

Autochthonous *Emergomycetes pasteurianus* pneumonia in an immunocompromised patient in Hong Kong: a case report

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Case

A 61-year-old man with end-stage renal failure secondary to immunoglobulin A nephropathy underwent a cadaveric kidney transplant. His post-transplantation course was uneventful until he had very poor compliance to the immunosuppressants since May 2017 and admitted that he had stopped all immunosuppressants in February 2018. He developed acute antibody-mediated graft rejection in May 2018. His medications were adjusted, tacrolimus 4 mg/day, mycophenolate 360 mg twice daily and prednisolone were started. He developed a chest infection in October 2018. He denied any travel history or significant contact history. On physical examination he had cushingoid features and right-sided crepitation. Investigations revealed a low white cell count ($1.8 \times 10^9/L$) and neutrophil count ($0.7 \times 10^9/L$). Chest X-ray showed right basal infiltrates (Fig 1a). Blood and sputum for bacterial culture was negative, as was sputum for acid-fast bacilli. The patient's condition deteriorated despite use of vancomycin, meropenem, azithromycin, and fluconazole. Computed tomography of the thorax revealed extensive collapse and consolidation over the right lower lobe and bilateral pleural effusions (Fig 1b). Bronchoscopy and transbronchial lung biopsy showed granulomatous inflammation, with granular eosinophilic material and narrow-based small budding yeasts grouped in clusters inside macrophages in the alveolar spaces. Both mucicarmine staining and immunohistochemical staining for *Pneumocystis*, *Cytomegalovirus*, and herpes simplex virus were negative (Fig 1d-g). In view of the histopathological findings, bronchoalveolar lavage was sent for fungal culture. After 7 days of incubation at 25°C, a tiny colony of mould was seen. 21 Days later, there was a white-coloured mould colony with a velvety texture, wrinkled surface, acquired splits on the surface with no diffusible pigment (Fig 2a and b). Lactophenol cotton blue stain of the wet mount revealed a classic floret pattern (Fig 2c and d). Subculture was performed at 35°C. After 10 days of incubation at 35°C, a 25-mm-diameter yeast colony was evident. Based on the characteristic growth morphology,

infection with the thermally dimorphic fungus was established. Subsequent molecular genetic analysis by sequencing of the internal transcribed spacer and D1/D2 regions was performed and identified *Emergomycetes pasteurianus*, a rare thermally dimorphic fungus not previously reported in our locality. Liposomal amphotericin B was commenced and continued for 8 weeks. The patient responded well both clinically and radiologically (Fig 1c) and treatment was switched to oral voriconazole 200 mg twice daily. The patient succumbed to his medical illness 10 weeks after being discharged home.

Discussion

Thermally dimorphic fungal pathogens cause a significant human disease but are rarely reported in our locality with the exception of *Talaromyces (Penicillium) marneffeii*. To the best of our knowledge, this is the first report of *Emergomycetes* infection in Hong Kong. *Emergomycetes* shares the characteristics of other thermally dimorphic fungi: filamentous forms at 25°C that becomes an invasive yeast-like form at 35°C.¹ *Emergomycetes pasteurianus* was previously known as *Emmonsia pasteuriana*. *Emmonsia* species are ubiquitous, soil-dwelling saprophytic fungi. Species such as *Emmonsia crescens* and *Emmonsia parva* may rarely cause adiaspiromycosis in rodents and humans.² Recently, *Emergomycetes* has been reclassified as a new genus within the family *Ajellomycetaceae*. *Emergomycetes* and *Emmonsia* have significant differences in microbiology, epidemiology, clinical manifestations, and treatment outcomes. Microbiologically, *Emergomycetes* is a thermally dimorphic fungus while *Emmonsia* does not undergo mould-to-yeast conversion at 37°C. *Emmonsia* is rarely cultivated from clinical specimens. Human infection with *Emmonsia* is relatively rare in clinical practice² but *Emergomycetes* can cause fatal and disseminated human infection and appropriate antifungal therapy is essential.

At the time of this report, five *Emergomycetes* species are described. These species differ in geographic distribution. *Es pasteurianus* has been reported in Europe, Asia, and Africa. *Emergomycetes*

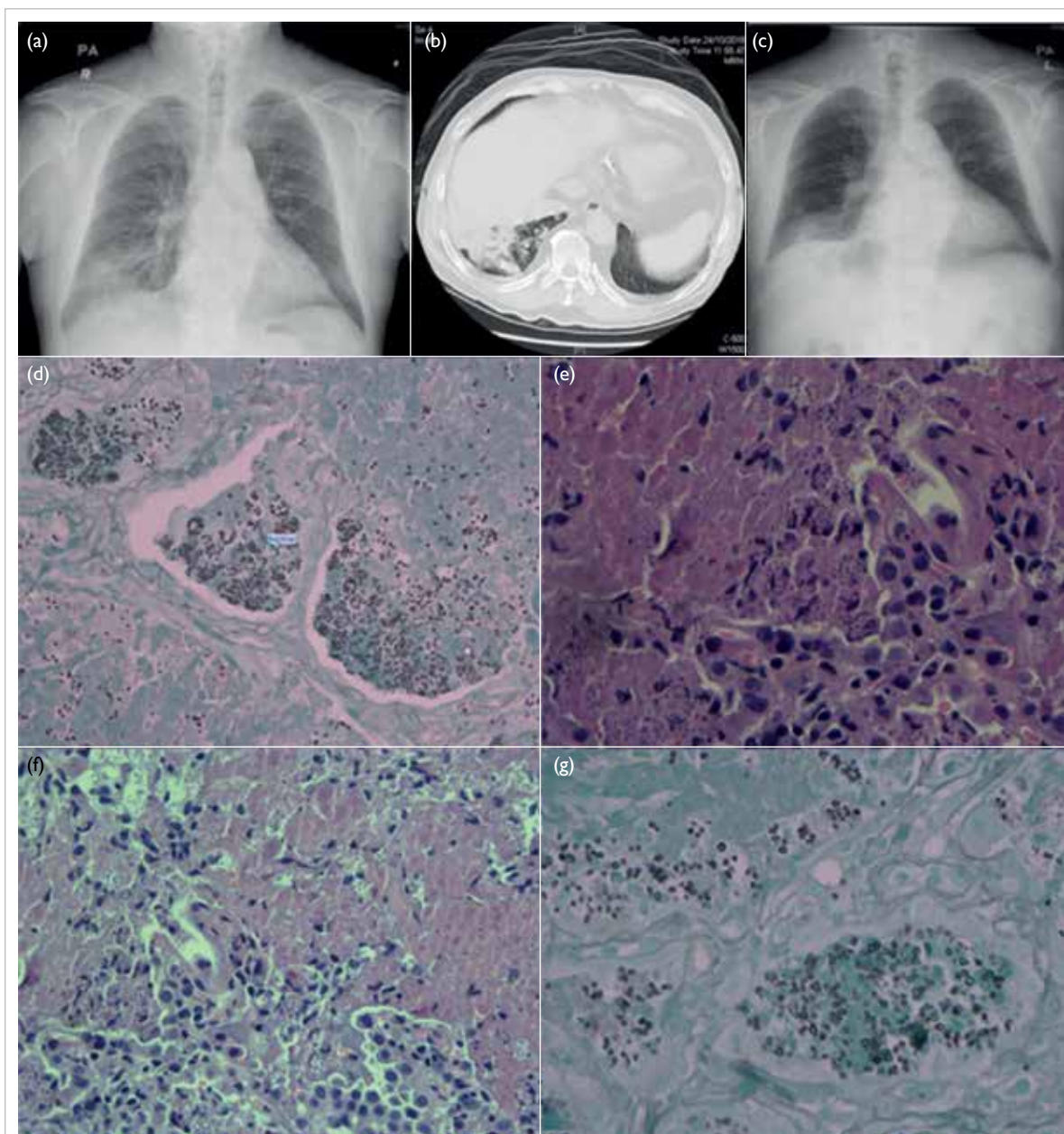


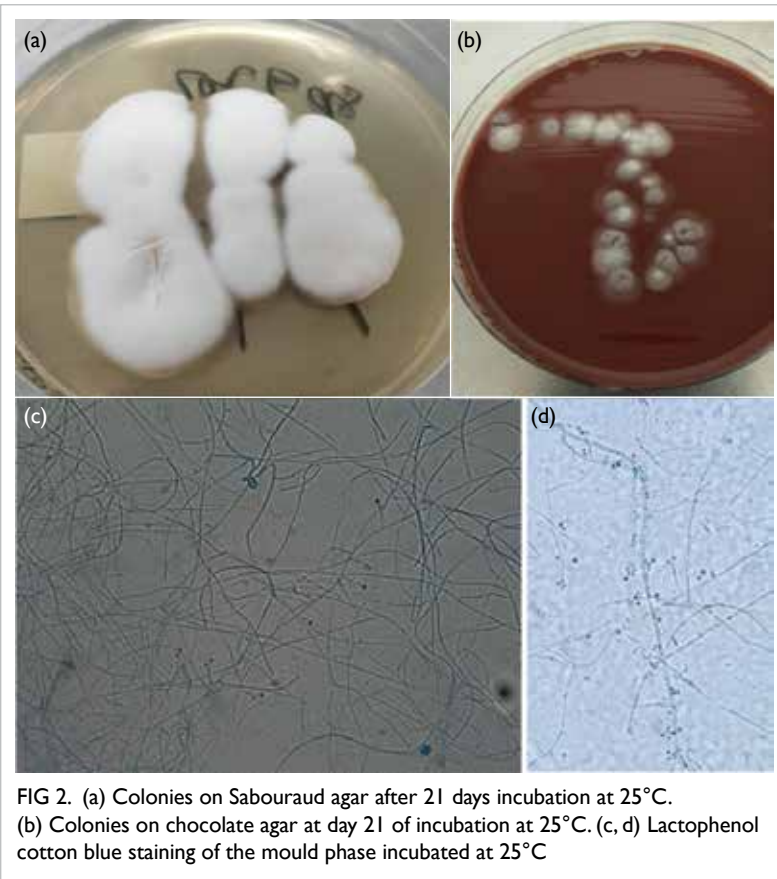
FIG 1. (a) Initial chest X-ray on admission. (b) Computed tomographic thorax scan on day 16 after admission. (c) Chest X-ray after 2 weeks of antifungal therapy. (d) Gomori methenamine silver stain, $\times 400$, showing small yeasts of 3-4 μm . (e) Haematoxylin and eosin stain, $\times 650$, showing intracellular yeasts. (f) Haematoxylin and eosin stain, $\times 400$, showing intracellular yeasts. (g) Gomori methenamine silver stain, $\times 650$

canadensis has been reported in Canada and the United States. *Emergomyces africanus* has been reported in South Africa, *Emergomyces orientalis* in China, and *Emergomyces europaeus* in Germany. The natural reservoir of *Emergomyces* is soil. Our patient presumably acquired the infection via inhalation of conidia in Hong Kong since he had no travel history outside of the region.

Emergomyces is a multi-system disease. According to case reports, patients most often present with fever, widespread skin lesions, weight

loss, and pulmonary disease.^{3,4} The diagnosis can be missed due to the slow-growing nature of the fungus. Histological examination of tissues can help diagnose the disease but on its own does not distinguish infection from other dimorphic fungi. Molecular diagnosis plays an important role in microbiological investigation. Using broad-range fungal polymerase chain reaction to sequence D2 large-subunit rDNA gene can help to confirm the diagnosis in a rapid, sensitive, and specific way.

There are no treatment guidelines for patients



with emergomycosis. Guidelines for blastomycosis and histoplasmosis recommend liposomal amphotericin B as initial therapy followed by itraconazole (or other newer azole). Our patient responded to liposomal amphotericin B and oral voriconazole but passed away due to his medical disease.

More patients are now rendered immunosuppressed by advances in treatment for a variety of diseases. Clinicians and microbiologists should be aware of the presence of rare invasive fungal infections among these susceptible patients.

Molecular techniques such as internal transcribed spacer polymerase chain reaction and sequencing can aid early and accurate identification of these rare fungal pathogens.

Author contributions

All authors contributed to the concept or design of the study, acquisition of the data, analysis or interpretation of the data, drafting of the manuscript, and critical revision of the manuscript for important intellectual content. All authors had full access to the data, contributed to the study, approved the final version for publication, and take responsibility for its accuracy and integrity.

Conflicts of interest

The authors have no conflicts of interest to disclose.

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Ethics approval

This case report was approved by the Hospital Authority Kowloon West Cluster Research Ethics Committee (Ref KW/EX-19-063(137-04)).

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