Pneumocystis carinii pneumonia and acquired immunodeficiency syndrome: an atypical presentation with lung cavitations

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ABSTRACT: The differential diagnosis of lung cavitations is very broad. We report a case of Pneumocystis carinii pneumonia (PCP) with lung cavitations on the chest X-ray in a patient with the acquired immunodeficiency syndrome (AIDS). We discuss the differential diagnosis of such an X-ray pattern and emphasize that multiple cavitations can be a roentgenographic presentation of PCP.


The roentgenographic pattern of Pneumocystis carinii pneumonia (PCP) is a diffuse pulmonary infiltration. This presentation has been described in approximately 80% of patients with the acquired immunodeficiency syndrome (AIDS) [1]. Unusual radiologic manifestations of PCP have been reported in up to 20% of cases, as lobar distribution, pleural effusion, atelectatic changes, hilar lymphadenopathy, pneumothorax, honeycomb appearance, and cavitations.

Although lung cavitations were not initially considered to occur in patients with AIDS and PCP [2], they represent a rare roentgenographic manifestation of PCP [3]. We report a case of multiple lung cavitations, which suggests that lung tissue destruction may occur during PCP [4].

Case report

A 30 yr old male intravenous drug addict, HIV positive for 2 yrs, was admitted to our hospital in 1989 for multiple lung cavitations. There was a past history of B-hepatitis in 1982, two episodes of bronchopneumonia in 1988, and a 12 kg body weight loss during the previous 5 mths. Two weeks before hospitalization, the patient presented with a productive cough, fever and nocturnal sweating. He progressively experienced dyspnoea and chest pain when coughing. The chest X-ray showed multiple thin-walled cavitations (fig. 1): a diagnosis of “multiple lung abscesses” was proposed, and the patient was treated with oral cotrimoxazole. This treatment was quite erratic due to poor compliance, and the patient was hospitalized because of worsening symptoms.

On examination the patient appeared thin. He had oral and genital candidosis, as well as right mandibular and

Fig. 1. – A: Chest radiograph showing thin-walled cavitations on the right middle and left upper pulmonary areas; B: computed tomography scan showing thin-walled cavitations with alveolar and interstitial infiltration.
bilateral inguinal lymphadenopathies. Temperature was 38°C, pulse 96 per min, blood pressure 130/70 mmHg, and respiratory rate 16 per min. Rales were heard over the right upper pulmonary area.

The haematocrit was 33%; the white cell count was 1,800 (therefore azidothymidine, which was prescribed since October 1988, was stopped), with 28% neutrophils, 14% band form, 14% monocytes, and 40% lymphocytes. The platelet count was 140,000·mm\(^{-3}\). Gamma-GT was 236 U·/·, alkaline phosphatase 242 U·/·, ASAT 84 U·/·, and LDH 663 U·/·. Immunoelectrophoresis showed increased immunoglobulin G (IgG) and IgA. CMV was negative for IgM but positive for IgG, the HIV serology showed positive anti-envelope antibody, negative anti-core, and positive p24 antigen; the CD4/CD8 ratio was 0.15 (n=1-2).

A fibreoptic bronchoscopic examination with bronchoalveolar lavage (BAL) was performed: neither *Pneumocystis carinii* nor fungi or mycobacteria were found. Blood cultures were negative. Echocardiography was unremarkable. Because of the presence of *Streptococcus pneumoniae* in the sputum, the patient received amoxycillin orally for 10 days. Three weeks later, a second bronchoscopic examination with BAL was performed, as the chest roentgenogram showed a progression of the pulmonary lesions (appearance of new thin-walled cavitations, size increase of pre-existent cavitations and an impressive progression of the alveolar and interstitial patterns). PC was identified in the bronchial lavage fluid, and cytological and bacteriological examinations, including mycobacterial cultures up to 8 wks, failed to reveal the presence of any other pathogen. The patient was therefore treated with intravenous cotrimoxazole. After a 10-day treatment, gastrointestinal side-effects appeared: cotrimoxazole was replaced by intravenous pentamidine for a further 11 days. The pulmonary symptoms improved and the interstitial and alveolar pattern decreased; the size of cavity lesions on chest X-rays remained unchanged. The patient was discharged after 50 days without any residual respiratory symptoms. One month later, a control chest X-ray showed a decrease in size of the cavitations.

**Discussion**

Many aetiologies may cause pulmonary involvement with cavitations in patients with AIDS. In the present case, an embolic problem (i.e. thromboembolic disease, aspetic embolus after drug injection) may be reasonably excluded. Furthermore, there is no evidence for vasculitis. As far as we know, there is no convincing description of cavitating tumours in patients with AIDS.
in the literature. Only infectious causes should therefore be considered here, i.e. mycobacteria (tuberculosis or atypical mycobacteria), anaerobic abscesses, septic emboli (staphylococci, streptococci, Gram-negative bacteria) and fungi. Our investigations failed to demonstrate any of these pathogens.

PC was the only microorganism that could be identified in BAL, which was a surprising finding, as cavitation is not a classical roentgenographic pattern of PCP. PC may be found in BAL of patients with AIDS after treated PCP without any apparent harmful consequence [5]. However, without a previous history of PCP, the presence of PC is considered as responsible for pulmonary pathological changes [1]. To our knowledge, there is no study demonstrating that PC may be present in the BAL of patients with AIDS without pulmonary involvement and pathological consequences.

The association between PC and cavitations has already been described, but it seems to be rare: up to now, only 18 cases have been described in the literature [3, 4, 6–11]. Sixty cases of PCP in patients with AIDS have been reported in our institution: our case is the only one with thin-walled cavitations without air-fluid levels. These cavitations related to PC are similar to those reported in the literature [3, 4, 6, 8–10].

By reviewing the only seven well-documented case reports in the literature (table 1), we were not able to determine if there is a specific clinical pattern of PCP with cavitations contrasting with the “classical” PCP.

The response to cotrimoxazole treatment, in our case, does not seem to differ from that in other cases of PCP. Therefore, although the pathogenesis of such pulmonary lesions (i.e. cavities) is still undetermined and even though their X-ray aspect suggests that PC may be an invasive microorganism that destroys lung parenchyma [4], this does not necessarily imply that a treatment failure and a poor prognosis are to be expected.

In conclusion, this report adds one more case to the 18 other previously reported cases in the literature. It also confirms that no particular clinical or laboratory patterns are related to PCP cavitary lesions; finally it demonstrates that this particular form of PCP responds quite well to standard PCP treatment regimens.

References