

Pulmonary embolism in a patient complaining of a cough symptoms**Youichi Yanagawa, Hiromichi Ohsaka, Kei Jitsuiki, Toshihiko Yoshizawa, Kouhei Ishikawa**

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Abstract: A thirty-six-year-old female, who complained of dry cough symptoms, was treated based on a diagnosis of flu or asthma. However, the cough did not improve. She subsequently developed dyspnea and felled into both a hypoxic state and shock and she was transferred to this department. Electrocardiograms revealed incomplete right bundle block with ST depression on the right precordial leads and a blood examination indicated high value of d-dimmer. Enhanced computed tomography showed pulmonary embolism. Thrombolytic therapy led to favorable outcome. Pulmonary embolism can be a lethal disease with various symptoms so physicians should consider pulmonary embolism when patients present with a refractory cough.**Keywords:** cough; pulmonary embolism; d-dimmer

INTRODUCTION

Pulmonary embolism presents with various symptoms. Stein et al. reported the frequency of symptoms of acute pulmonary embolism in 192 cases was 70% dyspnea at rest or exertion, 47% pleuralgia, 43% cough, 42% pain in a lower limb, 39% edema of the lower limbs, 36% orthopnea, 31% asthma and 17% chest pain [1]. A cough is not a rare symptom of acute pulmonary embolism. However, an isolated cough as the initial symptom of acute pulmonary embolism is rare. This report presents an educational case of pulmonary embolism in a patient with prolonged refractory cough that was initially been treated for flu and asthma.

CASE REPORT

A thirty-six-year-old female experienced a dry cough two weeks before she presented at this patient department. She had initially gone to local clinic was diagnosed to have the flu and was treated accordingly. However, her symptoms did not improve. Three days before arrival, she went to the same clinic again and was diagnosed and treated for asthma and prescription. However, her symptoms did not improve at all. She developed dyspnea one day before arrival. She felt chilliness and lethargy the following day and was unable to walk so that she called emergency services. As she was hypoxic and in shock, she was transported to the emergency department. She had a history of pyloric stenosis and patent ductus arteriosus requiring surgically as an infant. She had no family history. She was obese and had clear consciousness; however her radial artery was unpalpable. She had a pulse of 120 beats per min, tympanic temperature of 34.5 Celsius, and respiratory rate of 36 breaths per min.

Percutaneous oxygenic saturation could not be measured. Her blood pressure increased after rapid infusion and saturation became measurable. Five L/minute oxygen with a mask was necessary to maintain 100% saturation. An electrocardiogram revealed incomplete right bundle block with ST depression on the right precordial leads suggesting signs of right heart strain. Echocardiography revealed normal wall motion of left ventricular and loss of respiratory change in the inferior vena cava. A complete blood count showed 13100 /mm³ white blood cells, 37.5% hematocrit, and 24.2 x 10⁴/mm³ platelets. Serum biochemistry revealed 1.8 mg/dl c-reactive protein and 22 µg/ml D-dimmer. Enhanced computed tomography (CT) was performed because she had sign of right heart strain with high value of D-dimmer, and definite diagnosis of acute pulmonary embolism was obtained (**Figure 1**). She was infused with tissue plasminogen activator. Her heart rate decreased to 70-90 beats/minute and saturation increased to 96% with 2L/minute oxygen via a nasal cannula. Enhanced CT demonstrated the decreased in volume of the pulmonary embolism on the 4th hospital day, however it still remained despite the anticoagulant therapy. Her percutaneous saturation returned to 98% under room air on the 13th hospital day, and the refractory cough disappeared without medication. Enhanced CT on the same day revealed resolution of most of the pulmonary thrombus. She was discharged from the hospital on the 17th day without sequela.

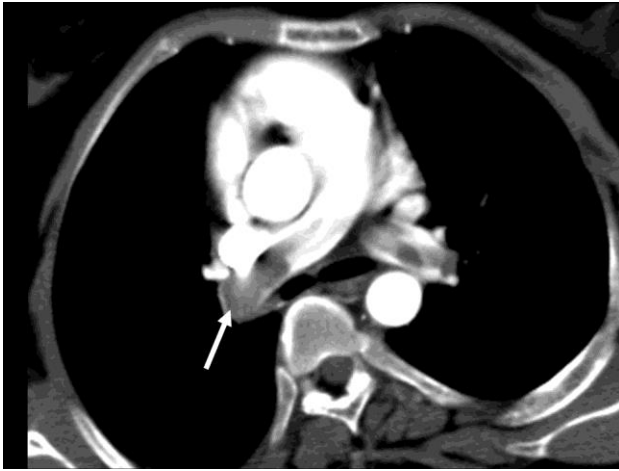


Fig-1: Enhanced chest CT on arrival, the CT shows pulmonary embolus in main duct of the pulmonary artery (white arrow).

DISCUSSION

This is the rare case of a pulmonary embolism in which the initial complaint was only a cough. A search of the literature revealed no similar reports. Although upper respiratory inflammation may have preceded pulmonary embolism in the present case, the cough may have been caused by pulmonary thrombosis because the patient presented with only a cough without fever, pharyngeal pain or nasal discharge. In addition, thrombolytic therapy was effective; however it did not provide an immediate complete resolution, suggesting that the thrombus was old with organized characteristics. Furthermore, the cough disappeared with the pulmonary thrombus.

The exact mechanism of cough induced pulmonary embolism is unclear. The cough receptor in the trachea may be stimulated by some chemical mediator originating from a pulmonary thrombus [2-4]. Pulmonary embolism can be a lethal disease with various symptoms so physicians should therefore consider pulmonary embolism in the differential diagnosis when patients present with a refractory cough [5].

CONCLUSION

A physician should therefore consider pulmonary embolism in the differential diagnosis when patients present with a refractory cough.

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