Fungal Endocarditis Caused by Pseudallescheria (Petriellidium) boydii in an Intravenous Drug Abuser

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We present a case of fungal endocarditis in a 42-year-old man with a history of intravenous drug abuse who required aortic valve replacement for severe aortic insufficiency. Cultures of the resected valve grew Pseudallescheria boydii. The patient subsequently developed persistent endocarditis of the prosthetic valve with systemic embolism. At autopsy, cultures of the prosthetic valve grew Monosporium apiospermum, an anamorph of Pseudallescheria boydii. Although fungal endocarditis is not uncommon in intravenous drug abusers, endocarditis caused by Pseudallescheria boydii is rare. It has been reported in only three other instances, and under circumstances that were different from ours. (Texas Heart Institute Journal 1987; 14:321-324)

Key words: Endocarditis, fungal; Pseudallescheria; aortic valve; substance abuse

SEUDALLESCHERIA boydii, formerly known as Petriellidium boydii and Allescheria boydii, is a soil-inhabiting true fungus and is the most common cause of mycetoma in the United States and Europe. The fungus can also on occasion be cultured from P. boydii infections of the lung, 1 cornea, 2 ear, 3 sinuses, 4,5 and meninges.^{6,7} The infection can be introduced or disseminated by surgery and other invasive therapeutic procedures. The fungus resists conventional antifungal agents such as amphotericin B but is susceptible to miconazole.

Until now, only three cases of fungal endocarditis attributable to P. boydii have been described in the literature. We report a fourth case, which involved a patient with a history of intravenous drug abuse who had not previously undergone cardiac surgery or invasive therapy.

CASE REPORT

A 42-year-old man with a history of intravenous drug abuse underwent urgent aortic valve replacement for severe aortic insufficiency. Cultures of the excised valve grew Pseudallescheria boydii, for which the patient was treated with miconazole for 5 weeks. He was later readmitted because of persistent fever and was felt to have recurrent fungal endocarditis on the prosthetic valve. He then developed left-sided hemiparesis, thought to be secondary to septic emboli. Repeat aortic valve replacement was ruled out because of inadequate myocardial tissue for attaching the valve. The patient's clinical course was complicated by renal insufficiency, respiratory difficulty, a deteriorating mental status, and

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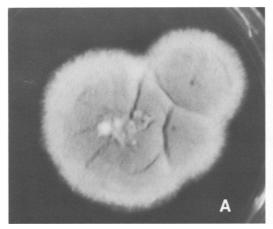
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Fig. 1 Occlusion of the prosthetic lumen by mycotic thrombus.



Fig. 2 Spores and branching hyphae of P. boydii (silver stain x200).



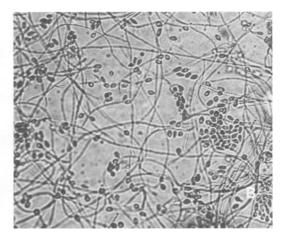


Fig. 3 (A) Fast-growing flocculent colony of tan to gray-white Monosporium apiospermum. (B) Slide from culture, showing branching septate hyphae with terminal pyriform conidia (cotton blue, x200).

an episode of cardiac arrest from which he was resuscitated. Despite efforts to stabilize his condition, he suffered a second cardiac arrest, and resuscitation attempts were unsuccessful. Interestingly, blood cultures had remained negative throughout his course.

At autopsy, the patient was seen to be a well-built, apparently well-nourished, younglooking man, weighing 77 kg and measuring 172 cm in length. The heart was grossly enlarged and weighed 900 g. The left ventricle was hypertrophied, with a thickness of 2.5 cm. The most important finding, however, was massive vegetation on the prosthetic aortic valve and a thrombus that measured 1.25 x 1.25 cm; together, these lesions almost completely occluded the aortic orifice (Fig. 1). The suture line, although intact, was covered by necrotic debris. Histologic sections of the thrombus and vegetation revealed abundant septated fungal hyphae with conidiophores and conidia (Fig. 2). Cultures of the same material grew Monosporium apiospermum, the asexual form of P. boydii (Figs. 3A and 3B).

Sections of the myocardium revealed focal pleomorphic infiltrates composed of lymphocytes, plasma cells, and eosinophils, but no hyphae. The lungs weighed 2,300 g. They were edematous and showed severe, chronic, passive congestion on microscopic examination.

Other significant findings included multiple infarcts of the kidney, spleen, and brain, caused by fungal emboli.

DISCUSSION

Although fungal endocarditis was almost unknown before 1940, it is no longer a rarity.8 Some of the factors that have contributed to its increasing incidence include cardiac surgery, 9,10 especially prosthetic valve placement, 11,12 cytotoxic drugs, and narcotic addiction. 13,14 Fungal endocarditis is particularly common following cardiac surgery or in association with use of infected central venous catheters, 15 and in immunocompromised hosts. Candida and Aspergillus species have been responsible in most recorded cases.

The first completely documented case of Candida endocarditis was reported in 1940 in a heroin addict. 16 Aspergillus valve endocarditis was reported in 1950, in a patient with rheumatic heart disease who was taking large doses of penicillin for a leg wound.¹⁷ Since then, fungal endocarditis has been identified with increasing frequency, and a variety of species in addition to Candida species, Aspergillus species, and Histoplasma capsulatum have been implicated.

Three cases of fungal endocarditis caused by Pseudallescheria boydii have previously been described. The first, which was reported in 1977 by Roberts and associates, 18 involved a 48-yearold Indian man who underwent porcine valve insertion for mitral stenosis and developed endocarditis of the prosthetic valve. He died 2 months later, and postmortem cultures of the porcine valve grew P. boydii.

In 1980, Davis and colleagues¹⁹ described a case of fatal P. bovdii endocarditis of the tricuspid valve in a 62-year-old woman with mixed connective tissue disease, who was receiving corticosteroid therapy. Massive vegetations nearly obliterated the tricuspid valve orifice, encasing a pacemaker catheter inserted 8 years earlier.

The most recent case, reported in 1985 by Gordon and Axebod, 20 involved a 52-year-old man who underwent aortic valve replacement for rheumatic heart disease and whose prosthetic valve subsequently developed a combined infection that involved both P. boydii and Clostridium limosum. The infected valve was replaced, but the patient died on the second postoperative day.

Our case is significant because P. boydii endocarditis originated in a previously healthy aortic valve not subjected to any cardiac surgery or procedure, and because the patient had a history of intravenous drug abuse. Although the organism is ordinarily susceptible to miconazole $(>1.0 \mu g/ml)$, this infection persisted.

As in other types of fungal endocarditis, the treatment of choice is surgical resection and débridement of infected tissue, in addition to treatment with antifungal agents. All the cases of P. boydii endocarditis reported so far, however, have had a fatal course.

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