is still uncertain whether treatment failures (recurrences) can be solely attributed to SCVs. Because the entry criteria included S. aureus–positive cultures, only patients whose gentamicin bead therapy failed were included.

To determine whether gentamicin beads should be used or avoided and how often SCVs are selected by gentamicin beads will require a large prospective study. Nevertheless, our data should alert physicians to consider SCVs when a treatment failure occurs in a patient who has received gentamicin beads. Furthermore, the fact that S. aureus SCVs play a role in osteomyelitis suggests that clinical laboratory personnel should carefully search for these organisms and that the clinician may need to alter therapy when they are present. Because SCVs are more resistant to aminoglycosides than S. aureus strains with normal phenotypes, failure to isolate the SCV strain leads to a major reporting error (i.e., the parent strain is susceptible, whereas the nonrecovered SCV is resistant).


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Phaeoacremonium parasiticum Infective Endocarditis Following Liver Transplantation

Phaeoacremonium species, formerly known as Phialophora species, are distributed worldwide, and infection usually occurs through traumatic inoculation. P. parasiticum has a predilection for infecting immunocompromised hosts [2], but infection in apparently immunocompetent hosts has been described [3, 4]. The clinical manifestations of P. parasiticum infection include subcutaneous abscesses and acute or chronic arthritis; there has been one case of disseminated infection [3–5]. Infective endocarditis and fungemia due to P. parasiticum have not yet been described. We report what we believe to be the first case of P. parasiticum (formerly Phialophora parasitica [1]) endocarditis and fungemia in the literature.

A 45-year-old man with liver failure secondary to alcohol abuse underwent orthotopic liver transplantation (OLT) in July 1995. Posttransplantation complications included respiratory failure, renal failure, venous thrombosis, wound dehiscence with ascitic fluid leakage, and bilateral pleural effusions. The immunosuppressive regimen included cyclosporine, azathioprine, and prednisolone. Infective complications included Klebsiella oxytoca bacteremia, Staphylococcus epidermidis bacteremia, and a pleural space Candida humicola empyema.

A second OLT was performed on day 55 of hospitalization because of allograft failure. Cytomegalovirus viremia was detected the next day and was treated with ganciclovir. On day 57, a lesion with a central brown-black eschar and surrounding induration was noted in the web space between the thumb and index finger of the left hand (figure 1). Microscopic examination of the fluid showed phaeoid fungal elements. A cottony-white mold grew on Sabouraud dextrose agar; this mold darkened with age. The colony texture was velvety-to-funiculose with darkened margins. The isolated mold was identified as P. parasiticum (Phialophora parasitica [1]).

Figure 1. Lesion between the thumb and index finger of the left hand of a patient with Phaeoacremonium parasiticum infective endocarditis following liver transplantation. Note the central pigmentation with associated induration.

References

The phialides were brown, thick-walled, and acicular. They were 15–50 μm long with both prominent and inconspicuous funnell-shaped collarettes. The phialides showed percurrent proliferation. The conidia were 3–6 μm in length, hyaline, thin-walled, cylindrical to sausage shaped, and aggregated in slimy heads at the apices of the phialides.

The organism was identified as *P. parasitica* and confirmed by David Ellis at the Mycology Reference Laboratory of the Women’s and Children’s Hospital in Adelaide, Australia. In vitro testing revealed that the organism was susceptible to all antifungals except 5-flucytosine. Therapy with iv amphotericin B (0.5 mg/[kg⋅d]) was started immediately and continued until the lesion healed on day 87 (cumulative dose, 600 mg).

The patient’s second OLT was complicated by biliary obstruction, renal failure, intraabdominal infection, and bilateral pleural effusions. Blood samples collected on day 111 of hospitalization showed fungal elements in the aerobic bottle after 7 days of incubation, and the organisms cultured were subsequently identified as *P. parasitica*. Parenteral amphotericin B therapy was restarted and continued until day 150 (cumulative dose, 1,150 mg), after which it was replaced with oral fluconazole (400 mg postdialysis). One week later, *P. parasitica* was isolated from blood samples collected 7 days previously. Fluconazole therapy was replaced with itraconazole therapy (400 mg/d). Six additional blood cultures were positive over the next week. Intravenous therapy with amphotericin B (1 mg/[kg⋅d]) was then added to the itraconazole regimen. A transesophageal echocardiogram showed a large tricuspid valve vegetation and mitral and aortic valve vegetations. Therapy with amphotericin B (total dose, 1,800 mg) and itraconazole was continued until day 181, at which time active therapy was discontinued; the patient died 2 days later. Autopsy revealed a 3.5-cm vegetation on the tricuspid valve and smaller vegetations on the mitral and aortic valves, from which *P. parasitica* was isolated. Histological examination of autopsy specimens revealed fungal myocarditis, fungal pneumonitis, and fungal microabscesses in the kidneys.

The optimal therapy for *P. parasitica* infection is unknown, but complete surgical resection of small localized cutaneous lesions is probably the treatment of choice. Antifungal agents that have been used to treat *P. parasitica* infection, with variable success, include amphotericin B, 5-flucytosine, ketoconazole, and terbinafine [5].

In summary, we present a case of *P. parasitica* endocarditis, fungemia, and disseminated infection following liver transplantation. As there are increasing numbers of immunosuppressed patients, the incidence of invasive fungal diseases—including infections with phaeoid fungi—will likely increase.

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References


**Invasive Cryptococcosis in a Family with Epidermodysplasia Verruciformis and Idiopathic CD4 Cell Depletion**

Epidermodysplasia verruciformis (EV) is a rare autosomal recessive disease that was first described in 1922 [1]. Patients with EV present with extensive flat and pityriasis-like warts in sun-exposed areas such as the face and hands [1, 2]. The onset of EV usually occurs in young adulthood, and the disease is associated with depressed cell-mediated immunity and a propensity for transformation of the warty lesions to squamous cell carcinoma [3–5]. Several human papilloma viruses have been associated with EV [2–4]. Other opportunistic infections have not been reported in patients with EV. We describe a consanguineous family with seven children in which two of three siblings who had EV developed disseminated cryptococcosis.

A 25-year-old woman was admitted to the hospital because of a 6-week history of headaches and intermittent fever. Her medical history was unremarkable except for multiple warts of different sizes that had appeared on her face, hands, and forearms over the 3 years before admission. Physical examination revealed a fever (temperature of 38.8°C), skin lesions, and bilateral papilledema. Lumbar puncture yielded CSF with a WBC count of 87/mm³; the CSF cryptococcal antigen titer was 1:1,024. Cultures of blood and CSF yielded *Cryptococcus neoformans* variety *neoformans* serotype A. The patient was treated with iv amphotericin B. After 2 weeks, her treatment was changed to oral fluconazole (400 mg/d), but all her symptoms, including papilledema, recurred. The patient was cured only after receiving another 6-week course of amphotericin B and several more weeks of oral fluconazole therapy.

The patient’s older brother had been admitted to the hospital 9 years earlier (at age 23) because of a 2-week history of headaches. A CT scan of the brain revealed a posterior fossa mass. His medical history was also significant for multiple flat warts of various sizes that covered his forehead and arms. Cultures of brain tissue and CSF yielded *C. neoformans* variety *neoformans* serotype A. The patient received a 6-week course of amphotericin B therapy and completely recovered; he has been well ever since. Papillomavirus type 3 and 10 were detected by PCR of biopsy material from skin lesions.