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Purpureocillium lilacinum as a Cause of Cavitary Pulmonary Disease: a New Clinical Presentation and Observations on Atypical Morphologic Characteristics of the Isolate

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The first case of cavitary pulmonary disease caused by *Purpureocillium lilacinum* is described. The isolate showed atypical microscopic characteristics similar to *Acremonium* and *Fusarium* spp., which necessitated molecular identification by sequencing of multiple conserved loci. The patient responded to voriconazole, reinforcing its therapeutic efficacy for *P. lilacinum* infections.

CASE REPORT

An 80-year-old asthmatic woman presented with a 3-week history of productive cough, associated with fever and pleuritic chest pain, which did not respond to a week of treatment with antibiotics and steroid therapy. She had a history of asthma, coronary artery disease, diabetes mellitus, hypertension, dyslipidemia, rheumatoid arthritis, and osteoporosis. Clinically, she presented with fever, tachypnea, tachycardia, and hypotension. Chest auscultation revealed bilateral scattered wheezes. The rest of the physical findings were unremarkable. Within 24 h of admission, her condition progressively deteriorated, requiring mechanical ventilation. As the patient was febrile, she was empirically started on ceftriaxone and clarithromycin. Her initial assessment revealed a normal white cell count, but the chest X-ray showed a consolidative lesion in the left upper lobe (LUL). The sputum culture showed heavy growth of *Pseudomonas aeruginosa*. Consequently, her antibiotic regimen was modified to include ciprofloxacin and meropenem for a course of 14 days, followed by a 3-week course of tazocin-ciprofloxacin. Despite an initial clinical improvement, the follow-up chest X ray showed persistence of the LUL lesion. Therefore, a computed tomography (CT) scan of the chest was performed on 7 October 2010, which revealed a cavitary lesion in the anterior segment of the LUL (Fig. 1A).

Since her condition deteriorated further, as assessed by her oxygen requirements, sputum specimens were collected on 11 and 18 October 2010 and were sent to the Mycology Reference Laboratory (MRL), Faculty of Medicine, Kuwait University. Both specimens showed septate hyphal elements when examined with calcofluor-potassium hydroxide (Fig. 2) and grew a white mold after 5 days on Sabouraud dextrose agar (Difco, Becton, Dickinson and Company, Sparks, MD). On 21 October 2010, a bronchoalveolar lavage (BAL) specimen from the LUL was obtained and sent to the MRL to establish the role of this white mold in the etiology of her cavitary lung lesion. The BAL specimen also showed septate fungal elements in the calcofluor-KOH mount (Fig. 2) and yielded a morphologically identical mold culture. The mold was provisionally identified as *Acremonium* or *Paecilomyces*. Although no defined antifungal susceptibility breakpoints exist for these organisms, the isolate demonstrated high MICs to amphotericin B (≥32 μg/ml), caspofungin (4 μg/ml), and itraconazole (≥32 μg/ml), suggesting resistance, but low MICs for posaconazole (0.5 μg/ml) and voriconazole (0.064 μg/ml), suggesting clinical efficacy. The patient was thus started on voriconazole, at 6 mg/kg of body weight at 12-h intervals for the first 24 h, followed by 4 mg/kg every 12 h. After 1 week of voriconazole therapy, the patient showed significant clinical improvement accompanied by regression in the size of the cavitary lesion (Fig. 1B). Since the patient continued to improve clinically, she was discharged on oral voriconazole (200 mg orally every 12 h). However, she died 2 weeks later apparently due to choking while she was being fed orally at home.

Colonies of our isolate (MF3411/10) on Sabouraud dextrose agar attained a diameter of 42 mm after 7 days of incubation at 30°C. The isolate showed restricted growth at 37°C, and it failed to grow at 40°C. Colonies consisted of a basal felt and sectors of floccose white aerial mycelium, which remained white after extended incubation. The colony reverse was colorless. Microscopic characteristics of the isolate are depicted in Fig. 3A to C. In slide culture at 30°C, straight or simply branched conidiophores of various lengths (6.2 to 52.0 by 1.8 to 2.6 μm) arising from the hyphae were observed. No verticillate branches, as seen in typical cultures of *Purpureocillium lilacinum*, were observed. Cylindrical, mostly *Acremonium*-like phialides, tapering toward the apex (3.2 to 22.4 by 1.5 to 3.2 μm) were observed (Fig. 3C). Hyaline conidia, mostly fusiform and ellipsoidal to subglobose and rarely globose (1.6 to 12.2 by 2.0 by 3.8 μm), were produced either singly or in clusters (Fig. 3B). Fusiform-shaped conidia were mostly formed on *Acremonium*-like conidiophores in a "slimy head" (Fig. 3A and C). The isolate has been deposited in the CBS-KNAW Biodiversity Center as CBS129077.

The Etest (bioMérieux, Marcy l'Etoile, France) was employed to determine MICs of five antifungal agents as described previously (17). Briefly, the test was performed on RPMI 1640 me-

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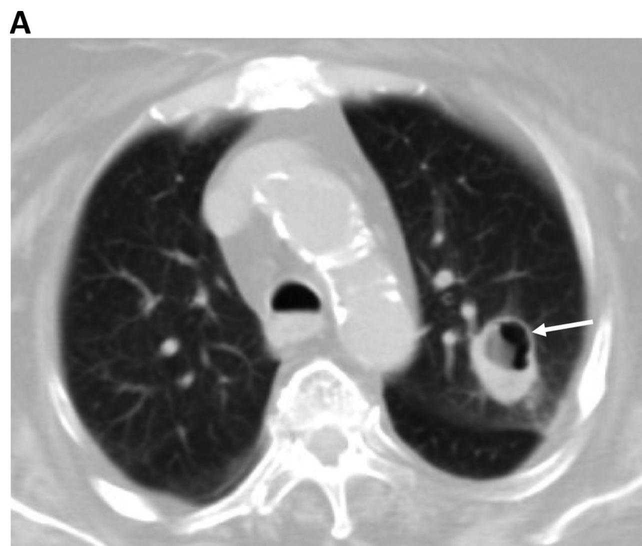
A



B

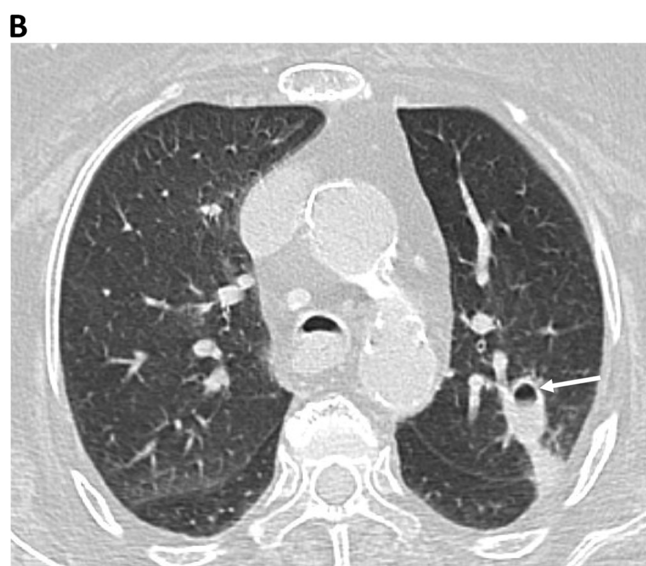


FIG 1 (A) Initial chest computed tomography (CT) imaging showing cavitating lesion in anterior segment of lung left upper lobe (arrow). (B) Follow-up chest CT image showing regression of cavitating lesion after 1 week of voroconazole therapy.

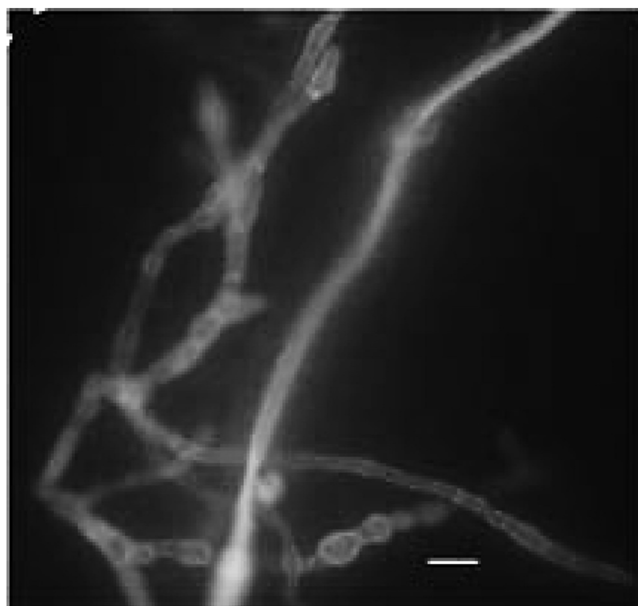


FIG 2 Potassium hydroxide (10%)-calcofluor (0.1%) mount of bronchoalveolar lavage showing septate hyphal elements. Bar, 5 μm.

dium supplemented with 2% glucose, and the pH was adjusted to 7.0 with 0.165 M morpholinepropanesulfonic acid buffer. The isolate was cultivated on a potato dextrose agar slant (Difco, Becton, Dickinson and Company, Sparks, MD) for 7 days at 30°C. Conidia were harvested in 2 ml sterile normal saline, and clumps were allowed to settle. The plates were inoculated by dipping a sterile swab into the conidial suspension and streaking it uniformly over the agar surface. Plates were allowed to dry at room temperature for 15 min before Etest strips were applied. MICs were read after 48 h of incubation at 35°C where the border of the inhibition ellipse intersected the scale on the antifungal strip. Microcolonies within the inhibition zone for caspofungin were ignored.

Genomic DNA from the patient's isolate (MF3411/10) was prepared as described previously and used as the template for PCR amplification (2). The divergent domains (D1/D2) of the 28S rRNA gene were amplified with the NL-1 and NL-4 primers, the internal transcribed spacer (ITS) region (ITS1, 5.8S rRNA, and ITS2) of ribosomal DNA (rDNA) was amplified with the ITS1 and ITS4 primers (3), and the variable region of the β-tubulin (*benA*) gene was amplified by using the BTUBF (5′-TGGTAACCAAATC GGTGCTGCTT-3′) and BTUBR (5′-GCACCCTCAGTGTAGT GACCCT-3′) primers, while the variable region of the calmodulin gene was amplified by using the Cmd5 (5′-GTCTCCGAGTACA AGGAGGC-3′) and Cmd6 (5′-TCGCCGATRGAGGTCATRAC GTG-3′) primers, and the amplicons were sequenced as described previously (3, 16, 17). The sequencing primers included BTUFS1 (5′-TAACCAAATCGGTGCTGCTTTCTG-3′) and BTURS (5′-C CTCAGTGTAGTGACCCTTGGC-3′) for the β-tubulin amplicon and CMDFS (5′-TCCGAGTACAAGGAGGCCTTC-3′) and CMDRS (5′-GATAGAGGTCATRACGTGRCGCA-3′) for the calmodulin gene fragment. GenBank basic local alignment search tool (BLAST) searches (http://blast.ncbi.nlm.nih.gov/Blast.cgi?CMD = Web&PAGE_TYPE = BlastHome) were performed for species identification.

The ITS sequence of our isolate exhibited 100% identity with the corresponding sequences from the type strain (CBS 284.36) and several other strains (ATCC 10114, CBS 431.87, CBS 432.87, CBS 226.73B, and CBS 100379) of *P. lilacinum*. The partial 28S rRNA gene sequence was also 100% identical to that of *P. lilacinum* CBS 284.36 and ATCC 10114 and differed at one nucleotide position from the sequences of CBS 431.87 and CBS 101068. The partial β-tubulin gene sequence also showed only 1 nucleotide difference from the sequence of *P. lilacinum* strains CBS 284.36 and CBS 432.87. The calmodulin gene sequence of our isolate also exhibited 100% identity to the sequence of the type strain of *P. lilacinum* (CBS 284.36).

The case described here is unique in three respects. First, it

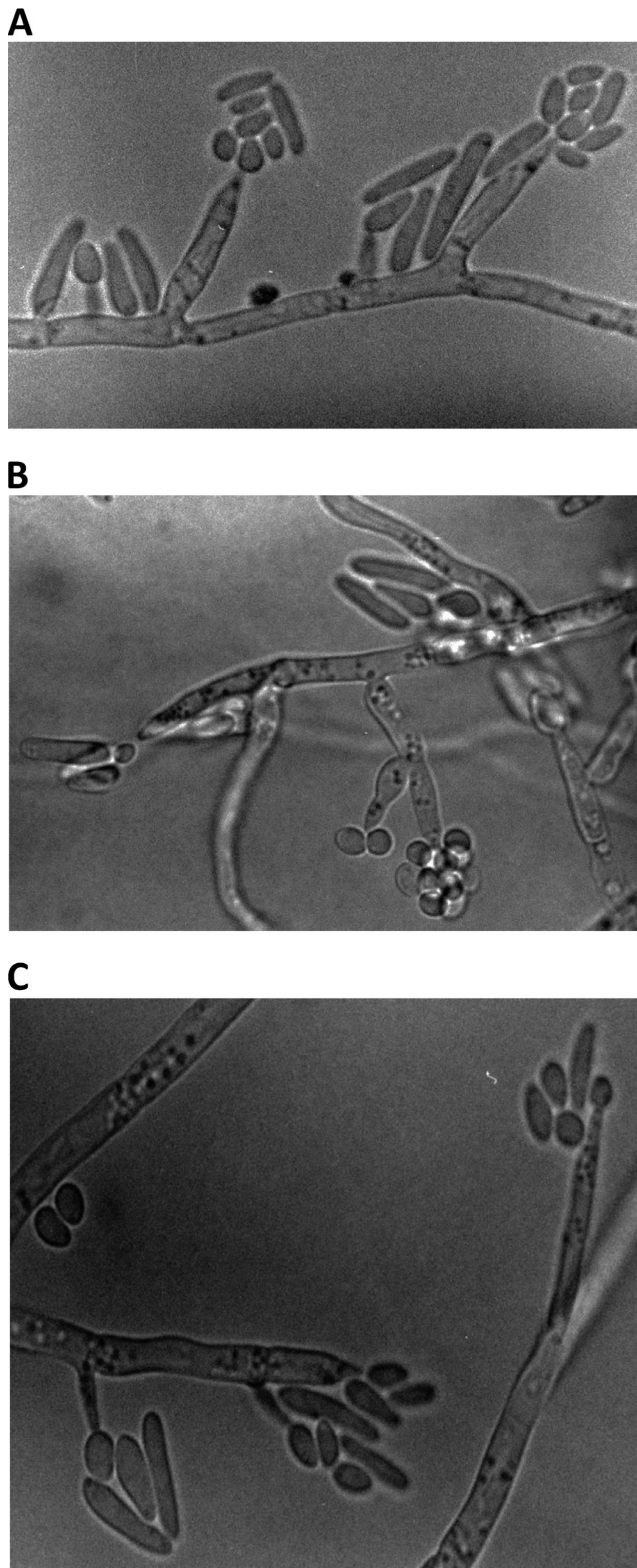


FIG 3 (A to C) One-week-old slide culture of *P. lilacinum* (MF3411/10) grown on Sabouraud dextrose agar at 30°C showing *Acremonium*-like phialides and fusiform conidia.

describes *P. lilacinum* as a cause of cavitary pulmonary disease, a clinical presentation, which to our knowledge, has not been described previously. Second, the isolate presented atypical morphological features, which were more akin to *Acremonium* spp. than to *P. lilacinum*, thus requiring molecular identification. Third, it documents the clinical efficacy of voriconazole in the treatment of a cavitary *Purpureocillium* infection.

Purpureocillium lilacinum (formerly *Paecilomyces lilacinus*) is a hyaline hyphomycete with a ubiquitous distribution (19). It occurs mainly in soil and decaying vegetable matter as a saprobe (7). *P. lilacinum* is an increasingly recognized agent of hyalohyphomycosis, capable of causing a wide spectrum of clinical manifestations in immunocompromised and immunocompetent individuals (4, 29, 34, 36, 39). Although ocular and cutaneous/subcutaneous infections are the most familiar clinical presentations, it is also encountered in cases of fungemia and deep-seated/systemic infections (28, 29). Infections with *P. lilacinum* present diagnostic and therapeutic challenges since its morphology in tissue is indistinguishable from those of *Aspergillus* and other agents of hyalohyphomycosis (36) and because it exhibits reduced susceptibility to amphotericin B (29).

Among upper respiratory tract infections, *P. lilacinum* has been implicated in the etiology of invasive rhinitis (6) and sinusitis (12, 23, 31, 32, 35, 36). Pulmonary infections due to *P. lilacinum* are rare. In this context, the present report is noteworthy as it describes the first case of cavitary pulmonary disease, thus extending the spectrum of clinical presentations known to be associated with *P. lilacinum.* So far, pulmonary infections due to *P. lilacinum* have been reported in four cases (Table 1) (9, 19, 22, 26). The first report of chest involvement was a case of empyema reported in 1972 in a 20-year-old male from Malta with no known predisposing condition (9). The second case was reported in a 58-year-old female with history of collagen lung disease who was receiving corticosteroid therapy (22). The fungus was isolated from pleural drainage. The third case involved a patient with acute lymphoblastic leukemia, where infection from the lung apparently disseminated to other organs and the fungus was isolated from blood (19). The fourth case was reported in a 57-year-old healthy man, who developed a coin lesion in the right hilum. A culture of pus obtained from the abscess following a right middle lobe lobectomy yielded *P. lilacinum*. The patient recovered without antifungal therapy (26). Two additional cases of pulmonary involvement, where the isolates were identified only to the genus level, have also been described (13, 37). One of these involved a 12-year-old Pakistani boy with chronic granulomatous disease in which the isolate was recovered from a lung biopsy specimen (37). The patient was treated with amphotericin B, followed by itraconazole and the withdrawal of prednisolone. The second case involved a 41-year-old female with a history of cough and hemoptysis (13). The isolate was cultured from a needle aspirate obtained from a pulmonary lesion with a radiologic diagnosis of mycetoma. Based on the DNA sequence of the ITS region, the isolate could only be identified up to the genus level due to poor sequence identity with available sequences of pathogenic *Paecilomyces* species, including *P. lilacinum* (13). The patient received voriconazole for 6 months, followed by resection of the fungal ball. The latter two reports underscore the difficulties in identifying *Paecilomyces* spp. Pulmonary infections due to *P. lilacinum* have also been reported in animals (30).

The etiologic significance of *P. lilacinum* in our patient is ap-

TABLE 1 Summary of salient findings in cases of pulmonary *P. lilacinum* infection

Case no.	Reference	Country	Age (yr)/sex[a]	Clinical presentation	Predisposing factor(s)	Treatment	Outcome
1	Fenech and Mallia (9)	Malta	20/M	Pleural effusion, no evidence of dissemination	Not known	Amphotericin B	Recovered
2	Mormede et al. (22)	France	58/F	Pleural effusion, diffuse reticonodular lesion	Interstitial lung disease, corticosteroids, recurrent pulmonary infections	Antibiotics, anti-inflammatory, no antifungal given	Died of hepatic complications
3	Liu et al. (19)	United States	Not available	Lung disseminated	Acute lymphoblastic leukemia	Not available	Recovered
4	Ono et al. (26)	Japan	57/M	Lung abscess	Not available	Lobectomy	Recovered
5	Present case	Kuwait	80/F	Fever, pleuritic chest pain	Asthma, diabetes, rheumatoid arthritis	Voriconazole	Improved but then died due to other causes

[a] M, male; F, female.

parent from both the fact that the same fungus was isolated from two sputum specimens and a BAL sample, all collected within a 10-day period, and the fact that the patient responded to voriconazole therapy. A noteworthy feature of our isolate is its atypical microscopic morphology characterized by the formation of *Acremonium*-like conidia in the absence of verticillate branches with whorls of phialides that characterize typical strains. This made a definitive morphological identification impossible. Unequivocal identification was established by DNA sequencing of four highly conserved genes. Consistent with our isolate, Okada et al. (25) demonstrated that *P. lilacinum* was able to form *Acremonium*-like conidiophores in submerged cultures, as well as on the agar surface, with dimorphic characteristics. *Acremonium*-like conidiophores, phialides, and conidia (formed in "slimy heads") were described by Luangsa-Ard et al. (20) when placing *Paecilomyces lilacinus* in the new genus *Purpureocillium*. This *Acremonium* state resembles members of the *Fusarium solani* species complex (FSSC), which are major agents of fungal keratitis. Interestingly, the most frequent manifestation of *P. lilacinum* is also keratitis (29), suggesting that these fungi may share similar pathogenesis or pathogenic mechanisms. Additionally, members of the FSSC and *P. lilacinum* may be misidentified in histopathological sections due to their similar morphological appearances (19). It is possible that like *Fusarium* spp., *P. lilacinum* may also form intravascular budding structures or phialoconidia through adventitious sporulation and thus facilitate its hematogenous dissemination to deeper tissues (19, 24). Thus, cutaneous lesions due to *P. lilacinum* may be caused not only by direct inoculation but may also result from hematogenous or lymphatic spread (15). It is worth noting that creams or lotions contaminated with *P. lilacinum* have resulted in outbreaks of cutaneous and disseminated disease (15, 27).

Purpureocillium lilacinum and *P. variotii* are the two clinically most important members of the genus (4, 14, 15, 29). Because of morphological similarities, their accurate identification is crucial as they exhibit different susceptibilities to antifungal agents (1). Recently, Castelli et al. (5) reported antifungal susceptibility profiles for *P. lilacinum* ($n = 27$) and *P. variotii* ($n = 31$) in which 20 of the isolates were identified by molecular methods. The results of this study indicated that amphotericin B, itraconazole, and echinocandins exhibit reduced susceptibility, whereas voriconazole and posaconazole show good activity against *P. lilacinum.* This finding is also consistent with the susceptibility profile of our isolate. In contrast, *P. variotii* isolates were susceptible to amphotericin B, itraconazole, and echinocandins, but showed reduced susceptibility to voriconazole (5). Similar susceptibility results were reported by Gonzalez et al. (11), where voriconazole, posaconazole, and ravuconazole demonstrated good *in vitro* activity against *P. lilacinum.* Pastor and Guarro (29) summarized available *in vitro* susceptibility data on *P. lilacinum.* Voriconazole and posaconazole were found to possess maximum activity, whereas amphotericin B was least active. Echinocandins have shown variable *in vitro* activity against *P. lilacinum* (5, 8, 29, 38). Voriconazole, consistent with its *in vitro* activity against *P. lilacinum*, has been used successfully for treatment of several cases with different clinical manifestations, including the present case (6, 10, 18, 21). The efficacy of voriconazole has also been demonstrated in an experimental murine model of *P. lilacinum* infection in comparison to amphotericin B (33). However, it is important to recognize that strains of *P. lilacinum* showing *in vitro* resistance to voriconazole have also been reported (5, 29). Clinical experience with posaconazole or other newer triazoles does not exist.

In conclusion, the first case of cavitary pulmonary disease caused by *P. lilacinum* is described. Since the isolate presented atypical, *Acremonium*-like morphological characteristics, a definitive identification was obtained by sequencing multiple loci. The patient was successfully treated with voriconazole.

Nucleotide sequence accession numbers. The DNA sequence data of our isolate (MF3411/10, CBS 129077, or UTHSC 11-93) have been deposited in the EMBL data bank under accession no. FR822391, FR822392, HE648327, and HE648328.

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