receiving corticosteroids or cytotoxic chemotherapy. Diabetic patients, such as our patient, were not identified as being at particular risk for disseminated disease [10].

Other than ethambutol, the common antituberculous agents are not effective against the rapidly growing mycobacteria. Within Runyon group IV, the various species have characteristic susceptibility patterns that may aid in their identification [1]. Clarithromycin administered for 6 months appears to be the most useful regimen against *M. abscessus* [2, 7]. Because of reports of resistance to clarithromycin monotherapy in *M. chelonae,* the use of combination therapy for treatment of disseminated infections due to rapidly growing mycobacteria has been advocated [8, 10]. Surgical debridement is essential for a good treatment outcome [7].

In conclusion, the rapidly growing mycobacteria are clinically important pathogens in the setting of surgical wound infections or infections associated with indwelling medical devices including, in our case, epicardial pacing leads. When slower-growing organisms resembling diphtheroids with unusual antibiograms are isolated in these settings, infection with rapidly growing mycobacteria should be suspected. Specific identification and susceptibility testing will allow the timely institution of appropriate antibiotic therapy.

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Basidiobolomycosis of the Rectum Masquerading as Crohn's Disease: Case Report and Review

Basidiobolomycosis caused by *Basidiobolus ranarum* Eidam, is a chronic inflammatory disease that is generally restricted to the subcutaneous tissue. Gastrointestinal zygomycosis due to Entomophthorales is a rare entity [1, 2], which can be distinguished from infections caused by Mucorales on the basis of mycological features of the etiologic agent and by characteristic histopathologic findings [3]. We describe the first culture-proven case of basidiobolomycosis of the rectum in a Bangladeshi male who apparently had no predisposing factors.

In January 1996, a 30-year-old Bangladeshi male was admitted to Al-Amiri Hospital, Kuwait, with a complaint of rectal bleeding often associated with constipation. On physical examination, a large polypoid mass was noted in the rectum, starting from the dentate line and extending ~10 cm into the lower third of the rectum. The circumferential mass appeared to involve the entire rectal wall as well as the perirectal tissues, and palpation of the mass revealed a very firm consistency and tightness around the examining finger.

A sigmoidoscopy was performed and revealed intact mucosa that was inflamed with many tiny ulcers that bled easily to touch. The preliminary diagnosis was carcinoma of the rectum. The results of laboratory studies, including a complete blood count, were normal except for persistent leukocytosis (WBCs, $18–22 \times 10^9$/L). Serological studies for HIV, hepatitis B surface antigen, hepatitis C virus, and syphilis were negative. There was a polyclonal increase in the γ-globulin level, consistent with chronic inflammation.

An ultrasonogram of the rectum revealed a tumor-like lesion; this was confirmed by a CT scan of the pelvis and lower abdomen, which showed a circumferential lesion ~17 cm in diameter. Abdominal radiographs obtained after administration of a barium enema showed narrowing of the rectal passage that extended down to the anal orifice. The rectal lesion was also seen on colonoscopy; the remainder of the colon was normal. Histopathologic examination of the first biopsy specimen of the rectal lesion showed epithelioid cell granulomas infiltrated with eosinophils, a few neutrophils, and lymphoplasmocytic cells. These findings led to the provisional diagnosis of active Crohn's disease.

Initially, treatment for Crohn's disease was instituted, and later an antituberculous treatment regimen was added. However, despite

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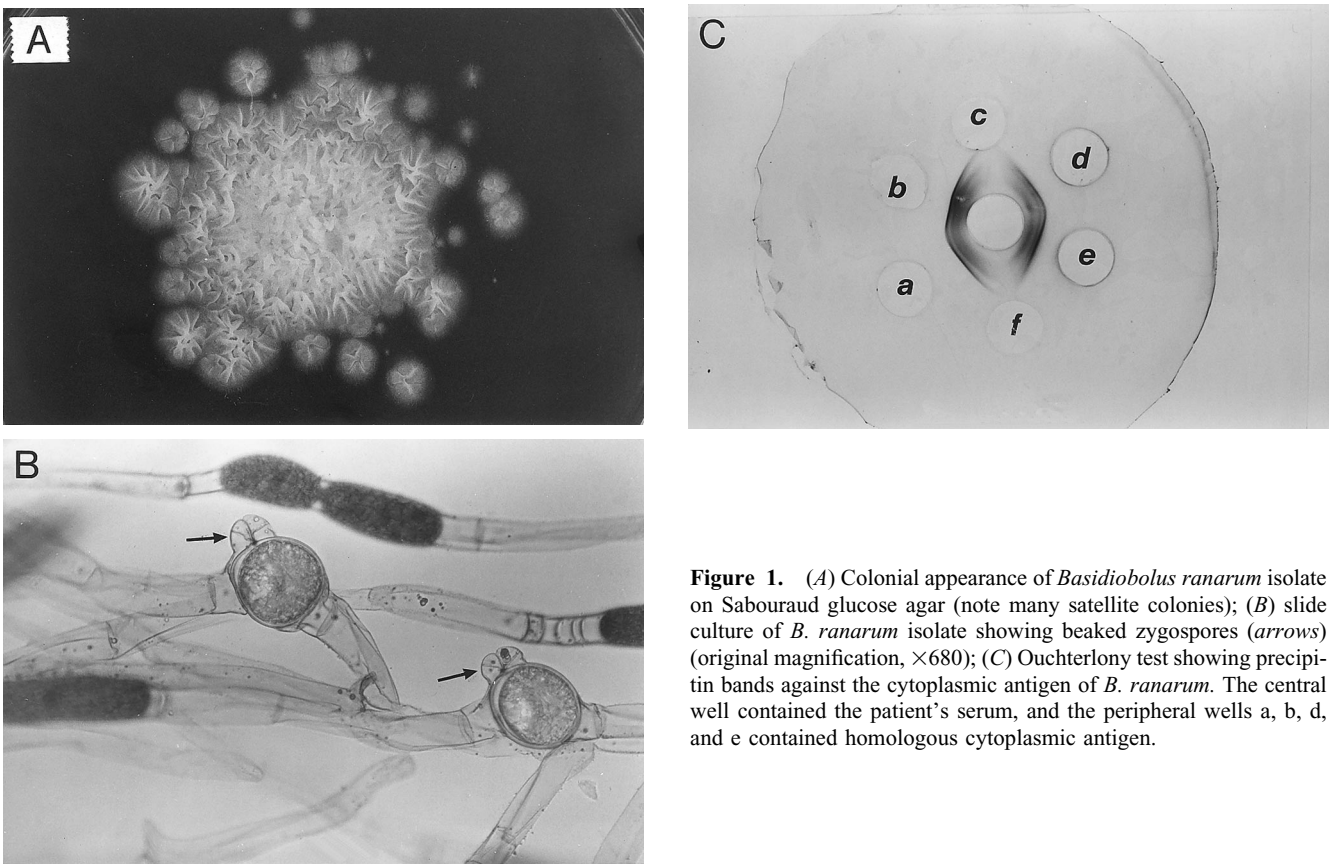

Figure 1. (*A*) Colonial appearance of *Basidiobolus ranarum* isolate on Sabouraud glucose agar (note many satellite colonies); (*B*) slide culture of *B. ranarum* isolate showing beaked zygospores (*arrows*) (original magnification, ×680); (*C*) Ouchterlony test showing precipitin bands against the cytoplasmic antigen of *B. ranarum.* The central well contained the patient's serum, and the peripheral wells a, b, d, and e contained homologous cytoplasmic antigen.

8 weeks of treatment with the above regimen, the rectal lesion did not regress. A transverse colostomy was performed, and this treatment was not helpful. Because the patient's condition continued to deteriorate, a second biopsy specimen was obtained with a tru-cut needle (Travenol Laboratories, Deerfield, IL), which, when cultured, yielded pure growth of *B. ranarum.* The identity of the isolate was confirmed by its characteristic colonial (figure 1A) and microscopic morphology (figure 1B) [3]. The hematoxylin-eosin-stained section of the biopsy specimen revealed the presence of broad, sparsely septate hyphae with marked eosinophilic infiltration. In addition, an Ouchterlony test of the patient's serum demonstrated multiple precipitin bands against the cytoplasmic antigen prepared from *B. ranarum* isolate (figure 1C).

The patient was treated with amphotericin B (1 mg/[kg · d]) for 3 weeks, and 1 week later ketoconazole (200 mg/d) was added to supplement the treatment. Because the patient also had severe gastritis (biopsy proven), addition of potassium iodide to the treatment regimen was deferred. The patient's condition showed marginal improvement, both clinically and on a CT scan, but he returned to Bangladesh without completing the full course of treatment.

We describe the first culture-proven case of basidiobolomycosis of the rectum. It is not clear where and how the patient acquired this infection at this unusual site. Culture-proven cases of gastrointestinal zygomycosis due to *B. ranarum* have been reported only twice [1, 2] (table 1). In both instances, the rectum was not affected, although there was extensive involvement of other intestinal segments, including the stomach in one case. In addition, there are two reports of invasive entomophthoromycosis involving the gastrointestinal tract [4, 5]. It is not known whether these cases were due to *Basidiobolus* species or *Conidiobolus* species, given that the characteristic histopathologic features produced by these organisms are indistinguishable.

The causative fungus in our case, *B. ranarum,* is found in decaying vegetable matter, soil, and the gastrointestinal tracts of reptiles, fish, amphibians, and insectivorous bats [3, 6, 7]. Occasionally the fungus has been isolated from insects [8]. Thus, stagnant water in ponds inhabited by fish or amphibians could be an important source of this fungus. Use of ponds as a water source for bathing or toilet purposes is not an uncommon practice in Bangladeshi villages. Of interest in our case is the localized nature of infection, restricted only to the rectum, which raises the possibility of some type of trauma to this region that might have predisposed the patient to this infection. Minor trauma and insect bites are considered the probable means by which *B. ranarum* gains access to human tissue causing subcutaneous disease [3].

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Table 1. Summary of culture-proven cases of gastrointestinal basidiobolomycosis.

Reference	Sex/ age (y)	Risk factor(s)	Symptoms	Diagnostic material	Histopathology	Organism cultured	Treatment (dosage), duration	Outcome
[1]	M/4	None	Abdominal pain, fever, heavy sweats, diarrhea	Tissue from gastrojejunal junction and colon	Epithelial cell and foreign body giant cell granuloma, Splendore-Hoeppli phenomenon around hyphae	*Basidiobolus ranarum* (as *Basidiobolus haptosporus*)	None	Died
[2]	M/69	Anergy, diabetes	Fever, nausea, constipation, bilious vomiting, lower quadrant pain	Tissue from cecum, ascending colon, part of duodenum	Prominent eosinophilic infiltration with histiocytes	*B. ranarum* (as *B. haptosporus*)	Amphotericin B, 2 w	Died
[PR]	M/30	None	Rectal bleeding, constipation	Biopsy specimen from rectal wall	Marked eosinophilic infiltration, few neutrophils and lymphoplasmocytic cells	*B. ranarum*	Amphotericin B (700 mg) and ketoconazole (200 mg/d), 2 w (incomplete treatment)	Left the country

NOTE. PR = present report.

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Severe Allergic Reaction After Repeated Exposure to Indinavir

To our knowledge, severe allergic reactions to indinavir with hypotension, rash, fever and elevation in liver enzyme levels (ELELs) have not been reported [1]. We describe a patient with a severe clinical picture of a drug-induced allergic reaction after exposure (repeated) to indinavir.

A 40-year-old male homosexual patient with AIDS ($CD4^+$ lymphocyte count, $20/mm^3$) started receiving a triple-therapy (TT) regimen with stavudine, lamivudine, and indinavir together with oral ganciclovir, itraconazole, pentamidine, and temazepam after being hospitalized for iv treatment of cytomegalovirus (CMV) retinitis with ganciclovir. He was rehospitalized 22 days later with fever (temperature, 39°C), icterus, hepatosplenomegaly, and ELELs. Two of six blood cultures yielded *Staphylococcus epidermidis,* and treatment with flucloxacillin was instituted. TT and itraconazole were stopped. Serology for CMV in the buffy coat was negative as was serology for hepatitis A and B. Mycobacterial blood cultures remained negative. Funduscopic examination showed stable disease.

After 5 days of treatment with flucloxacillin, there was normalization of the liver enzyme levels and disappearance of the fever and icterus; TT was reinstituted at 1500 hours. Five hours later the patient developed a diffuse rash, hypotension (blood pressure, 85/50 mm Hg), fever (temperature, 40.2°C), and diarrhea. Again there were ELELs and new neutropenia (200 cells/mm^3; previous values, $>1{,}200/mm^3$). Treatment with flucloxacillin was discontinued because of possible penicillin intolerance in the past and was replaced by vancomycin, amikacin, and granulocyte colony-stimulating factor (G-CSF). Because the fever persisted, TT was again discontinued 24 hours later. The fever and rash resolved after 12 hours, and the ELELs resolved 36 hours later, followed by a diffuse exfoliation. Blood cultures remained negative.

Finally, 5 days after withdrawal of the antibiotic treatment (the patient was receiving only oral ganciclovir, G-CSF, and topical

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