

Post-Diuretic Pulmonary Edema A Case Report

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Case report

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Abstract

Background Post-diuretic pulmonary edema (PDPE) is a special type of acute pulmonary edema, results from rapid blood volume loss caused by diuretics. Inappropriate administration of diuretics and inotropic agent, which is mainstay of treatment of pulmonary edema Classically, might have been a critical mistake in PDPE. Maintaining appropriate volume status and beta receptor blockers is the key to reversing the progress of PDPE. However, this condition remains unfamiliar to the medical community, making it an underdiagnosed and underreported condition. **Case representation** A 71-year-old patient who initially was diagnosed to have acute respiratory distress syndrome (ARDS) in intensive care unit (ICU) but got further dyspnea after rapid volume depletion. Point of care ultrasound (POCUS) examination showed worsened pulmonary edema with inferior vena cava collapsed during inspiratory period and a small but hyperdynamic left ventricular (LV). The patient subsequently improved with fluid, beta blocker, increased sedation, and paralysis. **Conclusion** We first put forward the concept of post-diuretic pulmonary edema, in order to remind medical staff to pay attention to this type of pulmonary edema and understand its pathophysiological mechanism.

Background

Although they have distinct etiologies, pulmonary edema may be difficult to distinguish because of their similar clinical manifestations, especially when they developed simultaneously. Diuresis is the most often used method for treating pulmonary edema. On the contrary, we reported a case of post-diuretic pulmonary edema (PDPE). PDPE is life-threatening if unrecognized timely and should be paid more alert on it. Point of care ultrasound is invaluable for accurate diagnosis of PDPE.

Case Presentation

A 71-year-old male presented to our hospital because of an incidentally discovered subpleural tubercle on right inferior lobe. Biopsy showed non-small cell lung carcinoma and had a right lower lobectomy under general anesthesia to remove the lesion. On the 5th day postoperative the patient was noted to be short of breath and dyspneic. On examination his heart rate was 122 beats per minute, respiratory rate 30 breaths per minute, and oxygen saturation 86 to 90% on 10 liters of oxygen by face mask. He was intubated and transferred to the ICU.

Upon ICU arrival, the patient was sedated with propofol, remifentanyl and mechanically ventilated using volume control mode, with low tidal volumes (6 ml per kilogram of predicted body weight), positive end-expiratory pressure (PEEP) of 7 cmH₂O, respiratory rate of 20 breaths per minute and the fraction of inspired oxygen (FiO₂) of 100%. With these settings, the patient continued to have respiratory distress. Physical examination revealed bilateral lung moist rale, normal heart sounds, and yellowish, viscous sputum in the endotracheal tube. His chest radiograph revealed bilateral diffuse and heterogeneous opacities (Fig. 1a).

Meeting the criteria of severe ADRS, he was treated with analgesia–deep sedation–neuromuscular blocking agents (NMBAs) and fluid restrictive management strategy. His respiratory status improved in the ensuing 5 days. Anteroposterior chest radiograph showed decreased pulmonary opacities (Fig.1b). Mechanical ventilation was weaned to a FiO_2 of 40%, respiratory rate of 20 breaths per minute and pressure support (PS) of 13 cmH_2O with PEEP of 5 cmH_2O . Cumulative fluid balance was negative about 3050ml during the ICU admission.

On the 6th day after ICU admission–the patient had increased sputum. On examination–the respiratory rate was 19 breaths per minute, oxygen saturation declined to 91% on mechanical ventilation with FiO_2 of 50% and his pulmonary exam demonstrated bilateral rales. A diagnosis of acute pulmonary edema was established and 40 mg of intravenous furosemide was delivered immediately. Fluid balance was negative about 970ml in the ensuing eight hours. However– the patient continued to have dyspnea and worsening hypoxemia. His respiratory rate was 31 breaths per minute, heart rate 129 beats per minute and oxygen saturation 91% on mechanical ventilation with FiO_2 of 70% and PS of 12 cmH_2O with PEEP of 5 cmH_2O . In addition–he was noted to be fever of 38.4°C.

Evaluation and Management

An immediate point-of-care-ultrasound (POCUS) showed a inferior vena cava (IVC) diameter was 0.92cm with inspiratory collapse(Fig.2a–video in supplementary file 1).Right ventricular(RV)/ LV end-diastolic area ratio was more than 0.6 in the apical four chamber view. The LV size was small (LV end-diastolic dimension, 33mm) with vigorous contraction (LV ejection fraction, 76%). Most notably, in systole–an aliasing of color flow doppler images was found across left ventricular outflow tract (LVOT) (Fig.3a left panel, video in supplementary file 3) and the maximal pressure gradient was 29mmHg estimated by a continuous-wave Doppler flow–Fig.3b left panel–implying increased resistance of LV ejection. Meanwhile, a paradoxical dynamic obstruction with a jet flow from the apex LV chamber to the basal in diastole was recognized–Fig.3c left panel, video in supplementary file 3–with an early diastolic mitral inflow velocity (E) of 1.1 meters per sec and an early diastolic mitral annulus velocity (e') at the lateral of 0.07 meters per sec(E/e' ratio 15.7)–implying that there is underlying LV diastolic dysfunction^[1]. Putting this all together, this shows that the left atrial pressure was elevated, and the LV filling was impaired. At this time, lung ultrasound evaluation showed increased B lines in both lungs and no A lines. This is consistent with a substantially increased amount of extra vascular lung water, despite diuresis. (Fig.1d)

Based on the result of the POCUS evaluation the low cardiac preload due to the rapid volume loss resulted in hyperdynamic LV contractions. This lead to increased resistance of LV ejection during systole and difficulty with LV filling during diastole. The increased LV end-diastolic pressure and left atrial pressure were then transmitted to the pulmonary capillaries and pushed fluid out of the vessels, which ultimate resulted in worsening of the patient's pulmonary edema. Although the pulmonary arterial wedge pressure estimated from E/e' was not high enough to induce hydrostatic pulmonary edema, the effect of hydrostatic pressure on the pulmonary edema was amplified by the high pulmonary capillary permeability due to ARDS and pneumonia. Eventually, the patient had worsening pulmonary edema and

worsening respiratory distress, which triggered negative pressure pulmonary edema (NPPE) attributed to the marked negative intrapleural pressure^[2-4].

If this mechanism is not recognized, then the typically treatment would involve extra diuresis and possibly vasodilators, inotropic agents, and increased mechanical ventilation support. Unfortunately, these treatment would result in worsening of the patient's suffer pulmonary edema.

Treatment

After determining that the patient required fluid despite pulmonary edema, the patient received fluid therapy, beta-blocker (intravenous esmolol), and increased sedation to mitigate LV stress. In order to eliminate the side effects of deep spontaneous breath, the patient was deeply sedated and paralyzed with NMBA (cisatracurium besylate, 5mg per hour). After 2 days, the fluid balance was positive about 1390ml. Meanwhile, the patient was stabilized again on mechanical ventilation requiring an FiO_2 of 45%, respiratory rate of 20 breaths per minute and PS of 12cmH₂O with PEEP of 6cmH₂O. Repeat POCUS showed the LV cavity enlarged and IVC diameter increased to 2.1cm (Fig.2b, video in supplementary file 2), with a laminar LVOT blood flow in systole (Fig.3a right panel) and a laminar LV cavity blood flow from base to apex in diastole (Fig.3c right panel) (video in supplementary file 4). The LVOT pressure gradient decreased to 9.0mmHg in systole (Fig.3b right panel) and the LV E/e' ratio decreased to 9. This indicated that the left atrial pressure decreased. Lung ultrasound examination showed decreased B lines compared to 48 hours before and A lines reappeared in both lungs. (Fig.1e)

Discussion

Diuresis is the most often used method for treating pulmonary edema. On the contrary, we first put forward the concept of post-diuretic pulmonary edema (PDPE).

Pulmonary edema results from increased hydrostatic pressure and increased permeability of the vessels^[5]. The general consensus in treating pulmonary edema is a restrictive fluid strategy in conjunction with diuresis. However if diuresis is too quick, hypovolemia will occur and result in the heart and the lung working harder to increase oxygen delivery. This will lead to a hyperdynamic LV and increased tidal volumes during spontaneous breathing. (Fig.4)

The hyperdynamic LV will increase LV end-diastolic pressures due to the elevated resistance of LV ejection in systole and LV filling in diastole. This increased resistance is transmitted to the pulmonary capillaries and will increase pulmonary capillary hydrostatic pressure which will worsen hydrostatic pulmonary edema. Particularly, this outcome can be aggravated in the condition of high pulmonary capillary permeability.

Deep and quick breaths from pulmonary edema will decrease thoracic pressure even to negative, which will worsen hydrostatic pulmonary edema by increasing pulmonary capillary hydrostatic pressure. Firstly, the negative thoracic pressure will increase RV output by drawing more venous blood return to RV^[3,4,6]

and will decrease LV output by elevating LV transmural pressure contributing to LV afterload increasing [7]. Together these two elements lead to pulmonary capillary hydrostatic pressure increased. Secondly, the negative thoracic pressure will further increase trans-pulmonary capillary pressure. The combination of all these elements in our patient resulted in worsening of hydrostatic pulmonary edema.

Negative thoracic pressure will also worsen pulmonary permeability edema by increasing transpulmonary pressure which will further exacerbate lung injury.

Putting this all together, our patient got worsening of pulmonary edema, despite volume depletion — Post-Diuretic Pulmonary Edema (PDPE).

Conclusions

PDPE is a potentially life-threatening, and under-recognized entity. A clear understanding of the pathophysiology of pulmonary edema and early recognition of interplay between the volume depletion and aggravated pulmonary edema are of paramount importance to make the diagnosis. POCUS is invaluable for accurate diagnosis of the unexpected PDPE.

Abbreviations

Post-diuretic pulmonary edema =PDPE; Acute respiratory distress syndrome =ADRS; Intensive care unit = ICU; Point of care ultrasound =POCUS; Left ventricular =LV; Positive end-expiratory pressure =PEEP; Fraction of inspired oxygen =FiO₂; Neuromuscular blocking agents =NMBAs; Pressure support =PS; Inferior vena cava =IVC; Right ventricular =RV; Left ventricular outflow tract =LVOT; Early diastolic mitral inflow velocity =E; Early diastolic mitral annulus velocity =e'; Negative pressure pulmonary edema =NPPE; Figure =Fig.

Declarations

Ethics approval and consent to participate

The manuscript was approved by the Ethical Committee of the fourth Hospital of Hebei Medical University. The participant signed an informed consent for information to be published.

Consent for publication

Written informed consent was obtained from the patient for publication of this article and any accompanying images and videos.

Availability of data and materials

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

L.L. and Q.Z. wrote this paper , contributed equally to this paper. T.Z., X.Wu, L.S. and B.L. as ward doctors participated in management of the patient. X.Wang and Y.C. and Z.H. reviewed and edited the manuscript. All authors read and approved the final manuscript.

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Figures

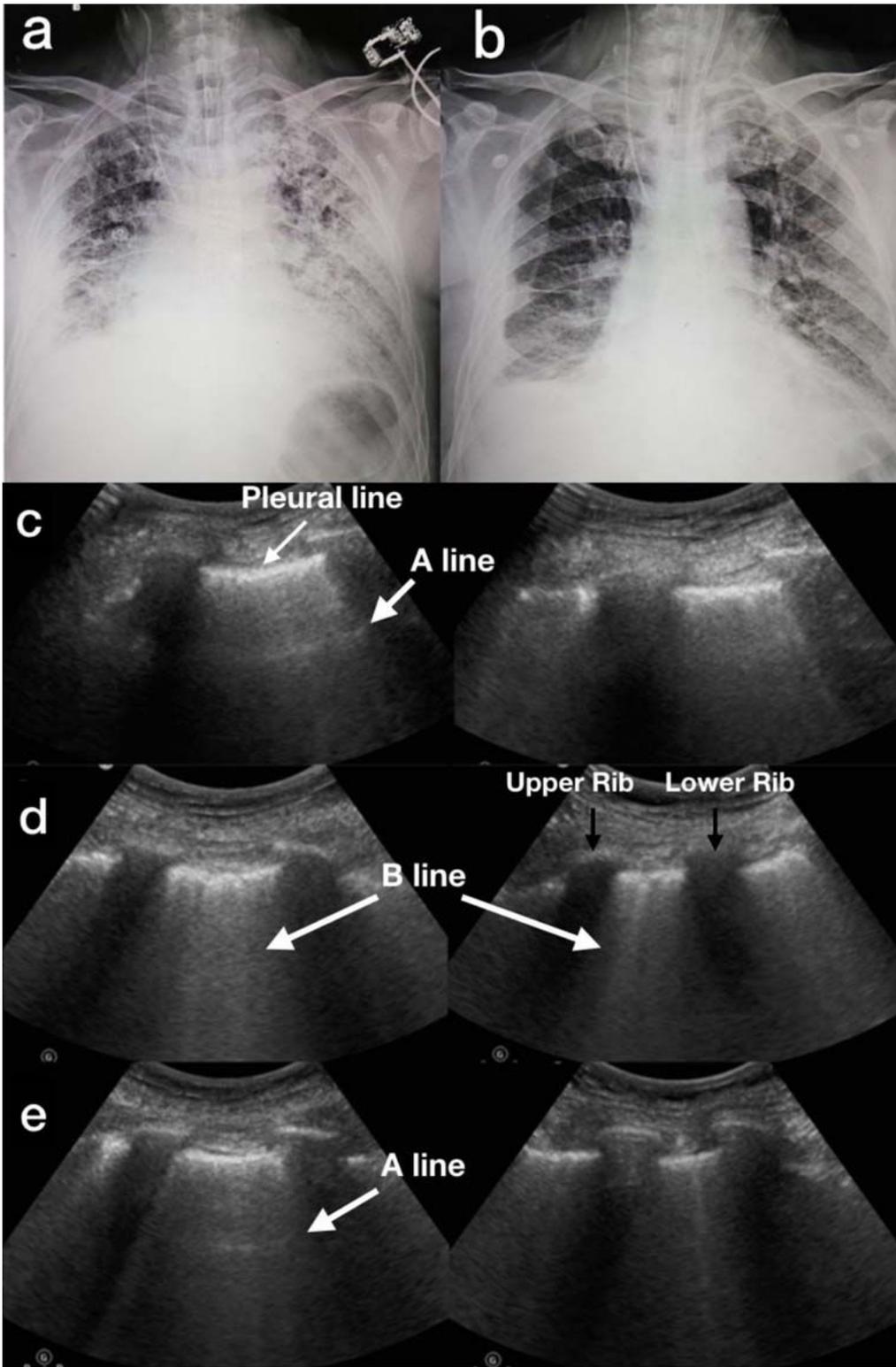


Figure 1

Imaging Studies of Chest X-ray and Lung Ultrasound. An anteroposterior chest radiograph showed bilateral diffuse and heterogeneous opacities on the first day of ICU admission (Panel A) And then opacities diminished markedly on 5th day after ICU admission (Panel B) with bilateral A-line on the anterior chest (Panel C, left panel) and a few B-line on the posterolateral chest wall (Panel C, right panel) evaluated by lung ultrasound examination. But one day later the examination showed marked increase

of B lines bilaterally and disappearance of A lines on anterior chest (Panel D—left panel: upper point—right panel—lower point). After 48 hours of treatment, a repeated lung ultrasound revealed marked decreased of B lines and A lines reappeared on anterior chest. (Panel E—left panel: upper point—right panel—lower point). (The A line—the horizontal lines arising from the pleural line are separated by regular intervals that are equal to the distance between the skin and the pleural line. The A-line indicates air. The B line—multiple vertical comet-tail artifacts arising from the pleural line are spreading to the edge of the screen without fading and moving with lung sliding. It reflects the coexistence of elements with a major acoustic impedance gradient, such as fluid and air. Several B lines indicate increasing pulmonary fluid.)

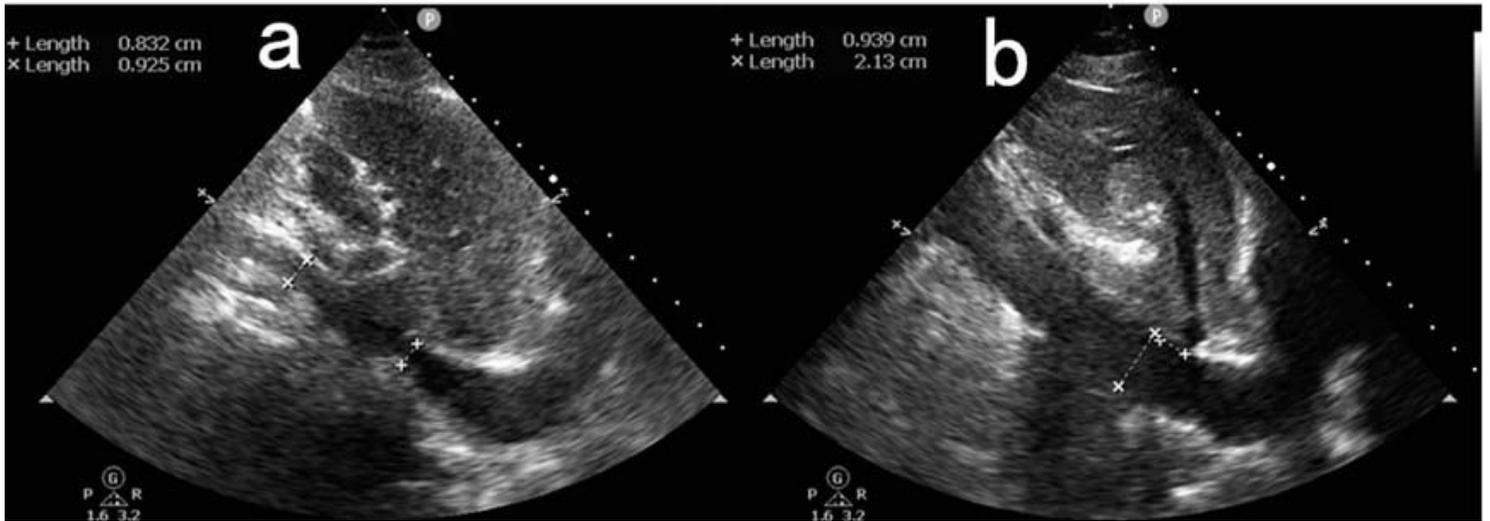


Figure 2

Inferior vena cava (IVC) diameter. IVC diameter was 0.92cm with inspiratory collapse(Panel A).(video in Supplement 1). At post-treatment, IVC diameter increased to 2.1cm(Panel B).(video in Supplement 2).

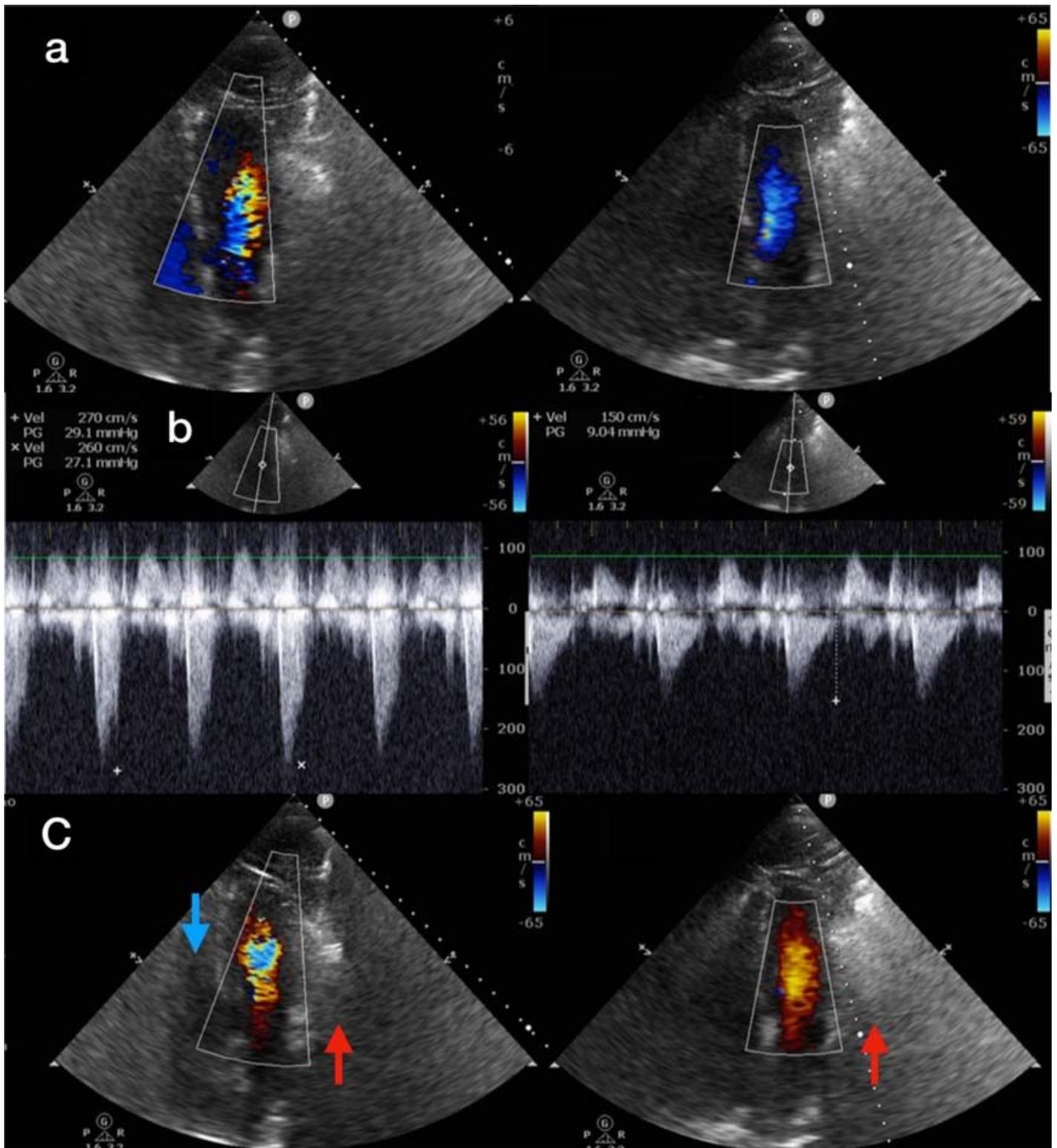


Figure 3

Echocardiographic imaging studies before [Left panel] and after treatment [Right panel]. In a small hyperdynamic LV cavity [an aliasing of color flow doppler images was found across LVOT [Panel A, left panel] with the maximal pressure gradient of 29mmHg estimated by a continuous-wave Doppler [Panel B, left panel] in systole, and a paradoxical dynamic obstruction with a jet flow from the apex LV chamber to the basal was recognized in diastole [Panel C, left panel] [video in Supplement 3]. At post-treatment [the

LV cavity enlarged with a laminar LVOT blood flow in systole (Panel A, right panel) and a laminar LV cavity blood flow from base to apex in diastole (Panel C, right panel) video in Supplement 4. Meanwhile the LVOT pressure gradient decreased to 9.0 mmHg (Panel B, right panel) in systole.

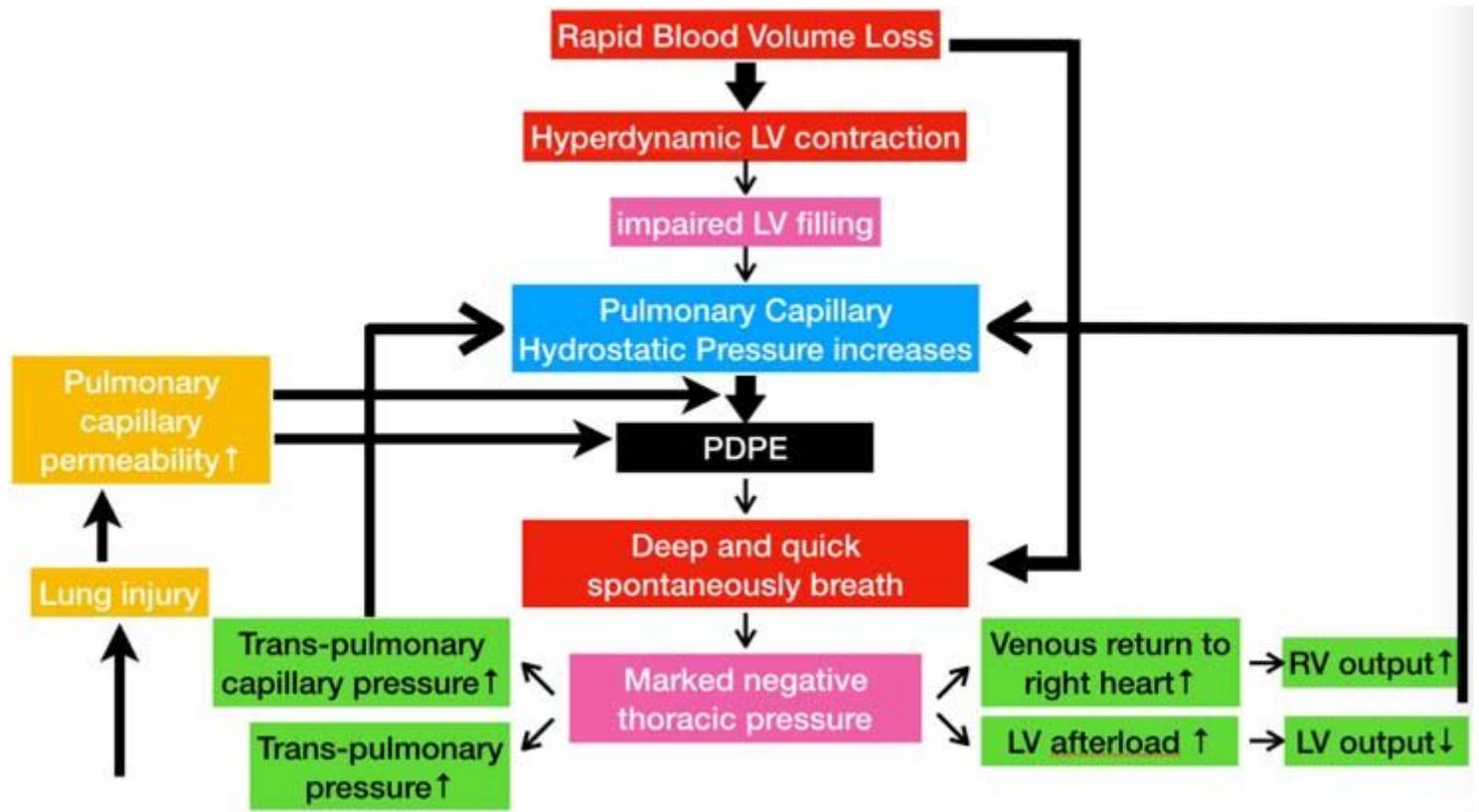


Figure 4

Pathophysiology of post-diuretic pulmonary edema (PDPE).

Supplementary Files

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