The mold isolated from pleural fluid and blood of the patient produced velvety, white colonies on Sabouraud dextrose agar (Figure, panel B). D1D2 rDNA sequencing identified the mold as *E. parva*. Because we found no previous reports of *E. parva* disseminated infections, we sent the isolate to a reference laboratory (University of Alberta Microfungus Collection and Herbarium, Edmonton, Alberta, Canada). Using culture characteristics and internal transcribed spacer and D1D2 sequences, the laboratory identified the fungus as a novel *Emmonsia* species not yet formally described (Figure 1 in Schwartz et al. [1]; L. Sigler, University of Alberta, Edmonton, Alberta, Canada, 2016, pers. comm.). When grown on different culture media incubated at 30°C, the fungus lacked coenidia but formed helically coiled, yellow-brown hyphae (Figure, panel C). When incubated on potato dextrose agar at 35°C, the fungus converted into a yeast-like form: clusters of small, irregularly shaped cells extending into short filaments.

Antifungal susceptibility testing of the mold phase was performed at the Fungal Testing Laboratory, University of Texas (San Antonio, TX, USA). The following MICs were obtained: amphotericin B, 0.125 μg/mL at 24 and 48 h; caspofungin, 0.5 μg/mL at 24 h and 2 μg/mL at 48 h; voriconazole 0.125 μg/mL at 24 and 48 h; and posaconazole, ≤0.03 μg/mL at 24 and 48 h.

A literature review of human *Emmonsia* infections is challenging because these organisms have undergone multiple taxonomic revisions (2). Most reports of adiaspiromycosis base the diagnosis solely on the appearance of adiaspores in histopathologic specimens (5,6), and some published *Emmonsia* cases might have misidentified the causative organism (1).

Disseminated *Emmonsia* infection appears to be a separate clinical entity from adiaspiromycosis (1). Human adiaspiromycosis is primarily a self-limited pulmonary infection caused by *E. crescens*, which is not associated with immunosuppression or fungemia. Disseminated *Emmonsia*...