

## Case report

# *Post partum* dyspnoea: look beyond the lungs

A 24-year-old woman (Para 1, Living 1) with no previous comorbidities presented to the emergency room (ER) with complaints of acute onset dyspnoea with swelling of both legs for the past 12 h and cough with frothy sputum for the past 4 h. She denied any history of chest pain, back pain, fever, sore throat, runny nose or rash. She had undergone a caesarean section 24 h ago, which was uneventful, and she was stable postoperatively. Her medical and obstetric history were insignificant.

The patient had had regular antenatal visits and her antenatal history was insignificant for any cardiac illness and/or preeclampsia. There was no history of any tocolytic administration or drug abuse.

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### Task 1

What is the differential diagnosis of *post partum* dyspnoea?

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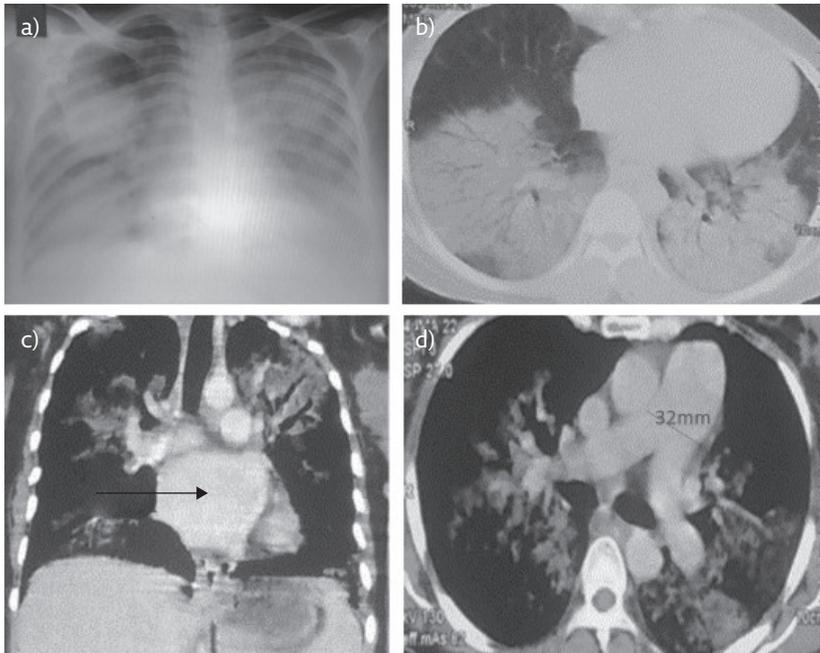
**The aetiology of acute-onset dyspnoea in the *post partum* period is diverse. However, subtle clinical and radiological findings assist in early diagnosis and definitive management, thereby conferring better prognosis and survival.** <https://bit.ly/361b4qm>



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**Figure 1** a) Chest radiograph showing bilateral alveolar infiltrates. b) CT thorax axial reformatted images showing bilateral consolidations with air bronchograms. c) CT thorax coronal section showing enlarged left atrium (arrow) with splaying of carina. d) CT thorax mediastinal window showing dilated main pulmonary artery.

### Answer 1

The differential diagnosis of *post partum* dyspnoea is broad and includes cardiac as well as non-cardiac causes. Cardiac causes include heart failure due to peripartum cardiomyopathy, myocardial infarction due to coronary artery dissection or an atheromatous plaque or coronary embolism, pulmonary embolism, amniotic fluid embolism, rheumatic valvular heart disease leading to heart failure, tocolytic induced pulmonary oedema, Takotsubo cardiomyopathy, and aortic dissection. Non-cardiac causes include pneumonia, *post partum* haemorrhage, thyroid disease and iatrogenic fluid overload [1].

On general examination, she was afebrile, restless, agitated, using her accessory muscles of respiration and in severe distress. Her jugular venous pressure (JVP) was elevated (15 cmH<sub>2</sub>O) and bilateral pitting pedal oedema was present. Both the calves were equal in size and non-tender on squeezing. Vitals were recorded, which revealed tachycardia (heart rate 130 beats·min<sup>-1</sup>, regular) and tachypnoea (respiratory rate 28 breaths·min<sup>-1</sup>). She was normotensive (130/84 mmHg) with a peripheral oxygen saturation (SpO<sub>2</sub>) of 74%. Respiratory system auscultation revealed bilateral basal fine end-inspiratory crepitations. Arterial blood gas analysis on ambient air revealed pH of 7.51, carbon dioxide tension 3.9 KPa, arterial oxygen tension 3.7 KPa, bicarbonate 21.8 mmol·L<sup>-1</sup> and arterial oxygen saturation of 70%.

Chest radiography showed bilateral alveolar infiltrates (figure 1a). ECG revealed sinus tachycardia with no ST/T changes. Troponin I was negative. A nasopharyngeal and throat swab was sent for COVID-19 RT-PCR as per local institutional protocol. Routine blood investigations (full blood count, liver and kidney function tests, prothrombin time/international normalised ratio, thyroid-stimulating hormone and serum electrolytes) did not disclose any abnormality. D-dimer was raised (700 ng·mL<sup>-1</sup>). Brain natriuretic peptide (BNP) was elevated (2934 pg·mL<sup>-1</sup>). Computed tomography (CT) of the thorax revealed bilateral alveolar consolidation with air bronchograms, cardiomegaly with an enlarged left atrium, and a dilated main pulmonary artery (figure 1b-d).

### Task 2

The most probable diagnosis in our case is?

- a) Acute respiratory distress syndrome (ARDS)
- b) Cardiogenic pulmonary oedema
- c) Viral pneumonia (COVID-19 pneumonia)
- d) Pulmonary haemorrhage

**Answer 2****b. Cardiogenic pulmonary oedema**

The clinical findings of an elevated JVP with pedal oedema and typical radiological findings (bilateral alveolar shadows, cardiomegaly and enlargement of the pulmonary vasculature) favours cardiogenic pulmonary oedema [2]. The diagnostic criteria of ARDS is given by the Berlin definition [3]. Fever is the most common symptom of COVID-19 pneumonia and it has classical radiological findings. These include peripheral ill-defined ground-glass opacities (GGO), consolidations, septal thickening, crazy paving and atoll sign [4]. Pulmonary haemorrhage is characterised by the triad of haemoptysis, falling haematocrit/haemoglobin and bilateral alveolar opacities. The radiological findings include bilateral GGO and consolidations with peripheral sparing. Interlobular septal thickening and crazy paving appearance occur due to fibrosis caused by repeated bleeding episodes and resolution [5]. Other causes of acute bilateral alveolar consolidations include pneumonia (fungal, bacterial, atypical and

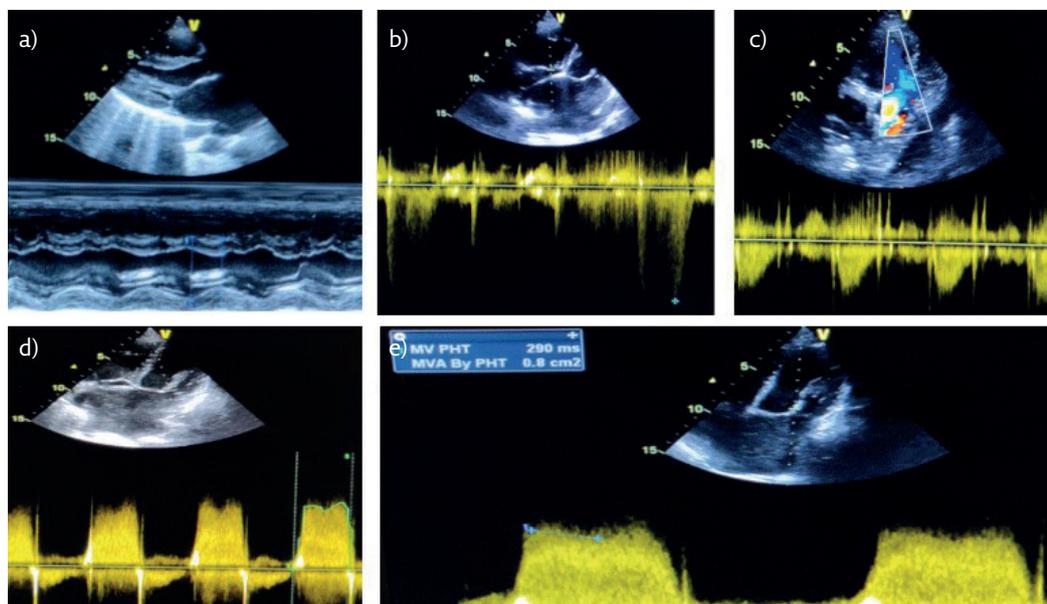
mycobacterial), acute eosinophilic pneumonia, acute interstitial pneumonia and radiation pneumonitis [6].

RT-PCR of the nasopharyngeal and throat swab for COVID-19 were negative. A CT pulmonary angiogram was carried out, which did not show any features of acute or chronic pulmonary embolism. Two-dimensional echocardiography revealed rheumatic valvular heart disease with severe mitral stenosis (valve area 0.8 cm<sup>2</sup>), severe tricuspid regurgitation, pulmonary hypertension with pulmonary artery systolic pressure (PASP) of 71 mmHg, mean diastolic gradient across mitral valve of 20 mmHg and left ventricle ejection fraction of 60% (figure 2a-f).

**Task 3**

Radiological findings of mitral valve stenosis include all of the following except:

- Straightened left heart border
- Double density sign
- Walking man sign
- Second mogul sign



**Figure 2** a) Doppler echocardiography parasternal long-axis view showing dilated left atrium and good left ventricular ejection fraction. b) The apical four-chamber view shows the measurement of PASP. c) Tricuspid regurgitation jet in apical four-chamber view. d) Measurement of the mean diastolic gradient across the mitral valve in the apical four-chamber view. e) Measurement of mitral valve area using the pressure half time method.

**Answer 3**

d. Second mogul sign.

In mitral valve stenosis there is an enlargement of left atrial appendage, which appears as a bulge in the left cardiomeastinal border just below the left main bronchus and is popularly known as “the third mogul sign” [7]. The second mogul refers to a bulge in the pulmonary artery bay and is seen in pulmonary hypertension. Double density sign (double right heart border) is due to the enlarged left atrium projecting beyond the right heart border [8]. Walking man sign is seen in a lateral chest radiograph and is due to posterior displacement of left main bronchus thereby the right and left main bronchus appear as the legs of a walking man [9]. Straightened left heart border (mitralisation) is formed by the alignment of the aortic knuckle, dilated pulmonary artery, prominent left atrial appendage, and left ventricle in a straight line. It is seen in severe mitral valve stenosis with pulmonary hypertension [10]. Other nonspecific radiological findings include upper lobe diversion (cephalisation) of the pulmonary veins, splaying of the carina, pulmonary oedema, pulmonary haemorrhage, pulmonary hemosiderosis, and pulmonary ossification [11]. Radiological findings of mitral valve stenosis in our case included a dilated left atrium, splaying of the carina, straightening of the left heart border, and pulmonary oedema.

She was propped up (High fowler’s position), put on continuous positive airway pressure (CPAP) therapy of 10 cmH<sub>2</sub>O, started on loop diuretics (furosemide), and other supportive measures like morphine and low-molecular-weight heparin were initiated. After 24 h, her general condition improved. Her dyspnoea subsided and she was weaned off CPAP therapy and oxygen support after 72 h.

**Task 4**

What is the role of high-flow nasal cannula (HFNC) in the management of the above patient?

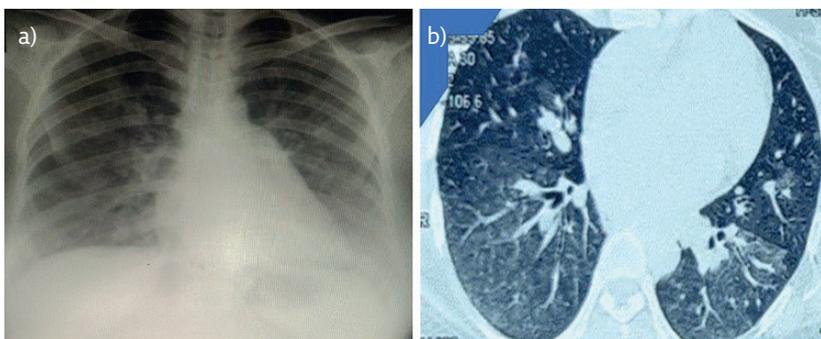
**Answer 4**

Acute hypoxaemic respiratory failure is considered to be an indication of HFNC. The FLORALI trial showed that the use of HFNC was associated with improved survival in patients with acute hypoxaemic respiratory failure. However, most of the patients in the FLORALI trial had pneumonia [12]. Data on the role of HFNC in cardiogenic pulmonary oedema is limited. In a prospective study, Roca *et al.* [13] showed that HFNC was associated with a decrease in respiratory rate in patients with heart failure. In a randomised controlled trial, MAKDEE *et al.* [14] showed that the use of HFNC was associated with a decrease in respiratory rate. However, they did not include patients with SpO<sub>2</sub> <90% and respiratory rate >35 breaths·min<sup>-1</sup>. We require high-quality randomised controlled trials to elucidate the role of HFNC in cardiogenic pulmonary oedema.

**Task 5**

Which of the following is not a beneficial effect of positive airway pressure (PAP) therapy in cardiogenic pulmonary oedema?

- a) Decrease in afterload
- b) Increase in preload
- c) Decrease in heart rate
- d) Increase in cardiac index



**Figure 3** a) Chest radiograph showing cardiomegaly, straightening of left heart border, and alveolar infiltrates have decreased in comparison with the previous chest radiograph (figure 1). b) CT thorax lung window axial section shows bilateral GGO (consolidation has resolved in comparison with the previous CT of the thorax).

**Answer 5**

b. Increase in preload.

PAP therapy increases the intrathoracic pressure, which in turn decreases the venous return (decrease in right and left ventricular preload) [15]. Other beneficial effects of PAP therapy include a reduction in afterload, decrease in heart rate, re-expanding flooded alveoli (thereby improving oxygenation), decrease in the work of breathing by unloading the inspiratory muscles, counteracting intrinsic positive end-expiratory pressure, prevention of micro-atelectasis, and increases in cardiac index and performance [15, 16]

Chest radiography and CT of the thorax were repeated at discharge, which showed almost complete resolution of alveolar consolidations (figure 3). BNP decreased to 128 pg·mL<sup>-1</sup>.

## Discussion

Acute pulmonary oedema in the *post partum* period is a medical emergency affecting 0.08% to 1.5% of women [17]. Physiological changes occurring immediately in the *post partum* period include a 60–80% increase in cardiac output due to the relief of the inferior vena cava obstruction by the fetus and redistribution of blood flow from placental to maternal circulation [18]. This increase in cardiac output is physiological and it returns to pre-labour levels by 12 h and pre-pregnancy level by 2 weeks. However, it can be catastrophic in those with underlying cardiac illness due to a sudden increase in venous return caused by autotransfusion leading to decompensated heart failure [19]. Cardiogenic pulmonary oedema in the *post partum* period is multifactorial. Among cardiovascular diseases, rheumatic heart disease is one of the most prevalent diseases in pregnancy [20], especially in developing countries and poses serious complications. Further, some patients have subclinical rheumatic heart disease and this is easily missed in the antenatal period [21]. A high degree of clinical suspicion is required for its diagnosis and management.

Apart from rheumatic valvular heart diseases, certain risk factors can also lead to pulmonary oedema. It is broadly classified into cardiogenic and non-cardiogenic pulmonary oedema. Cardiogenic origin includes structural heart cardiac disease, congenital heart disease, ischaemic heart disease, and peripartum cardiomyopathy. Whereas non-cardiogenic pulmonary oedema occurs secondary to pre-eclampsia/eclampsia, tocolytics use to prevent premature delivery, illicit drugs (cocaine) and iatrogenic fluid overload [17]. Other causes of acute onset dyspnoea include pulmonary embolism, air embolism, amniotic

fluid embolism and aspiration pneumonia [22]. Detecting underlying heart disease in patients with acute pulmonary oedema is crucial [17] and Doppler echocardiography is the preferred tool in diagnosing rheumatic valvular heart disease [23], which revealed severe mitral valve stenosis and pulmonary hypertension in our case.

Rheumatic heart disease is the most common cause of mitral valve stenosis [24], although nearly 50% of patients would have had a mild subclinical rheumatic fever without articular manifestations, as seen with our case [25]. As per the observations of Yazıcı *et al.* [26], a negative history of rheumatic fever does not rule out rheumatic heart disease. Among valvular heart diseases, mitral valve stenosis is better tolerated in pregnancy than regurgitant lesions like mitral regurgitation and aortic regurgitation [27]. The high cardiac output of pregnancy combined with the impaired diastolic filling leads to an increase in left atrial pressure and eventually congestive heart failure [28].

Even severe mitral valve stenosis can be asymptomatic and may manifest as acute pulmonary oedema in the *post partum* period [19]. Valvular heart disease diagnosed for the first time during pregnancy and in the *post partum* period is associated with poorer outcomes than heart failure due to congenital heart diseases [29]. In symptomatic mild-to-moderate mitral valve stenosis or asymptomatic severe mitral valve stenosis, surgery or valvuloplasty should be considered before planning pregnancy to avoid potential complications during the antenatal as well as *post partum* period [30].

Finally, management should be focused on the successful resuscitation of the patient. After the initial resuscitation, a rigorous evaluation should be carried out to identify the precipitating cause. Management measures include noninvasive ventilation and PAP therapy, pharmacotherapy (loop diuretics, and vasodilators), optimising comorbid medical conditions, and supportive measures like head-end elevation and fluid restriction with strict input/output charting, especially during acute decompensated heart failure [31].

## Conclusion

Acquired mitral valve stenosis due to rheumatic heart disease is an important yet preventable cause of *post partum* pulmonary oedema. Although Doppler echocardiography is required to confirm the diagnosis, chest radiology provides subtle clues to the diagnosis. Timely diagnosis and effective management are required irrespective of the cause of the pulmonary oedema. Doppler echocardiography should be included as part of routine antenatal screening to reduce maternal morbidity and mortality.

## Affiliations

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## Conflict of interest

None declared.

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