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CASE REPORT

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Bedside critical ultrasound as a key to the diagnosis of obstructive atelectasis complicated with acute cor pulmonale and differentiation from pulmonary embolism: A case report

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Abstract

Acute attack of dyspnea may be combined with acute cor pulmonale (ACP). Rapid and accurate identification of the etiology of ACP is the key to its diagnosis and treatment. Echocardiography is a better imaging tool in the assessment of right ventricular function. Under the guidance of the theory of cardiopulmonary interaction, ultrasonography can detect lung lesions, which causes ACP. We report the case of a 67-year-old man who received mechanical ventilation for acute respiratory failure. Right ventricular dysfunction was detected by echocardiography. Lung ultrasound showed a high risk of pulmonary embolism. However, obstructive atelectasis should not be ruled out after increasing back area ultrasonography. To avoid pitfalls, combined cardiac and lung ultrasound should be used carefully and strictly.

KEYWORDS
acute cor pulmonale, atelectasis, consolidation, critical ultrasound

1 | INTRODUCTION

The role of critical ultrasound in the emergency department and intensive care unit (ICU) is increasing.[1] As the most important part of critical ultrasound, lung ultrasound and echocardiography have accurate and rapid values in the etiological diagnosis of patients with acute dyspnea and acute hemodynamic disorders.[2,3] Based on the pathophysiology and hemodynamics of cardiopulmonary disease, we found that the effects of heart and lung function are complementary.[4] Knowledge on cardiopulmonary interactions may contribute to a better understanding of the disease. Ultrasound is "the third eye" of a clinician. The combination of cardiopulmonary interaction theory, echocardiography, and lung ultrasound technology is known as combined cardiac and lung ultrasound (CLUS), which can be used to explore the etiology of acute dyspnea or circulatory failure.

We herein report the case of a patient with sudden acute respiratory failure who was admitted to the emergency department and initially considered to have acute cor pulmonale (ACP) caused by pulmonary embolism (PE) on CLUS. However, after further lung ultrasound examination of the back lobe of the patient, obstructive pulmonary atelectasis could not be excluded, as confirmed by chest computed tomography (CT).

Our ultrasound examination is a routine examination for patients with emergency and critical cases in our hospital, which was performed after the request of emergency physicians and patients' families. Informed consent was obtained from patients' families.

TABLE 1 Echocardiographic and lung ultrasound features in patients with acute respiratory failure

	Size and function	Qualitative findings and significance
Inferior vena cava(IVC)	IVC diameter-max: 23.1 mm IVC diameter-min: 22.2 mm Variation of IVC: 3.9%	IVC dilation and fixation
Left heart	LA: 35 mm LV: 38 mm LA area: 12.0cm^2 LVEF: 67% *E*: 54 cm/s *A*: 70 cm/s *e′*: 4.08 cm/s	No expansion of LA and LV; On the contrary, LV "D-shape" appears due to interventricular septal compression
Right heart	RA: 45 mm RV: 44 mm RVEDA/LVEDA >1 TAPSE: 12.4 mm	RA and RV dilated; RV movement decreased; RV pressure increases and compresses the LV through the interventricular septum
Pulmonary artery	Main PA: 31 mm Left PA: 20 mm Right PA: 20 mm PASP: 85 mmHg	Pulmonary artery slightly dilated
Lung lobe	A-lines of anterior chest wall and lateral chest wall, and only left PLAPS point find B-lines	Only focal pneumonia in the left lung; Most of the lung areas are normally ventilated
pleura sliding	The pleura sliding of the left side was weaker than the right side	Decreased left lung activity
Lower limb deep veins	Thrombosis in left fibular vein and bilateral calf intermuscular vein	Venous thrombosis of deep venous

Abbreviations: *A*, late diastolic peak velocity of mitral flow; *e′*, peak velocity of early diastolic mitral annulus motion; *E*, early diastolic peak velocity of mitral flow; IVC, inferior vena cava; LA, left atrium; LV, Left ventricle; LVEDA, LV end-diastolic area; LVEF, LV ejection fraction; PA, pulmonary artery; PASP, pulmonary artery systolic pressure; PLAPS, Posterior lateral alveolar pleura syndrome; RA, right atrium; RV, right ventricle; RVEDA, RV end-diastolic area; TAPSE, tricuspid annular plane systolic excursion.

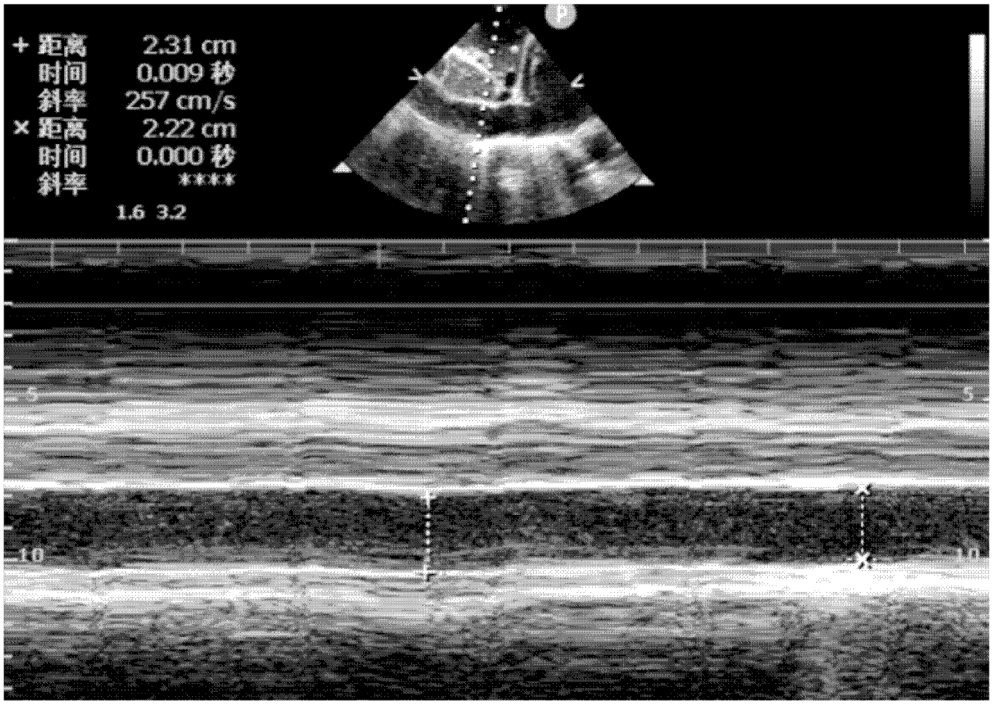

FIGURE 1 Inferior vena cava dilation and fixation, it is suggested that venous return does not match cardiac function

2 | CASE PRESENTATION

A 67-year-old man was admitted to our emergency department with strenuous breathing and wheezing. Rapid physical examination and a series of laboratory examinations were performed upon admission and the following were noted: temperature, 36.4 °C; respiratory rate, 33 bpm; heart rate, 111 bpm; and blood pressure, 106/71 mmHg. Previous systolic blood pressure was 150 mmHg. The results of blood gas analysis were as follows: pH, 7.33; PaO_2, 38.5 mmHg; $PaCO_2$, 105.2 mmHg; pulse oxygen saturation (SpO_2), 61%; oxygenation index (PaO_2/FiO_2), 183 mmHg; lactic acid, 2.6 mmol/L. Other important results were as follows: B-type natriuretic peptide, >9000 pg/mL; D-dimer, 2.56 mg/L FEU; creatinine, 89 μmol/L; glucose, 5.43 mmol/L; white blood cells, 3.75 $\times 10^9$/L; creatine kinase isoenzyme MB, 10.3 U/L; and troponin T, <40 ng/L. No myocardial infarction was found on the bedside electrocardiography.

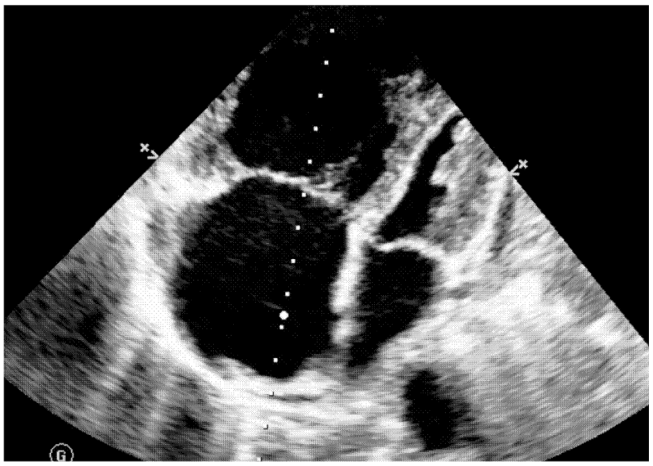

FIGURE 2 Obvious expansion of right heart can be found form the apical four chamber view

Acute left heart failure or PE was considered as the most likely cause of acute respiratory failure in patients by emergency physicians. Therefore, they invited us to perform an ultrasound examination. We arrived in 5 min and scanned the major organs. After the ultrasound results were integrated (Table 1), we described and analyzed them according to the path of blood circulation.

Through CLUS, we captured the following five important information: First, we found that the inferior vena cava (IVC) was dilated and fixed, indicating that the current cardiac function could not accommodate excessive venous return, and the two did not match (Figure 1). Second, right ventricle (RV) dilatation and systolic dysfunction indicated that the RV afterload may have significantly increased (Figure 2). Furthermore, a dilated pulmonary artery and increased pulmonary artery pressure indicated high pulmonary circulation resistance. Lung

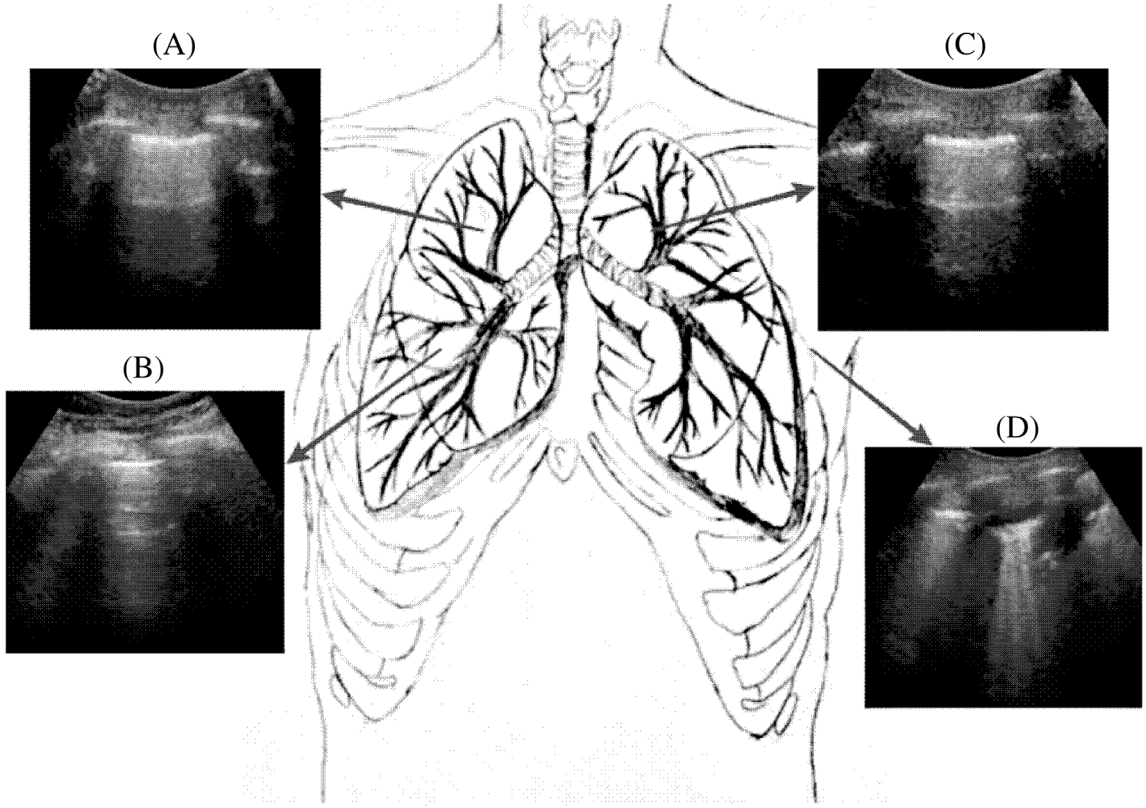

FIGURE 3 Lung ultrasound findings: (A) A-lines in right upper BLUE point. (B) A-lines in right lower BLUE point. (C) A-lines in left upper BLUE point. (D) B-lines in left PLAPS point

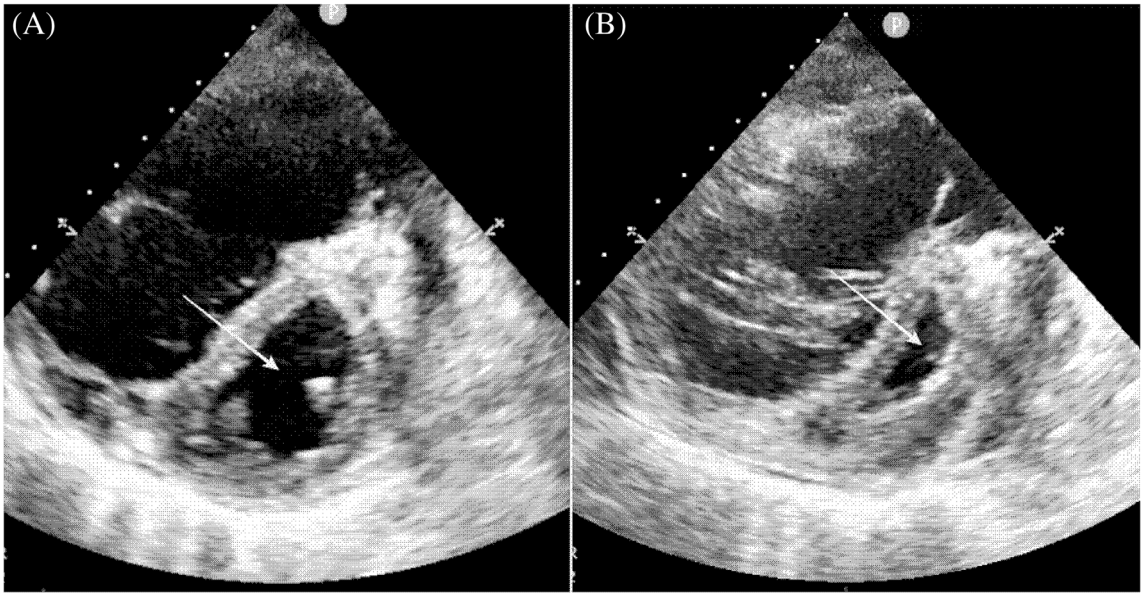

FIGURE 4 (A) diastolic phase of the heart. (B) systolic phase of the heart. The LV was compressed by the enlarged RV to form "D-shaped LV"(the two white arrow). "D-shaped LV" is more evident in systole than in diastole, which indicates that the afterload of RV (i.e., pulmonary vascular resistance) is higher

ultrasound examination based on bedside lung ultrasound in emergency (BLUE) protocol showed A-lines in the upper BLUE point, lower BLUE point, phrenic point of the bilateral lung, and posterior lateral alveolar pleura syndrome (PLAPS) point of the right lung, and only a few B-lines in the PLAPS point of the left lung (Figure 3). This result can exclude cardiogenic pulmonary edema.[5] Finally, the left atrium and left ventricle (LV) were not dilated, and the LV volume was smaller after compression by the dilated RV (Figure 4). The LV systolic function was normal. The diagnosis of LV diastolic dysfunction was insufficient (only two criteria for the diagnosis of LV diastolic function were met). Moreover, no abnormalities were found in the LV outflow tract.

According to the abovementioned ultrasonographic findings, no primary problems were found in the left heart. Therefore, we considered that pulmonary circulation dysfunction caused by lung disease led to right heart dysfunction. ACP can also be diagnosed.[6] According to the BLUE protocol,[5] although B-lines present at the left PLAPS point indicate that focal pneumonia should be diagnosed, such small-scale and limited B-lines were not consistent with the degree of respiratory failure and could not cause severe changes in RV function. At this time, we found a thrombus in the left fibular vein and bilateral calf intermuscular veins. Therefore, PE was more likely. We conducted detailed communication of the ultrasound findings with emergency physicians and recommended them to perform chest CT angiography (CTA). Emergency physicians contacted the imaging department immediately and prepared to perform anticoagulant or thrombolytic therapy.

However, at this time, a detail has attracted our attention: the pleura sliding at the position of axillary midline of both sides of the patient was inconsistent, and the left side was significantly weaker

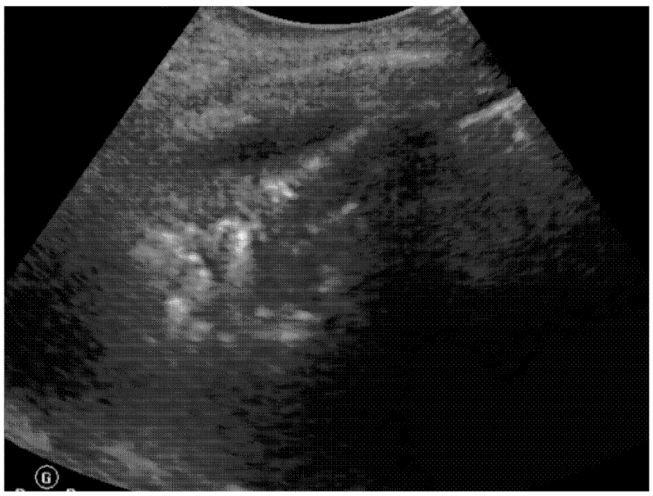

FIGURE 5 An additional scan of the left back area revealed lung consolidation with "air bronchogram", and no pleural effusion, indicating atelectasis

than the right side. In view of this phenomenon, it was also found that breath sounds in the left lung were fewer than those in the right lung by auscultation. We asked the family members whether the patient had a history of diaphragmatic injury caused by trauma, surgery, or central nervous system-related diseases, and all these were denied by them.

Therefore, we asked the emergency physician to try to turn over the patient to the right side recumbent, to check the back lobe of the lung. A large number of consolidation signs were found on the left side of the back near the spinal column, and "air bronchogram" (i.e.,

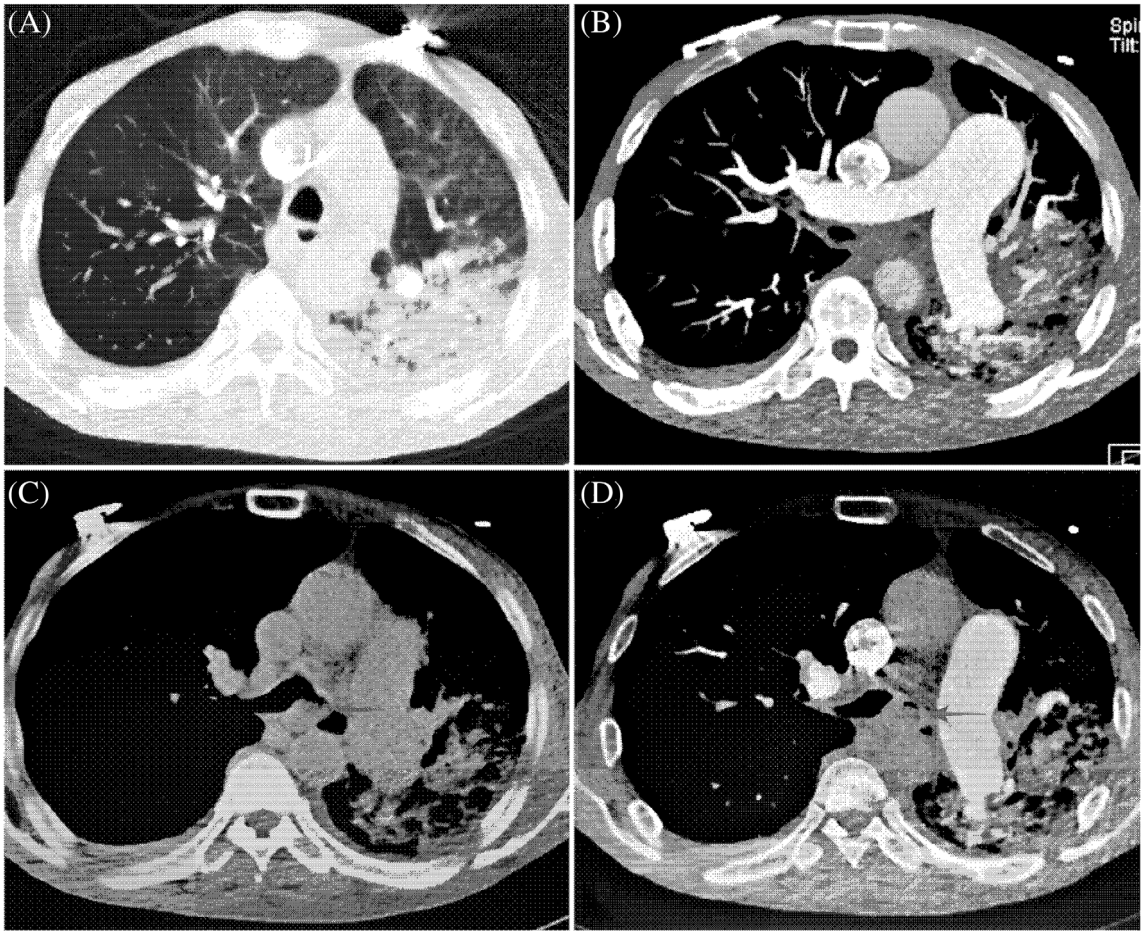

FIGURE 6 (A) Inflammatory focus in pulmonary window of chest CT. (B) No sign of PE in multislice helical CTA. (C,D) secretion obstruction (sputum mass)in bronchial in mediastinal window of chest CT

the bronchus is imaged by ultrasound because of internal gas absorption) was observed[7](Figure 5). This is a characteristic manifestation of obstructive pulmonary atelectasis.

The emergency physician prepared for bronchoscopy, but the patient's oxygen saturation was low and noninvasive ventilation was not possible, which did not meet the requirements of bronchoscopy. After slight improvement in breathing, the patients were transferred to the imaging department for CT examination accompanied by nurses using an oxygen inhalation device.

In ~1 h, the results of chest CT and CTA (Figure 6) showed the following: (1) There was no sign of PE in the bilateral pulmonary artery on CTA. (2) The left main bronchus was blocked by a sputum mass, indicating infection and atelectasis of the left lung. (3) A small amount of pleural effusion was observed on the left side.

Emergency physicians immediately discontinued the original therapeutic plan of anticoagulation and thrombolytic therapy and performed measures such as sputum suction, anti-infection therapy, and volume reduction. Blood gas analysis was performed again: pH, 7.30; PaO_2, 67.1 mmHg; $PaCO_2$, 122.5 mmHg; SpO_2, 92%; and lactic acid level, 1.7 mmol/L. Although the SpO_2 of patients increased (61–92%), airway obstruction was not relieved, and dyspnea persisted. We suggest that patients receive endotracheal intubation and transfer to the ICU for further treatment, but their family members refused. They asked to be returned to their local hospital for treatment.

3 | DISCUSSION

Lung ultrasound is a rapid diagnostic tool for acute and critical diseases and has the advantages of being fast, accurate, and convenient at the bedside.[1] As a diagnostic method for lung diseases, the accuracy of the BLUE protocol for acute dyspnea is as high as 90.5%.[5] Cardiopulmonary interaction is described as the relationship between cardiac function, respiratory function, and blood volume.[4] LV systolic or diastolic dysfunction can increase the pulmonary capillary hydrostatic pressure, leading to pulmonary edema.[8] In contrast, some serious pulmonary diseases (e.g., PE and ARDS) may cause changes in cardiac function due to the destruction of pulmonary vessels, especially the RV.[6] The enlarged RV can cause the LV to become smaller by compressing the interventricular septum, resulting in LV diastolic limitation. "D-shaped LV" is the characteristic manifestation of increased RV preload or afterload.[9] At this point, even if the LV

ejection fraction is normal, cardiac output still cannot meet the needs of maintaining blood pressure and tissue perfusion. IVC dilatation and fixation are associated with high central venous pressure, suggesting that cardiac function and venous return do not match.[10] In daily practice, echocardiography and lung ultrasound cannot be performed. Under the guidance of the theory of cardiopulmonary interaction, CLUS examination can evaluate respiratory function and hemodynamic status of patients more accurately.

This case involved a patient with sudden dyspnea. Blood gas analysis showed that the oxygen saturation and oxygenation indices were significantly lower than normal. The patient received noninvasive ventilation, but the treatment was unsatisfactory. According to our ultrasound results, the right heart function was severely impaired. The change in the left heart originates from the right heart rather than the primary heart. Therefore, the diagnosis of ACP is clear. Further scanning of the lung ultrasound revealed that B-lines can only be observed at the left PLAPS point, and focal pneumonia should be diagnosed. However, we considered that these limited B-lines did not match the current severe respiratory failure and RV dysfunction. Most lung areas showed A-lines, and thrombosis was found in the veins of both lower limbs, which seemed to be more consistent with the diagnosis of PE. PE cannot be detected by lung ultrasound because embolism in the arterioles and capillaries of the lungs is hardly detected by ultrasound.[11] The diagnostic procedures of PE in the BLUE protocol were as follows: (1) normal pleural sliding, (2) a profile of both lungs, and (3) thrombosis in DVT.[5] PE can also be indirectly indicated through echocardiographic changes. The increased afterload results in RV deformation, which can be obtained by measuring the size of the RV and evaluating the RV function.[12] Moreover, pulmonary hypertension and IVC dilatation are also suggestive of PE.

However, a detail that caught our attention was that the pleura sliding on the left side was weaker than on the right side. We need to exclude the possibility of diaphragmatic dysfunction.[13] In this case, a previous history of diaphragmatic dysfunction in the patient had been denied by family members. Therefore, we further examined the lobe of the left back. The patient was turned over with the help of an emergency physician, and a large area of consolidation and atelectasis was found in the left back lobe (near the spine) by lung ultrasound.

It is worth mentioning that there was no pleural effusion at this location, so lung consolidation was not considered to be caused by pleural effusion. Therefore, atelectasis was the most likely diagnosis.

Finally, atelectasis was confirmed by chest CTA, and PE was excluded. This result confirms our conjecture: obstructive atelectasis. The patient's left bronchus was blocked by a sputum mass, which resulted in the obstruction of inspiration in the left lung. The gas in the alveoli was gradually absorbed, leading to atelectasis. The pulmonary vessels were affected and destroyed when the alveoli collapsed completely, leading to ACP. The loss of alveolar ventilation in the left lung and arteriovenous shunts may be the cause of respiratory failure in the patient.

When most of the gas in the alveoli is absorbed, the lung tissue can be developed using ultrasound, known as lung consolidation.[14] Similarly, when the endobronchial gas is absorbed, "air bronchogram" may appear on ultrasound.[7] When pleural effusion does not exist, the presence of lung consolidation and air bronchogram is suggestive of atelectasis.

The consolidation in this case occurred on the lobes of the back; therefore, lung ultrasound was performed in the supine position, which may have resulted in the missed diagnosis. This reinforces the need to scan the back area as much as possible when performing a lung ultrasound. Moreover, according to the records of the ultrasound machine, it took ~7 min to perform CLUS. However, transferring patients to the CT room takes more time and has transport risks, such as respiratory failure, noninvasive ventilation, and potential hemodynamic disorders.

4 | CONCLUSION

Many types of pulmonary diseases can cause ACP. According to the traditional lung ultrasound diagnosis scheme, the lesions in the back area may be missed, which may lead to misdiagnosis as PE and incorrect guidance of treatment. The disease of the back lobes should not be ignored. The back area should not be ignored in lung ultrasound examination. CLUS can further reveal the etiology of ACP while identifying right heart dysfunction.

CLUS examination is a convenient and effective diagnostic tool for patients with respiratory or circulatory dysfunction. However, the ability to interpret ultrasound images and the differential diagnosis of diseases should be strictly studied and accurately mastered.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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