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Cavitary Pulmonary Infarct: The Differential Diagnostic Dilemma – A Case Report

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1. Introduction

Pulmonary infarction is localized destruction (necrosis) of lung tissue by blocking (obstruction of) the arterial blood supply. It follows an embolic event in ~10% of cases. Blockage of pulmonary artery by a clot or air bubble or other particle (called pulmonary embolism) leads to localized damage of lung tissue which results in pulmonary infarction (1, 2). The reasons for this low incidence of pulmonary infarct are the dual blood supply systems, as well as oxygenation of the lung tissues via ventilation (3).
The predisposing factors for pulmonary infarct include congestive heart failure, pleural effusion, pulmonary infection, atelectasis, hypotension, positive-pressure ventilation, chronic lung disease, central venous catheterization and an immunocompromised state. It is more common in people with chronic heart and lung diseases. Infarction condition may be mild and can be rapidly fatal (4).
Common symptoms include chest pain which may be because of difficulty in breathing, high pulse rate, mild fever, developing of fluid in the lungs, a productive cough (sputum may be blood-tinged). Blockage may also result into circulatory breakdown, like low blood pressure, presence of very little oxygen in the blood. Also, swelling of neck vein and leg, weakness, restlessness, and fainting. In the case of infection as developing complication, there is worsening of the clinical status, persistent fever, malaise, sweating, increasing pulse rate and leukocytosis (usually more than $20 \times 10^9/l$) (5).
Diseases that should be listed in the differential diagnosis include bacterial pneumonia, aspergillosis, tuberculosis, norcardia, actinomycosis, and granulomatous vasculitis. Other unusual etiologies that should be listed in the differential diagnosis include primary or metastatic angiosarcoma or leomyosarcoma and lung cancer invading the main pulmonary arteries (6,7).

2. Clinical presentation

Bacterial pneumonia and pulmonary infarction frequently mimic each other clinically, indicates that most methods for distinguishing between these illnesses are unsatisfactory. Both diseases may give rise to dyspnea, pleuritic pain, tachypnoea, fever, cyanosis, hypotension, cough, hemopthysis, jaundice, leukocytosis and similar radiographic abnormalities (8).
Shaking chills point strongly to bacterial pneumonia. Additional hints are a preceding upper respiratory tract infection followed by gradually increasing malaise and then cough, usually

productive of purulent sputum. Patients with pulmonary infarction more often become ill with dramatic suddenness, seldom have a cough and experience shaking chills only if the emboli are septic or the infarct become infected (8).
Physical signs are not specific for either diseases. Although high fever is more typical of bacterial pneumonia, it is occurs with sufficient frequency in pulmonary infarction to be unreliable as differential diagnostic sign. Patients with pulmonary infarction, as compared to those with bacterial pneumonia, generally are more dyspnoic and tachypnoic in relation to the extent of their physical and radiographic abnormalities and rarely exhibit classic signs of consolidation. They more often manifest hypotension, either transient or recurrent, and more commonly show signs suggesting pulmonary hypertension and right-sided congestive heart failure, e.g. a loud pulmonis component of the second heart sound and elevation of the jugular venous pressure (8).
A pleural friction rub helps in differentiation only when chest radiography shows no parenchymal disease. Then, infarction is more often do not cause radiographic changes early in the course of the illness (8).

3. Laboratory and other diagnostic tests

Sputum examination is one of the best ways of differentiating bacterial pneumonia from pulmonary infarction. In bacterial pneumonia the sputum classically is purulent, occasionally foul smelling and may contain bright red fleck of blood. Gram's stain typically shows many bacteria and polymorphonuclear leukocytes. In pulmonary infarction, sputum, when present, usually is frankly blood with few bacteria or inflammatory cells. If the infarct becomes infected the sputum may be indistinguishable from that in bacterial pneumonia. Blood cultures often reveal the causative microorganism in the patients with bacterial pneumonia but show no growth in cases of bland pulmonary infarction (8).
Cavitation after bland pulmonary infarcts may result from either aseptic necrosis of the infracted lung or from secondary bacterial infection with subsequent abscess formation. It is infective almost as often as it is aseptic (9).
Two types of infected pulmonary infarct have been proposed based on the mode by which infection sets in (10). One is called "primary" because the infection is from a septic embolus. The other is called "secondary" because the infection is bronchigenic origin. Some authors suggest that the development of fever and/or purulent sputum following a pulmonary infarct is highly suspicious for secondary infection. The spectrum of causative agents for infected pulmonary infarct is similar to that of nosocomial pneumonia (3).
Total leukocyte count has limited discriminatory value. It usually is normal or slightly elevated in pulmonary infarction, but there is reports of leukocytes count higher than 40 000 per mm^3 in patients with massive, bland necrosis of pulmonary tissue. Elevated serum lactic dehydrogenase (LDH) activity, normal aspartate aminotranspherase activity (AST) and increased serum bilirubin concentration forms a triad once considered a sensitive indicators of pulmonary embolism and infarction. However, subsequent studies have shown that these tests fail to differentiate pulmonary infarction from pneumonia and host of other disorders (8).
Electrocardiographic abnormalities that may appear are right ventricular conduction disturbances; right axis deviation; inverted T-waves with S-T segment deviation in the right precordial leads; peaked T-waves in leads II, III and AVF; various types of supraventricular and raraly ventricular arrhythmias and S1, Q3 or S1,S2, S3 patterns. Heart ultrasound may

reveals right ventricular dilatation, septal deviation of left ventricle, tricuspidal regurgitation or right ventricle hypokinesis with wall thinning (8, 11).
Pulmonary function test and arterial blood gas studies provide data that are too variable and nonspecific to differentiate bacterial pneumonia from pulmonary infarction (8).

4. Imaging studies

Radiographic similarities of bacterial pneumonia and pulmonary infarction are the chief source of diagnostic confusion between the two entities. Each is responsible for parenchymal infiltrates of varied size and shape, with or without pleural effusion, atelectasis or cavitation. In contrast to bacterial pneumonia, pulmonary infarcts always abut a pleural surface and predominate in lower lobes, especially the right. They also may appear in concert with dilatation of one or both main pulmonary arteries, decreased peripheral vascular markins in the affected portion of lung (oligemia) or engorged vassels in the non affected areas (pleonemia). Further radiographic clues to pulmonary infarction are infiltrates appearing first in one lung and then the other or "pneumonia" unresponsive to chemotherapy (8).
Spiral computed tomography and magnetic resonance angiography are helpful in establishing the difference between pneumonia and pulmonary infarct. Also, this imaging techniques could help tracking the resolution of the thrombo emboli, but they are expensive and unavailable in many hospitals (6,7).
Pulmonary arteriography is the most specific means of differentiating bacterial pneumonia from pulmonary infarction. In bacterial pneumonia the pulmonary arteries proximal to the subsegmental level show neither filling defect nor obstructive lesions, where in pulmonary infarction they contain filling defect, appear obstructed, or both (8).
The grater the size of infarct, more likely its centre will be hypoxic and nectrotic. Pulmonary infarct larger than 4 x 4 cm in size have a great tendency for cavitation (12).
The median time from the first detection of consolidation to cavity formation is 14 days (12).
Doppler sonography is a noninvasive and convenient tool for diagnosing pulmonary embolism and follow-up reperfusion of the lung. Dynamic changes in blood flow in consolidated areas provides information about the status of reperfusion (6).
Despite use of the aforementioned techniques, the question of infected versus infracted lung sometimes will persist. To minimize error, the physicians should think of both diseases when he considers either, particularly if the process involves the lower lobes, especially the right. Dangers of delaying treatment for pulmonary infarction rival the hazards of withholding specific chemotherapy in bacterial pneumonia. Thus, as long as the diagnosis remains in doubt, treatment for both disorders seems well advised (8).
Multiple complications have been associated with pulmonary infarct, including pneumonia, empyema, pneumothorax, lung abscess, bronchopleural fistulae and lethal haemorrhage. Large series of autopsies reveald cavitation in 4-5% of all pulmonary infarcts (5).
The mortality rate is as high as 41% and 73% for nonifected and infected cavitary pulmonary infarcts, respectively. Anticoagulants and antibiotics are the mainstay of therapy. Massive haemopthysis may persist even after discontinuation of anticoagulants. Possible explanation for this phenomenon are an overdose of anticoagulants and reperfusion of necrotic lung tissue. Anticoagulants use in cavitary pulmonary infarction, therefore, must be very carefully monitored and causation should be exercised in monitoring clinical conditions and the status of coagulation (10).

5. Case presentation

Patient was admitted to our clinic with a three week history of dyspnea, tachycardia, cough, expectoration of yellow colored sputum, fever, exhaustion and lower legs edema. Few days before admission to clinic he presented altered level of consciousness (mental confusion), caused by alcohol withdrawal. The patient suffered from congestive heart failure and chronic obstructive pulmonary disease for several years. There was no past medical history of predisposition factors for embolism or episodes of venous thrombo-embolism.

On examination he presented normal mental status, body temperature was within normal range, blood pressure 130/80 mmHg, pulse rate 90 beats/min; respiratory rate 26 breath/min; with signs of peripheral cyanosis. The chest expanded symmetrically and breathing sounds were clear. There were heart rhythm disorder (absolute arrhythmia) but no heart murmurs were detected. The liver and spleen were not palpable. Lower leg edema was noticed.

The initial laboratory blood test revealed elevated inflammatory parameters: white blood cells:15.7 x 10^9/L; with 87.6% neutrophils; 3.4% monocytes and 9.0% lymphocytes; C-reactive protein: 73 mg/L; D-dimer: 759 ŋg/ml. The prothrombin time, activated partial thromboplastin time (PTT) and platelets were within normal range. There were elevated activity of aspartate aminotranspherase (160.6 U/L); alanin aminotransferase (128.0 U/L); lactic dehydrogenase (745 U/L); serum bilirubin concentration (45 µmol/L);blood urea nitrogen (16.3 mmol/L) and creatinine (120.4 µmol/L). But, all this test has limited discriminatory value.

The arterial gas blood analysis showed the following: pH 7,5; carbon dioxide tension in arterial blood (PCO_2) 31 mmHg; oxygen tension in arterial blood (PO_2) 71 mmHg; HCO3 24 mmol/L; BE 1 mmol/L and oxygen saturation (SaO_2) 95%.

Lung functional test shown severe restrictive ventilation disorder (FEV1: 8%; FVC: 44% - 1.67 L; FEV1/FVC: 72%) which is nonspecific to differentiate bacterial pneumonia from pulmonary infarction as well as the arterial gas blood analysis too.

The electrocardiography (ECG) revealed atrial fibrillation only. The initial chest radiograph was showed enlarged cardiac shadow without pulmonary opacities (**Figure 1**) because infarction is more often do not cause radiographic changes early in the course of the illness. Congestive heart failure was diagnosed and the patient was treated with diuretics and inotropes.

However, a ten days later the intensity of dyspnea, fatique, weakness and exhaustion has increased. He was febrile (38.3^0 C). A contribution to literature data, in our case, clinical condition has got worsening suddenly. Chest radiography was significant since it showed round pulmonary consolidation with central cavitation on the apical segment of the right lower lobe (**Figure 2**). The patient was placed on antibiotic therapy (intravenous ceftazidine and ertapenem) for a presumptive diagnosis of bacterial pneumonia.

However, one week later, the treatment with antibiotics was not satisfactory and there were no clinical recovery. Also, there were no microbiologic confirmation of causative microorganism which made definitive diagnosis difficult. The clinical symptoms of congestive heart failure was dominant and the patient underwent a cardiac ultrasound which revealed tricuspidal regurgitation and elevated right ventricular pressure (systolic blood pressure of right ventricule - SPRV: 46 mmHg). The increased intensity of dyspnoea, fatique, weakness and exhaustion, despite the treatment with diuretics and inotropes was due to unconfirmed hemodynamic disorder.

Fig. 1. Initial chest PA (postero-anterior) radiography showing enlarged cardiac shadow without pulmonary opacities

Fig. 2. PA (postero-anterior) chest radiography ten days from admission showing round pulmonary consolidation with central cavitation on the apical segment of the right lower lobe

The most striking and unexpected findings was contrast-enhanced computed tomography (CT) scan of blood vessels and identification of filling defect in the main pulmonary arteries which presumptive diagnosis of pneumonia excluded. There were pulmonary consolidation with central cavitation on the right lower and left upper lobes too. The diameter of right and left consolidation was 63 x 75 mm and 85 x 70 mm respectively (**Figure 3a and 3b**). The size of pulmonary infarct , in our case, is grater than 40 x 40 mm which explain the appearance of cavitation. Also, the velocity of cavity formation presented in our case is significantly lower than literature data pointed out. Venous duplex ultrasound of lower extremitas was negative for deep-vein thrombosis. Low-molecular heparin were administered immediately after the findings of the CT scans were obtained.

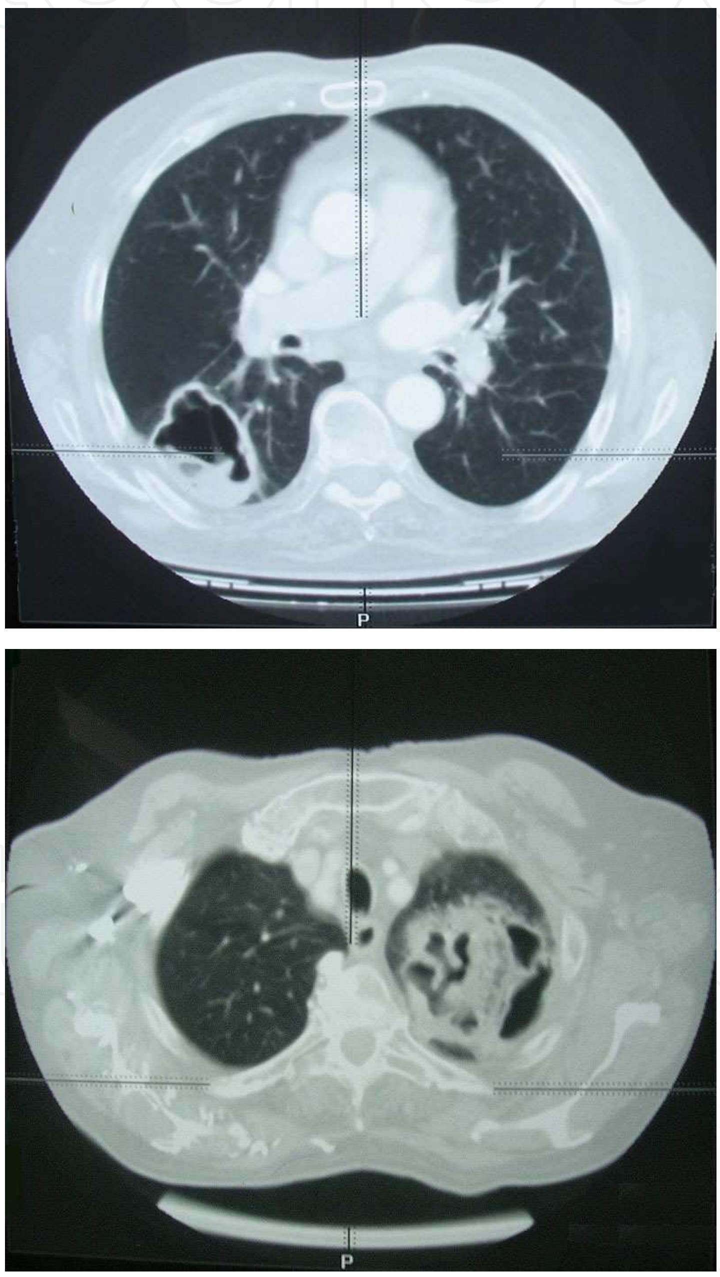

Fig. 3a. and 3b. Contrast - enhanced computed tomography scan of blood vessels showing filling defect in the main pulmonary arteries with central cavitation on the right lower and left upper lobes too

The follow-up chest radiograph (three weeks later) showed regression of mentioned pulmonary opacities (**Figure 4**). Due to anticoagulant therapy was administered immediately after the findings of the CT scans were obtained, the patient was clinicaly recovered and he was discharged with follow-up recommended.

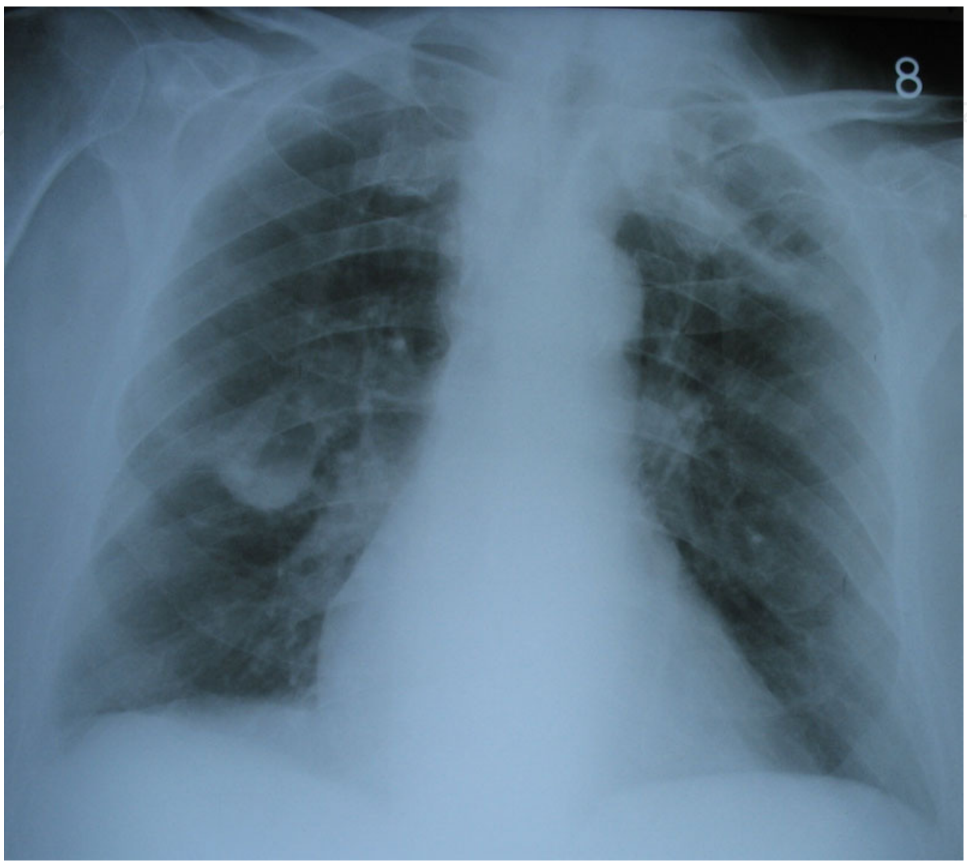

Fig. 4. Follow-up chest radiograph (three weeks later) showing regression of mentioned pulmonary opacities.

6. Conclusion

Cavitary pulmonary infarct is a rare but frequently misdiagnosed disease entity. Differentation between cavitary pulmonary infarct and multiple complications or other diseases can be a real challenge because of the similar radiographic abnormalities and clinical presentation of all this conditions. In the cases with clinical suspicion to "pneumonia" unresponsive to chemotherapy images studies are of great help. The best evidence of infarction is the angiographic demonstration of pulmonary thromboemboli. Anticoagulant and antibiotic treatment in the cases of infected cavitary pulmonary infarct must be started immediately after the diagnosis is established.

7. Acknowledgement

The authors wish to thanks all physicians from the clinic who participated in the medical care of the patient described in this report. Also, we are grateful to Marta Djordjevic for technical assistance in the computer processing of the images.

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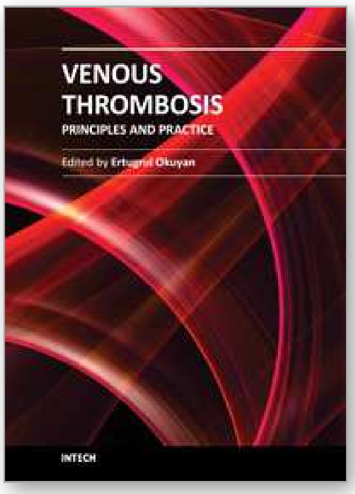

Venous Thrombosis - Principles and Practice
Edited by Dr. Ertugrul Okuyan

ISBN 978-953-307-885-4
Hard cover, 232 pages
Publisher InTech
Published online 05, January, 2012
Published in print edition January, 2012

According to Virchow's triad, venous thrombosis can occur as a result of one or more of three factors: changes in the dynamics of the blood flow, endothelial injury/dysfunction of the blood vessel and hypercoagulability. The blood in the veins is constantly forming microscopic thrombi that are routinely broken down by the body, and significant clotting can occur only when the balance of thrombus formation and resolution is altered. This book is a fresh synthesis of venous thromboembolism care and considers the opinions and studies from different fields of medicine. As venous thrombosis spectrum is wide and can affect many organ systems, from deep veins of the leg to the cerebral venous system, our intent is for this to be a comprehensive, up-to-date and readable book. We tried to present a synthesis of existing material infused with new ideas and perspectives and authors own clinical studies and even case-reports.

How to reference
In order to correctly reference this scholarly work, feel free to copy and paste the following:

Ivanka Djordjevic and Tatjana Pejcic (2012). Cavitary Pulmonary Infarct: The Differential Diagnostic Dilemma – A Case Report, Venous Thrombosis - Principles and Practice, Dr. Ertugrul Okuyan (Ed.), ISBN: 978-953-307-885-4, InTech, Available from: http://www.intechopen.com/books/venous-thrombosis-principles-and-practice/cavitary-pulmonary-infarct-the-differential-diagnostic-dilemma-a-case-report

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