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Case Report

Acute Pulmonary Edema in a Patient with Mitral Stenosis Presentation: Why Now?

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ABSTRACT

Pregnancy-associated pulmonary embolism remains the leading cause of direct maternal mortality globally. We will present a case of a young lady who was one month post-delivery via normal vaginal delivery. She presented with pulmonary edema and her clinical examination was consistent with severe mitral stenosis. Notably, her previous and recent pregnancies were uneventful. The key question was: why is there a sudden deterioration? "Why now?". This led to an industrious search of a precipitant for her presentation; a diagnosis of severe rheumatic mitral stenosis complicated by pulmonary edema precipitated by a pulmonary embolism was made. The patient later had a valve replacement with good outcomes.

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1. Case Report

Patient presentation: A 30-year-old G3P3 woman was referred to our unit 1 month after having had an uncomplicated vaginal delivery of a healthy baby. She was well until 2 days before referral, when she developed sudden onset of shortness of breath, which progressed rapidly over the subsequent 24 hours, to the point where she was short of breath at rest with PND. The accompanying symptoms included pleuritic chest pain and a non-productive cough. She denied palpitations, haemoptysis, fever, a productive cough, and lower limb pain or swelling.

Her prior pregnancies were uneventful, culminating in the vaginal delivery of full-term babies. She was HIV negative, and there was no history of previous surgeries. There was no known history of childhood rheumatic fever or congenital heart disease. Except for injectable contraceptives, she was not using any medications. She neither smoked nor consumed alcohol, and she denied recreational drug use. On arrival, she was mildly febrile with temp 37.6°Celsius, the blood pressure was 105/69mmHg, heart rate was 125 beats per minute, and respiratory rate was 30 breaths per minute. Her oxygen saturation was 99% on a non-rebreather mask, a significant improvement from 64% on room air. The physical examination was notable for a normal jugular venous pressure, a cardiac apex which was non-displaced with a palpable first heart sound, which was loud on auscultation. A diastolic murmur with pre-systolic accentuation was appreciated at the apex with the patient lying on the left side. At the left parasternal border, there was a pansystolic murmur of tricuspid regurgitation. The respiratory system

revealed bilateral crackles, and the abdomen was normal with no hepatomegaly, splenomegaly or ascites.

Her electrocardiogram (ECG) is illustrated in (Figure 1). Notable findings included sinus tachycardia, right axis deviation (90°), left atrial hypertrophy and a dominant R in V1. The laboratory studies showed an elevated white cell count of 30 x 10⁹/L with the predominance of neutrophils (87%). Her electrolytes and renal function were normal. Her chest X-ray was notable for a bat's wing pattern of bilateral airspace opacification indicative of pulmonary edema (Figure 2). Her echocardiography is presented in (Figure 3). It confirmed the clinical diagnosis of severe rheumatic mitral stenosis with a mean gradient of 16 mmHg, left atrial dilatation with echo 'smoke,' normal left ventricular dimensions with normal LV systolic function. The Wilkin's score was 9/16.

Clinical Course: Given the diagnosis of tight mitral stenosis complicated by acute pulmonary edema the patient received intravenous diuresis aggressively over the first 24 hours while she was continued on face mask oxygen.

Rheumatic severe mitral stenosis is a post-inflammatory fibrostenotic disease in which the left atrial pressure is chronically elevated. Therefore, an acute pulmonary edema presentation should always stimulate a search for precipitants. Because this was one month postpartum and was out of the window of cardiovascular vulnerability, pregnancy as a precipitant of her acute pulmonary edema was considered less likely.

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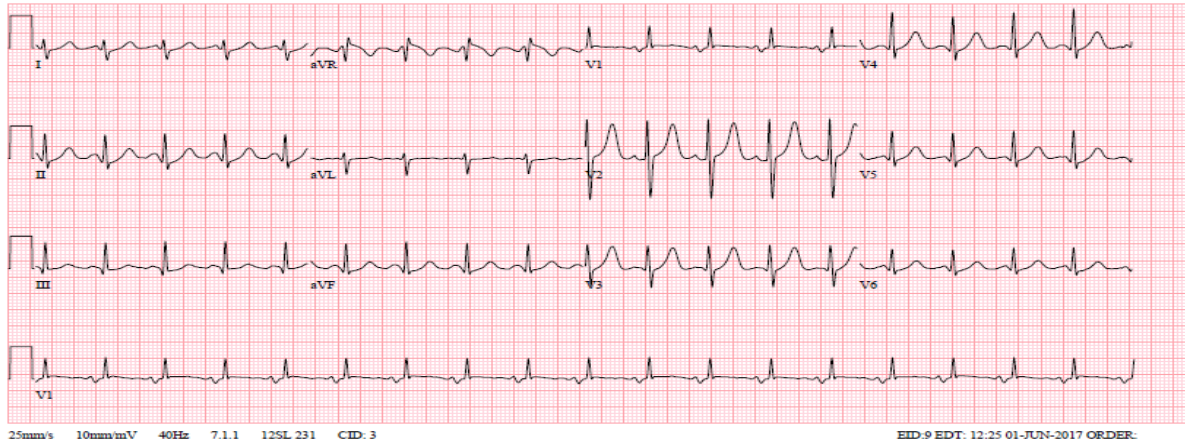


FIGURE 1: Electrocardiogram (ECG) shows sinus tachycardia at a rate of 114/minute, Right axis deviation, a dominant R wave in V1 suggestive of Right ventricular hypertrophy and Left atrial enlargement.

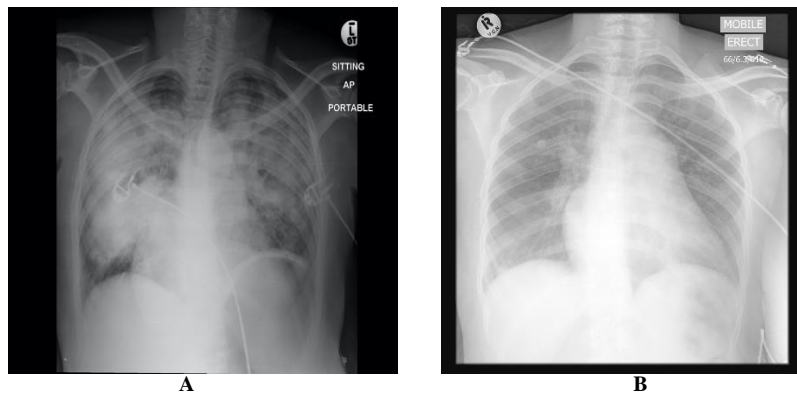


FIGURE 2: A) Chest X-ray B) Chest X-ray post IV diuresis.

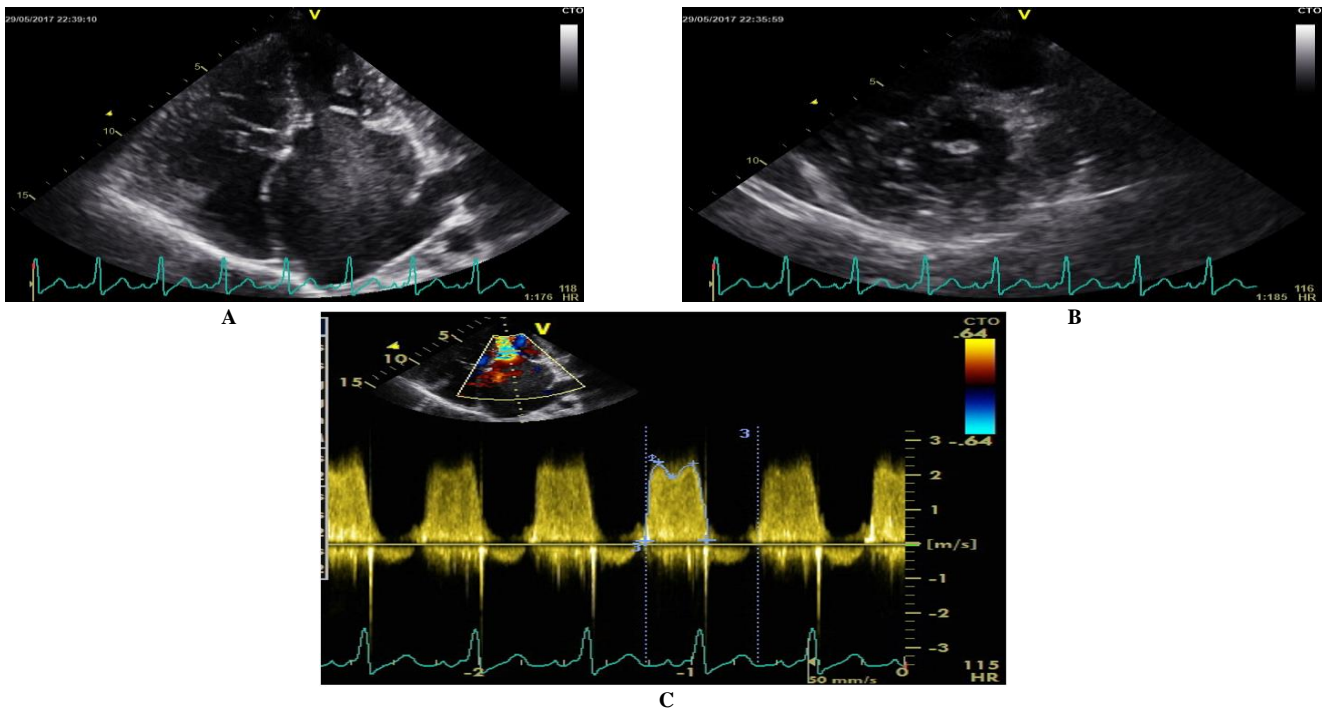


FIGURE 3: A) Apical four chamber view shows a large left atrium with spontaneous echo smoke, a non-dilated left ventricle. B) Parasternal short axis view at the level of the mitral valve shows a rheumatic mitral valve with commissural fusion and a reduced mitral valve area. C) Continuous wave doppler across the mitral valve in the apical four chamber view revealed a mean gradient of 16 mmHg.

Pneumonia was also considered unlikely despite the mild fever and leucocytosis, given the acuteness of the illness and lack of other characteristic symptoms such as sputum production. Tachyarrhythmia was considered a possibility, but the patient did not have palpitations, and there was no ECG and telemetry evidence of a rhythm disturbance. Finally, given the hypercoagulability of pregnancy and the hormone contraceptives she was using, she was treated empirically for an acute pulmonary embolism until she was well enough to either confirm or refute the diagnosis with appropriate testing. On day 3, after she had clinical resolution of her pulmonary edema, she went for a ^{99m}Tc lung ventilation-perfusion scan which revealed a perfusion mismatch in the entire right lower lung lobe as well as a matched perfusion defect in the superior basal segment of the left lung with perfusion being affected to a greater extent. Ventilation and perfusion to the lungs were inhomogeneous and more pronounced in the perfusion study. These findings were interpreted as high probability of a large PE.

2. Discussion

Pregnancy-associated pulmonary embolism remains the leading cause of direct maternal mortality globally, partly due to the fact that the clinical diagnosis of venous thromboembolism (VTE) in pregnancy is usually obscured by the maternal physiological changes that happen in pregnancy that may mimic symptoms and signs of VTE [1, 2]. VTE complicates 1 in 1000 pregnancies and is approximately 10 times more common in pregnant women compared with non-pregnant women [3, 4]. For example, in a study undertaken in Limpopo, South Africa, a total of 14,685 live births and 232 maternal deaths were reported over the five-year period of the study, resulting in a maternal mortality ratio of 1579 per 100 000 live births, 3 % (n=7) were due to pulmonary embolism [5]. Pregnancy-related VTE is one of the 10 most important potentially preventable causes of maternal death in South Africa (SA) [6].

During pregnancy, VTE events occur with equal distribution across all three trimesters [7]. By 20 weeks' gestation, more than half of women affected will have had a VTE event [8]. The risk of venous thromboembolism is also increased postnatally by approximately 20-fold compared to normal age-matched controls. Although the risk extends until at least 12 weeks into the postnatal period [9], most thromboembolic events occur in the first 3 weeks after delivery [9, 10].

While the association between valvular heart disease and pulmonary embolisms risk of VTE has only recently been confirmed [11], the association between mitral stenosis and pulmonary infarction was recognized as early as the 1930s [12]. The recognition and diagnosis of acute pulmonary embolus in patients with valvular heart disease and heart failure may be particularly difficult given the potential masking of "typical symptoms and signs" and difficulties distinguishing these from those of the underlying cardiac disease and heart failure [13]. In pregnant patients and those with valvular heart disease who present with pulmonary emboli, the predictors of a poor outcome are not dissimilar to other patients with pulmonary embolus [14]. Patients who present with hemodynamic instability are usually at increased risk of death [15]. The outcomes of the condition depend on early recognition and adequate therapy being instituted during the early stages. Early recognition and treatment have been reported to significantly decrease mortality rates [15, 16]. It is recommended that if there is clinical suspicion of

thromboembolism, treatment can be commenced pending confirmation of diagnosis [17].

The title of the article "Why now?" is a practical question that clinicians should aim to answer whenever there is a sudden deterioration of an otherwise "quiescent" mitral stenosis. When this occurs, a useful short list of precipitants to think about and rule out clinically or with appropriate testing is provided in (Table 1) below. In this particular patient, it was the pursuit of the answer to this question that allowed for confirmation of the diagnosis and treatment with both short term anticoagulant therapy and long-term warfarin, thus significantly reducing the likelihood of recurrence with even more dire consequences.

Cardiac precipitants	Non-cardiac precipitants
New tachyarrhythmia (a fib most common)	Infection, e.g., Pneumonia, UTI
Infective endocarditis	Acute or occult blood loss, e.g., GIT bleed
Pulmonary embolism	Pregnancy (most vulnerable at 24-36 weeks)
Acute ischemia (e.g., thromboembolism to coronary artery)	Thyrotoxicosis

3. Conclusion

We have presented a patient with a background of severe rheumatic mitral stenosis complicated by pulmonary edema precipitated by a pulmonary embolism in the early postpartum period. The case served as a very useful reminder of the importance of looking for potential precipitants in patients with an unexplained acute deterioration. In this particular incidence, the fact that she had not decompensated during pregnancy and the suddenness of her symptoms prompted a thorough search for pulmonary embolus, which was treated successfully with appropriate therapy. The patient was scheduled for valve replacement therapy after allowing a prolonged bonding period with her newborn baby daughter.

Conflicts of Interest

None.

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