

Streptococcus Intermedius Empyema Following Acute Pulmonary Embolism: Case Report

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Received: 13 Apr 2022

Accepted: 25 Apr 2022

Published: 30 Apr 2022

J Short Name: ACMCR

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Citation:

Sandio A, Streptococcus Intermedius Empyema Following Acute Pulmonary Embolism: Case Report. Ann Clin Med Case Rep. 2022; V9(1): 1-4

Keywords:

Streptococcus Intermedius; Pulmonary embolism; Empyema; Apixiban

1. Abstract

Pulmonary embolism is and has been an established medical diagnosis. The complications, however, seem to awe physicians and healthcare workers across the globe. The purpose of this report is to bring to awareness a case involving a 37-year-old female with pulmonary embolism which was complicated by development of right-sided empyema. This patient initially presented with shortness of breath, heart rate/pulse of 109 beats per minute and right sided pleuritic chest pain and was diagnosed with pulmonary embolism and started on apixaban. Upon discharge after three days, she continued to have persistent progressively worse right sided chest pain. She was readmitted five weeks later, and further investigation resulted in the diagnosis of right sided pleural effusion. CT guided drainage of the effusion was consistent with empyema with cultures growing streptococcus intermedius. The aim of this report is to present a readmission of a 37-year-old woman patient who developed streptococcus intermedius empyema after initial diagnosis and management for acute pulmonary embolism.

2. Introduction

Acute pulmonary embolism results in pulmonary infarct at a rate of approximately 16% to 31% [5]. The predisposing risk factors for pulmonary infarct are active smoking and increase body height. Patients are often of younger age groups without cardiopulmonary comorbidities [10, 11]. 4% to 7% of patients with pulmonary infarction develop cavitory lesions due to necrosis or superinfection

of the necrotic tissue [8, 12]. Pulmonary infarction is a wedge-shaped area that usually extends to the pleural surface and causes pleural effusion due to increase pulmonary capillaries permeability from ischemia or release of vasoactive cytokines [6, 9]. 23% to 52% of patients with Pulmonary embolism have pleural effusion on computed tomography [2]. The pleural effusion is usually exudative and hemorrhagic [2, 9]. Delayed onset of pleural effusion or late enlargement in the course are associated with either recurrent pulmonary embolism or superinfection [1].

Empyema is a collection of pus [fluid filled with immune cells, dead cells, and bacteria] in the pleural cavity [14]. The risk factors for empyema include pneumonia, chronic lung disease, diabetes mellitus, prolonged corticosteroid use, illicit drug use, alcohol abuse, aspiration, thoracic or esophageal surgery or trauma [3]. Empyema had rarely been associated with pulmonary embolism [3, 7]. The implicated pathogens in empyema include gram positive bacteria especially viridans strep species in community acquired settings while Staphylococcus aureus [Methicillin resistant Staphylococcus aureus] and Pseudomonas predominate in hospital-acquire cases [3, 14]. The mortality rate could be as high as 47% in hospital acquired and 17% in community acquired [13], thus prompt intervention is crucial in the management [14]. Management of empyema usually involves antibiotics in conjunction with surgical drainage of the fluid. Intrapleural agents like fibrinolytic and mucolytics are sometimes available as options but are not considered first or second line [14, 3].

3. Case Presentation

We present a case of a 37-year-old morbidly obese female with history of hypothyroidism, hypertension, uterine fibroids, and nicotine dependence initially presenting with right-sided pleuritic chest pain and shortness of breath with a diagnosis of pulmonary embolism complicated by development of right-sided empyema. A CT angiography showed right upper lobe anterior subsegmental pulmonary embolism with an adjacent pulmonary infarct (Figure 1). Patient was started on apixaban and discharged home. She presented to the emergency room again two weeks later with persistent right-sided chest pain, repeat CT angiography showed previously seen anteromedial right upper lobe pulmonary embolism; increased peripheral wedge like consolidation in the associated anteromedial right upper lobe, suggestive of an evolving pulmonary infarct; and dependent airspace opacity in the right lower lobe and small pleural effusion (Figure 2).

Rapid COVID-19 test was negative. She was administered analge-

sics with relief of her symptoms and was advised to follow up with hematology. However, her pain persisted; she developed worsening shortness of breath with a dry nonproductive cough and returned to the emergency room again three weeks later. On presentation, she was febrile with temperature of 99.2 F, tachypneic, tachycardic, in acute respiratory distress with oxygen saturation of 88% on room air. CXR was done which showed opacification of most of the right chest (Figure 3) and CT angiography showed massive right pleural effusion with mass effect compatible with tension hydrothorax; its density is mildly complex suggesting proteinaceous or hemorrhagic components; there was near complete collapse of the right lung; there was no pulmonary embolism identified. The Segmental and subsegmental right-sided pulmonary artery branches are not well assessed due to heterogeneous enhancement likely related to compression from the pleural effusion and lung collapse (Figure 4). CT chest done Day 2 after chest tube placement showed significant reduction in prior right pleural effusion, small residual right pleural effusion and extensive pleural thickening.



Figure 1: CT angiography chest showed right upper lobe anterior subsegmental pulmonary embolism with an adjacent pulmonary infarct.

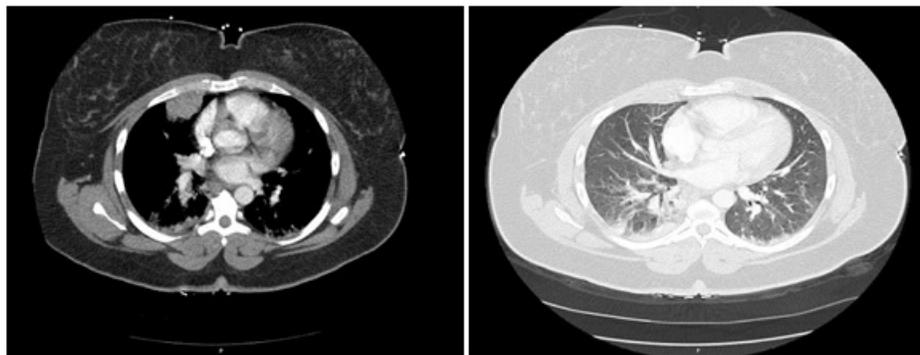


Figure 2: Repeat CTA chest on second presentation showed previously seen anteromedial right upper lobe pulmonary embolism; increased peripheral wedge like consolidation in the associated anteromedial right upper lobe, suggestive of an evolving pulmonary infarct. Dependent airspace opacity in the right lower lobe and small pleural effusion.

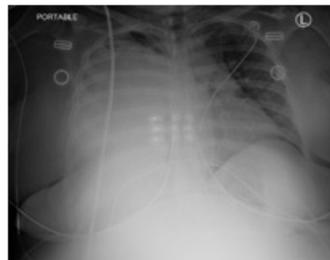


Figure 3: Chest x-ray done on third presentation showed opacification of most of the right chest suspicious for at least a component of pleural effusion.

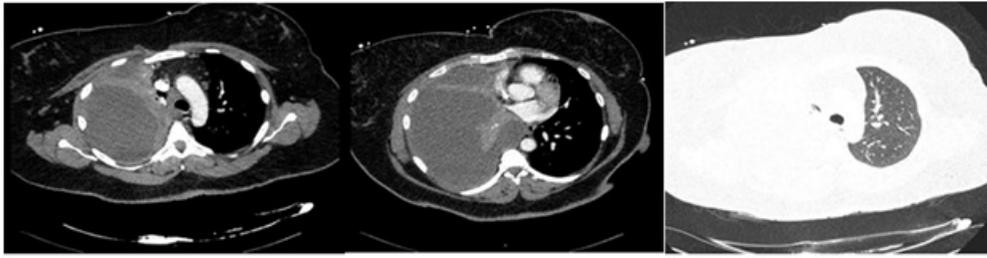


Figure 4: CT angiography done on third presentation showed massive right pleural effusion with mass effect compatible with tension hydrothorax. Its density is mildly complex suggesting proteinaceous or hemorrhagic components. Near complete collapse of the right lung. No pulmonary embolism is identified. Segmental and subsegmental right-sided pulmonary artery branches are not well assessed due to heterogeneous enhancement likely related to compression from the pleural effusion and lung collapse.

4. Results

Laboratory tests were remarkable for elevated white cell count of 14.8, she was anemic with hemoglobin of 7.1 g/dl, and complete metabolic panel was within normal limits. She was administered oxygen, and broad-spectrum antibiotics namely vancomycin, and meropenem as she was allergic to penicillin. Apixaban was held and she was started on a heparin drip. She was admitted to the intensive care unit, underwent urgent CT-guided chest tube placement (Figure 5), and approximately 800 cc of purulent fluid was

drained. Blood cultures showed no growth, pleural fluid cultures subsequently grew *Streptococcus intermedius*, and antibiotics were deescalated to ertapenem. CT chest done Day 2 after chest tube placement showed significant reduction in prior right pleural effusion, small residual right pleural effusion and extensive pleural thickening (Figure 6). Intrapleural tissue plasminogen activator was administered. She improved clinically, chest tube was removed, and she was discharged home to complete a 6-week course of intravenous antibiotics.

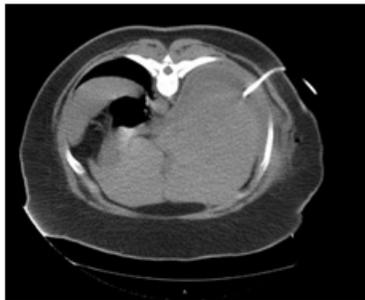


Figure 5: CT guided chest tube placement.

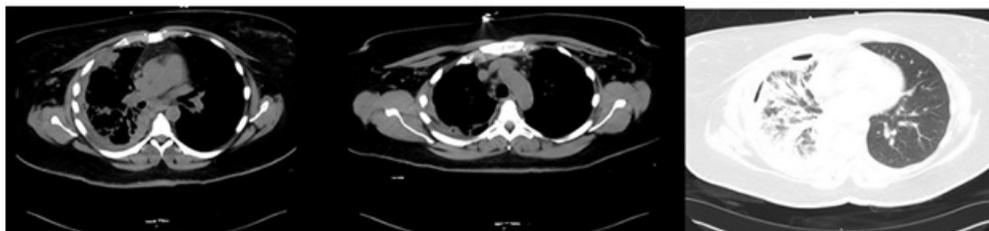


Figure 6: CT chest done Day 2 after chest tube placement showed significant reduction in prior right pleural effusion, small residual right pleural effusion and extensive pleural thickening.

5. Discussion

Pulmonary embolus can manifest with a diverse range of symptoms from being asymptomatic to sudden cardiac death. Approximately, 16% to 31% of pulmonary emboli [5] results in development of pulmonary infarct and the latter complication is more common in peripheral than in central PE. 4-7 % of pulmonary infarcts result in cavitation of the lung due to necrosis or infection involving the necrotic tissue [8, 12]. Rarely pulmonary infarction is associated with empyema [3, 6]. Hemorrhagic infarction is seen in the lung due to the dual blood supply and loose connective tissue of the

lung [6]. The pulmonary infarction is a wedge-shaped area that usually extends to the pleural surface and causes pleural effusion [6, 9]. Micro-organisms usually bacteria can colonize the pleural effusion and produce empyema. As seen in the case of the patient, she was initially diagnosed with pulmonary embolism with infarction, started on anticoagulation and presented later with empyema. Empyema is usually due to pneumonia. Although the second CT scan showed dependent airspace opacity in the right lower lobe which may be suggestive of pneumonia but because of the rapid interval change on the CT scan from small pleural effusion to mas-

sive empyema with mass effect compatible with tension hydrothorax, pulmonary infarction cannot be excluded as a major contributory factor to the development of this accelerated empyema.

Clinicians should consider pulmonary infarction as a cause of empyema especially in patient with recent pulmonary infarction presenting with fever, dyspnea, and cough and accelerated empyema. Prompt initiation of antibiotics should also be considered in cases like this due to likely presence of empyema on the background of recent pulmonary infarction because of high mortality [13-14].

6. Conclusion

Empyema is a rare complication of pulmonary infarction and it should be considered in patients with recent pulmonary infarction presenting with fever, dyspnea, cough, and hydrothorax. Prompt recognition, management with antibiotics and surgical drainage can help improve the declarations.

7. Contributions

- Patient seen as inpatient/outpatient in one hospital in the city of Poughkeepsie [Dr. Ajibola, Dr. Pang Lam, Dr. Nili Gujadhur, Dr. Oluwafemi Ajibola]
- Data collection of outpatient/inpatient, conducting research, collecting lab imaging, writing the manuscript [Dr. Rebecca Mazurkiewicz, Dr. Susan Collins, Dr. Smart Asare, Dr. Aubin Sandio].
- Interpreting results of lab imaging, results, discussions, and reviewing [Dr. Ajibola, Dr. Pang Lam, Dr. Nili Gujadhur; Dr. Mazin Shaikhoun, Dr. Aubin Sandio, Dr. Eric Ayers, Dr. Mayowa Apata, Dr. Smart Asare].

8. Ethics Declarations

8.1. Ethics approval

Not applicable.

8.2. Consent to participate

Not applicable.

8.3. Consent for publication

Patient has provided Written consent for publication of this case. The written consent form is available upon request.

9. Conflict of Interest

The authors declare no competing interests.

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