中文題目：一位以喘表現的單側肺水腫病人

英文題目：Dyspnea In A Patient With Unilateral Pulmonary Edema

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Case presentation:

This 50-year-old male driver with a history of gout and hyperlipidemia presented to emergent department (ED) because of progressed shortness of breath for 3 days.

He had fever, muscle soreness, rhinorrhea, productive cough with yellowish sputum in recent 3 days. Decreased appetite was also noted. His wife and son both caught cold recently. He denied chest pain, diarrhea, dysuria. Because progressive shortness of breath, he was sent to our ED, where physical examination revealed temperature of 37.3 degrees Celsius, pulse rate of 125 per minutes, respiratory rate of 28 per minutes, blood pressure of 130/81mmHg. Heart sound noted regular heartbeat, heart murmur (pansystolic murmur) and right crackle in breath sounds. On laboratory examination, biochemistry data revealed leukocytosis (WBC: 18500 (Neutrophilic Segment: 91.7%), hsCRP: 21.4 mg/dL, NT-pro BNP: 291 pg/mL, Troponin I <0.01 ng/mL, CKMB: 2.3 ng/mL, Procalcitonin: 0.48 ng/mL. EKG showed sinus tacycardia. The chest radiography revealed cental type alveolar pattern over right lung and cardiomegaly (Figure 1).

Initially, empiric antibiotics with CefTriAxone and MoxiFloxacin were prescribed for severe community-acquired pneumonia. Because hypoxemic respiratory failure, non-rebreathing mask was used. Because unilateral pulmonary was also suspected, echocardiography was arranged, which showed 1. corda tendon rupture and severe mitral valve regurgitation, stage C or D, with left atrium enlargement (Figure 2, 3) 2. preserved LV contractility, without regional wall motion abnormality. Hence, we consulted cardiovascular surgeon. Pre-operation coronary angiography was performed and it showed no significant lesion over coronary artery. Then he received the surgey of Robatic mitral valve repair(Physio II 30 mm, Neo chordae, P2 triangular resection). There was no fever or other complications after surgery and he discharged after 1-week of surgery.

Discussion:

Unilateral pulmonary edema (UPE) is an unusual manifestation of pulmonary edema that is frequently confused with other causes of unilateral alveolar and interstitial infiltrates. It is dangerous but easily missed phenomenon. Its ability to disguise itself as other pathologies, including pneumonia, tend to lead to delays in treatment. The prevalence of UPE was 2.1% of all cases of cardiogenic pulmonary edema. UPE was right-sided in 89%. All patients with UPE had severe mitral regurgitation.

UPE is usually seen in the right lung and differences in the lymphatic draining capacity are thought to play an important role. Extravascular fluid from the right lung drains through the small right broncho-mediastinal trunk (sometimes right lymphatic trunk), whereas fluids from the left lung drain through the much larger thoracic duct. Thus, fluid from the left lung is much more easily drained, whereas on the right, fluid overload can more easily exceed the capacity of the broncho-mediastinal trunk, causing right pulmonary edema.

History, absence of signs of infection, and elevated serum cardiac markers such as B-natriuretic peptide may help to differentiate UPE from other diagnoses. The key examination remains bedside transthoracic echocardiography, although transesophageal echocardiography can also provide additional information regarding the severity and mechanism of mitral regurgitation and documentation of the differential pressure between the right and left pulmonary veins.

The contralateral pulmonary edema usually occurs in the presence of a major perfusion abnormality in the non-edematous lung and may be associated with pulmonary thromboembolism, congenital absence/hypoplasia of a pulmonary artery, Swyer-James syndrome, localized emphysema, lobectomy or pneumonectomy, re-expansion of pneumothorax, systemic to pulmonary artery shunt (Pott's procedure), unilateral sympathectomy, and pleural diseases.

Ipsilateral pulmonary edema (same lung as the process that produces it) may be associated with unilateral veno-occlusive disease, systemic-to-pulmonary artery shunts for cyanotic congenital heart disease, bronchial obstruction, unilateral aspiration pneumonitis, pulmonary contusion, rapid thoracentesis of pleural fluid or air (lung re-expansion), and unilateral infusion of hypotonic saline via a catheter misplaced in the pulmonary vein.

Figure Legend

Figure 1.

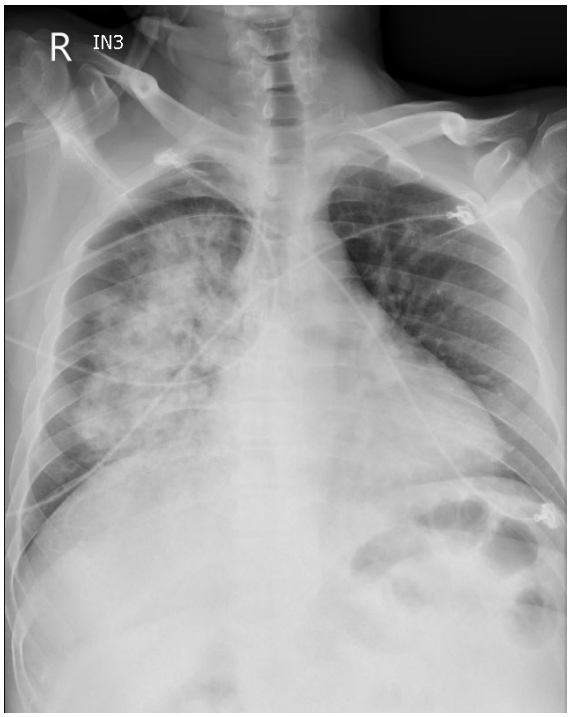

Figure 2. severe mitral valve regurgitation(white arrow)

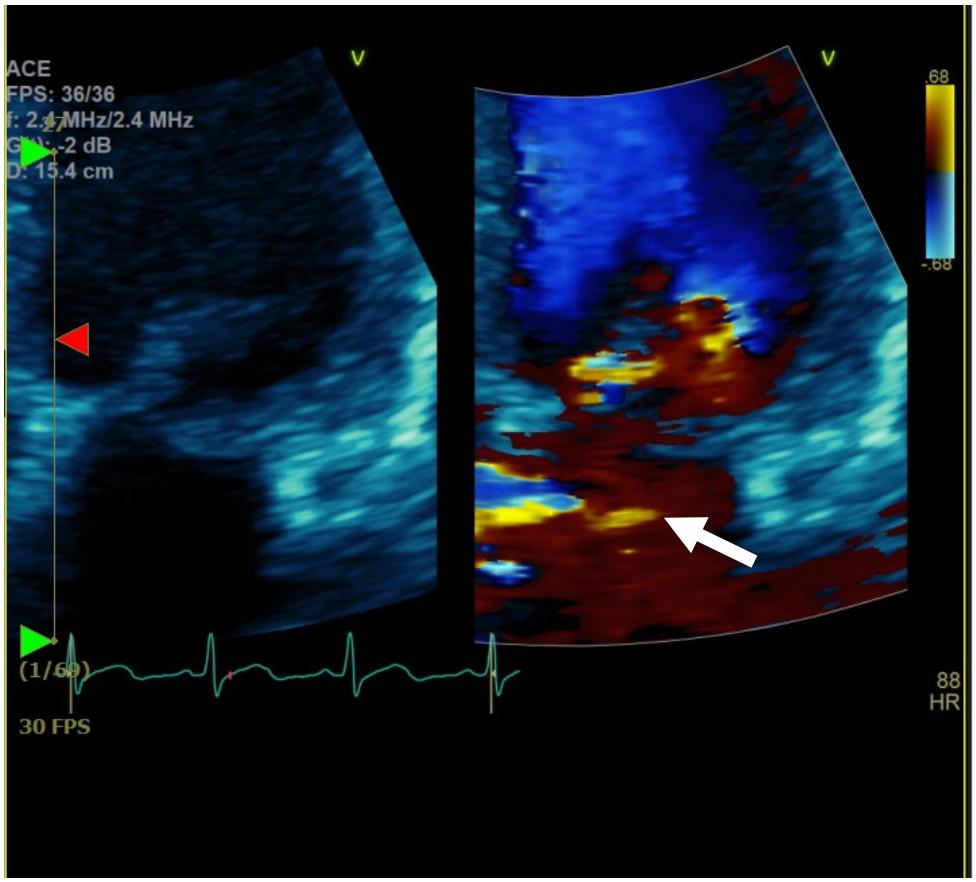

Figure 3. corda tendon rupture(white arrow)

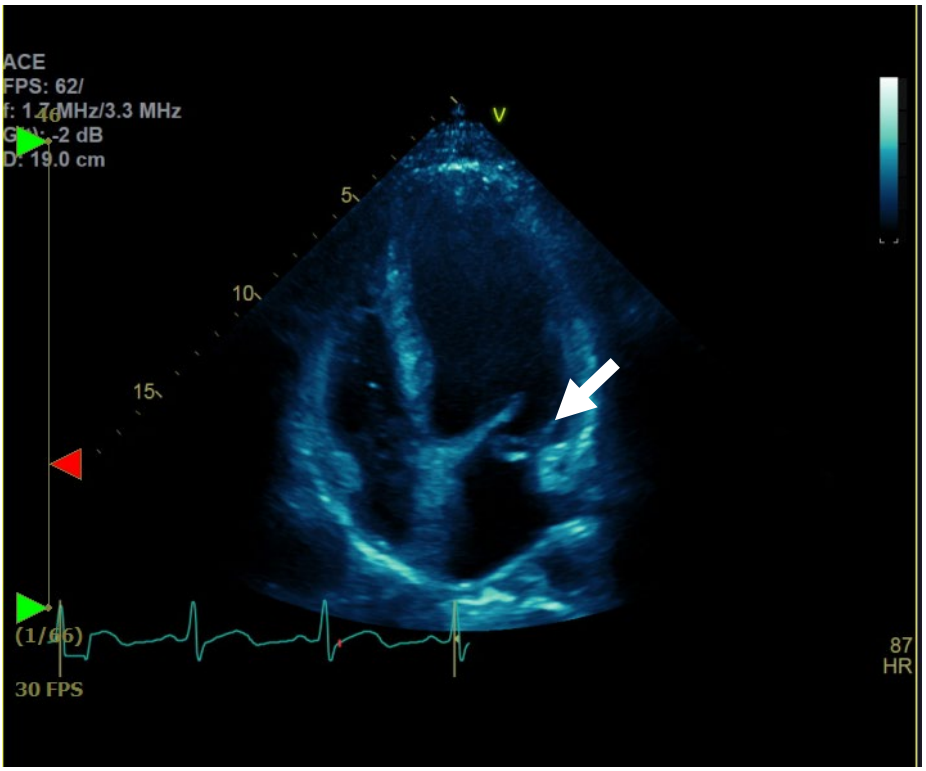

Figure 4. Robatic mitral valve repair without mitral regurgitation(white arrow)