Pulmonary histoplasmosis

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Introduction

Histoplasmosis is a systemic mycosis associated with numerous clinical syndromes, the most frequent of which is an influenza-like respiratory infection, asymptomatic or self-limited; however, it may present as chronic cavitary pulmonary disease, progressive disseminated disease affecting multiple organs, or immune-mediated disease of the mediastinum or the eye. Its causative agent is *Histoplasma capsulatum*, a dimorphic fungus with two varieties: *H. capsulatum var. capsulatum* (responsible for classic histoplasmosis) and *H. capsulatum var. duboisii* (the cause of African histoplasmosis).

The disease is acquired by inhaling dust particles from soil contaminated with bird or bat droppings, which contain small spores, the infectious form of the fungus.

The patient usually presents with cough, fever, malaise, weight loss and hilar adenopathy, with or without one or more areas of pneumonitis; occasionally, however, pathological cervical and axillary lymph nodes may be present, as well as erythema nodosum and other types of *rash*.

Histoplasmosis is typical of rural, tropical and temperate areas, and is also found along rivers and in regions rich in caves. In Brazil, the Amazon stands out as the most important area.

Histoplasmosis and tuberculosis may coexist in the same patient and, often, it is not possible to differentiate them radiologically. It is frequently associated with COPD (chronic obstructive pulmonary disease) and with situations in which cellular immunity is altered, such as infection with the human immunodeficiency virus, the use of corticosteroids or cytotoxic substances, and secondary to organ transplantation. Clinical disease in immunocompetent patients is rare, and its presentation outside rural settings is uncommon. The case reported here concerns pulmonary histoplasmosis in an urban area, affecting an individual known to be HIV negative.

Case presentation

D.R.P., male, 59 years old, white, married, born in Graúna (SP), living in Santo André for ten years, doorman at a Wolkswagen dealership for ten years. Chief complaint of chest pain for a year and a half. The patient reported that, about a year and a half earlier, a pain began in the sternal region, radiating to the whole chest, stabbing, of moderate to severe intensity, related to the left lateral decubitus position

and unrelated to physical exertion, accompanied by cold sweating, without nausea or vomiting, sometimes improving with aspirin, lasting 24 hours and with progressive worsening of intensity. Daily afternoon fever of 39 °C, profuse sweating in the early morning hours, with a weight loss of 1 kg in this period.

Six months earlier he had seen a cardiologist, where he underwent an ECG, a chest radiograph and blood tests. When he returned after 15 days, an abnormality was found on the chest radiograph, and he was advised to take aspirin (ASA) and to see a pulmonologist for investigation; chest CT, bronchoscopy with lavage and biopsy were inconclusive. In January 2000, he underwent a thoracotomy with nodule biopsy, with a diagnosis of chronic pulmonary histoplasmosis. He also reported shortness of breath, wheezing and occasional dry cough. He denied *diabetes mellitus*, hypertension, heart disease and lung disease. Hospital admission about 30 years earlier for a rib fracture (hit by a vehicle). He denied other admissions and treatments. He worked in the fields as a child; in a fireworks factory and a ceramics factory as an adolescent; twenty years at the Ford factory, in the paint shop (paints + solvents), and for ten years as a night watchman at the Volkswagen dealership. Removal of a nodule from the right leg 40 years earlier, but he did not know the histological type of the lesion. Smoker for 45 years, one and a half packs a day. He had stopped two months earlier. He did not drink alcohol. Mother died at 74 years of age of ischemic heart disease, and father at 76 years for the same reason. Brother with heart disease (50 years old). Healthy children.

On physical examination he was in good general condition, brevilineal, skin intact, adequate subcutaneous fat, no palpable lymph nodes, normotrophic musculoskeletal system, scar on the medial aspect of the right leg, blood pressure 120x70 mmHg. Vesicular breath sounds present, with mild rhonchi at the base of the left hemithorax. Regular heart rhythm, normal heart sounds, no murmurs. Distended abdomen, mild pain on palpation of the left flank. Conscious, oriented, no motor or sensory deficit. No edema; interdigital lesions on the right foot. With the diagnosis in hand, we were able to sharpen the history and arrive at the source of the infection. The patient, a night watchman at a car dealership, slept during his shift on a piece of cardboard that was stored where bats had free access to it.



Figure 1. Plain chest radiograph showing a diffuse, bilateral infiltrate in the alveolar interstitium, predominating in the middle-lower third, associated with poorly defined oval areas of increased density.

Bony thorax intact. A diffuse, bilateral alveolar infiltrate is observed, predominating in the middle-lower third, associated with poorly defined oval areas of increased density, of a nature yet to be clarified, which may be nodules. Costophrenic sinuses and diaphragmatic domes clear. Elongated aorta. Slight increase in the cardiothoracic ratio.

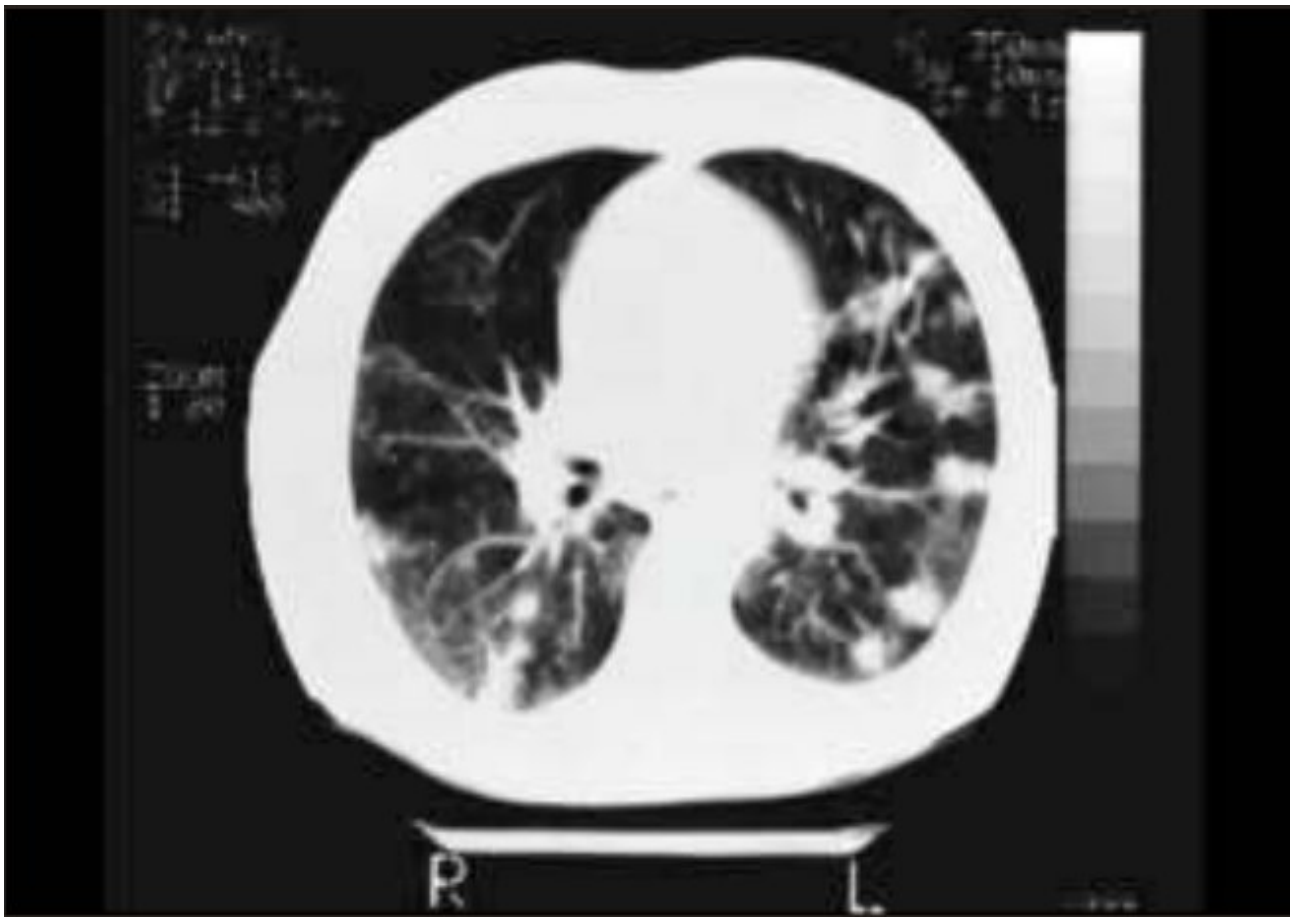


Figure 2. Computed tomography of the chest showing multiple nodular opacities in the parenchyma of both lungs.

The computed tomography study of the chest, performed without intravenous injection of iodinated contrast, with 10 mm thick axial slices at 10 mm increments, shows: extrathoracic soft tissues without abnormalities; no free fluid in either pleural cavity; trachea in the midline; main bronchi of normal caliber; ascending aorta, arch and descending aorta of normal morphology and dimensions; mediastinum in the midline, with no evidence of masses or lymph node enlargement; multiple nodular opacities with irregular contours, of varying sizes and with dense strands, peripheral, in the parenchyma of both lungs; hila preserved bilaterally; no pathological calcifications.

Conclusion: multiple nodular opacities in the parenchyma of both lungs (secondary?).

Biopsy

Two biopsies were performed, one on 26/11/99 and the other on 5/1/00. The result of the first, transbronchial biopsy, with testing for acid-fast bacilli (BK) and fungi, was: bronchial wall with mild chronic inflammatory reaction; lung parenchyma with swelling of the alveolar lining cells, mild fibrosis and mild chronic inflammatory reaction; no granulomas. Testing for fungi and acid-fast bacilli with special stains was negative, with no signs of malignancy. The stains used were hematoxylin-eosin, Ziehl-Neelsen and methenamine silver.

The result of the second biopsy, of the right nodule of the right lung, was: no signs of malignancy; histoplasmosis in lung tissue; round fungi about four micra in diameter, stained by the methenamine silver technique; the fungi are located inside granulomas formed by Langhans-type giant cells and epithelioid cells. There is diffuse fibrosis.

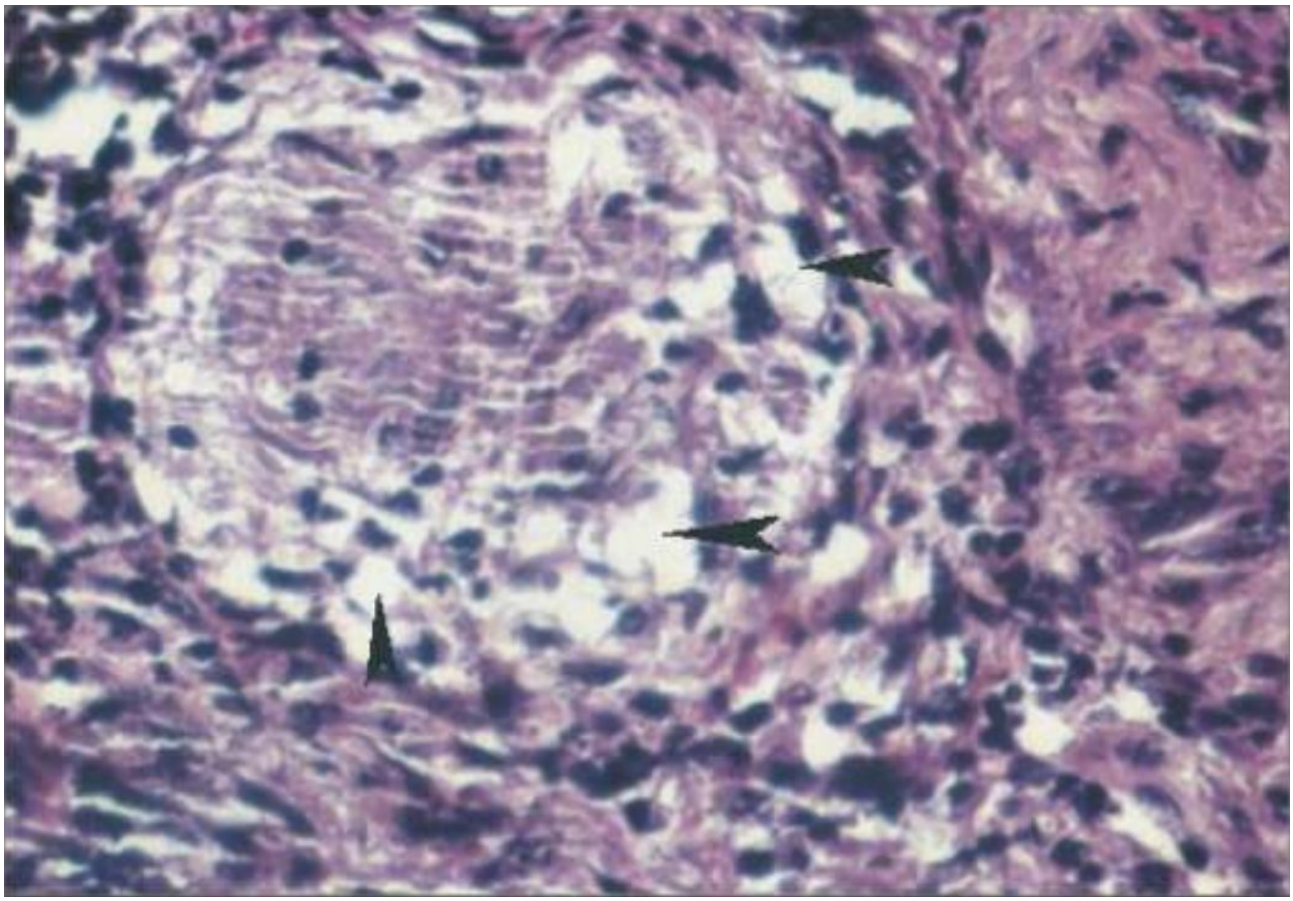


Figure 3. HE: core of a granuloma, where unstained structures can be seen.

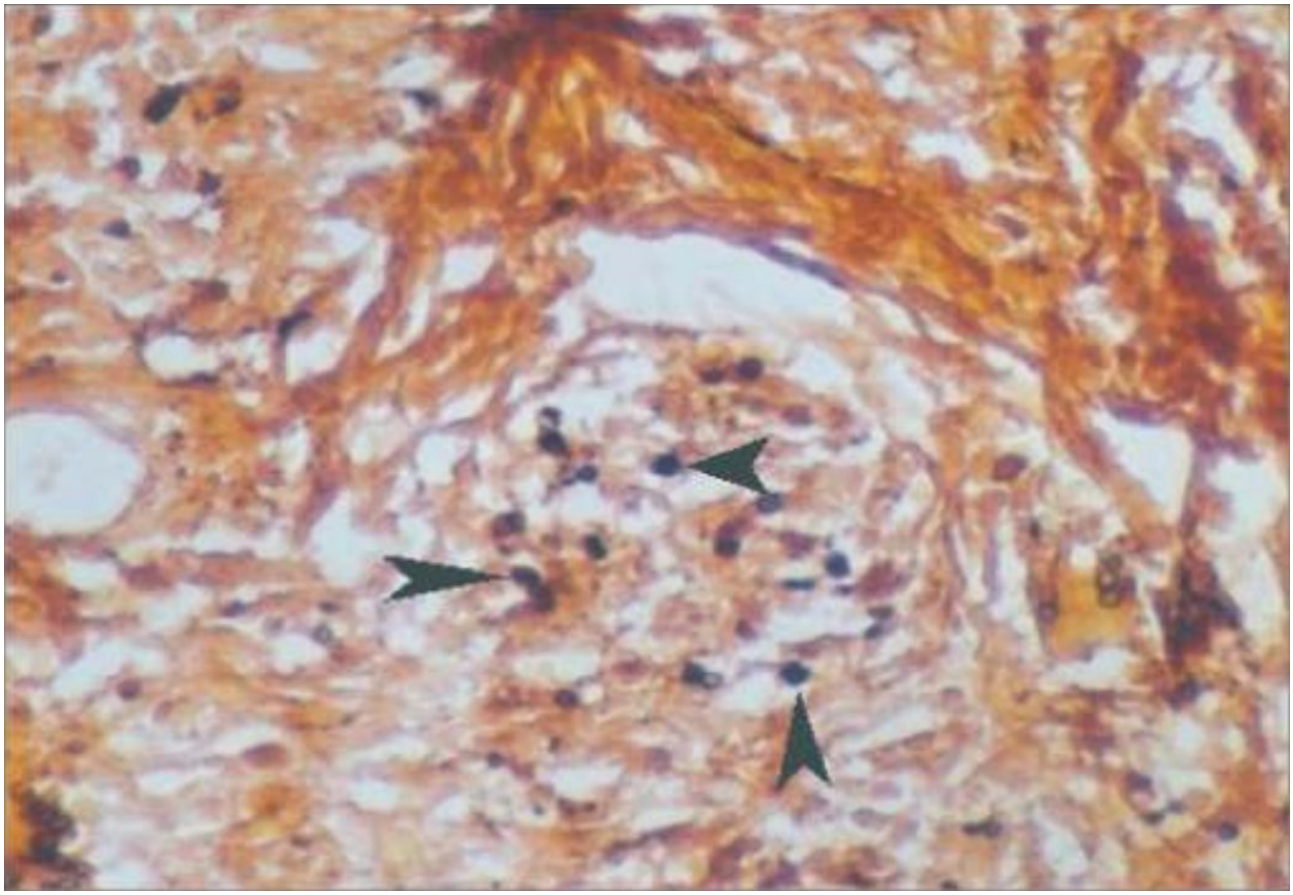


Figure 4. Methenamine silver: in this granuloma, structures stained brown, which are the fungi, can be seen.

Case discussion

In the biopsy of the right lung nodule, we can see round fungi about four micra in diameter, stained by the methenamine silver technique. This is consistent with the literature, which describes these fungi as ranging in size from two to five m. The fungi are located inside granulomas formed by Langhans-type giant cells and epithelioid cells. We know that *Histoplasma* infections produce epithelioid cell granulomas, which supports the diagnosis.[12]

Biopsy is essential to rule out other differential diagnoses, such as coccidioidomycosis, tuberculosis and sarcoidosis.[12]

On plain radiography, according to the current consensus, in the chronic pulmonary form there may be pleural involvement, fibrosis and contraction of the affected lobe or segment, with changes in the diameter of the chest and mediastinal shift. Involvement of the lymph nodes adjacent to the bronchi by histoplasmosis can cause collapse of the middle lobe or of other lung segments. The posterior apical segments are affected and cavitation is common, often persisting for a long period. These persistent cavities often enlarge gradually and may become very large. Involvement of the adjacent lung is common, and fibrosis may become extensive.[4] In this case, however, we did not find these changes.

Ill-defined patchy or nodular lesions are scattered throughout both lungs, which matches the case reported.[4]

According to the literature, computed tomography (CT) can show nodules located in the intralobular and interlobular regions of the lung, which may have a perivascular distribution. CT is an excellent method for demonstrating the effects of mediastinal fibrosis.[8] In this case, we observed multiple nodular opacities with irregular contours, of varying sizes and with dense peripheral strands, in the parenchyma of both lungs. Hila preserved bilaterally and no pathological calcifications. We did not see mediastinal fibrosis, but the nodules described in the literature are present.

Affiliations

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