中文題目:急性代償失調心臟衰竭患者之單側肺水腫

英文題目: Unilateral pulmonary edema in a patient with acute decompensated heart failure

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Background:

Unilateral pulmonary edema is a rare manifestation of acute decompensated heart failure secondary to severe eccentric mitral regurgitation (MR). It requires accurate diagnosis to prompt appropriate therapeutic and reduce the morbidity and mortality.

Case Report:

An 81-year-old male, with a past medical history of triple vessel coronary artery disease, moderate to severe eccentric mitral regurgitation (Fig.1), permanent atrial fibrillation, and heart failure with preserved ejection fraction, was admitted with *Listeria monocytogenes* sepsis. After a 14-day treatment of ampicillin, fever subsided with normalization of the laboratory examinations.

However, at one night, he had sudden-onset dyspnea. Physical examination unveiled jugular venous pressure beyond 16cm H₂O, grade 3/6 systolic murmur at mitral area and ventricular gallop, rales at bilateral lung fields, and limb hypoperfusion on palpation. Laboratory investigations revealed metabolic acidosis with high lactate level and respiratory compensation, acute kidney injury, elevated troponin-I and BNP, and leukocytosis. Chest radiograph disclosed dilated left atrium and ventricle superimposed by bilateral pleural effusion and right upper lung opacity (Fig. 2), which was a new lesion compared to previous chest radiograph (Fig. 3). Electrocardiogram detected atrial fibrillation with rapid ventricular response and non-specific STT change. Cefepime and furosemide were prescribed for management of pneumonia and acute decompensated heart failure, respectively. The family refused further aggressive management such as noninvasive positive pressure ventilation, intubation, intravenous vasodilator therapy under intensive hemodynamic surveillance, or even circulatory mechanical support like intraaortic balloon pump for decompensated heart failure and severe MR. They eventually signed do-not-resuscitate consent in light of advanced age and multiple comorbidities. The patient passed away soon owing to hypoxemic respiratory failure.

Discussion:

The patient initially had sepsis with *Listeria monocytogenes* bacteremia, which is a food-borne pathogen. Nevertheless, he developed sudden-onset

dyspnea few hours before his death. Chest radiograph showed right upper lung opacity, which couldn't be seen in previous one. As a result, a neoplasm seemed unlikely over a relatively short period of time. Lack of hemoptysis and anemia didn't support the diagnosis of pulmonary hemorrhage. The patient had been taking apixaban for thromboembolism prevention of permanent atrial fibrillation and dual antiplatelet therapy for avoidance of instent thrombosis after stent deployment 3 months ago. The possibility of pulmonary embolism seemed to be low. As a result, right upper lung opacity was probably attributed to unilateral pulmonary edema or pneumonia.

However, it is difficult to distinguish between pneumonia and unilateral pulmonary edema. Attias et al. reported that in 869 patients with cardiogenic pulmonary edema, approximately 2% of them had unilateral pulmonary edema, in which right upper segment being the most commonly involved. All patients with unilateral pulmonary edema had severe eccentric mitral regurgitation, as in our case. This is thought to happen owing to the regurgitant jet directing toward a single pulmonary vein, causing an increase in the hydrostatic pressure of the isolated pulmonary vein. Other possible explanations are increased capillary permeability, lymphatic obstruction, and increased plasma oncotic pressure.

It was also observed that patients with unilateral pulmonary edema tended to have higher rate of leukocytosis, though fever on presentation was uncommon. As a result, unilateral cardiogenic pulmonary edema is often misdiagnosed as pneumonia. Patients who exhibit unilateral pulmonary edema were prone to receiving antibiotic therapy, compared to those with bilateral pulmonary edema. In a retrospective study of 262 patients with severe eccentric mitral regurgitation, 60% of patients were initially misdiagnosed, with a maximum delay in diagnosis for 4 days. The mortality rate of unilateral pulmonary edema is twice higher than that of bilateral pulmonary edema because of misdiagnosis and delayed treatment.

In our case, the patient was treated empirically for both pneumonia and unilateral pulmonary edema, since pneumonia itself could lead to acute decompensated heart failure.

Conclusion:

Unilateral pulmonary edema is a rare presentation of acute decompensated heart failure and usually derives from severe eccentric MR. Mistaken diagnosis detains proper treatment planning and corresponding therapeutics, so mortality rate is much higher than that of bilateral pulmonary edema. Improvement of clinical outcome is substantially driven by timely identification

of the etiology underlying unilateral pulmonary edema.



Fig. 1 Echocardiogram showed moderate to severe mitral regurgitation with eccentric jet toward posterolateral side. The pressure gradient of tricuspid regurgitation was more prominent than before.



Fig. 2 Chest X-ray revealed right upper lung opacity.



Fig. 3 The chest X-ray was taken at the emergency room. There was no opacity over right upper lung.