

Cavitation Pulmonary Noninfectious In An Immunocompromised Host

A Sainz, L Navarro

Citation

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Abstract

Cavitary lung disease can be caused by a wide variety of pathologic conditions. Possible etiologies include infection, metastatic malignancies, septic pulmonary emboli, granulomatous vasculitides and rarely pneumoconioses and pulmonary sequestration (1,2). Cavitation resulting from bland (non-infected) pulmonary infarction is often not considered in this differential diagnosis (1,2,3,4). Cavitation pulmonary infarction accounts for about 1%-1,5% of all cavitating pulmonary lesions (1,2). Lung infarction occurs in 10-15% of pulmonary thromboembolism and in only 5-7% of these does the infarction process progress to cavitation either by aseptic necrosis or from secondary infection with subsequent abscess formation (1,2,5). Pulmonary disease in immunocompromised hosts is common (6,7), but cavitary lung disease is less common and is frequently associated with a fungal or mycobacterial infection (9). We report a case of aseptic cavitary infarction in an immunocompromised patient associated with pulmonary embolism what it disappeared after anticoagulant treatment.

CASE REPORT

A 83-year-old man was admitted to the hospital with a 16 weeks history of progressively increasing breathlessness, and leg swelling which inhibited him to walk. There was no history of cough, sputum or chest pain. He had a medical history of polymyalgia rheumatica 6 years before admission, high blood pressure and non-insulin-dependent diabetes mellitus. He had received prednisone 30 mg/d, digoxin 0,50 mg/d five times a week, enalapril 20 mg/d and furosemide 40 mg/day. On physical examination, he was tachyneic with a respiratory rate 26 breaths per minute. His temperature was 38°C; blood pressure was 120/50 mm Hg and the pulse rate was 100 beats per minute. The jugular venous pressure was normal. The heart examination revealed systolic blow.

The lung examination revealed crepitations in over the right lower zone. Examination finding of other systems and neurologic test were normal. Blood test gave the following results: white blood cell count 10900/ μ L (68 neutrophils), the hemoglobin level was 9 g/dL, hematocrit 28%; VCM 85 fl and the platelet count was 164×10^3 / μ L. An erythrocyte sedimentation rate was of 121 mm/h; C-reactive protein were 6,7 mg/dl (normal range 0,0-0,5). Glucose 121 mg/dL; creatinine 1,7 mg/dL. Serological markers for HIV virus and blood culture were negatives. Other laboratory investigations performed at his admission at hospital included electrolytes and liver function tests, coagulation studies and serum

protein electrophoresis were within all normal limits.

Results of arterial blood gas analysis on room air showed the following: pH 7,43; Pa CO₂ 46; Pa O₂ 65; HCO₃Na 30; Saturation 93% Electrocardiogram shown sinus rhythm a 100 beats per minute and left bundle branch block. Transthoracic echocardiography showed only left ventricle hypertrophic with adequate left ventricular ejection fraction. His chest radiograph showed cavitary lung lesion in the medium lobe (Figure 1). Subsequent computed tomography of the chest revealed a 4,5 cm cavitary lesion in the middle lobe which had developed progressively in pleura, bronchial and vessels. No associated mediastinal adenopathy was evident (Figure 2). An empirical treatment with intravenous cefotaxime 2 g every 6 hours, intravenous clindamycin 500 mg three times a day and subcutaneous low-molecular-weight heparin at a dose of 1mg/kg/12 hours was started.

Figure 1

Figure 1: Chest roentgenogram of 84 year-old man demonstrating cavitory pulmonary lesion in the medium lobe.



Figure 2

Figure 2: Computed tomography, showing a 4,5 cm cavitory lesion in the middle lobe, which affects to pleura, bronchial and vessels.



A bronchoscopy and a Doppler echocardiography of his legs were done. The bronchoscopy did not show any endobronchial lesion and test of bronchoalveolar lavage fluid for cytology, bacteria, mycobacteria, were negatives. Doppler echocardiography showed deep vein thrombosis in both iliac, femoral, and popliteal veins. On finished the antibiotic treatment and the oral anticoagulants were started. At follow-up 6 months later, the patient was asymptomatic and the cavitory pulmonary lesion and the deep vein thrombosis had disappeared.

DISCUSSION

Cavitory lung disease results from various causes (1,2,3,9). In our immunocompromised patient, infectious causes were certainly considered: mycobacterial, fungal, gram-negatives

necrotizing infection, anaerobic organisms were sought. Noninfectious causes such primary or metastatic neoplasms, pulmonary sequestration, granulomatous vasculitis must also be considered. However, in immunocompromised hosts, they would be of lower probability (6). Cavitory pulmonary infarction is rare, which produce significant morbidity and mortality (1,2,5,8,10).

The infarction is uncommon because the lung has two blood supplies, through the bronchial and pulmonary arteries (6). Our case, the computed tomography of the chest revealed invasion of the bronchial and pulmonary vessels for the lung cavitory lesion. After infarction occurs, cavitation can follow as a result of either aseptic liquefaction or secondary infection, which occurs by direct infection with bronchial organisms, aspiration of orally administered fluid, or hematogenous spread (5,9). Clinical symptoms and signs of cavitory infarct are nonspecific. Cavitory infarcts have been resected as suspected bronchogenic carcinoma, tuberculosis, complicated bacterial infection and anaerobic abscess (6,9,10). However, pulmonary infarction is other likely cause, overcoat in immunocompromised hosts (6,8,9). The appearance of a cavity in an infarcted lung, especially when a fluid level is present, raises the suspicion of abscess formation. However, the absence of symptoms of infection, as well as the negative sputum culture made de diagnosis of a septic process unlikely (9).

We think that aseptic necrosis with cavitory formation may not be as rare as been believed hitherto; it should be considered in the differential diagnosis of pulmonary cavitation.

CORRESPONDENCE TO

Araceli Jimeno Sainz. C/ Luis Guedea n° 19, 3A. 50300 Calatayud (Zaragoza). Spain Tfno +34-660298525 E-mail: ajimenos@salud.aragon.es

References

1. Libby LS; Kin TE; Lafroce FM; Schwarz MI. Pulmonary cavitation following pulmonary infarction. *Medicine*. 1985;64:342-348.
2. Griego MH; Ryan SF. Aseptic cavitory pulmonary infarction. *Am J Med*. 1968;45:811-816.
3. Booth H; Wildon J; Snow M. Bourke S. Cavitating lung lesion and a cerebral mass. *Chest*. 1995; 108: 1737-1738.
4. Lim KH. Tan LH. Liam CK. Mee-Ming Wong C. An unusual cause of secondary spontaneous pneumothorax in a 27-year-old man. *Chest*. 2001;120:1728-1731.
5. Vaideeswar P. Cavitory pulmonary infarction- a rare cause of spontaneous pneumothorax. *J Postgrad Med*. 1998;44:99-100.

6. Rosenow EC III. Wilson WR. Cockerill FR III. Pulmonary disease in the immunocompromised host I. Mayo Clin Proc. 1985;60:473-485.
7. Wilson WR. Cockerill FR III. Rosenow EC III. Pulmonary disease in the immunocompromised host I. Mayo Clin Proc. 1985;60: 610-631.
8. Scharf J. Nahir AM. Mank J. Lichtig CH. Aseptic cavitation in pulmonary infarction. Chest. 1971;59: 456-457.
9. Morgenthaler TI. Ryu JH. Utz JP. Cavitory pulmonary infarct in immunocompromised hosts. Mayo Clin Proc. 1995;70:66-68.
10. Aslam PA. Eastridge CE. Hughes A. Pulmonary cavitation after embolism. Int Surg. 1970;53:157-161.

Author Information

A. Jimeno Sainz, M.D.

Department of Internal Medicine, Hospital of Ernest Lluch

L. Guerrero Navarro, M.D.

Department of Internal Medicine, Hospital of Ernest Lluch