in pregnancy [2]. Transthoracic echocardiography (TTE) is a valuable tool in diagnosing rheumatic MS in the absence of a history of rheumatic fever [3,4]. Transoesophageal echocardiography (TOE) is vital in identifying complications of valvular heart disease, such as a left atrial appendage (LAA) thrombus prior to planned valvular intervention [5]. Our patient, unfortunately, had a miscarriage, which could be secondary to her decompensated rheumatic MS, which highlights the increased morbidity associated with this condition [2]; other differentials such as spontaneous miscarriage and drug side effects were also considered equally possible.

Case Presentation

A 36-year-old Indian origin mother of two presented to the hospital with non-specific abdominal pain and palpitations. A pregnancy test (beta human chorionic gonadotrophin (beta hCG) and ventilation/perfusion scan were both negative, but her electrocardiogram (ECG) showed AF with a fast ventricular response. She was clinically well and discharged on apixaban and bisoprolol with a plan for an outpatient TTE and possible direct current cardioversion.

She had a background of iron deficiency anaemia and bilateral breast fibroadenomas. She had two healthy children, aged three years and 18 months. Her previous pregnancies were uneventful via spontaneous vaginal delivery, and there was no history of previous miscarriage. She denied any childhood infections.

She re-presented to the ambulatory clinic, 10 weeks later, with a four-day history of worsening breathlessness, peripheral oedema, and vaginal bleeding. She had been visiting her family in India for eight weeks prior to presentation, and during this time, had been taking her bisoprolol regularly but was only intermittently compliant with her apixaban, initiated during her previous presentation. Outpatient TTE had been performed; however, she re-presented to the hospital prior to her planned clinic review.

Physical examination revealed an irregularly irregular pulse and a low-pitched, mid-diastolic murmur. The ECG done on this presentation showed AF (Figure *1*), and the chest radiograph revealed mild cardiomegaly and pulmonary oedema. Blood work confirmed pregnancy on this presentation with a positive serum beta hCG (1900 IU/L), which she was unaware of, and had a raised N-terminal pro B-type natriuretic peptide (NT-

pro BNP) at 4375 pg/ml. A summary of the laboratory results with their interpretation is available in Table *1*. Outpatient TTE was reviewed, which showed severe MS (mitral valve area (MVA) 0.7 cm^2, mean pressure gradient (PG) 11 mmHg) with a hockey-stick appearance of the anterior leaflet (Figure *2*, Video *1*), restricted movement of the posterior leaflet, and tenting consistent with rheumatic valve disease. There was mild left ventricular systolic dysfunction, mild mitral regurgitation, dilated left atrium, and a mild pericardial effusion.

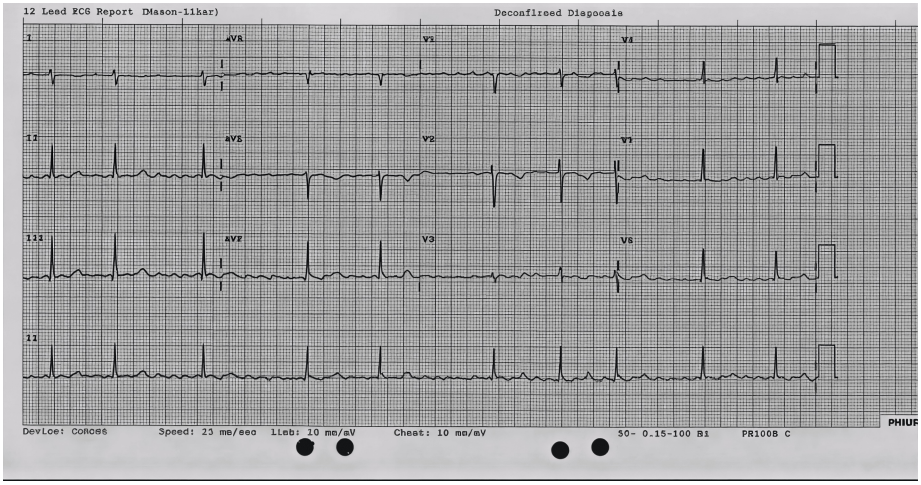


FIGURE 1: Electrocardiogram demonstrating atrial fibrillation at a rate of 60 beats per minute.

Test	Result	Reference range (units)	Interpretation
N-terminal pro-B-type natriuretic peptide	4375 pg/ml	<400 pg/ml	Raised, consistent with decompensated heart failure
Serum beta–human chorionic gonadotrophin (initial presentation)	<5 IU/L	<5 IU/L if not pregnant	Negative for pregnancy
Serum beta-human chorionic gonadotrophin (subsequent presentation 10 weeks later)	1900 IU/L	<5 IU/L if not pregnant	Consistent with early pregnancy
Serum beta-human chorionic gonadotrophin (48 hours after the above test)	1000 IU/L	<5 IU/L if not pregnant; no single reference range applies in pregnancy	Inappropriate fall over 48 hours, consistent with miscarriage
C-reactive protein	1.3 mg/L	0–5 mg/L	Normal
White cell count	4.50 $\times 10^9$/L	3.7–11.0 $\times 10^9$/L	Normal
Neutrophils	2.34 $\times 10^9$/L	1.7–7.5 $\times 10^9$/L	Normal
Lymphocytes	1.52 $\times 10^9$/L	0.9–4.0 $\times 10^9$/L	Normal
Monocytes	0.45 $\times 10^9$/L	0.2–1.0 $\times 10^9$/L	Normal
Eosinophils	0.15 $\times 10^9$/L	0.0–0.5 $\times 10^9$/L	Normal
Basophils	0.04 $\times 10^9$/L	0.0–0.1 $\times 10^9$/L	Normal
Haemoglobin	142 g/L	120–165 g/L	Normal
Red blood cell count	4.53 $\times 10^{12}$/L	3.80–5.00 $\times 10^{12}$/L	Normal
Haematocrit	0.429 L/L	0.360–0.460 L/L	Normal
Mean corpuscular volume	94.7 fL	83.0–101.0 fL	Normal
Mean corpuscular haemoglobin	31.3 pg	27.0–32.0 pg	Normal
Mean corpuscular haemoglobin concentration	331 g/L	315–360 g/L	Normal
Platelets	289 $\times 10^9$/L	150–400 $\times 10^9$/L	Normal
Albumin	42 g/L	32–50 g/L	Normal
Alanine aminotransferase	16 IU/L	10–35 IU/L	Normal
Alkaline phosphatase	92 IU/L	30–130 IU/L	Normal
Bilirubin	11 µmol/L	0–21 µmol/L	Normal
Total protein	71 g/L	60–80 g/L	Normal
Globulin	29 g/L	20–39 g/L	Normal
Sodium	142 mmol/L	133–146 mmol/L	Normal
Potassium	4.6 mmol/L	3.5–5.3 mmol/L	Normal
Urea	6.6 mmol/L	2.5–7.8 mmol/L	Normal
Creatinine (enzymatic)	68 µmol/L	49–90 µmol/L	Normal
Estimated glomerular filtration rate (CKD-EPI)	>90 mL/min/1.73 m^2	>90 mL/min/1.73 m^2	Preserved renal function

TABLE 1: Laboratory results with interpretation.

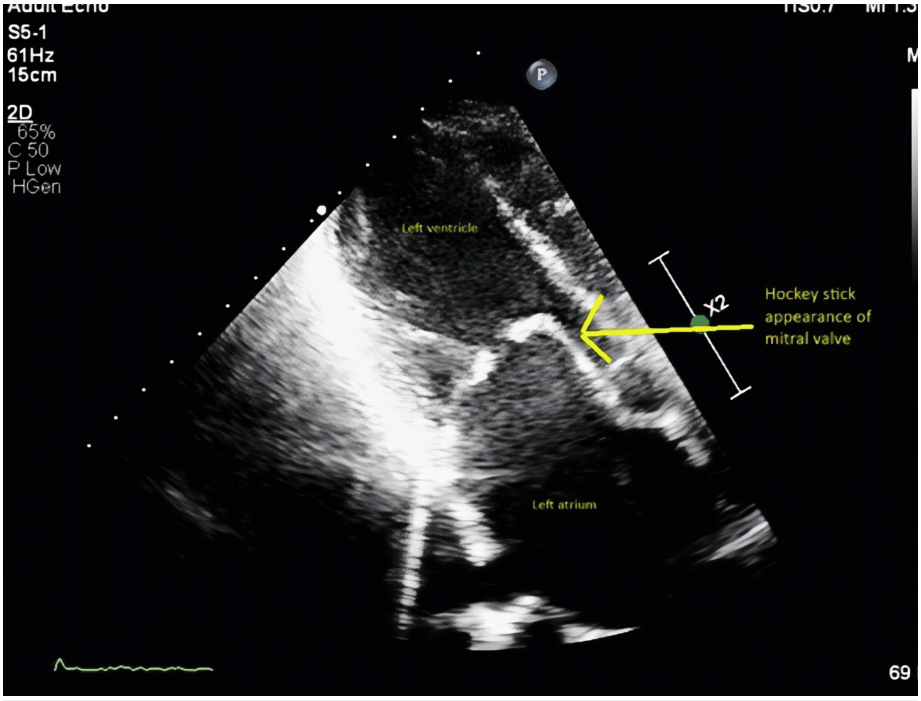
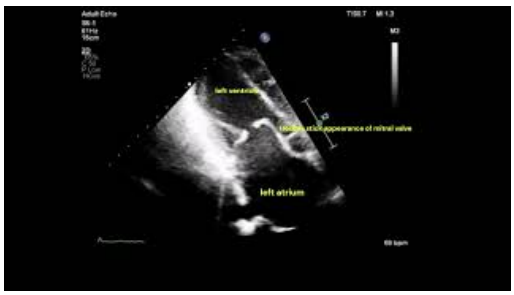


FIGURE 2: Labelled transthoracic echocardiogram showing the 'hockey stick' appearance of the mitral valve (two-chamber view).



VIDEO 1: Labelled transthoracic echocardiogram showing the 'hockey stick' appearance of the mitral valve (two-chamber view).

View video here: https://www.youtube.com/watch?v=l6LMZxeRWkk

She was admitted under cardiology and treated as AF with decompensated heart failure (HF) with intravenous furosemide. Apixaban was switched to low-molecular-weight heparin in line with prescribing guidelines for pregnancy [6]. The Obstetrics team repeated the serum beta-hCG and found a significant drop (from 1900 IU/L to 1000 IU/L) over 48 hours. A transvaginal ultrasound (TVUS) undertaken 72 hours after presentation showed an empty uterus and no adnexal mass or free fluid consistent with pregnancy of unknown location. This acute change in beta-hCG levels and normal TVUS represented a miscarriage, which was confirmed by a further decline of serial serum beta-hCG levels to <5 IU/L. A TOE was performed as part of her workup for valvular intervention, which confirmed severe MS (MVA 0.3 cm^2, mean PG 12.6 mmHg, peak PG 18.3 mmHg) and a severely dilated left atrium. There was spontaneous echo contrast in the dilated left atrium, which represents a stagnated or low-velocity blood flow state, and a multilobulated, large LAA thrombus (Figure *3*, Video *2*). The diagnosis was made: HF due to decompensated MS with LAA thrombus. The aetiology was presumed to be rheumatic due to the characteristic TTE findings, and the decompensation was likely secondary to the hemodynamic effects of AF. The miscarriage could be a complication of the decompensation leading to reduced uterine perfusion; however, other differentials such as coincidental spontaneous miscarriage and the effects of apixaban, a drug contraindicated in pregnancy, were also considered equally possible. Anticoagulation with warfarin was indicated for valvular AF and the left atrial thrombus, with a target international normalised ratio (INR) of 3.0. She was discharged following optimisation of her INR and HF medications with a plan for a repeat TOE to assess for clot resolution and outpatient workup for valvular intervention.

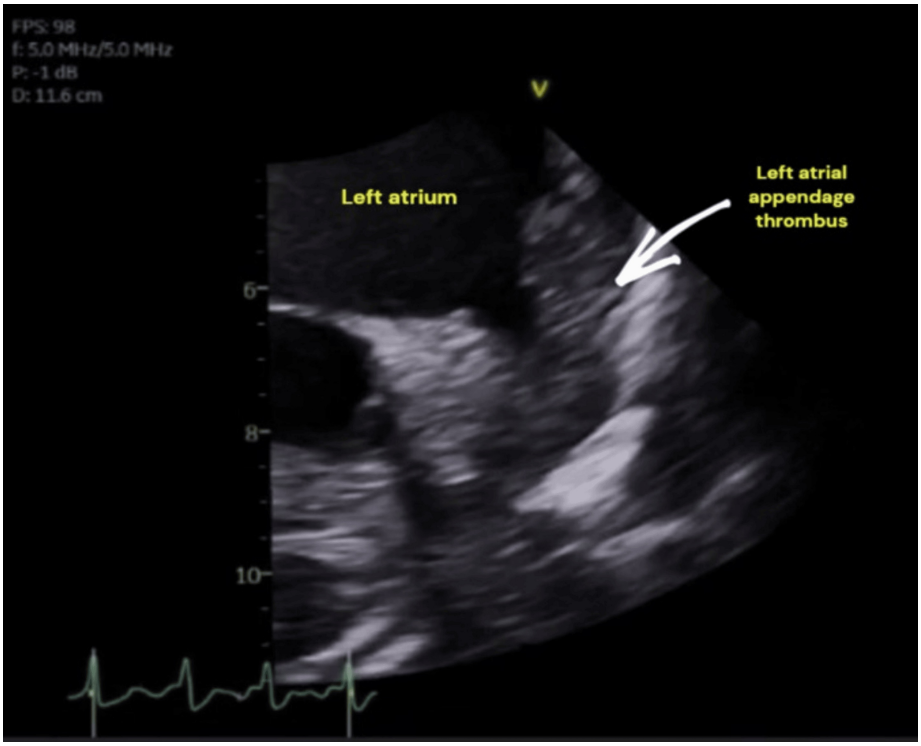


FIGURE 3: Labelled transoesophageal echocardiogram showing left atrial appendage thrombus.



VIDEO 2: Labelled transoesophageal echocardiogram showing left atrial appendage thrombus.

View video here: https://www.youtube.com/watch?v=2RIEE6MVGko&time_continue=0&source_ve_path=NzY3NTg&embeds_referring_euri=https%3A%2F%2Fwww.cureus.com%2

Following discussion at our local Cardiac Surgery Multidisciplinary Team (MDT) meeting, it was agreed that she would be a candidate for percutaneous mitral commissurotomy (PMC); however, due to the presence of a left atrial thrombus, PMC was to be deferred until the resolution of the thrombus. Following a Maternal Medicine MDT meeting, it was agreed that any further pregnancies would pose a severe risk to both mother and child. Hence, it was recommended that she use strict contraception pending definitive management of her MS.

At six-week follow-up, a repeat TOE showed persistence of the LAA thrombus, which was organising. Following further discussion at the local tertiary centre valve MDT, it was agreed that the clot was sufficiently laminated and deep within the LAA to be able to proceed with the PMC and is currently awaiting the procedure. She remains on warfarin with a therapeutic INR.

Discussion

In the Western world, the incidence of RHD is low due to widespread antibiotic use altering Group A *Streptococcus* strains and improved living standards [1]. Our case serves as a reminder that the incidence of RHD is currently on the rise in the Western world, aided by global migration, and remains the leading cause of MS worldwide [1].

Although there was no evidence of childhood rheumatic fever, the echocardiographic findings of hockey stick appearance and tenting of mitral valve leaflets are in keeping with rheumatic pathology [3,7] and consistent with the 2023 World Heart Federation guidelines for the TTE-based diagnosis of RHD [4]. Echocardiographic features suggestive of rheumatic MS are described in Table *2*. According to the European Society of Cardiology guidelines, TTE provides sufficient information to guide routine management; however, TOE is recommended to exclude an LAA thrombus and to characterise the mitral valve prior to planned intervention or if TTE is suboptimal [3,5].

Echocardiographic features of rheumatic mitral stenosis
Leaflet fibrosis and thickening predominantly confined to the tips and commissures
Chordal thickening, fusion and shortening
Commissural fusion with thickened rolled free edges (best visualised in parasternal short axis)
Funnel-shaped stenotic orifice
Restrictive motion of the mitral leaflet tips compared to the base
Characteristic ‘hockey stick’ appearance of mitral valve (best visualised in parasternal long axis)

TABLE 2: Echocardiographic features of rheumatic mitral stenosis

Sources: [3,7]

MS is clinically significant when MVA < 1.5 cm^2 and severe when MVA < 1.0 cm^2 [3, 5]; in our case, MVA was 0.7 cm^2 on outpatient TTE and 0.3 cm^2 on TOE during decompensation. This discrepancy is likely due to methodological variation between TTE and TOE and haemodynamic factors such as heart rate, intravascular volume and AF rather than true disease progression over a short period of time [8]. MVA measurement on TTE typically uses two-dimensional planimetry, and on TOE uses three-dimensional planimetry [8]. While the MVA obtained by both techniques are comparable, two-dimensional TTE planimetry tends to overestimate MVA compared with three-dimensional TOE planimetry [8]. The magnitude of the overestimation is mainly associated with left atrium dimension and user expertise in obtaining optimal short-axis images of the mitral valve tips. In situations when there is a discrepancy between the two imaging modalities, TOE should be considered as more accurate, especially in the context of a large left atrium [8].

Pregnancy and AF are important causes for decompensation in MS [6]. MS remains the leading cause of acquired valvular disease in pregnancy [9]. There is a 50% increase in transvalvular gradient during pregnancy with MS, especially from the first to second trimester, which increases the risk of maternal and foetal complications such as maternal death, intrauterine growth retardation, prematurity and foetal loss [2, 6]. AF further intensifies the haemodynamic burden on the heart and is the most common arrhythmia associated with RHD in pregnancy [2]. Acute decompensation and HF in pregnant women with MS can be seen in around 50% of patients with an MVA of < 1.5 cm^2 [9].

Unfortunately, our patient suffered a miscarriage as a possible complication of MS decompensation with consequent reduction in uterine perfusion [2]. Alternative explanations, including coincidental spontaneous miscarriage and the effects of apixaban, a drug contraindicated in pregnancy [6], were also considered equally possible due to lack of clear evidence pointing to one cause over the other. Apixaban and other novel oral anticoagulants are not recommended in rheumatic AF [5,10] and contraindicated in pregnancy [6].

A further point of interest is that the patient's two previous pregnancies were uncomplicated, the most recent being 18 months ago, at which time it can be assumed that she already had RHD with a comparable valve area. We, therefore, believe that the adverse outcome on this occasion could be attributable to her being in AF rather than sinus rhythm, in addition to her severe MS with pregnancy, a possible minor contributor. Family planning in RHD should involve a Maternal Medicine MDT approach.

Conclusions

Our case adds to existing literature on rheumatic MS by demonstrating maternofoetal complications associated with decompensated rheumatic MS in the context of pregnancy and AF. It serves as a reminder that the incidence of RHD is currently on the rise in the Western world, aided by global migration. It highlights the diagnostic TTE findings in rheumatic MS while also emphasising the value of TOE in excluding an LAA thrombus and characterising the mitral valve prior to planned intervention.

The key clinical takeaway from our case is that RHD in the context of pregnancy and AF is fraught with complications, and multidisciplinary management between cardiology, cardiac surgery and maternal

medicine is vital to improve patient outcomes.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Rohith Narayan Narayanaswamy Yechanahally

Acquisition, analysis, or interpretation of data: Rohith Narayan Narayanaswamy Yechanahally, Timothy Swinn, Laisha Gogola, Amardeep G. Dastidar

Drafting of the manuscript: Rohith Narayan Narayanaswamy Yechanahally

Critical review of the manuscript for important intellectual content: Rohith Narayan Narayanaswamy Yechanahally, Timothy Swinn, Laisha Gogola, Amardeep G. Dastidar

Supervision: Amardeep G. Dastidar

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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