Bilateral multilobar cavitary pneumonia in an immunocompetent host. A case report of invasive pulmonary *Scedosporium spp* infection

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ABSTRACT

Background: *Scedosporium spp* is an established cause of multisystemic clinical disease in immunocompromised patients. Invasive lung disease has been reported in immunocompetent hosts, the majority of whom have pre-existent structural lung disease.

Case Presentation: We present a case of a 31-year-old African American man who presented with cough and hemoptysis for four months. Lung imaging revealed bilateral multilobar cavitary infiltrates. Bronchial alveolar lavage fungal cultures resulted in positive for *Scedosporium spp*. Work up for alternative causes of cavitary pneumonias, including *Mycobacterium tuberculosis*, as well as immunodeficiency syndromes, was negative. He achieved complete symptom resolution with radiological improvement on voriconazole therapy.

Conclusion: *Scedosporium spp* is emerging as a possible rare cause of cavitary pneumonias in immunocompetent patients without pre-existing lung disease. An accurate diagnosis is important as treatment involves a long course of antifungal therapy.

Keywords: *Scedosporium spp,* cavitary pneumonia, immunocompetent host, voriconazole.

Type of Article: CASE REPORT **Specialty:** Infectious disease

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Background

Scedosporium spp. is an established cause of clinical disease in immunocompromised patients, including chronic granulomatous lung disease, keratitis and endophthalmitis, brain abscesses, as well as soft tissue infections [1]. Lung disease has been reported in immunocompetent hosts, the majority of whom have pre-existent lung disease like chronic obstructive pulmonary disease, concurrent pulmonary tuberculosis, bronchiectasis, or cystic fibrosis [2-4].Click or tap here to enter text. Establishing an accurate diagnosis is important as drug choices are limited, as well as the need for prolonged therapy spanning over months to years.

We present a case of a 31-year-old African American man who presented with a persistent cough and hemoptysis, with lung imaging showing bilateral cavitary infiltrates with eventual bronchial cultures positive for *Scedosporium spp* (Table 1).

Case Presentation

A 31-year-old African American man presented with concerns of a worsening cough with hemoptysis that had been ongoing for up to 4 months. He denied any past medical conditions or long-term medication use.

He reported subjective low-grade fevers as well as chills that he described as randomly occurring. He denied weight loss, though he acknowledged being 'small bodied' for the whole of his life'. He had a normal appetite, had no night sweats, and had not noticed any swollen glands. He reported no history of pulmonary tuberculosis (TB) treatment or contact with tuberculosis patients or suspects. He also reported no history of pre-existent lung disease, history of body rash, joint swelling or pain, or family history of auto-immune diseases. He reported no history of recurrent sinus and ear infections. He had immigrated from the Virgin Islands to New York State 2 years prior to his presentation. He had never smoked cigarettes though admitted to a remote history of smoking marijuana

Table 1. *Timeline of the patient's clinical course.*

TIMELINE	PROCEDURES	EVENTS
Admission		-Cough and hemoptysis for 4 months with subjective low-grade fevers -Imaging showed multilobar cavitary pneumonia with diffuse infiltrates
	Bronchoscopy	-Bronchial lavage samples resulted positive for *Scedosporium spp* *-Started on voriconazole therapy*
3- months after discharge		- Complete symptom resolution - Resolution of infiltrates with residual bronchiectasis on chest CT scan

Table 2. Summary of laboratory tests performed at hospital admission.

TEST	RESULT	NORMAL RANGES
Anti-nuclear antibody	Negative	
HIV antibody/ antigen	Negative	
Myeloperoxidase Antibody	< 0.2 U/ml (normal)	
Proteinase 3 Ab-	<0.2 U/ml (normal)	
Qantiferon TB gold	Negative	
Acid fast bacilli (AFB) sputum smear and Xpert MTB/RIF	Negative	
Bronchial alveolar lavage sample MTB culture	Negative	
T Cell subset analysis	- CD3+- 827 cells/ul (60%) - CD8+ - 301 cells/ul (22%) -CD4+ - 494 cells/ul (37%) -CD16 +CD56- 425 cells/ul (30%) -CD19+ 132 cells/ul (9%)	-CD3+:1,000-2,300 cells/ul, 63%-84% -CD8+: 200-1,000 cells/ul, 11%-38% -CD4+: 600-1,600cells/ul, 34%-56% -CD16 +CD56:100-430 cells/ul,4%-21% -CD19+:140-600cells/ul, 5%-21%
Immunoglobulin A, M and G	Within normal limits	
Aspergillus (galactomannan) antigen	0.585	<0.5 index
White blood cell count	9,000 cells/ul	4.5-11× 10^3 cells/ul
Hemoglobin	12.3 g/dl	13.5-18 g/dl
Platelet count	372,000 cells/ul	150,000-450,000 cells/ul
Serum sodium	136 mmol/l	135-145 mmol/l
Serum potassium	4.3 mmol/l	3.5-5.3 mmol/l
serum bicarbonate	29 mmol/l	21-32 mmol/l
Anion gap	7	8-12
Blood urea nitrogen	8 mg/dl	7-23 mg/dl
Serum creatine	0.7 mg/dl	0.5-1.2 mg/dl
Random blood glucose	102 mg/dl	70-140 mg/dl
Serum calcium	8.8 mg/dl	8.4-10.4 mg/dl
Aspartate transaminase (AST)	30U/l	<59U/l
Alanine transferase (ALT)	21U/l	<35U/l
Serum albumin	4.1 g/dl	3.5-5 g/dl
Glycated hemoglobin (HBA1C)	6.4%	<5.7%- Normal 5.7%-6.4%- pre-diabetes ≥ 6.5%- diabetes mellitus
ESR	42 mm/hour	< 20 mm/hour
Nasopharyngeal swab	-SARS-COV-2 negative -Influenza A/B negative -Respiratory syncytial virus- negative.	
Urine drug screen	Negative for: amphetamines, benzodiazepines, cannabinoids, cocaine, barbiturates, opiates	

while still in the Virgin Islands. He did not take alcohol, did not report any past episodes of convulsions or loss of consciousness, and reported no previous episodes of dysphagia. He had tested negative on previous HIV screens,

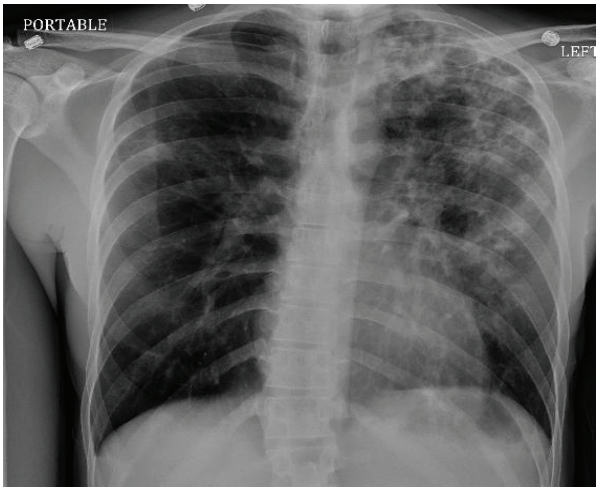

Figure 1A

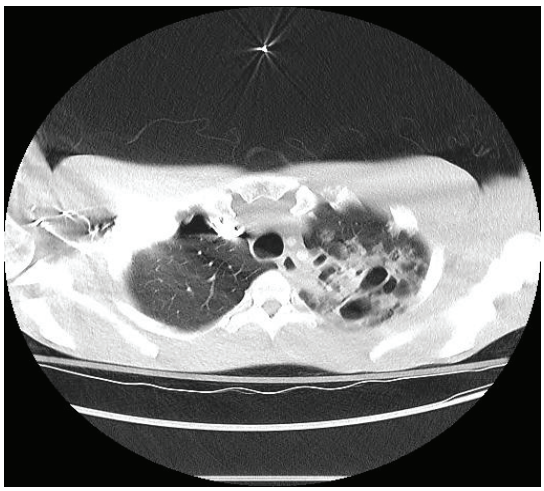

Figure 1B

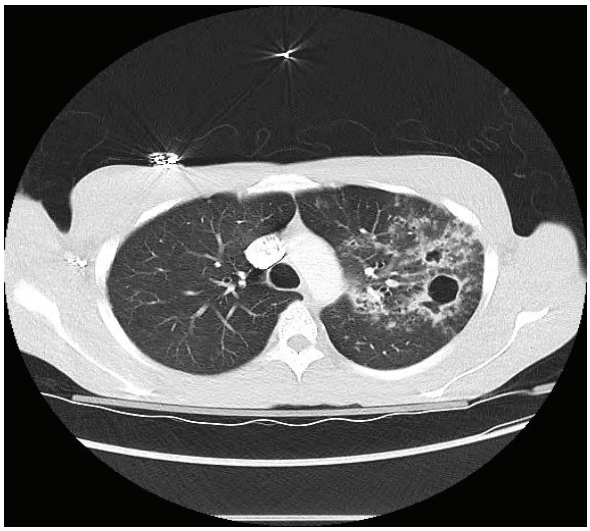

Figure 1C

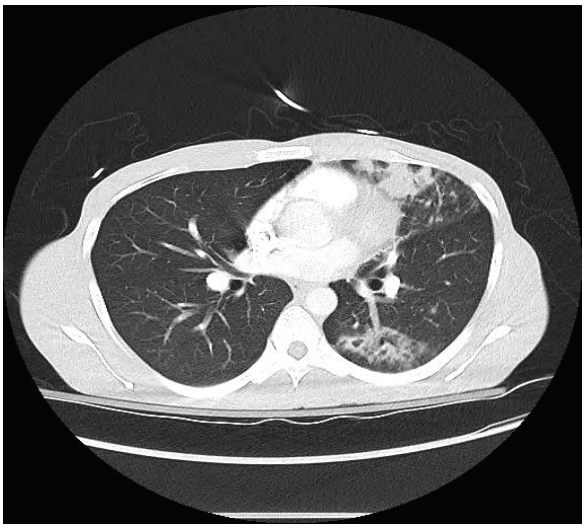

Figure 1D

Figure 1. *Chest X-ray and non-contrasted chest CT scan findings at admission. (A) Chest X-ray showing left upper and middle lung zone as well as right middle- lung zone heterogenous infiltrates. Multiple left lung cavitary lesions demonstrated. (B) Non contrasted Chest CT scan showing bilateral apical cystic and cavitary infiltrates. (C) Non contrasted Chest CT scan showing left middle lung zone infiltrates with two cavitary lesions. Increased lung markings in the right middle lobe. (D) Non contrasted Chest CT scan showing left lower lobar multifocal infiltrates.*

had one sexual partner, had no history of incarceration or working in the armed forces.

At admission, his oxygen saturation by pulse oximetry was 98% on room air, his blood pressure was 105/60mmHG with a heart rate of 105beats/minute and a respiratory rate of 16 breaths/ minute. He was afebrile with a temperature of 36.9^0C. He was underweight with a body mass index of $17.16 kg/m^2$.

On general examination, he was mildly wasted, was in fair general condition, and in no acute distress. He had no visible rash, digital clubbing, swollen lymph nodes, nail changes, or visible joint swelling. He was fully oriented,

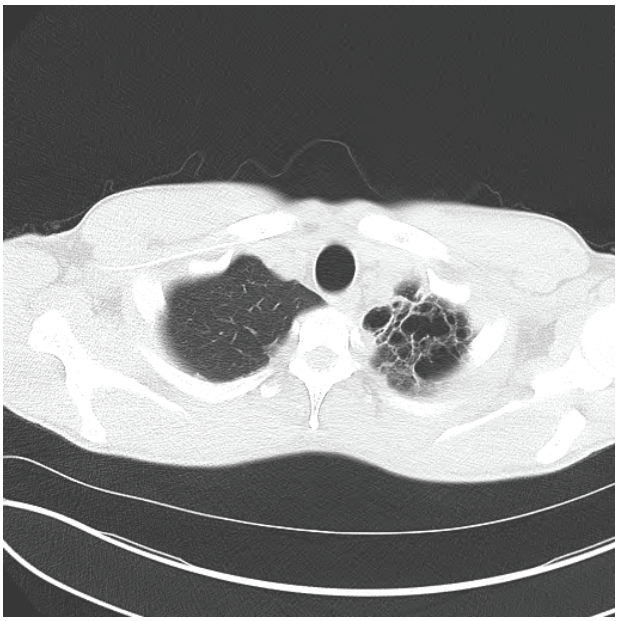

Figure 2A

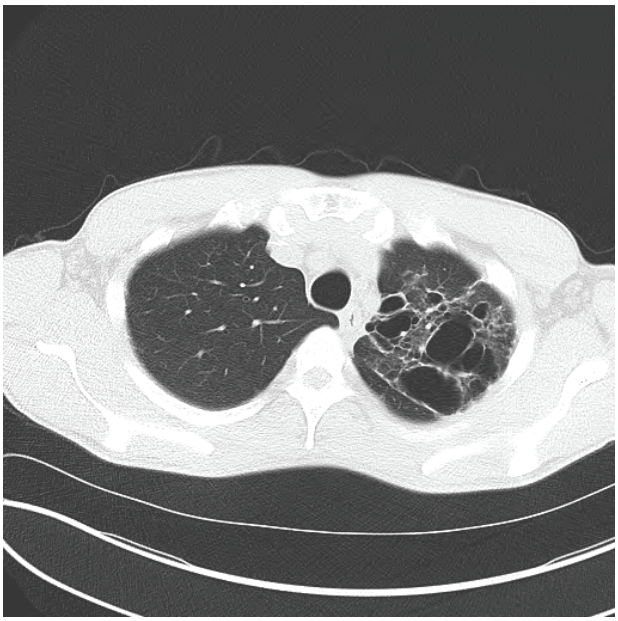

Figure 2B

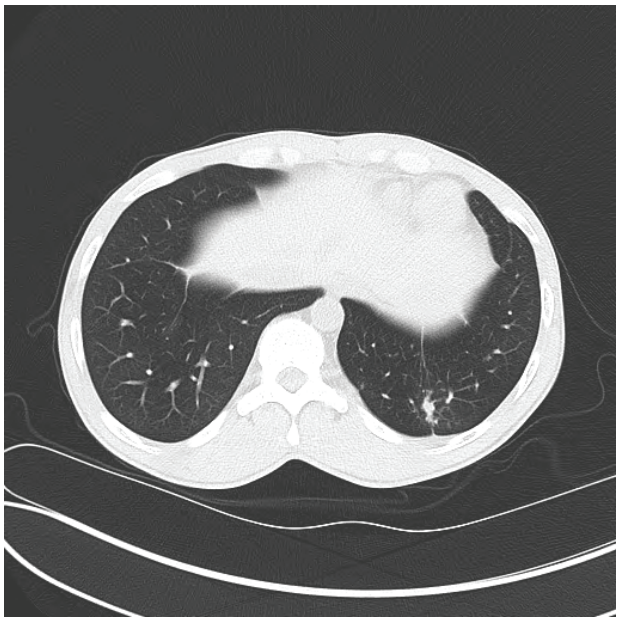

Figure 2C

Figure 2.*Non-contrasted chest CT scan findings after 4 weeks on voriconazole. (A) Resolution of right upper lobar infiltrates. Residual bronchiectasis and cystic changes in the left apical region. (B) Residual bronchiectasis and cystic changes in the left middle lung zone. (C) Resolution of the left basilar consolidative infiltrates.*

able to give a full history with a soft neck and normal motor, sensory, and coordination examination.

He had diffuse coarse crackles bilaterally, most prominent on the left apical and mammary regions, with no rhonchi. He was tachycardic with a normal volume pulse, no jugular venous distention with a point of maximal impulse in the fifth intercostal space, mid-clavicular line, and normal heart sounds without murmurs. His abdomen was soft, moving with respiration, non-tender with no palpable liver, spleen, or other masses. The musculoskeletal examination was unremarkable with no changes in joint temperature, tenderness, or impaired range of motion of both large and small joints. Admission laboratory tests are summarized in Table 2.

A chest X-ray and a non-contrasted chest computed tomography scan (CT scan) were done (Figure 1).

Hospital course

He was admitted and started on IV ampicillin – sulbactam and IV linezolid to empirically cover for possible aspiration pneumonia and Staphylococcal pneumonia much as his clinical history did not seem very commensurate with the two clinical syndromes.

When his serum *aspergillus* (galactomannan) antigen resulted slightly raised (0.585 (NR: <0.5 index), oral

voriconazole was added to his therapy. Subsequently, bronchoscopy was done, which demonstrated old blood in the left upper lobe anterior segment. Lavage ruled out alveolar hemorrhage. The conclusion was that he had normal airways on gross examination. Broncho-alveolar lavage samples were sent for conventional bacterial cultures, TB cultures, fungal cultures, as well as Gram and fungal staining. Bacterial cultures as well as pneumocystis jirovecii polymerase chain reaction were negative. Fungal cultures were positive for *scedosporium spp* but susceptibility testing was unable to be done from the isolates. The left upper lobe bronchial wash was negative for malignant cells, showed a few squamous cells, and hemosiderin-laden macrophages. He was discharged on oral voriconazole with a final diagnosis of *Scedosporium spp* bilateral multilobar cavitary pneumonia.

Follow up

He was reviewed in the outpatient clinic, 4 weeks post discharge. He reported complete symptom resolution, and a follow up CT scan showed resolution of consolidation with residual bronchiectasis (Figure 2). He continues to follow up with the infectious disease clinic on oral voriconazole.

Discussion

The studied global distribution of *Scedosporium* spp includes Europe, Australia, South America, the United States, Thailand, Morocco, and Taiwan. Human infection often results from inhalation of spores from the environment into the lungs or paranasal sinuses or through direct inoculation, as in a skin puncture. It is the second most common colonizer of cystic fibrosis patient airways with a potential to be a stimulus for inflammatory responses similar to allergic bronchopulmonary aspergillosis [4-6]

Common causes of cavitary lung disease include pulmonary tuberculosis (*Mycobacterium tuberculosis* and non-tuberculous mycobacteria), necrotizing bacterial pneumonias often caused by *Staphylococcus aureus*, gram negative bacteria, and anaerobes, fungi including *Aspergillosis, Blastomycosis, Coccidioidomycosis*, septic emboli commonly seen in the setting of right-sided infective endocarditis. Other noninfectious causes include Granulomatosis with polyangiitis, rheumatoid arthritis, sarcoidosis, lung infarctions, and malignancies [7].

The patient we present did not seem to have any underlying immunodeficiency state, a common predisposition to invasive lung disease. A few case reports of symptomatic lung disease have been reported in immunocompetent hosts. One such report summarized 25 previously published cases [8]. Out of 25 cases, 19 had underlying lung disease, including tuberculosis, chronic obstructive pulmonary disease, cystic fibrosis, and bronchiectasis. The patient we report had no lung symptoms prior to the 4 months of disease presentation, did not report the previous history of smoking, and had negative TB, auto immune, and lung malignancy studies. Unfortunately, we did not have prior lung imaging to compare our lung imaging finds to, to conclusively rule out pre-existent asymptomatic structural lung disease.

Clinical reasoning usually inclines clinicians to think along the usual fungal and bacterial cavitary pneumonia causes when faced with immunocompetent patients. The case we present highlights the importance of avoiding tunnel vision and considering work up for pathogens that are known largely known to cause pneumonia in immunocompromised hosts but are emerging as possible causes of clinical disease in immunocompetent hosts. The drug of choice for invasive *Scedosporium spp* is voriconazole, with treatment duration potentially lasting months to even years depending on clinical and radiological response [9]. In appropriate cases, such as osteomyelitis, soft tissue infections, localized pneumonias, and cerebral abscesses, surgical debridement to reduce pathogen load has been shown to be beneficial.

Conclusion

Scedosporium spp is emerging as a possible rare cause of invasive lung disease, including cavitary pneumonias in immunocompetent patients without pre-existing lung disease. Appropriate diagnosis is imperative as treatment response is variable with voriconazole use, and treatment duration can potentially last months to even years.

What's new?

Scedosporium spp is an established cause of clinical disease in immunocompromised patients and patients with pre-existent structural lung disease. This case involved an immunocompetent patient who presented with clinical disease, highlighting Scedosporium spp as a rare but possible cause of clinical disease in immunocompetent hosts.

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We acknowledge the patient for allowing us to write up the case.

Conflict of interest

The authors declare that they have no conflict of interest regarding the publication of this case report.

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Ethics approval and consent to participate

The patient provided consent for the case write up. The United Health Services Institutional review board (IRB) approved the publication of this case report; IRB number-14862 (June/18/2025).

Author contribution

JZ participated in the care of the patient. FM, HW, MR, SC, YS, and JZ reviewed the patient's records as well as participated in manuscript writing.

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References

1. Panackal AA, Marr KA. Scedosporium/Pseudallescheria infections. Semin Respir Crit Care Med. 2004;25(2):171–81. https://doi.org/10.1055/s-2004-824901
2. Seidel D, Meißner A, Lackner M, Piepenbrock E, Salmanton-García J, Stecher M, et al. Prognostic factors in 264 adults with invasive *Scedosporium* spp. and *Lomentospora prolificans* infection reported in the literature and FungiScope®. Crit Rev Microbiol. 2019;45(1):1–21. https://doi.org/10.1080/1040841X.2018.1514366
3. Álvarez-Uría A, Guinea JV, Escribano P, Gómez-Castellá J, Valerio M, Galar A, et al. Invasive *Scedosporium* and *Lomentosora* infections in the era of antifungal prophylaxis: a 20-year experience from a single centre in Spain. Mycoses. 2020;63(11):1195–202. https://doi.org/10.1111/myc.13154
4. Bronnimann D, Garcia-Hermoso D, Dromer F, Lanternier F, Maulin L, Leprince Y, et al.; French Mycoses Study Group; Characterization of the isolates at the NRCMA. Scedosporiosis/lomentosporiosis observational study (SOS): clinical significance of Scedosporium species identification. Med Mycol. 2021;59(5):486–97.
5. Neoh CF, Chen SC, Crowe A, Hamilton K, Nguyen QA, Marriott D, et al. Invasive *Scedosporium* and *Lomentospora prolificans* infections in Australia: a multicenter retrospective cohort study. Open Forum Infect Dis. 2023;10(2):ofad059. https://doi.org/10.1093/ofid/ofad059
6. Rodriguez-Tudela JL, Berenguer J, Guarro J, Kantarcioglu AS, Horre R, de Hoog GS, et al. Epidemiology and outcome of *Scedosporium prolificans* infection, a review of 162 cases. Med Mycol. 2009;47(4):359–70. https://doi.org/10.1080/13693780802524506
7. Gadkowski LB, Stout JE. Cavitary pulmonary disease. Clin Microbiol Rev. 2008;21(2):305–33. https://doi.org/10.1128/CMR.00060-07
8. Liang HY, Han CH, Xu WJ, Sun J, Wang Q. Pulmonary scedosporiosis in an intractable immunocompetent host: a case report and literature review. Clin Respir J. 2024;18(3):e13739. https://doi.org/10.1111/crj.13739
9. Troke P, Aguirrebengoa K, Arteaga C, Ellis D, Heath CH, Lutsar I, et al. Global *Scedosporium* Study Group. Treatment of scedosporiosis with voriconazole: clinical experience with 107 patients. Antimicrob Agents Chemother. 2008;52(5):1743–50. https://doi.org/10.1128/AAC.01388-07

Summary of the case

1	Patient (gender, age)	31-year-old, male patient.
2	Final diagnosis	*Scedosporium spp* bilateral multilobar cavitary pneumonia
3	Symptoms	Cough and hemoptysis for four months
4	Medications	Oral voriconazole
5	Clinical procedure	Bronchoscopy
6	Specialty	Internal medicine, Infectious diseases