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Filamentous Basidiomycetes in the Clinical Laboratory

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Abstract

Filamentous basidiomycetes are difficult to identify in the clinical laboratory, mostly due to lack of sporulation, and their role as agents of fungal infection can be difficult to assess. More cases of infection with these agents are being reported as more laboratories gain proficiency with the recognition of their subtle morphologic features and the use of DNA-based methods for identification. Most infections occur in the respiratory tract and sinuses, although brain infection has been reported. Susceptibility testing suggests that these agents will respond well to azole drugs other than fluconazole.

Keywords

Basidomycete fungus; *Schizophyllumcommune*; *Hormographiella*; *Coprinus*; Fungal respiratory infections; Filamentous basidiomycetes

Introduction

The identification and assessment of filamentous basidiomycete fungi from clinical specimens has long been difficult. First, many of these isolates are sterile in the clinical laboratory. Their lack of sporulation renders them difficult to identify without molecular-based testing. Second, their pathogenicity is difficult to assess either because many of these species were recovered from non-sterile body sites or because the species involved are not routinely considered human pathogens.

A number of basidiomycete fungi have been reported recently as agents of human infection or as agents of hypersensitivity and allergic reactions. This paper is intended as a review of case reports and a discussion of the role of these agents in human disease. This review will not cover basid-iomycete yeasts such as *Cryptococcus* species.

Basidiomycete is a commonly used informal term for all members of the phylum *Basidiomycota*. They are further subdivided taxonomically into three subphyla, 16 classes,

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and 52 orders, and include organisms as diverse as mushrooms, stinkhorns and barberry-wheat rusts [1]. Basidiomycota reproduce sexually through the production of specialized club-shaped cells called basidia, in which basidiospores are produced through meiosis. There are many variations in their life cycles. Some basidiomycetes also reproduce asexually. In the clinical laboratory, most basidiomycete fungi are fast-growing, cottony to fluffy colonies, hyaline to slightly brownish in color, and may produce crystals [2]. Some genera also produce arthroconidia, which causes them to be mistaken for the thermally dimorphic ascomycete fungus *Coccidioides*. Most basid-iomycetes are tolerant to benomyl (2–10 μg/ml) and sensitive to cycloheximide, allowing them to be distinguished from *Coccidioides*, which is resistant to cycloheximide. Many basidiomycetes are sterile and will not sporulate in the laboratory, although some colonies may display clamp connections and/or fruiting bodies such as basidiocarps (mushrooms). A clamp connection is a hyphal structure allowing a connection between two cells, allowing transfer of daughter nuclei, and resulting in a dikaryon, or a cell with two distinct haploid nuclei.

Some basidiomycete fungi have been implicated in hypersensitivity syndromes such as fungus-associated chronic cough [3]. Ogawa et al. studied 17 patients with fungus-associated chronic cough. When challenged with an antigenic solution from the basidiomycete *Bjerkandera adusta*, 11 of these patients showed a positive response. They concluded that *B. adusta* may play a role in enhancing the cough severity of patients who are sensitized to this fungus. Another fungus, *Sporotrichum pruinosum*, was recovered in repeated culture of the respiratory tract of three patients with cough, dyspnea, and sputum production. Two of these patients also demonstrated hypersensitivity to *S. pruinosum* when challenged in a skin test. The authors concluded that this basidiomycete fungus could display pathogenic potential [4].

Probably the most notorious human pathogen in this group is *Schizophyllum commune*, which was first reported as a human pathogen many decades ago in a nail infection [5]. This hyaline fungus is widely distributed in the environment and can be found in decaying organic matter such as rotten wood. In the laboratory, it may produce basidiocarps containing basidiospores, and thus can be identified by recognition of these structures. However, many isolates do not produce these structures and manifest as hyaline nonsporulating molds. The types of infection caused by this fungus can range from asthma to brain lesion, and can occur in both immunocompetent and immunocompromised patients. In their 2013 paper, Chowdhary et al. reviewed the existing literature and found 71 reported cases of *S. commune* infection [6••]. Most cases [22] were sinusitis, followed by allergic bronchopulmonary disease [20], fungus ball [2], and asthma [2]. Only four cases were extrapulmonary, and were one case each of onychomycosis, brain abscess, meningitis, and palate ulceration. Most cases [33] were reported from Japan, followed by Iran [7], and United States [6••]. The authors ascribed the predominance of cases in Japan to greater awareness of the disease and the agent, although climate and weather factors may also be involved.

Ogawa et al. reported two patients who both demonstrated bronchial asthma due to *S. commune* [7]. In both cases, their elevated levels of specific *S. commune* IgG decreased during a course of antifungal therapy. A case of bronchopneumonia due to *S. commune* was

reported in a patient immunocompromised due to gastric carcinoma [8]. This patient was cured after 6 weeks of intravenous fluconazole. In their recent report, Chowdhary et al. reviewed 143 cases of allergic bronchopulmonary mycosis and found that *S. commune* was reported in 11 % of cases, mostly from Japan [9].

S. commune has also been reported as an agent of sinusitis. In one unusual case from Japan, a recipient of a cord blood transfusion for treatment of leukemia developed sinusitis 13 days after the transplant. This patient was successfully treated with 2 months of liposomal amphotericin followed by oral voriconazole [10]. Allergic fungal sinusitis caused by *S. commune* was reported in a young Korean girl who presented with nasal obstruction and purulent discharge. *S. commune* was recovered from culture of allergic mucin recovered at endoscopic surgery [11]. In another case from Korea, this organism was the causative agent of a sino-orbital infection diagnosed in an immunocompetent patient who presented with sinusitis and an orbital tumor [12]. Ethmoido-maxillary sinusitis caused by *S. commune* was also reported in an immunocompetent patient in France. The patient recovered well after sinus surgery and did not require antifungal treatment [13].

This organism can produce deeper respiratory infection. Two cases were reported from India, one of allergic-bronchopulmonary disease in a patient taking inhaled steroids, and one case of pulmonary fungus ball in a patient with a previous history of tuberculosis [6••]. *Schizophyllum commune* has also been reported as an agent of eye infections. A case of keratitis was reported from India [14], where the fungus was recovered in culture of corneal scrapings. Topical voriconazole and intraocular amphotericin B as well as surgery were required to treat the infection in this case.

The most serious manifestation of fungal infection with *S. commune* is brain abscess. Two cases have been reported in the literature. The first was reported in 1996, as a pulmonary infection that migrated to the brain [15]. The patient was originally misdiagnosed with lymphoma and steroid therapy was initiated. At presentation, culture of multiple bilateral lung masses revealed a white nonsporulating mold that was thought to be *Aspergillus* species. A magnetic resonance image (MRI) of the brain showed a right frontal lobe lesion, from which fungal elements thought to be *Aspergillus* were detected. The patient received amphotericin B, itraconazole, and fluconazole before expiring of respiratory failure and sepsis due to bacterial pneumonia. A white mold later identified as *S. commune* was recovered from lung and brain tissue at autopsy. The second case was reported from Austria [16]. A patient with diabetes presented with severe headache, and MRI revealed three abscesses in the right frontal brain and bilateral sinusitis. Sinus surgery was performed and fungal elements were detected. Culture of brain abscess drainage grew *S. commune*. Liposomal amphotericin was administered for 5 weeks, and the patient was discharged with oral posaconazole.

Another basidiomycete organism reported as an agent of human infection is *Hormographiella aspergillata*, the anamorph of *Coprinopsis cinerea* (formerly called *Coprinus cinereus*). This organism was reported in three invasive infections in leukemia patients [17]. All patients had pulmonary infiltrates, and one patient also had cerebral and ocular disease. All of these patients died despite antifungal therapy. A French group also

reported *H. aspergillata* infection in two neutropenic patients who developed pulmonary infections with this organism while receiving empiric caspofungin therapy for fungal pneumonia [18]. One patient died, but the other recovered after therapy was changed to liposomal amphotericin B. Further investigation showed that 19 breakthrough fungal infections had occurred in three French university hospitals while patients were receiving caspofungin [19••]. One of these infections was caused by *Hormographiella*. In these patients, caspofungin had been prescribed either as prophylaxis or therapy for various fungal infections. In a further case, a patient who had undergone allogeneic bone marrow transplant for treatment of leukemia developed a lesion on the right forearm. The lesion was empirically treated with caspofungin, and the lesion when biopsied 12 days later showed fungal elements. *H. aspergillata* was recovered in culture. Although the patient ultimately underwent surgical resection of the lesion and received treatment with amphotericin and voriconazole, she had persistent fever and ultimately expired from respiratory failure and septic shock [20]. In this case, the patient appeared to have developed the lesion prior to the administration of caspofungin, although the drug was used for empiric therapy. These case reports should serve as a caution to clinicians about the unintended consequences of administering echinocandin drugs to high-risk patients.

Several other basidiomycete genera have been reported as agents of infection. In a report from India, the wood rot organism *Ceriporia lacerata* was recovered from respiratory samples in four patients with preexisting lung damage caused by tuberculosis or chronic obstructive pulmonary disease [21]. In one case the fungus appeared as a commensal and probably did not cause disease. In the other three cases, the fungus was probably the agent of bronchopneumonia. Two patients received itraconazole, but the outcome could not be assessed because patients were lost to follow-up. A case of soft tissue infection was reported to be caused by the basidiomycete fungus *Phellinus undulatus* [22]. The organism was identified using DNA sequencing, as it did not produce any diagnostic features in culture. Another wood-decaying fungus, *Irpex lacteus*, was recovered from a pulmonary abscess in an immunosuppressed child [23]. The basidiomycete *Inonotus (Phellinus) tropicalis* was recovered from multiple specimens, including a bone marrow aspirate and paraspinal abscess tissue, in a patient with chronic granulomatous disease [24]. A case of pulmonary fungus ball due to *Perenniporia* species was reported from India, in a diabetic patient with a history of tuberculosis [25]. The patient was not treated with antifungals. Finally, a patient from India who was receiving an umbilical cord blood transplant for Hodgkin's lymphoma developed pulmonary and brain infection [26]. A white mold was recovered from bronchial washing and brain biopsy samples. The mold was identified as *Volvariella volvacea*, a paddy straw mushroom, commonly found in tropical regions of the world, including China and India. Despite aggressive treatment, the patient expired. It was assumed that the patient had been exposed to the fungus sometime earlier in India, and her profound immunosuppression permitted the fungus to begin replicating actively. All of these cases demonstrate that filamentous basidiomycetes can be agents of infection in compromised hosts. Patients can be compromised by immunosuppression or by physical changes to the lung, such as in damage caused by prior tuberculosis lesions.

Microbiology

Filamentous basidiomycetes are difficult to identify in the routine clinical laboratory. They usually present as white, orange, golden, or tan, floccose to wooly colonies [2, 27]. They grow rapidly at 25 °C and 37 °C, filling the plate in 2– 3 days. Some genera will grow at higher temperatures including 40–42 °C. Basidiomycetes are tolerant to benomyl [28], and sensitive to cycloheximide, although Sutton et al. report one *Inonotus* strain that grew in the presence of 0.04 % cycloheximide [24]. Because so many basidiomycetes are recovered from respiratory samples, one important requirement is to distinguish basidiomycetes from dimorphic fungi such as *Coccidioides*, *Histoplasma* and *Blastomyces*, which have similar colonial morphology. This can be determined because dimorphic pathogens usually grow in the presence of cycloheximide, and usually fail to produce a mold phase at 37 °C. A nonsporulating hyaline mold that grows at 37 °C, fails to grow on cycloheximide, and grows on benomyl agar can be suspected to be a basidiomycete. It is important to appreciate that many such isolates have been presumptively identified as *Aspergillus* species or other molds, and this may not be the case.

Some basidiomycetes will produce morphologic characteristics that provide clues to their taxonomic origin. Many strains of *Schizophyllum commune* produce a distinctive bleach-like smell. Some isolates produce crystals, which can be seen on the culture plate. Some isolates will produce clamp connections or spicules on the hyphae, which can be seen microscopically, and some isolates may produce basidiocarps (mushrooms) after exposure of the culture plate to alternating cycles of light and darkness (culture slant can be left on the bench for several weeks). In a recent study Chowdhary et al. reported that only four of 26 isolates of *S. commune* produced basidiocarps after 4–5 weeks at 28 °C with exposure to periodic light [6••]. Some isolates (particularly *Bjerkandera adusta*) produce arthroconidia, which require attention to distinguish them from *Coccidioides* arthroconidia, usually by assessing growth on cycloheximide as described above. However, most isolates do not sporulate or produce any diagnostic structures allowing an identification to be made morphologically.

DNA-based methods are particularly helpful in confirming the identification of basidiomycete fungi. Usually the ribosomal ITS (intervening transcribed spacer) region is used for this purpose [29]. Pounder et al. sequenced the ITS region from 50 isolates of nonsporulating molds received in their reference laboratory, and identified five isolates of *Schizophyllum commune*, one isolate of *Coprinus* sp., and 32 isolates of other filamentous basidiomycetes using a 93 % identity cutoff for identification to the genus level [30]. A cautionary note was raised in a later analysis of ribosomal sequences [31••]. In this study, comparison of the BLAST results in the GenBank database for 168 ITS and ribosomal large subunit D1-D2 region sequences showed that the same species identification was demonstrated by both regions with only 48 isolates (28 %). At the genus level, only 82 isolates (48 %) showed the same genus identification using both regions. In 119 cases, (70.8%) the authors were unable to assign a conclusive identification at the species level. In many cases, either a corresponding ITS region or a D1-D2 region deposit in GenBank was lacking. Only 8 % of the isolates in this study had a complete ITS region deposit and only 10 % had a complete D1D2 deposit. Thirty percent of the species that were identified had either

an ITS or a D1D2 sequence that matched a deposit in GenBank, but not both. Other problems associated with the GenBank database, such as the continued appearance of incorrect or obsolete nomenclature, have been noted by others [32, 33].

Antifungal Susceptibility

The antifungal susceptibility profiles of basidiomycete fungi have been determined by several investigators. It should be appreciated that there is no standardized method for testing filamentous basidiomycetes, and no interpretive breakpoints exist. However, determination of minimum inhibitory concentration (MIC) values can provide some estimate of that drug's success in treatment, particularly if the MIC is very high. In an early study, 44 clinical isolates of basidiomycete fungi were tested using a modification of the macrobroth dilution method CLSI M-38P [34]. They found that the geometric mean (GM) for amphotericin, itraconazole, voriconazole, and posaconazole were consistently low, and the values for fluconazole and flucytosine were somewhat higher. Chowdhary et al. tested 30 isolates of *Schizophyllum commune* using a modified version of the CLSI M-38A2 microbroth dilution method [35••]. These isolates had a low geometric mean (GM) for amphotericin (0.29 μg/ml) and several azoles (itraconazole 0.2 μg/ml; voriconazole 0.24 μg/ml), but a high GM for fluconazole (19.39 μg/ml) and flucytosine (17.28 μg/ml). They also describe cases of invasive pulmonary infection and fungus ball that were successfully treated with voriconazole and/or itraconazole. In contrast, in some individual case reports, high MICs to itraconazole have been reported along with treatment failure [36]. It appears that, in general, amphotericin and azoles other than fluconazole may be useful for treatment of basidiomycete infections. It is not clear if different fungal genera show differing susceptibility to antifungal agents.

Conclusions

Filamentous basidiomycetes are common environmental organisms, and some genera have demonstrated pathogenic potential in humans. These organisms are usually agents of respiratory and sinus infections, reflecting inhalation as a portal of entry, and can cause infections in both immunocompetent and immunosuppressed hosts [37]. Basidiomycetes can also be recovered from respiratory sources without signs of infection, suggesting that these agents can also colonize the respiratory tree. Recent studies have suggested a link between use of caspofungin drugs and acquisition of basidiomycetes as agents of secondary infection in highly immunosuppressed hosts. These isolates are difficult to identify in the clinical laboratory because they usually present as hyaline nonsporulating molds with no specific diagnostic features. They can be confused with other hyaline molds, especially *Aspergillus* species. DNA sequencing tools are usually required in identifying these organisms, but public DNA databases may not contain comprehensive basidiomycete sequences. Treatment can be successful when azole agents other than fluconazole are used, with or without amphotericin B.

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