# Coccidioidomycosis in a 38-year-old man: a case report

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**Abstract:** The present article describes a case of acute pulmonary coccidioidomycosis in a 38-year-old man, a research worker. The disease started during the patient stay in Arizona, USA, and clinical symptoms persisted after his return to Poland. Acute coccidioidomycosis is one the clinical manifestations of *Coccidioides immitis* strain endemic infections occurring in the south-western regions of USA including California (mainly San Joaquin Valley), Western Texas, New Mexico and the desert areas of Arizona, and Central and South America. The native environment of *Coccidioides immitis* is soil penetrated by rodents. People, domestic and wild animals suffer from coccidioidomycosis. The infection rate in endemic areas is about 2–4% a year in the healthy population. Coccidioidomycosis can be observed in non-endemic areas due to population mobility and in immunocompromised patients. The *Coccidioides immitis* infection is caused by inhaled airborne fungal spores and it may occur as primary pulmonary (acute or chronic) asymptomatic form, meningitis, or disseminated disease. The clinical symptoms of coccidioidomycotis like acute pulmonary manifestations may resemble typical, resistant to empiric antibiotic treatment of bacterial pneumonia. In healthy subjects, pulmonary coccidioidomycosis may occur as asymptomatic infection, which resolves spontaneously without medication. Sometimes, slight shadows like local fibrosis and cavities may be visible on the chest X-ray. The *Coccidioides immitis* infection in people with immunological deficiency syndromes, e.g. HIV/AIDS, manifests itself as disseminated disease and may lead to severe complications including death.

**Key words:** acute pulmonary, *Coccidioides immitis*, coccidioidomycosis

## INTRODUCTION

Coccidioidomycosis (San Joaquin fever, Valley fever) is caused by endemic fungi *Coccidioides immitis*. Endemic regions include southwestern USA with California (mainly San Joaquin Valley), western Texas, New Mexico and the desert areas of Arizona, and Central and South America [1]. Immunosupression patients and inhabitants of endemic regions constitute the infection risk group [2].

## CASE REPORT

A 38-year-old man, a research worker, was admitted to the Department of Lung Diseases at Medical University in Poznań, Poland, because of pulmonary coccidioidomycosis which was diagnosed during a 6-week stay of the patient in Arizona. The first clinical symptoms related to infection occurred 2 weeks after his arrival to Arizona. They were: severe fatigue, a high fever of up to 40°C, profuse night sweating, severe myalgia, headache, chest pain aggrevating during dry cough and deep breathing, skin hypersensitivity on the legs, and anorexia.

The patient had not had any health problems before the admission to hospital. The chest X-ray showed left lung pneumonia (interstitial infiltration in the left lower lung field) (Fig. 1). Laboratory examinations showed elevated blood leukocyte count (14.6 G/I). Nonsteroidal anti-inflammatory drugs (NSAID) as the treatment of choice, were administered. After 10 days chest pain subsided. However, high fever, cough, headache, anorexia and fatigue persisted. Blood leukocyte count decreased (12.2 G/I). Erythrocyte sedimentation rate was 63 mm after 1 hour. Serum titer of *Coccidioides immitis* antibodies in immunoglobulin M (IgM) class was 1:8. During next visits, a gradual drop in body temperature and less intense clinical symptoms were observed. After 4 weeks from the onset of the disease, laboratory examinations showed normal blood leukocyte count. The control chest X-ray revealed consolidation of inflammatory infiltrates in the left lower field. Despite normalization of body temperature (37°C), the patient suffered from cough and fatigue. In the course of the disease body weight loss about 5 kilograms was observed. At that time the patient returned to Po-

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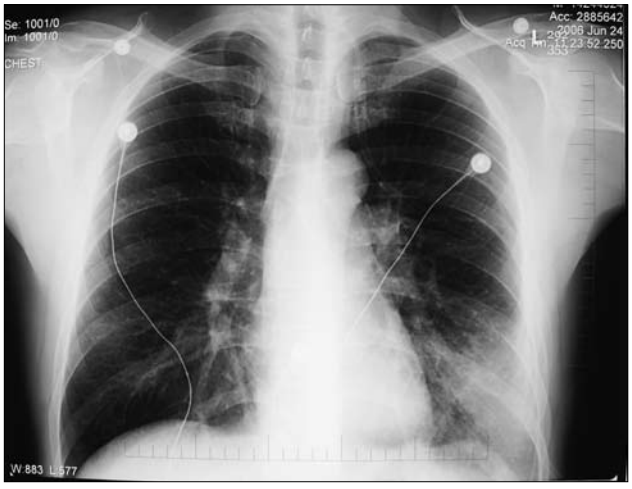


**Fig. 1.** Chest X-ray of a 38-year-old man with acute pulmonary coccidioidomycosis (from the archives of the Department of Lung Diseases, Medical University, Poznań)

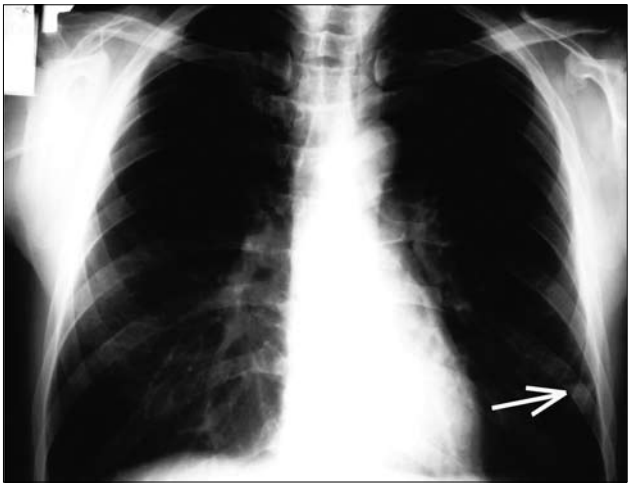

**Fig. 2.** Chest X-ray of the same patient taken several months later when the clinical symptoms of acute pulmonary coccidioidomycosis subsided, but the thin-walled cavity persisted (arrow) (from the archives of the Department of Lung Diseases, Medical University, Poznań)

land and was admitted to the Department of Lung Diseases at Medical University in Poznań. Slightly intensified fatigue persisted and skin hypersensitivity on the legs occasionally occurred. Physical examination showed no abnormalities. Symptomatic treatment with NSAID was continued. After several weeks, clinical symptoms disappeared and body mass increased by about 4 kilograms. The results of control laboratory examination (blood cell count and erythrocyte sedimentation rate) were normal. The titer of anti-*Cocciodioides immitis* antibodies was 1:16. The chest X-ray showed the cavitation and small areas of fibrosis in the left lower field (Fig. 2). At present, the patient is under control of the outpatient clinic and the follow-up was uneventful with unaltered abnormalities on the chest X-ray.

## DISCUSSION

Soil penetrated by rodents is the native environment for fungi of the *Coccidioides immitis* species. People and domestic and wild animals may suffer from fungi-related diseases. Inhalation of fungal spores leads to the infection [3,4]. The duration of incubation is about 7–21 days. In 60% of infected subjects clinical symptoms are absent. In about 40% of the subjects, pulmonary symptoms of acute coccidioidomycosis are present [5] and they resemble acute respiratory infection. As in the current case, high body temperature, dry cough, dyspnea, fatigue and pleural chest pain are the most common symptoms. In some patients, skin lesions like papulous erythema, multiform erythema and erythema nodosum may occur. Abnormalities revealed on physical examination are not specific. Rhonchi, wheezes and dull respiratory sounds may occur. In the majority of cases the chest X-ray is normal. Segmental or lobar infiltrations, in 20% of patients enlargement of the lung hila and pleural effusion, in 5% of patients singular or multiple nodular lesions, thin-walled or thick-walled cavities and enlarged lymph nodes of the mediastinum, as in the case described here, may be observed (Fig. 1). In the majority of cases radiological abnormalities resolve spontaneously or in 5–10% of subjects persist, like in the described case (Fig. 2). When the diameter of the cavity in the lung exceeds 6 cm, surgical resection ought to be considered because of the risk of the bronchial fistula formation. Diagnosis of acute pulmonary coccidioidomycosis is based upon a specific history of a patient, which confirms his/her stay in endemic areas, possible host defense mechanism injury, specific clinical symptoms, X-ray abnormalities and positive serological tests in previously healthy individuals. Serological tests in our patient are the commonly used diagnostic methods. The immunodiffusion method involves the measurement of IgM antibodies, which appear in serum 4 weeks after the infection and disappear about 2–6 months later. The detection of IgG antibodies, present in serum during the first 4–12 weeks of the infection, is available by the complement fixation test. Titers 1:32 or higher occur in about 60% of patients with disseminated disease. Repeated measurements of antibodies titer as the response to treatment and progression of the disease are useful. In difficult diagnostic cases microbiological examination is recommended. In the direct examination of sputum, bronchoalveolar lavage, joint and cerebrospinal fluid and sometimes the blood, the big, thick-walled endospores with small spheric bodies inside of them, are visible under the light microscope. Staining with the 10% KOH solution and Parker's ink, PAS-digestion (periodic acid Schiff reaction) and Grocott or Gram staining are used. The time of incubation of the culture is 2–7 days at the temperature of 25–30°C.

Like in the discussed case, acute pulmonary coccidioidomycosis most often resolves spontaneously. After several weeks clinical symptoms regress [6,7]. In these patients symptomatic treatment with NSAID is used. Severe acute pulmonary coccidioidomycosis which leads to development of chronic pulmonary or disseminated disease rarely occurs. This clinical condition concerns patients with host defense mechanism injury, for example HIV infected or diabetic people [8]. When clinical symptoms persist, i.e. chest X-ray radiological changes progress (visible enlargement of lung hila and lymph nodes of the mediastinum), and antibodies titer is elevated, anti-fungal treatment is recommended. Amfotericin B in a daily dose of 0.5–0.7 mg/kg body mass and after clinical stabilization 0.8–1.0 mg/kg body mass on every second day (total dose 0.5–1.5 g) is the treatment of choice. In the mild course of the disease, intraconazol in a daily dose of 200 mg or fluconazol in a daily dose of 400 mg during 2–6 months is recommended [5]. Although coccidioidomycosis is an endemic disease which rarely occurs in Poland, it ought to be taken into account in the differential diagnosis of pneumonia resistant to standard medication, with small cavities visible in the chest X-ray. A detailed patient's history including a question about possible travels to endemic areas is important. Nowadays, the knowledge of rare in Poland endemic fungal diseases (including coccidioidomycosis) may be useful for general practitioners because of increased population mobility and number of immunocompromised, including HIV infected, patients.

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