

Figure. Onset dates of 34 cases of microsporidial keratoconjunctivitis among rugby players from Hong Kong, People's Republic of China, who were exposed to contaminated soil and mud during a tournament in Singapore, April 21–22, 2012. Three cases (onset May 4, 5, and 7) were diagnosed in players by positive PCR testing; all other cases were diagnosed by the presence of eye redness and 1 of the following ocular signs or symptoms since April 21: pain, discharge, swelling, or itchiness.

This outbreak provided an opportunity to study the incubation period of *V. corneae* keratoconjunctivitis in otherwise healthy persons. Time from soil exposure to development of ocular symptoms of microsporidial keratitis has been reported to be 2–21 days (median 14 days) (2) and 5–14 days (mean 6.8 days) (3). The incubation period during this outbreak was 5–31 days (median 15 days).

An investigation by the Singapore Ministry of Health of this outbreak revealed that microsporidial spores are probably ubiquitous in soil in Singapore (4). All interviewed rugby players from Hong Kong were exposed to soil or muddy water in Singapore before the outbreak, but we could not demonstrate the doseresponse relationship because of the long incubation period and difficulty quantifying exposure to soil and muddy water.

This was an uncommon keratoconjunctivitis microsporidial outbreak. Advice about hygiene should be given to athletes who are exposed to dirt and mud on playing fields to minimize their risk for infection. Instructions should be given for safe and thorough washing of eyes, especially after dirt/mud exposures on waterlogged playing fields. Clinicians public health professionals and must consider microsporidial

keratoconjunctivitis as a differential diagnosis for conditions of field athletes who exhibit eye redness accompanied by eye pain, discharge, swelling, or itchiness after exposure to soil or mud.

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Mycobacterium fortuitum Endocarditis Associated with Cardiac Surgery, Serbia

To the Editor: Mycobacterium fortuitum is a member of the group of rapidly growing nontuberculous mycobacteria. It is a well-known causative agent of skin and soft tissue infections, postsurgical wound infections, and other health careassociated infections (1). Only sporadic cases of endocarditis caused by this bacterium have been reported (2–4). We describe a cardiac surgeryrelated outbreak of endocarditis caused by M. fortuitum in 3 children.

Over a 3-week period during 2009, eight children consecutively underwent surgery for correction of ventricular septal defect (VSD) by insertion of a bovine pericardial patch at the University Children's Hospital in Belgrade, Serbia. None of them had previous cardiac surgery.

The same patch, SJM Pericardial Patch with EnCap Technology (St. Jude Medical, St. Paul, MN, USA), was used as a source for smaller, tailored patches for all patients. Sterile scissors and forceps were used to tailor a piece of the patch needed for a corresponding VSD closure. During repeated performances of this procedure and between surgeries, the patch had been continuously stored in 2% propylene oxide (PO) provided by the manufacturer. Each tailored piece of the patch had been immersed into freshly prepared sterile saline for 6 min before defect patching. The postoperative course had been uneventful for all patients, and they were discharged 7 days after the procedure. However, 3 patients were readmitted to the hospital because of prolonged fever and increasing fatigue. Patients 1, 2, and 3 (Table) had been the fourth, sixth, and eighth patients undergoing VSD repair, respectively. Diagnosis of infective endocarditis in these patients was established by transthoracic echocardiography findings and blood cultures positive for acid-fast bacteria (Table). Acidfast bacteria also were recovered from the patch and vegetation taken during reoperation in patient 3 (Table). The isolates were identified as M. fortuitum by the GenoType Mycobacterium CM assay (Hain Lifescience, Nehren, Germany) (5). Empiric treatment with vancomycin and ceftriaxone was switched to amikacin, ciprofloxacin, and imipenem. After 6 weeks of treatment, the patients were discharged, and all were asymptomatic 12 months later.

The cultural characteristics and susceptibility patterns of all the isolates obtained were indistinguishable. To explore their possible clonal relatedness, we genotyped 3 *M. fortuitum* strains isolated from blood cultures (1 isolate per patient) and 2 *M. fortuitum* isolates recovered from samples taken during reoperation in 1 of the patients. The enterobacterial repetitive intergenic consensus PCR was used (6), and all isolates produced identical patterns.

Nosocomially acquired M. fortuitum endocarditis has been reported but only sporadically in adults, and these cases usually were fatal (3,4,7). In contrast, we describe 3 related cases of M. fortuitum endocarditis in children who recovered. The relatedness of the cases is strongly supported by the following. First, epidemiologic links are obvious because the 3 patients underwent surgery in the same operating room, and the same patch was used in all of them. Second, M. fortuitum strains isolated from the 3 patients were phenotypically and genotypically identical.

Repeated use of the same patch in multiple surgeries strongly suggests

the contaminated patch was the source of M. fortuitum infection in the 3 patients. This possibility could not be corroborated by bacteriologic examination of the patch because the remaining unusable fragments had been discarded after the surgeries (i.e., \approx 3 months before the outbreak became evident). Although contamination of the patch during manufacture is possible (8), it seems more reasonable to assume that the contamination occurred intraoperatively. The common factor in nosocomially acquired M. fortuitum infections is presumed to be exposure to a liquid contaminated with this organism (1,9). The patch was not exposed to solutions other than the PO in which it had been stored and the sterile saline used during the rinsing procedure. Because only a piece of the patch tailored for a particular patient was exposed to a saline freshly prepared for each surgery, contamination of the PO by M. fortuitum presumably led to contamination of the patch. Liquid PO is used as a chemical sterilant for bioprostheses intended for single use. However, multiple use of the same patch implied repeated exposure of the PO solution to the environment and prolonged storage at 4°C between surgeries. Because PO effectiveness is markedly reduced at temperatures <16°C (10), the specific circumstances could have compromised the sterilizing capacity of the PO solution and enabled

Table. Characteristics of patients in an outbr	eak of Mycobacterium fortuitun	n endocarditis, Serbia*	
Characteristic	Patient 1	Patient 2	Patient 3
Age, y/sex	12.0/F	2.0/F	0.5/M
Comorbidity	No	Down syndrome	No
Time between surgery and readmission, d	86	97	76
Position of vegetation	Septal cusp of tricuspid valve	Septal cusp of tricuspid valve and VSD patch	Septal cusp of tricuspid valve and VSD patch
Hemodynamic consequence	Moderate tricuspid valve regurgitation	Moderate tricuspid valve regurgitation	VSD patch dehiscence
No. blood cultures collected, aerobic/anaerobic	3/3	10/4	6/3
No. blood cultures positive for acid-fast bacteria, aerobic/anaerobic	2/0	5/0	2/0
Time of collection of first positive/last positive blood culture, d after readmission	8/11	1/21	4/16
Outcome	Resolved after antimicrobial drug treatment	Resolved after antimicrobial drug treatment	Resolved after antimicrobial drug treatment and reoperation

^{*}VSD, ventricular septal defect.

contamination by ubiquitous *M. fortuitum*.

We are well aware that the patch was intended for single use only and that application of the same patch in multiple patients is not a practice in industrialized countries. However, it is a practice in some resource-limited countries. The outbreak of *M. fortuitum* endocarditis we describe is a clear warning that such practice is associated with high risk and thus should be discontinued.

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Cryptococcus gattii, Florida, USA, 2011

To the Editor: Cryptococcosis is a systemic mycosis most commonly caused by 2 species of encapsulated Cryptococcus yeast: neoformans and C. gattii. C. gattii is a globally emerging pathogen. In the United States, an outbreak of *C. gattii* infection caused by molecular type VGII has been ongoing since 2004, primarily in the Pacific Northwest (1). In addition, sporadic cases caused by molecular types VGI and VGIII have been reported in other areas, including North Carolina, Rhode Island, New Mexico, Michigan, Georgia, and Montana (2). We report a case of disseminated C. gattii VGIIb infection in the United States outside of the Pacific Northwest in an otherwise healthy Florida native who had no known travel to C. gattii endemic areas.

In May 2011, a 50-year-old man sought care for 6 months of progressive pain, swelling, and deformity of the left thigh and stiffness of his left knee. His only recent trauma was a minor left lower extremity injury 2 years earlier when a horse rolled on him. However, he had no fracture, and the injury eventually healed without medical care. He also reported occasional productive coughing and smoking 1 pack of cigarettes per day for 30 years. The patient was born and raised in Pasco County, Florida, and had not traveled outside of Florida in 20 years. He reported working on a dairy farm and having regular exposure to horses and pigs. Imaging showed a possible fracture of his left femur at the same site as the horse-related injury 2 years earlier. Computed tomographic scan of the chest demonstrated mediastinal lymphadenopathy and multiple pulmonary nodules bilaterally.

The man underwent open biopsy and fixation of the left femur fracture. Arthrocentesis was performed on his