Case report

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Acute decompensated cor pulmonale: When holistic approaches meet early tracheostomy, a case report

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Abstract

Acute decompensated cor pulmonale is a clinical syndrome manifested by acute signs of severe congestive heart failure in chronic pulmonary disease with a high mortality rate. A patient with a history of uncontrolled bronchiectasis presented with generalized edema, severe shortness of breath, cardiogenic shock, and gradual loss of consciousness. Laboratory findings showed severe uncompensated respiratory acidosis and signs of infection. An echocardiography study showed signs of pulmonary hypertension with right ventricular (RV) dysfunction. Early tracheostomy in combination with the holistic approach (infection control, increased RV afterload prevention, RV contractility improvement, and RV preload optimization) were performed and were able to control the disease progression. This case report showcased the beneficial effect of early tracheostomy in managing acute decompensated cor pulmonale.

Key words: Decompensated cor pulmonale, pulmonary hypertension, right heart failure, cardiogenic shock, early tracheostomy.

Background

Cor pulmonale is defined as the presence of pulmonary arterial hypertension caused by structural pulmonary disease, which causes right ventricular (RV) dysfunction. Uncompensated increased pulmonary vascular resistance (PVR), which leads to a decompensated condition, is typically instigated by an exacerbation of the chronic lung disease. As we know, decompensated cor pulmonale is an irreversible terminal condition that frequently needs prolonged mechanical ventilation and ICU care yet has a high mortality rate. (1,2) Early tracheostomy will be beneficial in ventilator-dependent patients to reduce the mechanical ventilation day and increase the survival rate. (3) Here, we present a case report of the successful management of acute decompensated cor pulmonale with the combination of a holistic approach and early tracheostomy.

Case illustration

A 53-year-old female patient presented with severe respiratory failure (oxygen saturation [SpO2] 80% on room air), cardiogenic shock (mean arterial pressure [MAP] 51 mmHg), and gradual loss of consciousness. The patient had a history of uncontrolled bronchiectasis, which was followed by a week of intermittent shortness of breath and worsening generalized edema (**Figure 1**). On physical examination, we found excessive purulent sputum from tracheal aspirates accompanied by rhonchi and wheezing on both sides of the lungs. Her laboratory findings revealed leukocytosis (19,360/µl), increased procalcitonin (1.09 ng/ml), hyperkalemia 6.1 mmol/l, azotemia (creatinine serum 2.06 mg/dl), hypoalbuminemia 2.73 g/dl, and hyperglycemia 229 mg/dl (glycated hemoglobin [HbA1c] 8.5%). Her

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initial blood gas analysis indicated severe uncompensated respiratory acidosis (pH 7.12; pCO2 104; pO2 213; HCO3 33.8; BE +4.5; FiO2 80%). The chest x-ray denoted cardiomegaly with an enlarged right heart and bilateral infiltrates on the lungs (**Figure 2**). Trans-thoracic echocardiography found RV dilatation with intermediate probability of pulmonary hypertension (mean pulmonary arterial pressure [mPAP] 36.25 mmHg and pulmonary capillary wedge pressure [PCWP] 11.2 mmHg) and RV dysfunction (tricuspid annular plane systolic excursion [TAPSE] 1.6 cm).

She was then intubated and managed with the targeted mechanical ventilation (maintaining SpO2 90-95%, low tidal volume of 6 ml/kg, low peak pressure, and etCO2 between 55-60 mmHg), empirical antibiotic, nebulizing with beta 2 agonist and corticosteroid, and hemodynamic support using a combination of dobutamine, norepinephrine, and milrinone. After the hemodynamic was stabilized, sildenafil and furosemide were initiated. On the 3rd day, we decided to do an early surgical tracheostomy to speed up the weaning process. Multidiscipline management combining medications, fluid deficit program, psychology support, low carbohydrate nutritional support, breathing exercise, pulmonary and chest physiotherapy, as well as gradual mobilization was successfully weaned down the ventilator support, and on the 12th day, she was weaned off from the mechanical ventilator. On the 20th day in the ICU, the patient could cough adequately and take an oral diet. After the hemodynamic support had been turned off, she was decannulated and discharged from the ICU with oxygen support from a nasal cannula of 2 liters/min.

Discussion

Cor pulmonale is a condition in which RV failure occurs due to increased pressure in the pulmonary arteries caused by pathologies in lung parenchyma which cause an increase in PVR in which cardiac output and PCWP are normal. (2,4) Here, the patient was a female 53 years old, with a history of uncontrolled bronchiectasis and a severe episode of exacerbation, which initiated the episode of acute decompensated cor pulmonale that manifested by anasarca edema and high predicted pulmonary artery pressure accompanied by RV failure (decrease TAPSE 1,6).

Cor pulmonale with pulmonary hypertension and RV failure often need prolonged mechanical ventilator support and ICU care. Early tracheostomy (tracheostomy before the 7th day) has been proposed as a tool to speed up the weaning process and is associated with shorter mechanical ventilator duration and less sedation requirement. (3) Although there are many debates regarding the beneficial effect of early tracheostomy towards hospital mortality, length of ICU and hospital stay, we all can agree that tracheostomy will help in decreasing the death space, breathing effort, and helping with the bronchial toilet. Furthermore, extended mechanical ventilation prior to tracheostomy is associated with a high mortality rate in a time-dependent manner. (5) In our case, tracheostomy was done surgically early on the 3rd day (percutaneous approach was too risky due to the anasarca edema) considering her clinical condition with bronchiectasis exacerbation combined with RV dysfunction which ultimately prone to be mechanical ventilator dependent. After the tracheostomy, we were able to wean her off on the 12th day.

Management of patients with decompensated cor pulmonale is preventing an increase in RV afterload, improving RV contractility, and optimizing RV preload. In our case, afterload reduction was done by targeted ventilation management (limiting driving pressure, low to normal PEEP, etCO2 between 55-60, tidal volume 6 ml/kg, and SpO2> 95%), exacerbation control (in our case infection control with antibiotic), and administering bronchodilator and phosphodiesterase-5 (PDE5) inhibitor. Improving RV contractility was managed by ino-dilator agents like dobutamine and milrinone. Preload reduction was achieved with loop diuretic like furosemide. (4,6) Finally, early enteral nutrition with high fat and low carb, psychological support, and rehabilitation are also fundamental in the recovery process, which needs to be considered early in the therapy process.

Conclusion

Acute decompensated cor pulmonale is frequently viewed as a difficult-to-treat terminal disease in the intensive care setting. This case showed holistic approaches in controlling exacerbation and alleviating the RV burden, combined with early tracheostomy, are proven to be beneficial and must be considered in patients with acute decompensated cor pulmonale.

Figure 1. Patient's clinical condition from the 1st day to the 12th day

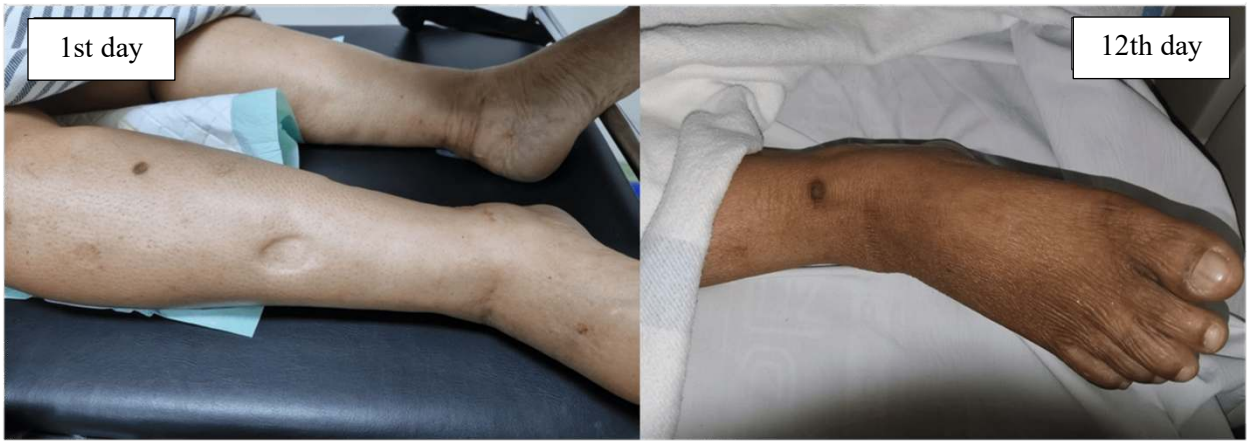
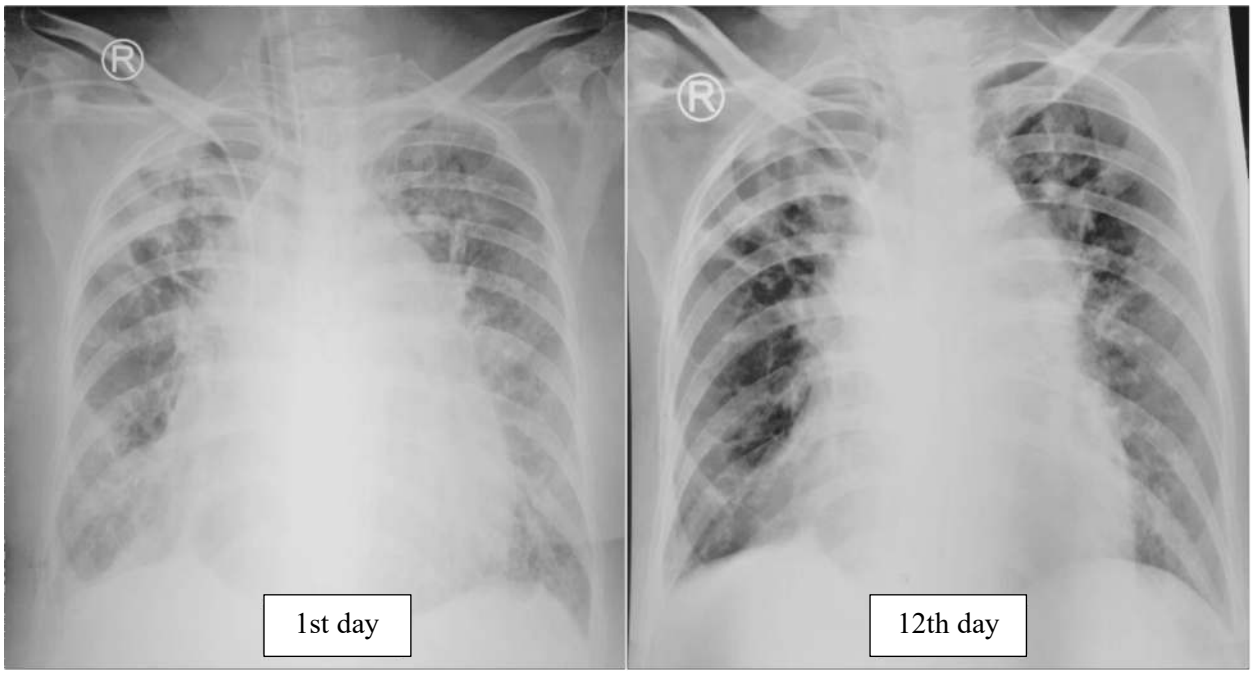


Figure 2. Patient's chest X-ray from the 1st day to the 12th day



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