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Brief Report

Rare presentation of *Candida albicans*: infective endocarditis and a pulmonary coin lesion

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Abstract We present a case of a rare association of infective endocarditis and a coin lesion in the lung caused by *Candida albicans*. The lesion disappeared after 6 weeks of treatment with 5 mg/kg/day amphotericin B.

Keywords: *Candida albicans*; infective endocarditis; pulmonary coin lesion; general cardiology

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FUNGAL ENDOCARDITIS IN CHILDREN IS MOST frequently attributed to *Candida* species, which account for 63% of reported cases. This infection usually affects the right side of the heart, most often the right ventricle.[1] Echocardiographic characteristics of intracardiac fungal lesions include bright, smooth margins with homogeneous echogenicity. Most involve the ventricular apex, atrial appendages, or intravascular catheters. Clinical signs of cardiac involvement depend on the size, location, and consistency of the mass. Small intracardiac thrombi often produce no clinical symptoms, and clinical suspicion can be delayed, as standard microbiological studies take about 3 days to establish a diagnosis of fungaemia.[2,3]

Echocardiographic findings of intracardiac vegetation associated with candidaemia and negative bacterial blood cultures are highly suggestive of *Candida* infective endocarditis.[4]

Candida pneumonia typically develops as local or diffuse bronchopneumonia. It spreads by endobronchial inoculation or haematogenous dissemination. At the beginning, it is quite difficult to differentiate nodular or diffuse infiltration from congestive heart failure and pneumocystis pneumonia. Less often, it may cause necrotising pneumonia, pulmonary mycetoma, or empyema.[5]

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We have not found in the literature that *Candida albicans* causes coin lesions in the lung; therefore, we report a 7-year-old boy with infective endocarditis and a pulmonary coin lesion caused by *C. albicans.*

Case

A 7-year-old boy, who underwent surgery with the diagnosis of tetralogy of Fallot at 5 years of age, was hospitalised for placement of a pulmonary bioprosthesis and tricuspid valve repair due to severe pulmonary and tricuspid insufficiency. The surgery and the postoperative course was uncomplicated and the patient was discharged at postoperative day 7. Intra-atrial reentrant tachycardia was observed 1 week after the discharge and the patient was rehospitalised, and β-blocker therapy were initiated. The patient converted to sinus rhythm within 24 hours; however, fever developed 2 days later.

On physical examination, the patient was conscious and cooperative. Body temperature was 39°C, respiratory rate was 45/minute, and respiratory sounds were normal. Laboratory results were as follows: total leucocyte count: 15,070/μL; haemoglobin: 8.2 g/dL; platelet count: 357,000/μL; erythrocyte sedimentation rate: 50 mm/hour; C-reactive protein: positive (9 mg/dL). Electrocardiography showed normal sinus rhythm. Two vegetative areas running from the apex of the right ventricle towards the outflow tract were detected on transthoracic echocardiography (Fig 1, Supplementary video 1). Blood

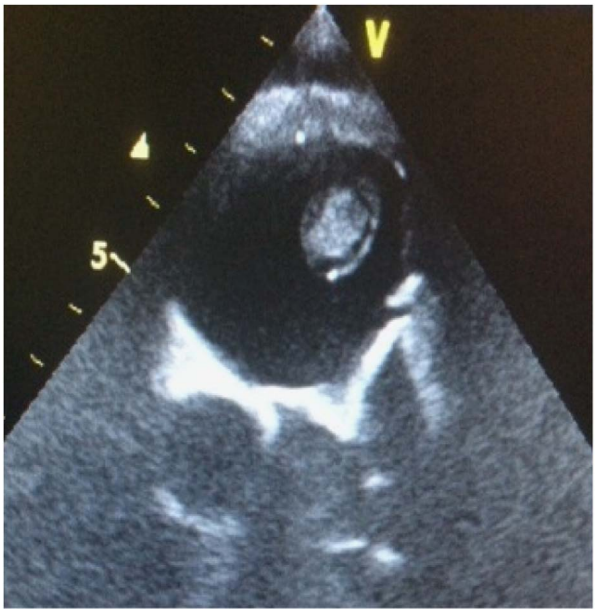

Figure 1.
Echocardiographic image showing vegetation in the right ventricule.

cultures were obtained and non-specific antibiotherapy (vancomycin and amikacin) was initiated. Rifampicin was added 5 days later owing to the lack of response. However, the fever persisted. An increase in the vegetative areas was observed on echocardiography. *C. albicans* was isolated in a blood culture and fluconazole was added according to the antibiogram susceptibility. Non-specific treatment was discontinued 3 days later. However, fever persisted and 3 mg/kg/day amphotericin B was added. *C. albicans* was isolated in a second blood culture, and one of the vegetative areas detached and localised within the pulmonary bioprosthesis. The size of the vegetation increased on day 5 of treatment, and the fever persisted. As a result, the patient underwent a second surgery. The vegetation and the bioprosthetic valve were removed, and a pulmonary homograft was implanted. Amphotericin B therapy was continued at a dose of 5 mg/kg/day postoperatively. Body temperature was subfebrile on postoperative day 3, and tachypnea and cough developed in the patient. A coin lesion was detected in the left lung on a follow-up chest X-ray (Fig 2). Body temperature returned to normal and clinical recovery was observed during postoperative week 1. As *C. albicans* was isolated in the bioprosthesis, which was surgically removed, it was planned that the treatment would continue for 6 weeks. No additional problems were observed during the follow-up, and the coin lesion in the left lung disappeared at the end of postoperative week 5. Treatment was continued for 6 weeks, and after that the suppression treatment started with 5 mg/kg/day fluconazole, and it was

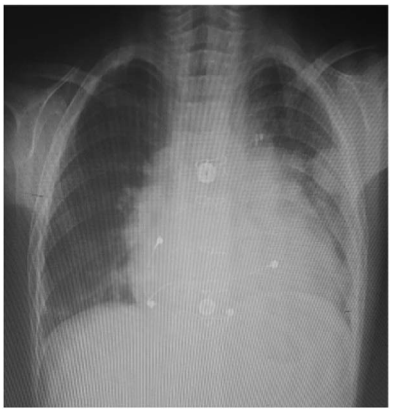

Figure 2.
Chest X-ray showing a coin lesion.

recommended that it be continued lifelong.[6] The patient was uneventfully discharged on day 42.

Discussion

The most common cause of fungal infective endocarditis is *Candida* species, and *C. albicans* is the most common agent. To date, six clinical risk factors associated with *Candida* endocarditis have been described, including underlying valvular disease, drug abuse, chemotherapy in patients with a malignancy, prolonged use of intravenous catheters, and underlying bacterial endocarditis. About half of the cases were admitted following cardiac surgery.[5] It occurs more frequently in patients with systemic candidiasis or in those with a central venous catheter.

Candida species can affect all layers of the heart; however, it frequently affects the endocardium with high mortality and morbidity rates. It frequently involves the aortic and mitral valves, as well as prosthetic valves. Valvular involvement can rarely be visualised using two-dimensional echocardiography. The diagnosis is difficult and embolisation, obstruction of large arteries, necrosis, and microabscesses can develop owing to endocardial involvement. Although the mortality rate was about 90% before surgical methods were used, this rate has decreased to 45% with the use of surgical treatments and sophisticated antifungal therapies.[5]

Candida pneumonia typically develops as local or diffuse bronchopneumonia. It spreads by endobronchial inoculation or haematogenous dissemination. At the beginning, it is quite difficult to differentiate nodular or diffuse infiltration from congestive heart failure and pneumocystis pneumonia. Less often, it may cause necrotising pneumonia, pulmonary mycetoma, or empyema. Mycetoma with air crescent and increased fibrotic changes is a round to oval-shaped mass of fungi situated within a cavity in the lung. Mycetoma with this feature is separated

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from coin pneumonia.[7] Radiographic findings are non-specific and the incidence of respiratory colonisation is high. *Candida* species can also cause bronchial infections, laryngitis, epiglottitis, and infection of the laryngeal prosthesis.[5]

Prosthetic valve endocarditis due to *C. albicans* is an extremely severe condition that develops following nasocomial candidaemia. One patient was treated with amphotericin B and fluconazole for 16 months without any need for surgical treatment.[8] A fungal ball is rarely detected in implantable cardioverter defibrillators[9], but several *C. albicans*-related cases have been described, including embolic materials and a fungal ball in the left atrium[10], pansinusitis due to a fungal ball[11], and pulmonary mycetoma in a case of uncontrolled diabetes and tuberculosis that resolved with antifungal therapy.[12]

In conclusion, infective endocarditis and a coin lesion in the lungs caused by *C. albicans* is a rare condition and this case showed that it resolves completely after medical therapy directed for the agent.

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Conflicts of Interest

None.

Supplementary material

To view supplementary material for this article, please visit https://doi.org/10.1017/S1047951117002645

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