

# A challenging case of pulmonary fusariosis superimposed on coronary artery bypass surgery complications in an uncontrolled diabetic patient

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## ABSTRACT

A 68-year-old man with poorly controlled type 2 diabetes mellitus, recent non-ST-elevation myocardial infarction (NSTEMI), and coronary artery bypass grafting was readmitted for purulent mediastinal wound discharge. Methicillin-sensitive *Staphylococcus aureus* grew from wound cultures and intravenous cefazolin was initiated. During hospitalization the patient sustained an acute ST-segment-elevation myocardial infarction (STEMI) requiring percutaneous coronary intervention. Post-procedure imaging revealed left lung collapse from a large pleural effusion on computed tomography (CT) scan, also confirmed through bronchoscopy. The effusion was drained through left chest tube thoracostomy after which the lung re-expanded. Bronchial lavage cultures grew pan-sensitive *Pseudomonas aeruginosa*, prompting intravenous ciprofloxacin. Seven days later, fungal cultures from the same bronchoalveolar lavage yielded colonies of mold which were apricot coloured. Microscopy demonstrated sickle-shaped conidia consistent with *Fusarium* species. The patient was clinically stable and discharged on oral cephalexin and ciprofloxacin; outpatient voriconazole was added once the mold was identified. At a two-month follow-up he remained asymptomatic with a clean sternotomy wound and clear chest radiograph. This report highlights the importance of pursuing fungal culture in persistent pulmonary or postoperative infections, recognising *Fusarium* as an emerging pathogen in diabetics and post-cardiac surgery patients, and initiating timely azole therapy despite initial clinical improvement.

**Keywords:** Fusariosis, *Pseudomonas aeruginosa*, Pleural effusion, postoperative infection, diabetes mellitus, NSTEMI.

## INTRODUCTION

Invasive fusariosis often affects neutropenic or transplant recipients, but its spectrum now spans immunocompetent hosts with metabolic or structural lung disease.<sup>1</sup> *Fusarium* species inhaled as airborne conidia can colonise damaged airways, cause necrotising pneumonia or disseminate hematogenously.<sup>2,3</sup> Mortality has been documented to be approximately 50–80% in immunosuppressed cohorts.<sup>4,5</sup> Cardiothoracic surgery further raises

infectious risk through prolonged ventilation, mediastinal wounds, and pleural instrumentation. We describe an unusual convergence of bacterial, fungal, and ischaemic complications in a diabetic post-coronary artery bypass graft (CABG) patient, underscoring diagnostic vigilance and multidisciplinary management.

## CASE

A 68-year-old man with 20-year poorly controlled type 2 diabetes mellitus (HbA1c 9.1%) underwent CABG two months earlier after non-ST-elevation myocardial infarction (NSTEMI). He presented with a 4-day history of purulent sternal discharge and right-sided chest pain radiating to the arm, unrelated to exertion.

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**Table 1. Timeline of Patient's Clinical Management**

Hospital Day and Event		Microbiology	Intervention	Outcome
0	Sternotomy debridement	Methicillin-sensitive <i>Staphylococcus aureus</i> (MSSA) from wound	Intravenous cefazolin	Local control
3	ST-segment-elevation myocardial infarction (STEMI)	N/A	Emergency percutaneous coronary intervention (PCI)	Haemodynamic stability
4	Left lung opacity	Sterile pleural fluid	Tube thoracostomy	Lung re-expansion
5	Bronchoscopy	Bronchoalveolar lavage (BAL) positive for <i>Pseudomonas aeruginosa</i>	Add intravenous ciprofloxacin	WBC count decreased
12	BAL positive for fungal culture	<i>Fusarium</i> species	Started oral voriconazole	Maintained
14	Discharge	None	Oral cephalixin, oral ciprofloxacin and voriconazole	Asymptomatic

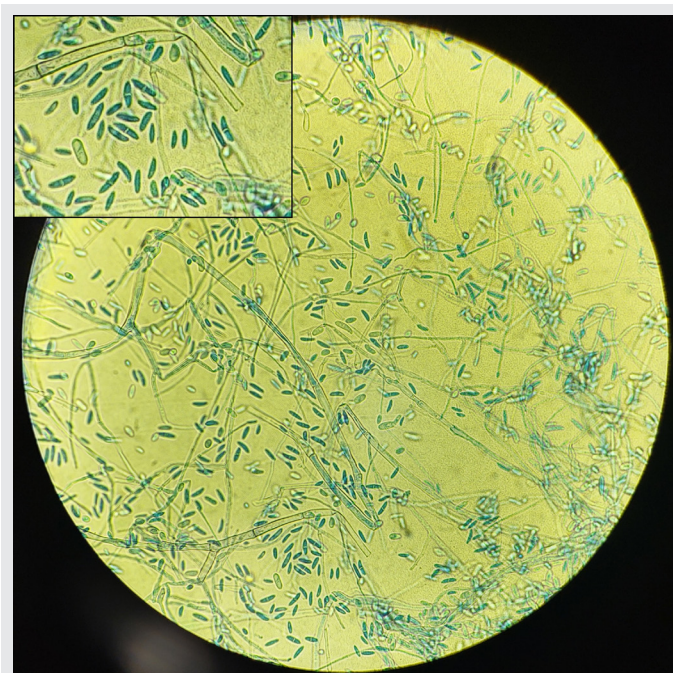
Vital signs showed temperature 37.9°C, blood pressure 145/86 mmHg, pulse 102 beats/min and SpO<sub>2</sub> 95% on room air. Examination revealed an erythematous, draining mid-sternal wound; breath sounds were decreased over the left base. Laboratory investigations revealed WBC count of 18.4 × 10<sup>9</sup>/L, neutrophils 85% and serum glucose 298 mg/dL. On electrocardiography (ECG), sinus rhythm without acute changes was observed. Chest x-ray showed clear lung fields bilaterally and intact mediastinal wires.

Management timeline is summarized in Table 1. Surgical debridement with wire rewiring was performed, during which pus and tissue cultures grew methicillin-sensitive *Staphylococcus aureus* (MSSA). Intravenous cefazolin 2 g q8h was started. On day 3 the patient developed retrosternal pain with anterior ST-elevation, and urgent percutaneous coronary intervention (PCI) of an occluded graft was successfully undertaken. The patient developed left lung collapse post-PCI. Bronchoscopy was done for evaluation of any internal obstruction. On bronchoscopy, the airways were clear with no secretions, suggesting the impression that left lung collapse was likely due to outside compression. Computed tomography (CT) scan confirmed massive pleural effusion compressing the left lower lobe. Subsequently, left chest tube thoracostomy was performed and 1,200 mL sero-haemorrhagic fluid was drained that yielded no growth on culture.

Bronchoalveolar lavage was sent for bacterial and fungal culture. *Pseudomonas aeruginosa* sensitive to amikacin, aztreonam, ciprofloxacin, meropenem, imipenem, piperacillin/tazobactam, ceftazidime, and ceftazidime/avibactam grew after 48 hours of incubation, and ciprofloxacin 400 mg IV q12h was added. Fungal culture was positive and grew colonies of mold after 7 days of incubation at 25°C and 37°C. The colonies of mold were apricot-coloured with a lighter periphery (Figure 1). On lactophenol cotton blue staining, septate hyphae were observed showing long and at places short conidiophores bearing small, oval, one- to two-celled sickle-shaped conidia that were present in clusters (Figure 2). Hence, identification of *Fusarium* species was reported. Subsequently, oral voriconazole was added to the treatment. The patient remained afebrile with improving labs and was discharged on oral cephalixin, oral ciprofloxacin, and oral voriconazole with outpatient follow-up. At a two-month review he reported no respiratory or wound symptoms. Chest x-ray was clear; voriconazole was continued to complete 12 weeks.

## DISCUSSION

Invasive fusariosis has traditionally been regarded as a complication of profound neutropenia or allogeneic transplantation, yet recent literature confirms that



**Figure 1.** Microscopic morphology of *Fusarium spp* (inset: septate hyphae with scattered small, oval, one- to two-celled sickle-shaped conidia)

metabolic and structural lung disorders independently reduce host resistance. Diabetes mellitus causes functional neutrophil defects, altered cytokine profiles and microvascular compromise, increasing the relative risk of invasive mycoses by 1.3- to 1.8-fold compared with euglycaemic controls.<sup>6</sup> Hyperglycaemia also promotes free-iron release and spore germination, mechanisms well described for mucormycosis and increasingly extrapolated to *Fusarium spp*.<sup>7</sup> Our patient's long-standing, poorly controlled diabetes therefore created an immunologic milieu comparable to the classic neutropenic setting even before cardiothoracic stressors were added.

CABG amplifies this risk profile. Up to 89% of patients develop a postoperative pleural effusion detectable by ultrasound or CT, while approximately 10% accumulate fluid occupying  $\geq 25\%$  of the hemithorax by 4 weeks.<sup>8,9</sup> Early removal of intercostal drains, mechanical ventilation at the time of tube withdrawal and concomitant pericardial effusion are modifiable predictors of effusion requiring drainage.<sup>10</sup> The sero-haemorrhagic exudate evacuated from our patient on day 4 is entirely consistent with these



**Figure 2.** Colony morphology of *Fusarium spp* on Sabouraud agar.

epidemiological observations and underscores that a large effusion alone should not be attributed to infection without further microbiology.

Radiology seldom discriminates fusariosis from other fungal pneumonias. In two CT series enrolling haematology patients, 82% of *Fusarium* cases presented with nodules or masses, whereas the classic halo sign seen in early aspergillosis was absent.<sup>3</sup> Our patient's left-lung collapse resulted from compressive effusion rather than parenchymal nodularity, illustrating that radiographic silence does not exclude angio-invasive fungi. Accordingly, the European Society of Clinical Microbiology and Infectious Diseases (ESCMID) and the European Confederation of Medical Mycology (ECMM) guidelines stress routine fungal culture of bronchoalveolar lavage or pleural fluid whenever bacterial isolates fail to fully explain clinical findings.<sup>5</sup>

Therapeutic evidence remains limited to retrospective series and composite case reviews. In the largest multinational analysis of 206 fusariosis cases, 90-day survival was significantly higher with voriconazole (53%) or lipid amphotericin B (48%) than with amphotericin B deoxycholate (27%).<sup>11</sup> Our patient's prompt clinical recovery on oral voriconazole monotherapy aligns with these outcomes and supports early de-escalation from parenteral agents when gastrointestinal absorption is assured.

Cardiac-surgical mycoses are typically *Candida* mediastinitis; among 73,688 operations only 4 cases of post-sternotomy aspergillosis were captured with none due to *Fusarium*, highlighting the rarity of our presentation.<sup>12</sup> Nevertheless, sternotomy wires, mediastinal dissection and transient ischaemia collectively impair local perfusion, facilitating mold adherence and invasion. Strict peri-operative glycaemic control, minimization of broad-spectrum antibiotics and judicious timing of chest-tube removal therefore represent pragmatic preventive strategies. Routine antifungal prophylaxis cannot be justified given the low incidence and variable in-vitro susceptibility of *Fusarium* isolates; instead, clinicians should maintain a low threshold for diagnostic bronchoscopy and repeat imaging when postoperative pleuro-pulmonary complications deviate from the expected bacterial trajectory.

Finally, multidisciplinary collaboration proved critical where cardiology managed graft occlusion, surgery addressed sternal sepsis, pulmonology performed bronchoscopy, and infectious disease specialists integrated microbiologic data with guideline-driven therapy. This coordinated approach mirrors ESCMID-ECMM recommendations that emphasize combined medical and surgical management wherever feasible.<sup>5</sup> The favourable two-month outcome therefore reinforces that (i) uncontrolled diabetes can predispose to fusarial infection even in non-neutropenic hosts, (ii) absence of typical radiological signs of fungal infection does not preclude invasive disease and (iii) early culture-directed anti-fungal therapy remains the cornerstone of therapy, with surgical source control when indicated. Heightened diagnostic vigilance in similar clinical intersections will be essential as the population of metabolically compromised surgical patients continues to expand.

## CONCLUSION

Pulmonary fusariosis is an emerging consideration in diabetic post-cardiac surgery patients presenting with pleural complications, even in the absence of classical imaging findings. Early multidisciplinary intervention, comprehensive microbiological assessment including fungal cultures, and timely initiation of anti-fungal therapy are pivotal to improving outcomes.

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**Conflicts of interest:** none

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