Sphingomonas mucosissima
Bacteremia in Patient with Sickle Cell Disease

To the Editor: The genus Sphingomonas was proposed by Yabuuchi et al. in 1990 (1) and amended by Takeuchi et al. in 1993 (2). It now has been subdivided into 4 separate genera: Sphingomonas sensu stricto, Sphingobium, Novosphingobium, and Sphingopyxis. The bacteria of the genus Sphingomonas are yellow-pigmented, nonfermenting, gram-negative bacilli with a single polar flagellum; they are widely distributed in the natural environment, especially in water and soil (3). These bacteria are characterized by the presence of a unique sphingoglycolipid with the long-chain base—dihydro sphingosin, ubiquinone 10 (Q-10), and 2-hydroxy-myristic acid (2-OH C14:0)—and the absence of 3-hydroxy fatty acids (4). S. mucosissima was isolated and identified in 2007 by Reddy and Garcia-Piché from biologic soil crust samples collected from sandy arid soil in the US Colorado Plateau (5). Sphingomonas spp. are opportunistic pathogens and have recently been implicated in a variety of community-acquired and nosocomial infections, considered to originate from contaminated hospital equipment or manipulation of some medical devices (3). The survival of Sphingomonas spp. in indoor dust particles as aerosols and their resistance to many disinfecting and toxic chemicals may explain their ability to colonize medical devices such as mechanical ventilators, catheters, and bronchofiberoscopes (6). In the past few years, these organisms, in particular S. paucimobilis, have been implicated in a variety of community-acquired and nosocomial infections.

We report a case of S. mucosissima bacteremia in a patient with sickle cell disease. In February 2008, a 17-year-old woman with homozygous sickle cell anemia was hospitalized when her condition suddenly became worse. The patient had undergone a splenectomy in 1992 and a cholecystectomy in February 2007. Four days after admission, she had a fever of 38.7°C. Two aerobic blood specimens, drawn on the fifth day of her hospitalization, yielded gram-negative bacilli after a 24-hour incubation. The gram-negative bacilli were positive for catalase and oxidase but remained unidentified by API 20NE strip (bioMérieux, Marcy l’Etoile, France). MICs of antimicrobial drugs were determined for the gram-negative bacilli by using an Etest assay (AB BIODISK, Solna, Sweden) on Mueller-Hinton medium. MICs were 1 μg/mL for cefotaxime, 1 μg/mL for amoxicillin–clavulanic acid, 2–3 μg/mL for vancomycin, 0.064 μg/mL for imipenem, 4–5 μg/mL for ceftazidime, 1 μg/mL for amikacin, 3 μg/mL for ciprofloxacin, and 0.047 μg/mL for trimethoprim-sulfamethoxazole.

DNA was extracted from 1 colony by using a QIAamp Tissue kit (QIAGEN, Hilden, Germany) as described by the manufacturer. A 16S rDNA sequence was obtained (1,410 bp) by using the fD1 (5′-AGAGTTTGATCCTGGCTCAG-3′) and rP2 (5′-ACGGCTACCTTGTTACGACTT-3′) primer pair (7,8). Using BLAST version 2.2.9 software (www.ncbi.nlm.nih.gov/BLAST), we determined that this sequence showed 98% similarity with the 16S rDNA sequence of S. mucosissima (GenBank accession no. AM229669). A phylogenetic neighbor-joining tree resulting from comparison of sequences of the 16S rDNA genes of Sphingomonas spp. was made with the MEGA 3.1 software (www.megasoftware.net). This analysis confirmed that the isolate belonged to S. mucosissima.

Initial treatment of intravenous administration of ceftriaxone was begun. The fever resolved after 1 day and the patient’s condition improved. Treatment was stopped after 5 days, and the patient remained afebrile. Two S. mucosissima isolates were recovered from 2 different blood-culture samples drawn 24 hours apart, which suggests that S. mucosissima was not just a transient organism but indeed was responsible for the patient’s septicemia. Phenotypic identification of the gram-negative bacilli failed because the definite bacterial species S. mucosissima was not included in the API database (http://industry.biomerieux-usa.com/industry/food/api/apiweb.htm) used for the phenotypic identification. However, the isolates’ biochemical characteristics were consistent with those previously reported for S. mucosissima (5) (Table). Final identification was achieved by comparing the almost complete 16S rDNA sequence with homologous sequences deposited in GenBank.

Table. Biochemical characteristics of the previously reported Sphingomonas mucosissima isolate (AM229669) and the isolate from this study

| Characteristic | S. mucosissima | Isolate from this study |
|---------------|----------------|------------------------|
| Oxidase       | +              | +                      |
| Catalase      | +              | +                      |
| Phosphatase   | +              | +                      |
| β-galactosidase | –             | –                      |
| Gelatinase    | –              | –                      |
| Nitrate reduction | –         | –                      |
| Assimilation of carbon compounds | | |
| Alanine       | +              | +                      |
| Glucose       | +              | +                      |
| Glutamic acid | –              | –                      |
| Mannitol      | –              | –                      |
| Sucrose       | +              | +                      |
We believe that the patient’s intravenous catheter was the source of the infection because she did not have wound infections, and cultures of her urine were negative for infectious agents. Antimicrobial drug treatment, selected on the basis of an in vitro S. mucosissima susceptibility profile, facilitated the patient’s recovery. This case report illustrates that the pathogenic potential of S. mucosissima should be considered in diagnosis in such cases because the organism can cause bacteremia in patients, primarily in those with underlying debilitating conditions and those who have undergone medical interventions.

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**LETTERS**

WU Polyomavirus in Fecal Specimens of Children with Acute Gastroenteritis, China

To the Editor: WU polyomavi-

r us (WUPyV) is a recently described
PyV found in patients with acute re-
spiratory tract infections (1). The role
of the virus in disease pathogenesis
remains unclear. The ability to detect
it in clinical specimens would help in
the determination of its replication
sites and its routes of transmission and
dissemination. WUPyV has been
found in specimens from the respira-
tory tract only (1).

Previous studies of other PyVs, including BK virus, JC virus, and
the newly identified KIPyV, demonstrat-
ed their presence in fecal specimens
(2,3), which suggests their potential
for transmission through the gas-
троintestinal (GI) tract (2). Because
some children (6.8%–27.7%) who
had WUPyV results in previous stud-
ies (1,4,5) displayed respiratory and
GI clinical signs, we speculated that
WUPyV might also be transmitted
through the GI tract.

In this study, we tested for the pres-
ence of WUPyV in children with acute gastroenteritis. A total of 377 fecal
specimens were collected from children
with acute nonbacterial gastroenteritis
at the Outpatient Clinic Department of
the Beijing Children’s Hospital from
March 2006 through November 2007.
Patients with nonbacterial gastroen-
teritis were defined as 1) those who
had acute, watery, but not bloody, di-
arrhea, accompanied by other clinical
signs and symptoms such as fever, ab-
dominal cramps, nausea, vomiting, and
headache; and 2) those who had nega-
tive test results for any known bacteria
that might cause gastroenteritis, such as
Salmonella spp., Shigella spp., Staphy-
lococcus spp., Campylobacter jejuni,
Clostridium spp., Escherichia coli, and
Yersinia spp.

All patients, whose ages ranged
from 1 month to 13 years (mean age
11.7 months, median age 9 months),
did not exhibit apparent clinical respira-
tory signs. Fecal specimens from patients
were diluted in phosphate-buffered sa-
line (pH 7.2) by using a 10% wt/vol
ratio and were cleared of cell debris by
centrifugation (2,500 × g, 5 min). Virus
nucleic acids were extracted by using the
NucliSens miniMAG and isolation
reagents according to the manufacturer’s
instructions (bioMérieux, Marcy
l’Etoile, France). Samples were subse-
quently screened for group A rotavirus
(RVA) by using the rotavirus ELISA
diagnostic kit (Lanzhou Institute for