

Case report

Approaching cavitory lung lesion in a patient with pulmonary embolism

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Abstract

The pulmonary cavity may reflect the presence of a broad spectrum of lung pathological processes, with surprising etiology at times. Bacteria, fungi, viruses, parasites, embolism, malignancy, and autoimmune disease may be involved in its formation. We present the case of a 78 year-old patient who was hospitalized 7 days prior to admission to our hospital with grade IV mMRC dyspnea and dry cough, the chest CT indicating pulmonary embolism and the presence of a pulmonary cavity, suggestive of neoplastic etiology. An infection with *Staphylococcus aureus* complicated with pulmonary embolism, two pathological entities which can lead to cavitory pulmonary damage, can prove challenging to any physician in the given context.

Keywords: lung cavity; *Staphylococcus aureus*; pulmonary embolism

Introduction

The cavity, a gas filled space in a pulmonary consolidation area, mass or nodule, is produced by the expulsion of the necrotic content in a bronchus. It may or may not contain a liquid level and is surrounded by a wall of variable thickness, being a manifestation of a broad spectrum of lung pathological processes [1].

A rather rare finding in pulmonary imaging, the cavity formation may result from bacterial infections such as microaerophilic streptococci, viridans streptococci, *Klebsiella pneumoniae*, *Staphylococcus aureus*, *Pseudomonas aeruginosa*, *Haemophilus influenzae*, *Acinetobacter species*, *Escherichia coli*, *Legionella species*, *Fusobacterium necrophorum*, *Nocardia asteroides*,

mycobacteria such as *Mycobacterium tuberculosis*, nontuberculous mycobacteria and *Mycobacterium avium* complex, fungi such as *Cryptococcus neoformans*, *Coccidioides*, *Aspergillus* and mucormycosis, parasites such as *Paragonimus westermani* and *Echinococcus genus*, and viral infections such as papilloma virus [1,2]. Moreover, the cavity may be the result of a septic pulmonary embolism, such as Lemierre's syndrome, or a primary or secondary malignancy (lung cancer, lymphoma, squamous cell carcinoma, sarcoma), autoimmune disease (rheumatoid arthritis, granulomatosis with polyangiitis, ankylosing spondylitis, histiocytosis) [1-5].

We present the case of a patient with cavitory pulmonary disease on plain chest radiography, an entity with a challenging diagnosis in the context of rich pathological history and significant family history.

Received: July 2018; Accepted after review: December 2018; Published: December 2018.

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Case report

A 78-year-old non-smoker female patient was transferred to our clinic from a regional



hospital with the suspicion of a right broncho-pulmonary neoplasm. The patient presented to the hospital seven days prior to admission in our clinic with dry cough and sudden onset, grade IV on the modified Medical Research Council (mMRC) dyspnea scale, fatigue and palpitations; she was hospitalized in the cardiology department for massive bilateral pulmonary embolism confirmed by computed tomography (CT), followed by a treatment with intravenous heparin in continuous infusion. The patient was then transferred to our department in an influenced, but stable state. There was no recorded history of fever, hemoptysis, weight loss, tuberculosis exposure or venous thrombosis prior to admission.

The patient has a personal history of malignancy, breast cancer, for which the patient underwent with chemo- and radio-therapy and surgery three years prior the current admission, uterine fibromatosis operated six years before - total hysterectomy and bilateral oophorectomy), with no relapse of breast cancer and no hormonal treatment. The complex cardiovascular pathology included past paroxystic atrial fibrillation, ventricular extrasystolic arrhythmia, aortic atherosclerosis, NYHA class II heart failure, grade III tricuspid regurgitation, grade I mitral regurgitation, low pulmonary hypertension, previously treated with propafenone/ amiodarone and acetylsalicylic acid, currently under treatment with bisoprolol 2.5 mgX2/day. She had 5 pregnancies, only one delivered on term, the others recorded as spontaneous abortions. Her family history highlighted the death of her father at the age of 66 due to broncho-pulmonary neoplasm, two deceased brothers with lung cancer and a sister deceased probably due to cardiovascular complications.

Upon admission, patient's clinical examination revealed influenced general state, pallor, multiple bruises on the upper limbs, abdomen and a second grade decubitus ulcer on the right external malleolus. On inspection, two straight post-quadrantectomy scars were

present on the anterior side of the right hemithorax. Her breathing rate was 15 breaths/minute, with dullness of the basal right hemithorax on percussion and abolished vesicular breathing sounds at the base of the right hemithorax on auscultation. The saturation of peripheral oxygen (SpO₂) was 92%. Recorded cardiac rhythm normal, at 80 beats/minute and the blood pressure was 108/62 mmHg. On admission, the performed lab tests included a white blood cell count of 10480/ μ L, erythrocyte sedimentation rate (ESR) of 140 mm/1 hour, thrombocytosis of 525000/ μ L and normocytic normochromic anemia with Hb of 10 g/dl, urea of 10,1 mg/dL, creatinine of 0,55 mg/dL, glycemia of 89,1 mg/dL, ALAT of 19,5 U/L, ASAT of 19,6 U/L, total protein count of 69,26 g/L were also recorded. The patient had previously received antibiotic treatment with amoxicillin / acidum clavulanicum 1.2 g every 12 h for seven days.

The chest CT revealed lesions suggestive of pulmonary embolism in the distal segments of the right pulmonary artery and the proximal left inferior lobar artery, a cavitary image with anfractuous wall in the upper right lobe, right basal pleural fluid (Figure 1). Vertebral bone hemangiomas were identified at D7 and D8 level.

The chest radiograph on admission revealed an area of hyperlucency containing air-fluid level in the lower 1/3 of the right lung area and an area of opacity in the lower 1/2 of the right hemithorax, with concave superior interface border (Figure 2).

The pleural ultrasound performed on the second day of admission showed a finely septated pleural effusion of approximately 3.5 cm depth, which maintained the same aspect until the 13th day of hospitalization. Diagnostic thoracentesis was performed, revealing an exudate of uncertain origin.

The ear, nose and throat examination performed on second day of admission revealed paresis of the left recurrent laryngeal nerve, with no identifiable primary upper respiratory tract tumor.

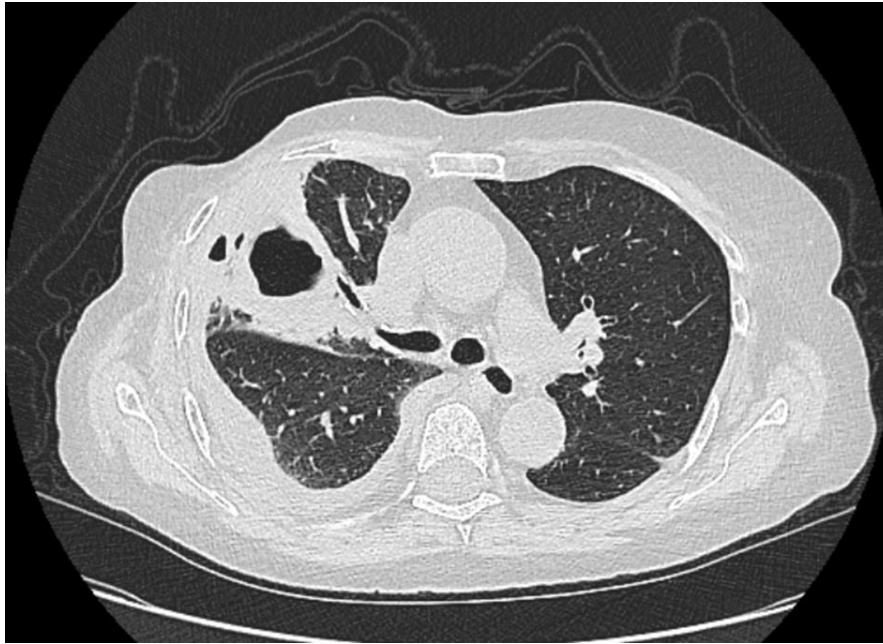


Fig. 1. CT scan image showing a thick-walled cavity. Right basal pleural fluid.

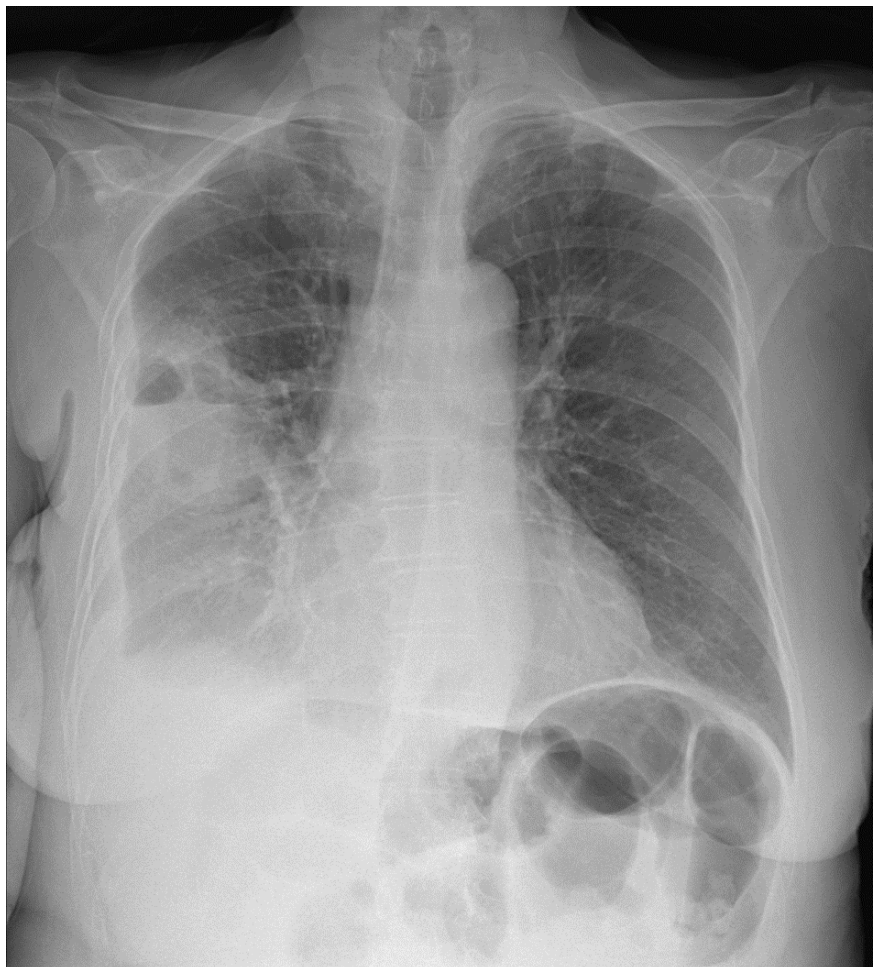


Fig 2. Hyperlucent area containing an air-fluid level in lower 1/3 of the right lung and chest opacity in lower 1/2 of right hemithorax, with concave superior interface border.

The fibro-bronchoscopy examination revealed purulent secretions of white-viscous appearance adherent to the bronchial mucosa in the upper right lobe, probably from the middle lobe or basal pyramid. Samples were sent to the laboratory for cultures on specific media for common bacteria, *Mycobacterium tuberculosis* and fungi. Bronchial aspiration cytology noted the presence of a moderate inflammatory reaction with neutrophils, Gram positive bacteria, with cultures revealing methicillin-sensitive *Staphylococcus aureus* (3+). The patient's antibiotic therapy was then adapted according to the antibiogram. She was started with 1g of ceftriaxone every 8 hours (intravenous administration) and 500 mg of metronidazole every 6 hours (per os) for five days, later switched on 600 mg of clindamycin every 8 hours (intravenous administration) for 10 days, followed by per os administration for two weeks after discharge.

The patient's condition improved slowly. Under targeted antibiotic regimen, oxygen therapy, low molecular weight heparin therapy, cough suppressants, and analgesia, her clinical,

biological and imaging parameters steadily improved.

With adequate wound dressing and topical cutaneous creams, the decubitus ulcer healed. Follow-up examination revealed the following parameters: white blood cell count of 7010/ μ L, hemoglobin of 12.7 g/dL, platelet count of 323000/ μ L, ESR of 88 mm/1, urea of 18.6 mg/dL, creatinine of 0.52 mg/dL, glycemia of 109 mg/dL, ALAT of 12.5 U/L, ASAT of 7 U/L, total protein count of 75 g/L.

She was discharged of the 15th day of hospitalization with recommendations for two weeks of antibiotic therapy, continuous anticoagulation and regular check-ups. The follow-up at one, two and three months showed steady improvement, confirmed by imaging studies. Chest x-ray showed the accentuation of the peribronchovascular interstitium in 1/3 of the right hemithorax at three months (Figure 3) and the persistence of a lower left lobe arterial thrombus, with complete medium lobe artery obstruction on CT scan.

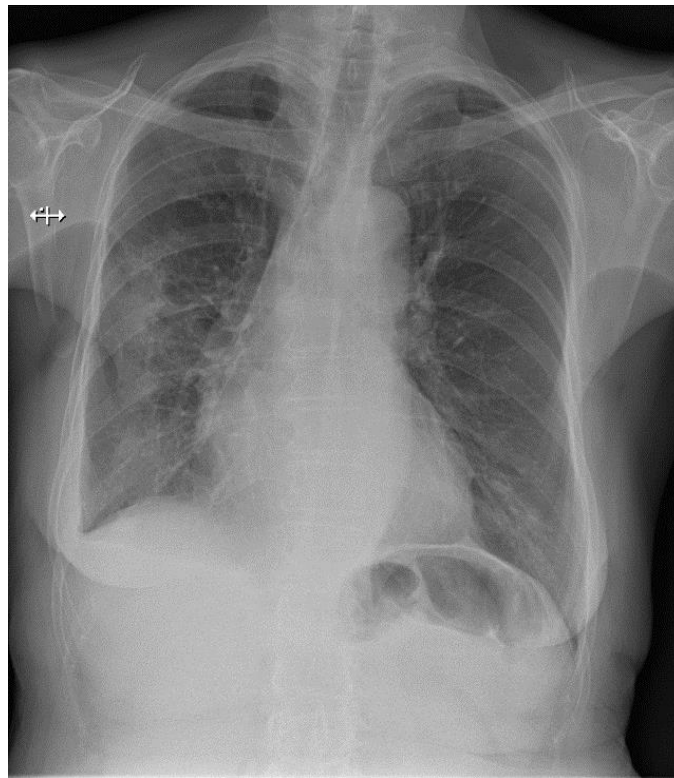


Fig. 3. Chest radiography at three months. Accentuating peribronchovascular interstitium 1/3 of the right lung area.

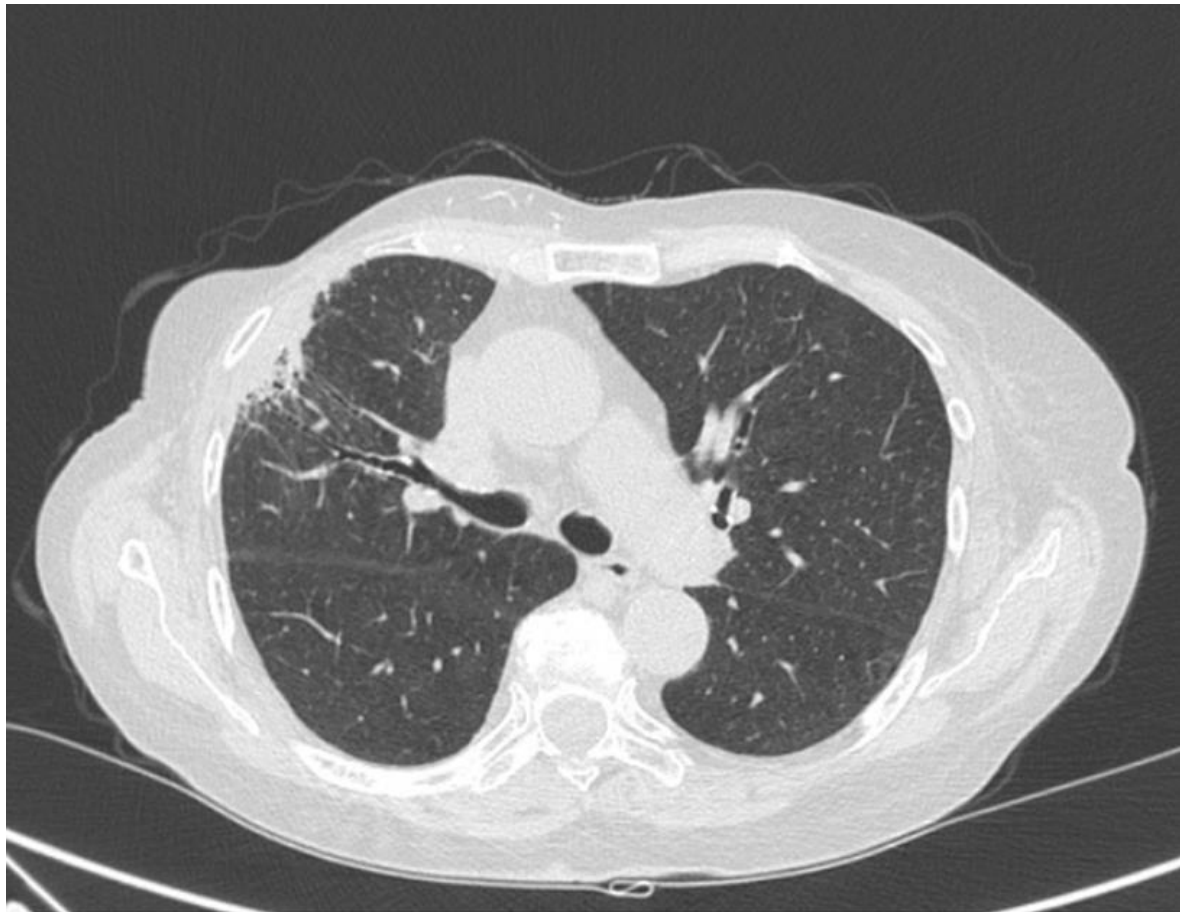


Fig. 4. Chest CT scan at three months. Resolution of cavity. Right pachypleuritis.

The topography of the former abscess showed a small residual air-filled cavity communicating with the bronchial tree and bronchiectasis in the medium lobe. Ground-glass appearance at the level of the left basal pyramid and the right posterior basal segment were observed (Figure 4). At three months, the blood parameters showed normal complete blood count, ESR of 47 mm/1h, normal renal and liver function. At seven months after discharge the patient's general condition significantly improved.

Discussions

In regards to cavitary pulmonary disease, although radiological imaging may be highly suggestive of a diagnosis, only through integrating the clinical context and targeted paraclinical investigations can one achieve a complete and correct diagnosis.

Before approaching a cavitary lesion, clinicians must exclude the great mimics of

such an entity, such as cysts, emphysema bullae, infected lung bullae, cystic bronchiectasis [1, 2].

Recent studies propose an algorithm to address cavitary pulmonary disease, in respect to the duration of clinical symptomatology and the radiological findings, as well as the supplementary investigations. Thus, acute and subacute cavities (<12 weeks) may be determined by common bacteria that produce lung abscess, necrotizing pneumonia, septic emboli and fungal infections [2].

Hemocultures in patients with septic emboli frequently highlight staphylococcus, *Fusobacterium necrophorum*, *Klebsiella pneumoniae* and *Streptococcus viridans*. Among the more common fungal infections present, literature notes the ones caused by *Cryptococcus neoformans* and *Aspergillus species*.

When facing a lesion such as an acute cavity, clinicians should always take a detailed history, accompanied by parameters such as a

complete blood count, procalcitonin, blood culture, sputum culture and integrate the resulting data with the CT scan features of the lesion. The same conduct applies in the case of a chronic cavity, albeit a biopsy should be done if the complete diagnosis remains unclear [2].

Chronic cavities (> 12 weeks) can be determined by three categories of disease: chronic infection, malignancy and autoimmune disease. Chronic infections include *Mycobacterium tuberculosis*, nontuberculous mycobacteria (with a particular risk group such as elderly women without structural pulmonary disease), chronic necrotic aspergillosis, histoplasmosis, blastomycosis, paragonimiasis, echinococcosis and tracheobronchial papillomatosis. A cavity wall >15 mm thick on chest X-ray is highly suggestive for malignancies (in 90% of the cases) [1, 4, 6]. The autoimmune disease most likely leading to the formation of a pulmonary cavitory lesion is granulomatosis with polyangiitis, with 40-70% of patients having nodular lung lesions and 25-50% of these cases, in which nodules are >2 cm presenting with cavities. In 20% of patients with rheumatoid arthritis, imaging studies highlight lung rheumatoid nodules, which may develop central necrosis. The main differential diagnosis in all mentioned cases is lung cancer [2, 5].

The patient history for the presented case raised the suspicion of an acute cavity, although the diagnosis of bronchopulmonary neoplasm could not be initially ruled out. *Staphylococcus aureus* is rarely isolated in cultures from such lesions; it may be found in patients with a certain background, and causing necrotizing pneumonia, a rare but very severe diagnosis with some cases originating from uncomplicated skin and soft-tissue infections [7, 8]. The most prevalent microorganisms isolated are *Haemophilus influenzae* and *Streptococcus pneumoniae* followed by *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*. Community acquired pneumonias and cavities caused by *Staphylococcus aureus* are uncommon and usually occur after influenza or from staphylococcal bacteremia [9-11]. In our case, a possible source of can point in the direction

of the decubitus ulcer, from which samples for culture were not taken.

A non-malignant cavitory lesion can mimic malignant lesion on most radiological examinations. Also, for pulmonary thromboembolism, the causes are various, including a state of hypercoagulability induced by sepsis. Virchow's triad suggests that the pathogenesis of venous thrombosis consists of stasis (stagnant flow), vessel wall damage, and disorders of coagulation (hypercoagulability). Damage to vessel wall can be due to infection, infiltration or trauma. However, the more common issues are coagulation defects that cause a hypercoagulability state which can result in sudden death from silent emboli [12].

The three month follow-up CT scan confirmed one of our hypotheses, namely the presence of bronchiectasis, which is a predisposing factor for pneumonitis. Appropriate long-term treatment was indicated. Anticoagulant therapy initiated by continuous intravenous infusion was replaced with subcutaneous treatment, required for lifetime. At the six month follow-up, the presence of thrombophilia (factor V Leiden, plasma antithrombin, protein C, and protein S activity measurements) can be assessed, given the patient's history. However, further findings will not change the current treatment or prognosis of the patient [13].

Conclusions

Identifying the etiology of a cavitory pulmonary disease is a challenge due to the wide range of factors and large variety of clinical manifestations. A particular diagnosis can be favored in the context of clinical and radiological data, but the patient must always be fully investigated, following all diagnostic paths in order to exclude other causes.

Consent

Written informed consent was obtained from the patient for publication of this case report.

Competing interests

The authors declare that they have no competing interests.



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