Unknown Rheumatic Cardiac Disease as Cause of Acute Onset Post-Partum Dyspnea: a Case Report

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Case Report

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Abstract

Background: Acute post-partum dyspnea configures an obstetric challenge with multiple differential diagnosis.

Case Presentation: We present a case of a previous healthy woman with preeclampsia who developed severe dyspnea 30 hours after delivery. She complained of cough, orthopnea, and bilateral lower extremities oedema. She denied headaches, blurry vision, nausea, vomiting, fever or chills. Auscultation revealed a diastolic murmur, and was compatible with pulmonary oedema. A timely bedside echocardiogram showed moderate dilated left atrium with severe mitral insufficiency suggestive of an unknown rheumatic disease. She was managed with noninvasive ventilation, loop diuretics, vasodilators, thromboprophylaxis, head-end elevation, and fluid restriction with progressive improving.

Conclusions: Previously silent cardiac diseases constitute intriguing causes of post-partum dyspnea with pulmonary oedema. A timely and multidisciplinary approach is required to manage these situations.

Background

Early puerperium is a complex period with readaptation processes occurring in the female organism and redistribution of blood volume. The investigation of an acute onset dyspnea in this period (even during the COVID-19 era) should include a complete evaluation for causes, such as previously silent cardiac diseases [1].

Case Report

A 28-year-old pregnant woman (3 Para, 1 Living) of South African nationality with no previous chronic comorbidities was diagnosed with mild preeclampsia (de novo hypertension plus proteinuria) at 36 weeks of gestation. The patient had had regular antenatal visits, and her obstetric history revealed a previous diagnosis of preeclampsia and preterm labor, with absence of a post-partum revision consultation. Labor was induced at 37 weeks with an uncomplicated eutocic delivery.

Approximately 30 hours after delivery, she presented chief complaint of increasing shortness of breath, and chest pressure. Patient also reported cough, orthopnea, and bilateral lower extremities oedema. She denied headaches, blurry vision, nausea, vomiting, fever or chills. During observation, she was in severe distress, restless, agitated, and using her accessory muscles of respiration. Vitals were recorded, which revealed normal temperature, tachycardia (120bpm), tachypnea (28cpm), and mild hypertension (143/86mmHg). She had a peripheral oxygen saturation of 83%. Cardiac auscultation revealed tachycardia and a diastolic murmur more perceptible in the mitral area, and respiratory auscultation revealed fine crackles heard bilaterally with decreased basilar breathing sounds. Bilateral pitting pedal oedema was noted. Therefore, a multidisciplinary team approach with Obstetrics, Internal Medicine and Cardiology team was implemented.

Arterial blood gas analysis on Venturi mask 28% revealed pH of 7.46, carbon dioxide tension 33mmHg, arterial oxygen tension 51mmHg, bicarbonate 23.5 mmol/L, and arterial oxygen saturation of 88%. Routine blood investigations (full blood count, liver and kidney function tests, prothrombin time/international normalized ratio, serum electrolytes, and urine analysis) were drawn. This study showed no abnormalities, besides mild leukocytosis. Brain natriuretic peptide (BNP) was raised - 1011pg/mL. Cardiac enzymes were negative. Serial 12 leads electrocardiogram showed sinus tachycardia, with T-wave inversion in V1-V3. SARS-CoV2 Real Time-PCR was negative. A 2D bedside echocardiogram was performed revealing moderate dilated left atrium with severe mitral insufficiency suggestive of rheumatic disease, normal systolic function, and no other alterations.

After initial stabilization, patient was admitted to the ICU for further supervision. She was managed with noninvasive ventilation, loop diuretics, vasodilators (nifedipine), thromboprophylaxis, head-end elevation, and fluid restriction with progressive improving. Four days after delivery, the patient was stable and was discharged.

A Cardiology and Obstetrics post-partum revision consultation was conducted, 6-8 weeks after delivery. By that time, the patient was asymptomatic without need of any anti-hypertensive or diuretic drug. A new echocardiogram was conducted showing mild mitral disease with a rheumatic pattern, and no other relevant changes. Patient was referred for dental evaluation and informed of the impact of her rheumatic cardiac disease in the present pregnancy and the need for adequate follow-up in case of eventual future pregnancies.

Discussion and Conclusions

Many conditions can result in acute onset post-partum dyspnea [2] (Figure 1). These conditions include pathologies not associated with pulmonary oedema, such as pulmonary embolism, amniotic fluid embolism, pneumonia (e.g. bacterial, COVID-19), sepsis, and pathologies associated with pulmonary oedema [3]. In the last group, the causes may be cardiogenic, including peripartum cardiomyopathy, preeclampsia-induced cardiomyopathy, myocardial ischemia, and underlying structural heart disease or valvular heart disease (as in this clinical case). Examples of noncardiogenic pulmonary oedema are acute respiratory distress syndrome (ARDS), iatrogenic fluid overload (e.g., during labor), thyroid disease, rheumatologic conditions, and drug-induced pulmonary oedema (e.g., by oxytocin).

Mitral valve disease due to rheumatic heart disease is an important yet rare cause of post-partum cardiogenic pulmonary oedema, and dyspnea. This condition may be silent and present for the first time in the peripartum period. A complete physical examination and doppler echocardiography were the key to confirm the diagnosis in this case and should be included as part of a routine investigation of an acute onset post-partum dyspnea. Timely diagnosis and effective multidisciplinary management are required irrespective of the dyspnea´s cause in order to reduce morbidity and mortality. Clinicians should keep in mind a broad differential diagnosis for post-partum dyspnea.

Declarations

Ethics approval and consent to participate:

Written informed consent was obtained from patient.

Consent for publication:

Written informed consent was obtained from patient.

Availability of data and materials:

The datasets used and analyzed during the current study is available from the corresponding author on reasonable request.

Conflicts of interest:

All authors do not have conflict of interest.

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Author´s contributions:

António De Pinho – conception, acquisition and interpretation of data, manuscript writing. All authors: reading and revision of the final manuscript.

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Figures

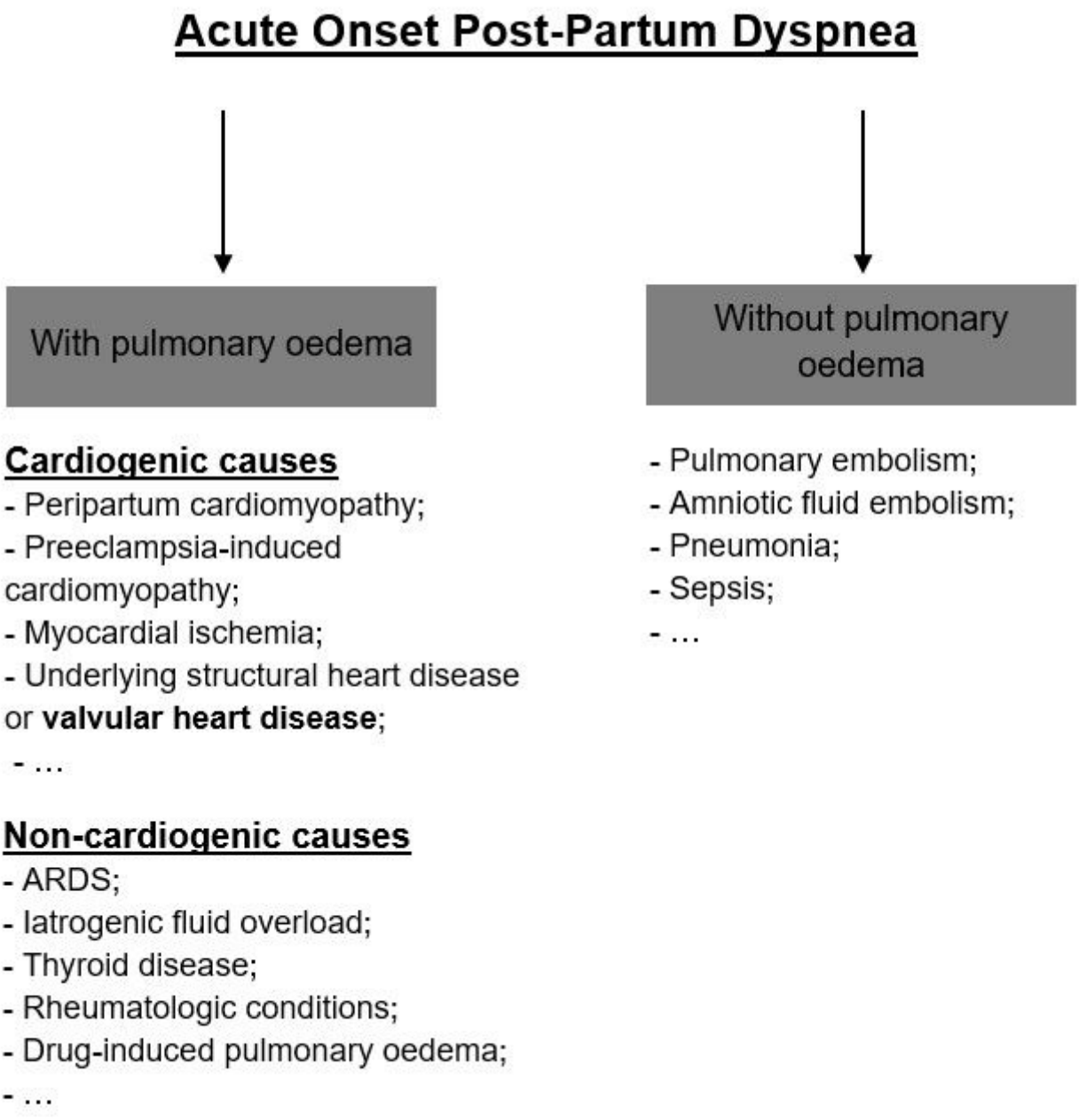

Figure 1

Main Causes of Acute Onset Post-Partum Dyspnea