and vesical veins, as well as in the liver and the portal system.

In a more comprehensive study (*9*), *Bulinus* snails were found in all of Corsica's coastal rivers, except for those in the northwestern-most part of the island. However, of the 55 bodies of water where *Bulinus* snails were found, only 1 contained gastropods with *Schistosoma* cercariae, and results of a search for blood flukes in 220 small rodents (known for being susceptible to *S. bovis* and captured near bodies of water where *Bulinus* snails had been observed) were negative.

We have found no other documentation on bovine schistosomiasis in Corsica between 1966 and the present time. Has this disease disappeared since the 1960s? Is it still present as an enzootic disease with silent transmission? It should be noted that the disease produces few or no clinical signs and that slaughterhouse detection requires dissection of the circulatory system of the abdominal cavity. In any case, the discovery of human cases of schistosomiasis proves that a human–*Bulinus* parasitic cycle exists in Corsica, and therefore an animal–*Bulinus* cycle may exist as well. For the sake of scientific interest, an investigation into the presence of *S. bovis* in ruminants in Corsica would be worthwhile. Moreover, the fact that both *Schistosoma* species use the same intermediate host, *Bulinus contortus* snails, could cause problems with differential diagnosis.

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References

1. Holtfreter MC, Moné H, Müller-Stöver I, Mouahid G, Richter J. *Schistosoma haematobium* infections acquired in Corsica, France, August 2013. Euro Surveill. 2014;19:20821 http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=20821.
2. Berry A, Moné H, Iriart X, Mouahid G, Abbo O, Boissier J, et al. Schistosomiasis haematobium, Corsica, France [letter]. Emerg Infect Dis. 2014;20:1595–7. http://dx.doi.org/10.3201/eid2009.140928.
3. Brumpt E. Cycle évolutif du *Schistosoma bovis* (*Bilharzia crassa*); infection spontanée de *Bulinus contortus* en Corse C.-R. Acad. Sci. 1929;CLXXXXI:879.
4. Brumpt E. Cycle évolutif complet de *Schistosoma bovis*. Infection naturelle en Corse et infection expérimentale de *Bulinus contortus*. Ann Parasitol Hum Comp. 1930;VIII:17–50.
5. Pandey VS, Ziam H. Helminthoses circulatoires. In: Lefèvre P-C, Blancou J, Charmette R, editors. Principales maladies infectieuses et parasitaires du bétail, Europe et régions chaudes. Paris: Tec & Doc Editions Lavoisier; 2003. p. 1485–99.
6. Dollfus PH. Sur la présence en France et en Corse du *Bullinus contortus* (Michaud), hôte intermédiaire de *Schistosoma hæmatobium* (Bilharz): note préliminaire. Bull Soc Pathol Exot. 1922;15:208–12.
7. Arfaa F, Massoud J, Chu KY. Susceptibility of Portuguese *Bulinus contortus* to Iranian strains of *Schistosoma haematobium* and *S. bovis*. Bull World Health Organ. 1967;37:165–6.
8. Gretillat S. Epidémiologie de certaines affections à trématodes des animaux domestiques en Corse (bilharziose bovine et distomatose bovine et ovine). Ann Parasitol Hum Comp. 1963;38:471–81.
9. Doby J-M, Rault S, Deblock S, Chabaud A. Bulins et bilharzioses en Corse. Répartition, fréquence et biologie de "*Bulinus truncates*" [in French]. Annal. Parasitol. 1966;4:337–49.

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HIV-Associated Disseminated Emmonsiosis, Johannesburg, South Africa

To the Editor: *Emmonsia* spp., dimorphic fungi found worldwide, cause disease mainly among lower-order mammals (*1*). Although emmonsia rarely infect humans, the fungi can cause localized granulomatous pulmonary disease (adiaspiromycosis) in immunocompetent persons (*1–4*). Before 2013, no association was known between emmonsia and HIV, and there was no indication that emmonsia were endemic to sub-Saharan Africa.

In 2013 a novel *Emmonsia* sp. that is closely related to *E. pasteuriana* was described. The fungus caused disseminated disease in 13 HIV-infected persons in South Africa (12 in Cape Town, 1 in Bloemfontein) (*5*). Two additional cases of disseminated emmonsiosis caused by this novel species were identified in HIV-uninfected persons (1 immunocompetent, the other immunosuppressed for renal transplantation) in Cape Town (*6*). Because these cases clustered geographically, it was suggested that this novel *Emmonsia* sp. occupies a microenvironment around Cape Town (*7*). We report 3 additional cases of disseminated emmonsiosis from Johannesburg, South Africa, 403 km from Bloemfontein and 1,400 km from Cape Town. All patients were HIV-infected and reported no travel to Bloemfontein or Cape Town.

The 3 patients were admitted to Helen Joseph Hospital between August 2012 and August 2014; all patients were male and had CD4 counts of ≤5 cells/μL at admission. Patient 1 had never received antiretroviral therapy; patients 2 and 3 had defaulted antiretroviral treatment for several months before admission. All patients had disseminated skin rash, pneumonia, anemia, and substantial weight loss; chest radiographs suggested pulmonary tuberculosis. The rash appeared as disseminated hyperpigmented scaly papules and plaques (online Technical Appendix Figure 1, http://wwwnc.cdc.gov/EID/article/20/12/14-0902-Techapp1.pdf). Patients 1 and 2 also had diarrhea and exhibited delirium.

Laboratory investigations for patient 1 showed normocytic anemia,

hyponatremia, renal insufficiency, and elevated liver enzyme levels. Patients 2 and 3 had pancytopenia, hyponatremia, metabolic acidosis, and elevated liver enzyme levels. Lumbar puncture results were unremarkable for patients 1 and 3; patient 2 had normal lumbar puncture results during a previous admission (online Technical Appendix Table 1). Automated laboratory identification systems initially misidentified the *Emmonsia* sp. on blood culture as *Trichosporon* spp. (patients 1 and 3) and *Histoplasma capsulatum* (patient 2). Co-infection with *Mycobacterium avium* (patients 1 and 2) and *M. tuberculosis* (patient 2) was observed (online Technical Appendix Table 2). Subsequent histologic examination of skin biopsy specimens from all patients showed granulomas and yeast-like organisms.

Antifungal drug therapy consisted of fluconazole for patient 1 and conventional amphotericin B for patients 2 and 3; itraconazole was co-administered to patient 2 (online Technical Appendix Table 3). Clinical and biochemical parameters improved for all patients during the first 2 weeks of hospitalization. However, hospital-acquired pneumonia developed in patient 1, who subsequently died on hospitalization day 21, and patient 2 died of an unknown cause on day 17. Permission for autopsy was not granted for either patient. At the time of this report, patient 3 was recovering well.

The fungal isolates were not identified molecularly before patients 1 and 2 died. Sequencing of the ribosomal DNA internal transcribed spacer region of isolates from all 3 patients showed 97%–99% homology with the previously described novel *Emmonsia* sp. (GenBank accession nos. KM199781–83 and KM492927) (*5*). At admission, clinical features for patients in our study were similar to those for patients with the previously reported cases of HIV-associated emmonsiosis (*5*): all patients had rash, anemia, low CD4 count, abnormal liver enzyme levels, and chest radiographs compatible with pulmonary tuberculosis.

The patients in our study received an initial misdiagnosis. Kenyon et al. (*5*) also encountered diagnostic ambiguity in invasive fungal infection cases: over an 8-year period in South Africa, 39 cases were diagnosed as histoplasmosis on the basis of histologic findings, and only 1 was confirmed by using molecular techniques. Because laboratory services in Africa are generally weak (*8*), this trend of misdiagnosis could continue. The high death rate among patients with HIV-associated emmonsiosis (31%, 5/16 patients) may partly be explained by misdiagnoses (*5*), and it also raises questions regarding optimal treatment. Amphotericin B will likely remain the optimal empiric induction therapy for suspected cases of disseminated fungal infection among HIV-infected persons in sub-Saharan Africa, given the phylogenetic proximity of *Emmonsia* spp. to *Histoplasma* spp., the antifungal minimum inhibitory concentrations of emmonsia reported thus far (*5*), and the potential for laboratory misdiagnosis of fungal infection cases. Supplementary itraconazole may be beneficial if dimorphic fungal infection, specifically emmonsia, is clinically suspected. Regardless, early confirmatory diagnoses based on culture and histopathologic results should be aggressively pursued.

Although HIV-associated emmonsiosis was suggested to be geographically isolated to the Western Cape Province, South Africa (*7*), the ecologic niche occupied by this novel *Emmonsia* sp. probably extends throughout southern Africa. Of the 13 previously reported patients (*5*), 12 lived near Cape Town (Western Cape Province) and 1 lived in Bloemfontein (Free State Province; in the center of the country) at the time of diagnosis. The 3 additional patients reported in our study resided in Johannesburg, a geographic setting distinctly separate from the other locations. A region-wide surveillance program is needed to enhance disease identification within South Africa and to determine the environmental presence of this organism beyond South Africa's borders.

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References

1. Anstead GM, Sutton DA, Graybill JR. Adiaspiromycosis causing respiratory failure and a review of human infections due to *Emmonsia* and *Chrysosporium* spp. J Clin Microbiol. 2012;50:1346–54. http://dx.doi.org/10.1128/JCM.00226-11

2. Dot JM, Debourgogne A, Champigneulle J, Salles Y, Brizion M, Puyhardy JM, et al. Molecular diagnosis of disseminated adiaspiromycosis due to *Emmonsia crescens*. J Clin Microbiol. 2009;47:1269–73. http://dx.doi.org/10.1128/JCM.01885-08
3. Peterson SW, Sigler L. Molecular genetic variation in *Emmonsia crescens* and *Emmonsia parva*, etiologic agents of adiaspiromycosis, and their phylogenetic relationship to *Blastomyces dermatitidis* (*Ajellomyces dermatitidis*) and other systemic fungal pathogens. J Clin Microbiol. 1998;36:2918–25.
4. Wellinghausen N, Kern WV, Haase G, Rozdzinski E, Kern P, Marre R, et al. Chronic granulomatous lung infection caused by the dimorphic fungus *Emmonsia* sp. Int J Med Microbiol. 2003;293:441–5. http://dx.doi.org/10.1078/1438-4221-00281
5. Kenyon C, Bonorchis K, Corcoran C, Meintjes G, Locketz M, Lehloenya R, et al. A dimorphic fungus causing disseminated infection in South Africa. N Engl J Med. 2013;369:1416–24. http://dx.doi.org/10.1056/NEJMoa1215460
6. Heys I, Taljaard J, Orth H. An *Emmonsia* species causing disseminated infection in South Africa. N Engl J Med. 2014;370:283–4. http://dx.doi.org/10.1056/NEJMc1314277
7. Latgé JP. Oh, to be new. N Engl J Med. 2013;369:1464–6. http://dx.doi.org/10.1056/NEJMe1309132
8. Petti CA, Polage CR, Quinn TC, Ronald AR, Sande MA. Laboratory medicine in Africa: a barrier to effective health care. Clin Infect Dis. 2006;42:377–82. http://dx.doi.org/10.1086/499363

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Ecosystem Effects of Variant Rabbit Hemorrhagic Disease Virus, Iberian Peninsula

To the Editor: In this investigation, we found evidence for the apparent effects that a new variant of the rabbit hemorrhagic disease virus (RHDV) is having on native wild European rabbit (*Oryctolagus cuniculus*) populations on the Iberian Peninsula, and how this virus could threaten the conservation of endangered predators.

Historically, European rabbits were extremely abundant on the Iberian Peninsula, which is in their native range. However, during the 20th century, the number of rabbits on the peninsula has declined >90%, mainly because of diseases (*1*). The first notable crisis among rabbits occurred during the 1950s concurrent with the arrival of myxomatosis among rabbit populations, which caused mortality rates of ≈90% (*1*), as registered in other regions. During the late 1980s, a calicivirus, RHDV, caused infections that made a strong impact on rabbit populations, causing initial mortality rates of 55%–75% in Iberia (*1*). Since their initial outbreaks, both diseases have become enzootic, and related mortality rates have decreased, in part because of increased host resistance, although the infections still play a major role in the dynamics of rabbit populations (*2*).

In 2011, a new variant of RHDV, which appears to be closely related to an isolate originating in France that was described in 2010 (*3*), caused high mortality rates in some rabbit farms in Spain (*4*) and was also identified in an experimental wild rabbit plot in northern Spain (*5*). Since 2012, the new variant of RHDV has been detected in most rabbit farms in Spain (*6*), and in several wild populations distributed across Spain and Portugal (*7*), suggesting that it has rapidly spread throughout the Iberian Peninsula. This variant affects both of the wild rabbit subspecies (*O. cuniculus cuniculus* and *O. c. algirus*), and unlike the classical form of RHDV, it kills rabbits as young as 11 days of age and rabbits that have been vaccinated against classic RHDV (*6*,*7*). This scenario has raised concern for the survival of wild rabbit populations and its predators in this region.

Data regarding rabbit trends seem to sustain this concern. For example, a long-term monitoring program in Aragón in northern Spain shows a notable decline in rabbit numbers during 2013 in populations that showed both long-term increasing and decreasing trends over the monitoring period (Figure, panels A, B, respectively). A similar trend has been observed in the main areas inhabited by the highly endangered Iberian lynx (*Lynx pardinus*). The lynx relies on rabbits for survival, because they represent >85% of the lynx's diet (*9*). For instance, in Coto del Rey, the area within Doñana National Park in southern Spain that traditionally held the highest rabbit densities and therefore represents the core of Iberian lynx populations in this national park, there was a decline in rabbits of >80% during 2012–2013 (Figure, panel C). Similar declines have been detected in low-density rabbit populations surveyed within Doñana National Park (Figure, panel C). Rabbit numbers have also been progressively dropping in the proximity of the Yeguas River in Andújar and Cardeña Natural Parks in southern Spain, where the largest Iberian lynx population currently lives: rabbit density was >3.5 rabbits/hectare in 2010 and <1 rabbit/hectare in 2013, a decline of ≈75% (*10*). In accordance with field surveys, hunters throughout Iberia claim that the number of rabbits harvested this season has decreased dramatically, pointing to a 70%–80% decline compared to the previous hunting season in some estates (A. Linares, pers. comm.).

The European rabbit is a multifunctional keystone species of the Iberian Mediterranean ecosystem, where it serves as prey for >30 predatory animals, alters plant species composition and vegetation structure through grazing and seed dispersal, its excrement and urine have an effect on soil fertility and plant growth and provide feeding resources for invertebrates, and its burrows provide shelter for different

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Technical Appendix

Technical Appendix Table 1. Laboratory results at admission for 3 patients with HIV-associated disseminated emmonsiosis, Johannesburg, South Africa*

Laboratory investigation	Case 1	Case 2	Case 3	Reference range
CD4 count	5 cells/μL	3 cells/μL	0 cells/μL	50–2010 cells/μL
Leukocyte count	16.91 × 10^9/L	1.52 ×1 0^9/L	1.84 × 10^9/L	4.00–10.00 × 10^9/L
Hemoglobin	8.7 g/dL	11.7 g/dL	7.8 g/dL	14.3–18.3 g/dL
Mean cell volume	91 fL	90 fL	87.6 fL	83–101 fL
Platelets	523 × 10^9/L	74 × 10^9/L	122 × 10^9/L	150–400 × 10^9/L
Sodium	121 mmol/L	111 mmol/L	128 mmol/L	136–145 mmol/L
Potassium	3.7 mmol/L	4 mmol/L	4.8 mmol/L	3.5–5.1 mmol/L
Chloride	77 mmol/L	79 mmol/L	101 mmol/L	98–107mmol/L
Bicarbonate	30 mmol/L	16 mmol/L	16 mmol/L	23–29 mmol/L
Urea	31.4 mmol/L	5.5 mmol/L	4.8 mmol/L	2.1–7.1 mmol/L
Creatinine	590 μmol/L	55 μmol/L	64 μmol/L	64–104 μmol/L
Total bilirubin	5 μmol/L	12 μmol/L	7 μmol/L	5–21 μmol/L
Conjugated bilirubin	3 μmol/L	9 μmol/L	5 μmol/L	0–3 μmol/L
Total protein	51 g/L	56 g/L	46 g/L	60–78 g/L
Albumin	11 g/L	22 g/L	13 g/L	35–52 g/L
Alkaline phosphatase	400 U/L	301 U/L	131 U/L	40–120 U/L
γ-glutamyl transpeptidase	396 U/L	228 U/L	92 U/L	0–60 U/L
Alanine transaminase	37 U/L	53 U/L	40 U/L	10–40 U/L
Aspartate transaminase	266 U/L	94 U/L	145 U/L	15–40 U/L
Hepatitis A IgM antibody	Neg	Neg	ND	–
Hepatitis B surface antigen	Neg	Neg	ND	–
Hepatitis B core IgM antibody	Neg	Neg	ND	–
Hepatitis C antibody	Neg	Neg	ND	–
Cryptococcal serum antigen	Neg	Neg	Neg	–
CSF polymorphonuclear cells	0	0†	0	0
CSF lymphocytes	0	0†	0	0
CSF erythrocytes	0	18†	30	0

*CD4, CD4+ T-cell; CSF, cerebrospinal fluid; ND, not done; Neg, negative; NG, no growth.
†Lumbar puncture results reported are from a hospitalization in June 2013; meningeal disease was not suspected during the August 2013 hospitalization when the patient was diagnosed with emmonsiosis.

Technical Appendix Table 2. Culture data for 3 patients with HIV-associated disseminated emmonsiosis, Johannesburg, South Africa*

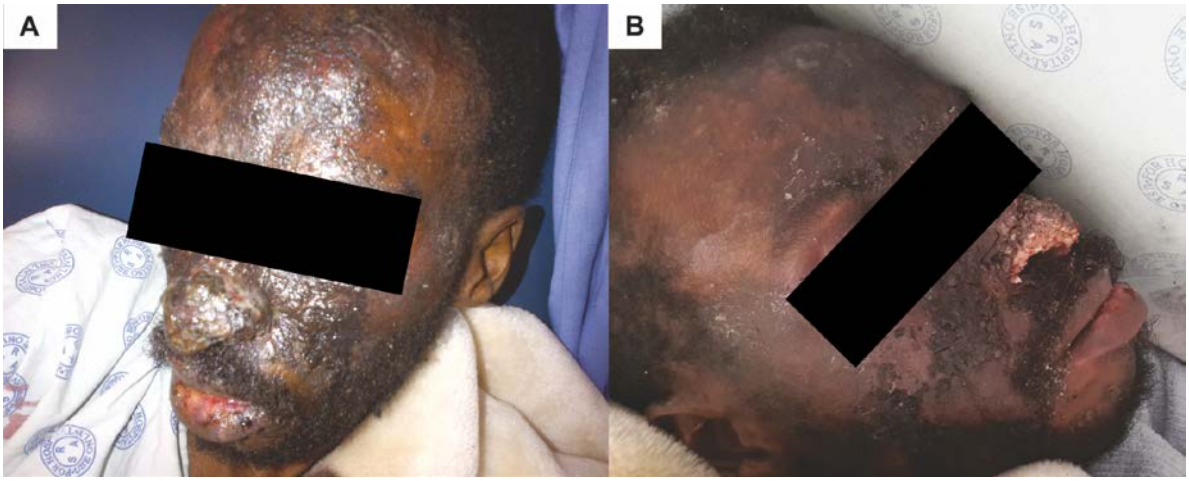
Case	Source	Initial Identification	Final Identification	Time to Positivity
1	Blood	NTM	NTM	14 d
	Blood	*Trichosporon* spp.	*Emmonsia* spp.†	157.7 h
	Blood	*Trichosporon* spp.	*Emmonsia* spp. †	124.9 h
2	Blood	MTB and MAC	MTB and MAC	36 d
	BMA	*Histoplasma capsulatum*	*Emmonsia* spp.†	45.0 h
3	Blood	Trichosporon spp.	*Emmonsia* spp.†	168.0 h

*BMA, bone marrow aspirate; MAC, *Mycobacterium avium* complex; MTB, *Mycobacterium tuberculosis*; NTM, unspeciated nontuberculous mycobacteria.
†Final identification of *Emmonsia* spp. was accomplished by sequencing of the internal transcribed spacer of the ribosomal DNA from the fungal isolate in each case.

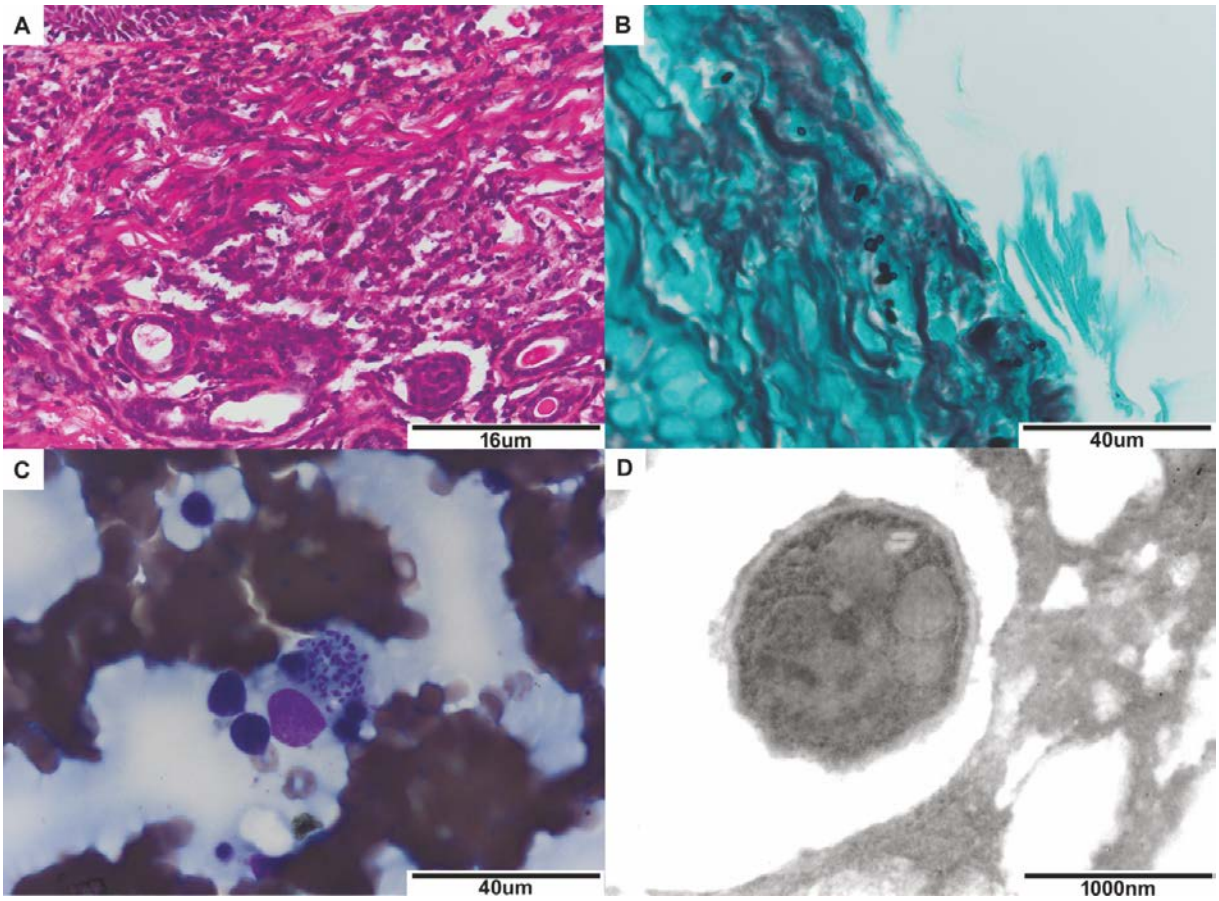
Technical Appendix Table 3: Summary of inpatient antimicrobial drug treatment administered

Case	Antifungal agent	Antimycobacterial agent	Antibacterial agent
1	Fluconazole†	Rifampin/isoniazid/pyrazinamide/ethambutol combination*	Amoxicillin/clavulanic acid†
2	Amphotericin B† and itraconazole*	Rifampin/isoniazid/pyrazinamide/ethambutol combination* and azithromycin*	Amoxicillin/clavulanic acid†
3	Amphotericin B†		Amoxicillin/clavulanic acid,† azithromycin,† and trimethoprim/sulfamethoxazole†

*Orally administered.
†Intravenously administered.



Technical Appendix Figure 1. A, Disseminated hyperpigmented scaly papules and plaques of the face with relative sparing of the eyelids is shown before treatment. B, Clinical response of the rash is shown after 10 days of treatment.



Technical Appendix Figure 2. A, Hematoxylin and eosin stain (original magnification × 40) of the skin biopsy demonstrates patchy inflammatory foci within the dermis (superficial and deep), that are predominantly suppurative, but also an occasional poorly formed granuloma. Bar represents 16 μm. B, Grocott stain (original magnification × 100) showing narrow-based budding yeasts within the dermis. Bar represents 40 μm. C, Bone marrow aspirate (100×) demonstrating a macrophage with multiple engulfed intracellular yeasts. Bar represents 40 μm. D, Transmission electron micrograph (original magnification × 40,000) of the dermis showing a free -lying organism in the yeast-phase. Bar represents 1,000 nm.

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