

# Unilateral pulmonary oedema: a case report and literature review

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## Abstract

Acute myocarditis is often secondary to an acute virus infection, which can be the first manifestation of upper respiratory tract symptoms, followed by chest tightness, shortness of breath, palpitations, chest pain and other non-specific symptoms. In severe cases, it can quickly progress to serious complications such as heart failure, shock and respiratory failure. Laboratory examinations can show an increase of myocardial injury markers, infection and inflammatory indicators. Cardiac ultrasound can detect the weakening of the myocardial contraction and valve regurgitation. On imaging, bilateral pulmonary oedema demonstrates symmetrical infiltration along the hilum of lung, called the “butterfly shadow”. This current case report describes a patient with unilateral pulmonary oedema caused by myocarditis that was initially misdiagnosed and treated as pneumonia. The patient was subsequently treated with the application of extracorporeal membrane oxygenation and he made a full recovery. A review of this case highlights that when a patient’s symptoms are not typical, a comprehensive examination and evaluation are required to avoid incorrect treatment.

## Keywords

Unilateral pulmonary oedema, myocarditis, extracorporeal membrane oxygenation

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## Introduction

Pulmonary oedema is a common clinical syndrome, but unilateral pulmonary oedema is a rare event that accounts for 2.1% of cardiogenic pulmonary oedemas.<sup>1</sup> Most unilateral pulmonary oedemas are

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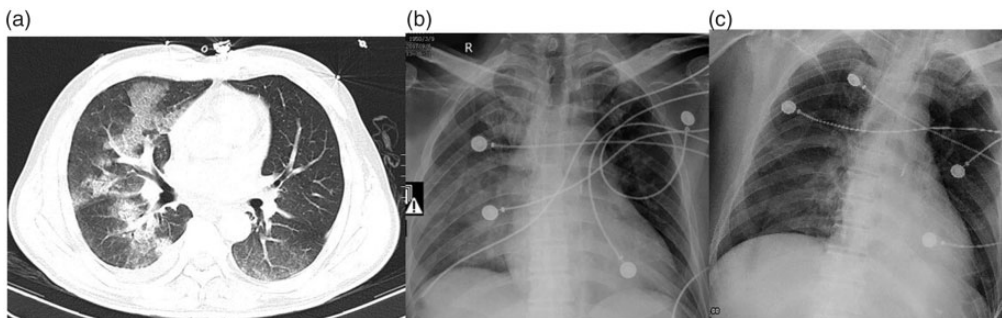
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caused by mild-to-severe mitral regurgitation, which commonly occurs secondary to acute coronary artery disease.<sup>2,3</sup> This current case report describes a patient with unilateral pulmonary oedema secondary to acute severe myocarditis without any evidence of mild-to-severe mitral regurgitation on ultrasound.

## Case report

On 5 September 2017, a 67-year-old male patient with hypertension for 10 years was brought to the emergency department of Suzhou Hospital Affiliated to Nanjing Medical University with cough and dyspnoea persistent for 2 days, accompanied by a fever of 38°C. His initial vital signs were as follows: heart rate was 152 beats per min, respiratory rate was 40 breaths per min, blood pressure was 90/56 mmHg and body temperature was 37.8°C. His oxygen saturation was 90% with a nasal cannula oxygen rate of 3 l/min. There were crackles on both lower sides of the lungs and gallop on auscultation. The laboratory examination revealed a white blood cell count of  $23.3 \times 10^9/l$ , brain natriuretic peptide (BNP) was 3070 ng/l, troponin I was 15.4 µg/l and procalcitonin was

0.1 ng/ml. The arterial blood gas analysis (ABG) demonstrated the following: an oxygen partial pressure of 63 mmHg, carbon-oxidation partial pressure of 28 mmHg, lactic acid of 3.6 mmol/l and residual alkali of 17 mmol/l. The electrocardiogram showed ST segment depression and T wave inversion in the II/III/AVF and V4–V6 leads. He was suspected of acute myocardial infraction, but there were no obvious abnormalities in subsequent coronary angiography (CAG). However, bedside echocardiography revealed a slightly dilated left atrium, mild mitral regurgitation and decreased left ventricular motion with an ejection fraction of 22%. The patient initially had a fever of nearly 38°C, followed by hypoxia, dyspnoea, crackles in both lungs and gallop rhythm, and the laboratory tests revealed myocardial damage, but there was no typical evidence of pulmonary embolism or Tako-Tsubo cardiomyopathy according to echocardiography or CAG. As a consequence of these findings, he was initially diagnosed with acute myocarditis and cardiogenic shock. He was admitted to the intensive care unit immediately. Chest computed tomography (CT) showed infiltration mainly in the right lung (Figure 1a).



**Figure 1.** Representative imaging of a 67-year-old male patient with hypertension for 10 years that presented with cough and dyspnoea persistent for 2 days, accompanied by a fever of 38°C: (a) chest computed tomography showed infiltration mainly in the right lung; (b) bedside chest radiography demonstrated increasing consolidated exudation in the mid and lower right lobe of the lung; (c) the right lung infiltration resolved after treatment as demonstrated by the bedside chest radiography.

The patient subsequently accepted basic oxygen therapy support and an intra-aortic balloon pump (IABP) in the right femoral artery in addition to other managements to maintaining haemodynamic stability. However, the patient was suffering from persistent tachycardia, dyspnoea and refractory hypotension, despite high-dose norepinephrine and fluid resuscitation. Finally, mechanical ventilation was undertaken after tracheal intubation. In addition, antibiotics were administered to the patient because it was difficult to rule out infectious diseases based on the chest CT imaging. The patient's condition did not improve significantly. Veno-arterial extracorporeal membrane oxygenation (ECMO) was administered via the left femoral artery and the right femoral vein. The initial parameters were as follows: the rotary speed was 2500 rpm, the blood flow rate was 2.5 l/min and the ratio of sweep gas flow to the blood flow was 1:1, 100% oxygen. On the fourth day, the patient's oxygenation index suddenly deteriorated, meanwhile the ABG suggested type II respiratory failure. The attending physicians hesitated to increase the anti-infection treatment, but the bedside chest radiography identified much more exudation in the middle and lower lung on the right side (Figure 1b). An electronic bronchoscope was performed to collect the alveolar lavage fluid for examination. However, only increasing white foamy sputum in the right airway was observed. After consultation between the treating physicians, the patient was diagnosed with unilateral pulmonary oedema that might have been caused by overload fluid for resuscitation at the beginning. Oliguria due to acute kidney injury could have been another reason. Subsequently, continuous renal replacement treatment was undertaken to optimize the volume and then the infiltration on the right side of the lung disappeared on the seventh day, which was

confirmed by the bedside radiography (Figure 1c). ECMO was removed on the eighth day and the patient was discharged from hospital on day 14.

The reporting of this case report conforms with the CARE guidelines.<sup>4</sup> The patient and his family provided consent for all treatments received.

## Discussion

According to clinical reports, unilateral pulmonary oedema accounts for 2% of congestive heart failure and 1.4% of those that received mitral valve replacement.<sup>1,5</sup> Unilateral pulmonary oedema does not have the typical imaging features of bilateral pulmonary oedema, which demonstrates symmetrical infiltration along the hilum of lung, called the "butterfly shadow". Therefore, unilateral pulmonary oedema is often initially misdiagnosed as other diseases, such as pneumonia or alveolar haemorrhage, which leads to inappropriate treatment.<sup>6</sup> Upon hospital admission, the current patient was treated with antibiotics because of his laboratory test results, the presence of pyrexia and his CT features, which were all suggestive of pneumonia. However, the antibiotics did not lead to any clinical improvement. The bedside chest radiography demonstrated a ground glass shadow on the right lung. An electronic bronchoscope provided a clue because there was increasing white foamy sputum in the right airway and bacteriological analyses were negative. Those signs strongly suggested to the treating physicians that he had unilateral pulmonary oedema.

Most cardiogenic unilateral pulmonary oedemas present in the right lung, especially the upper lobe.<sup>2,7</sup> The pathogenesis is not completely clear, but moderate or severe mitral regurgitation is considered to be a key factor.<sup>8-10</sup> When eccentric blood flow jets toward the right superior pulmonary vein, this leads to a predilection for

oedema formation in the right upper lobe.<sup>11,12</sup> However, there were no similar representations on the current patient's echocardiography. Previous research has found that transient mitral regurgitation secondary to acute coronary artery disease may cause unilateral pulmonary oedema in patients without severe mitral regurgitation.<sup>6,13</sup> It would appear that the current patient's myocardial contractility was initially weak, as the echocardiography showed, which induced an increased left heart volume and pressure. With the help of ECMO and other therapies, the contraction of the left ventricle improved. However, the mitral valve's function, which was caused by ischaemia, was not correspondingly improved, resulting in mitral regurgitation. Unfortunately, bedside echocardiography cannot provide a clearer image, but the treating physicians were unable to undertake a transoesophageal ultrasound or valvular angiography in the emergency department.

Other possible causes of unilateral pulmonary oedema include increased pulmonary capillary permeability and elevated hydrostatic pressure, which are similar to that of bilateral pulmonary oedema.<sup>14</sup> Some researchers found that the permeability change caused by ischaemia–reperfusion injury played an important role in the occurrence of unilateral pulmonary oedema in patients with rapid recruitment of the lung, such as pneumothorax and pleural effusion.<sup>15–17</sup> Elevated hydrostatic pressure of the pulmonary capillaries might also contribute to unilateral pulmonary oedema. Some conditions, such as pulmonary embolism, left ventricular pseudo aneurism, left atrial myxoma, aortic dissection and Swyer–James syndrome, might express or impede the flow of the ipsilateral or contralateral pulmonary vessels, leading to escalation of intravascular pressure, and eventually causing unilateral pulmonary oedema.<sup>18,19</sup> The current patient's bedside

echocardiography revealed enlargement of the left atrium, which contributed to the rise in the left atrial pressure and the pulmonary venous pressure. Unfortunately, pulmonary artery catheter or Swan-Ganz catheter were not undertaken in the current case to evaluate the pressure of the pulmonary circulation. Some researchers believe that the lymphatic drainage of the right lung is less than that of the left because of the large calibre of the thoracic duct.<sup>1,19,20</sup> This may have also contributed to the unilateral pulmonary oedema in the current patient.

To the best of our knowledge, there are no reports of unilateral pulmonary oedema caused by myocarditis, especially describing patients helped by the administration of ECMO. ECMO is an important cardiopulmonary support technique that is applied to save patients with severe pulmonary embolism, cardiac arrest, systemic inflammatory response syndrome and other cardiopulmonary failure complications.<sup>21</sup> However, the establishment of ECMO may often need more liquid to initially maintain haemodynamic stability, as in the current patient. The overloading of fluid might contribute to the occurrence of unilateral pulmonary oedema in those with poor heart function. Elevated BNP and pro-BNP levels might help to differentiate cardiogenic unilateral pulmonary oedema from other infectious conditions,<sup>22</sup> and thus provide a better prognosis. For the current patient, the initial BNP level did not suggest unilateral pulmonary oedema, because his primary disease and acute kidney injury can cause an elevation in BNP. However, the elevated BNP level should have caught the attention of the treating physicians. Moreover, continuous haemodynamic monitoring would have helped in the early detection of changes in the pulmonary circulation pressure, so that unilateral pulmonary oedema could have been distinguished from other comorbidities.<sup>23</sup> Unfortunately, pulse

indicator continuous cardiac output (or pulse index continuous cardiac output) was not established because both of the current patient's femoral arteries were occupied by ECMO and IABP catheters. There was also no opportunity to install a Swan-Ganz catheter.

The infiltration of the patient's right lung resolved quickly after managing the preload. Echocardiography confirmed better kinesis of the whole heart and there was still no obvious evidence of mitral regurgitation.

In conclusion, cases of unilateral pulmonary oedema that are associated with acute myocarditis are extremely rare and their pathophysiology remains unclear. Physicians must be aware of the need for rapid diagnosis and timely therapy in order to avoid mortality and morbidity.

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### Declaration of conflicting interest


The authors declare that there are no conflicts of interest.

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### Reference

1. Attias D, Mansencal N, Auvert B, et al. Prevalence, characteristics, and outcomes of patients presenting with cardiogenic unilateral pulmonary edema. *Circulation* 2010; 122: 1109–1115.

2. Schnyder PA, Sarraj AM, Duvoisin BE, et al. Pulmonary edema associated with mitral regurgitation: prevalence of predominant involvement of the right upper lobe. *AJR Am J Roentgenol* 1993; 161: 33–36.
3. Birnbaum Y, Chamoun AJ, Conti VR, et al. Mitral regurgitation following acute myocardial infarction. *Coron Artery Dis* 2002; 13: 337–344.
4. Gagnier JJ, Kienle G, Altman DG, et al. The CARE guidelines: consensus-based clinical case reporting guideline development. *Headache* 2013; 53: 1541–1547.
5. Moss E, Halkos ME, Binongo JN, et al. Prevention of unilateral pulmonary edema complicating robotic mitral valve operations. *Ann Thorac Surg* 2017 103: 98–104.
6. Shin JH, Kim SH, Park J, et al. Unilateral pulmonary edema: a rare presentation of cardiogenic shock due to acute myocardial infarction. *J Korean Med Sci* 2012; 27: 211–214.
7. Gudinchet F, Rodoni P, Sarraj A, et al. Pulmonary oedema associated with mitral regurgitation: prevalence of predominant right upper lobe involvement in children. *Pediatr Radiol* 1998; 28: 260–262.
8. Gurney JW and Goodman LR. Pulmonary edema localized in the right upper lobe accompanying mitral regurgitation. *Radiology* 1989; 171: 397–399.
9. Pena C, Jaquet M, Salgado J, et al. Asymmetric pulmonary perfusion causing unilateral pulmonary edema as a complication of acute myocardial infarction. *Rev Esp Cardiol* 2005; 58: 875–877 [Article in Spanish, English abstract].
10. Agarwal R, Aggarwal AN and Gupta D. Other causes of unilateral pulmonary edema. *Am J Emerg Med* 2007; 25: 129–131.
11. Young AL, Langston CS, Schiffman RL, et al. Mitral valve regurgitation causing right upper lobe pulmonary edema. *Tex Heart Inst J* 2001; 28: 53–56.
12. McNinch RW and McNamara MJ. An unusual cause of right upper- and mid-zone infiltrates on chest x-ray. *Crit Care Resusc* 2007; 9: 256–258.
13. Stingo FE, Sallam T, Govindu R, et al. It Is Not Pneumonia! A Case of Unilateral

- Pulmonary Edema. *Am J Med* 2020; 134: e38–e39.
14. MacIver DH and Clark AL. The vital role of the right ventricle in the pathogenesis of acute pulmonary edema. *Am J Cardiol* 2015; 115: 992–1000.
  15. Papakonstantinou DK, Gatziooufas ZI, Tzegas GI, et al. Unilateral pulmonary oedema due to lung re-expansion following pleurocentesis for spontaneous pneumothorax. The role of non-invasive continuous positive airway pressure ventilation. *Int J Cardiol* 2007; 114: 398–400.
  16. Childress ME, Moy G and Mottram M. Unilateral pulmonary edema resulting from treatment of spontaneous pneumothorax. *Am Rev Respir Dis* 1971; 104: 119–121.
  17. Mayeur N, Groyer S, Vimeux S, et al. Severe Respiratory and Hemodynamic Failures following Successful Spontaneous Pneumothorax Drainage. *Case Rep Emerg Med* 2021; 2021: 6677656.
  18. Inotani S, Kubokawa SI, Nakaoka Y, et al. Unilateral cardiogenic pulmonary edema. *J Cardiol Cases* 2018; 17: 85–88.
  19. Handagala R, Ralapanawa U and Jayalath T. Unilateral pulmonary edema: a case report and review of the literature. *J Med Case Rep* 2018; 12: 219.
  20. Hirata K, Ishimine T, Nakayama I, et al. Unilateral Left Pulmonary edema caused by contained rupture of the ascending aortic dissection. *Intern Med* 2021; 60: 751–753.
  21. Richardson ASC, Tonna JE, Nanjaya V, et al. Extracorporeal Cardiopulmonary Resuscitation in Adults. Interim Guideline Consensus Statement From the Extracorporeal Life Support Organization. *ASAIO J* 2021; 67: 221–228.
  22. Ray P, Arthaud M, Birolleau S, et al. Comparison of brain natriuretic peptide and probrain natriuretic peptide in the diagnosis of cardiogenic pulmonary edema in patients aged 65 and older. *J Am Geriatr Soc* 2005; 53: 643–648.
  23. Jiang X, Cheng X and Zhang W. Unilateral cardiogenic pulmonary edema caused by acute mitral valve prolapse: a case report. *Medicine* 2021; 100: e24622.