and anaerobic bacteria); no cases were reported to be due to *Pseudomonas* species [1]. Bacterial invasion of the muscle is thought to be secondary to transient bacteremia [3].

It has been proposed that local trauma (such as filariasis in our case) may predispose to pyomyositis and bacteremia, but this has not been proven. In conclusion, polymicrobial pyomyositis due to catalase-producing pathogens may be found among patients with AIDS. Our patient's case was also unusual because his disease recurred after an asymptomatic period of 15 months [3].

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Vibrio cholerae O139 Synonym Bengal in Hong Kong

SIR—The discovery of *Vibrio cholerae* O139 synonym Bengal in Bangladesh and India last year [1] established the epidemic potential of certain non-O1 serotypes of *V. cholerae*. We present a case of infection due to *V. cholerae* O139 that was contracted in southern China and that was related to the Indian O139 strain.

In May 1993 a 60-year-old man was admitted to the hospital for evaluation of abdominal pain, vomiting, and severe watery diarrhea after returning from the Guangdong Province in southern China. The patient was in hypovolemic shock and was anuric on admission. He was successfully resuscitated when 10 L of intravenous fluid were given over 24 hours. Culture of his stool on thiosulfate citrate bile salts sucrose agar yielded a sucrose-utilizing *Vibrio* species after 18 hours. Biochemical reactions were compatible with those of *V. cholerae*, although the organism was resistant to the vibriostatic compound O/129 (Oxoid, Basingstoke, United Kingdom). The strain was found to be susceptible to tetracycline when the Kirby-Bauer disk diffusion test was performed. Serotyping with *V. cholerae* O1 antiserum (Burroughs Wellcome, London, United Kingdom) was negative but positive with the O139 antiserum (courtesy of Dr. T. Shimada and Professor Y. Takeda, National Institute of Health, Kyoto, Japan). The patient was treated with oral tetracycline (500 mg four times a day) and subsequently recovered.

The characteristics of this "Guangdong" strain were compared with those of the Indian and Bangladesh outbreak strains (provided by Dr. G. B. Nair, National Institute of Cholera and Enteric Diseases, New Delhi, India). The Guangdong and Indian strains had a biochemical profile number of 7000610027 using the AutoMicrobic System GNI card (Vitek Systems; bioMérieux Vitek, Hazelwood, MO), but the Bangladesh strain had a different profile number of 7000610227. All three strains were resistant to cotrimoxazole, polymyxin B (50 units) and O/129. All three strains produced enterotoxin, as demonstrated with the use of the rabbit ileal loop model and latex agglutination assay by VET-RPLA Toxin detection kits (Oxoid) [2]. DNA fingerprinting was also performed by digestion with *Hpa*II and *Pst*I. The Southern-blotted restriction fragments were hybridized with a ^{32}P-labeled cholera toxin gene (*ctx*A) probe [3]. The Guangdong strain and Indian strains had an identical pattern that was distinct from the Bangladesh strain (figure 1). Whereas the O139 strains tested produced two bands after hy-

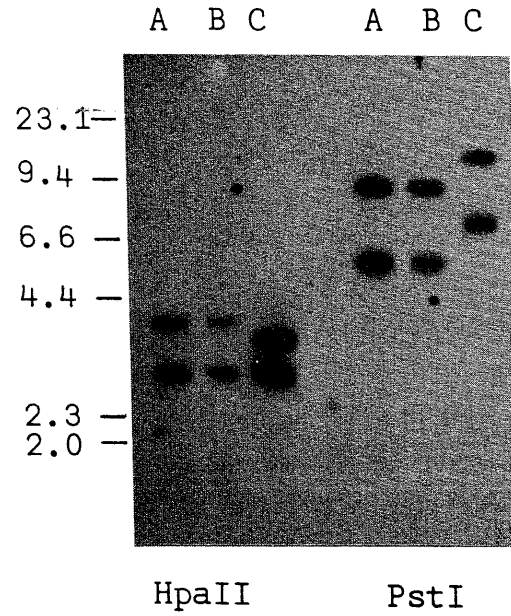

Figure 1. Chromosomal DNA of strains of *V. cholerae* O139 seen after digestion with *Hpa*II and *Pst*I, electrophoresis, Southern blotting, and hybridization with ^{32}P-labeled cholera toxin gene probe. Lane A, "Guangdong" strain; lane B, Indian strain; lane C, Bangladesh strain. Molecular weight markers are in Kb.

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bridization with the *ctx*A gene probe, all previous Southeast Asian isolates of *V. cholerae* produced a single band [4].

Since the initial isolation of *V. cholerae* O139 in India and Bangladesh, the strain has rapidly spread to many parts of the world. The spread of the India strain of *V. cholerae* O139 to southern China was confirmed here by both serological and molecular means. This is not unexpected, given the increasing cross-border trading in southwestern China and poor hygienic conditions made worse by the influx of millions of workers from northern China every year.

The India and the Bangladesh strains of *V. cholerae* O139 were distinguished with use of molecular typing and biotyping methods. The Guangdong strain described herein was identical to the India strain with respect to biotype, serotype, antibiogram, enterotoxin production, and the hybridization pattern of the *ctx*A cholera toxin gene. Nevertheless, the cholera toxin gene pattern in *V. cholerae* is known to be an unstable trait. Ribotyping with a broad-spectrum rRNA probe revealed no difference so far in hybridization pattern among all the O139 strains (unpublished data), despite the presence of different *ctx*A gene patterns. It is therefore possible that all of the O139 strains belonged to the same clonal linage, with subsequent divergence in terms of cholera toxin gene arrangement. The presence of two copies of cholera toxin gene in the O139 strains was also documented in this case. Our results agree with those of previous investigators who identified tandem repeats of the cholera toxin gene in *V. cholerae* O139. This characteristic probably accounts for the huge quantities of toxin produced by this organism that result in infections of the same severity as *V. cholerae* O1.

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Does Hospitalizing Patients with Tuberculosis Ensure Compliance with Drug Regimens?

SIR—Physicians in some developing and industrialized countries are facing a formidable failure rate in the treatment of tuberculosis because of poor compliance with treatment regimens, which has led to the emergence of drug-resistant organisms [1, 2]. The primary factors that govern compliance with a drug regimen are the attitudes of the patient, the physician, and the nursing staff; the nature of the illness; and the type of medication prescribed [3]. Direct observation of patients who are receiving short-course chemotherapy greatly increases levels of compliance; this is especially true in the case of outpatient treatment programs, under which the vast majority of patients with tuberculosis are treated [4].

Since the 1970s, it has been the standard practice of Hong Kong's Chest Service at Grantham Hospital to fully supervise administration of chemotherapy for tuberculosis to outpatients, whether the patient receives treatment as a study participant or through a treatment program [5]. Administration of medication in the hospital setting has been used to improve patients' level of compliance with drug treatments even if the disease is not severe enough to warrant hospitalization. However, equating hospitalization with compliance is problematic, and the cost of inpatient treatment can be prohibitive [3]. We report the case of a patient with tuberculosis who was hospitalized for treatment, yet still did not comply with his treatment regimen.

A 44-year-old male drug addict and recovering alcoholic had several courses of supervised outpatient chemotherapy for pulmonary tuberculosis from 1977 to 1986. His attendance had been irregular and he had never completed any of the courses of treatment. In 1986 cultures of his sputum still yielded *Mycobacterium tuberculosis* that was susceptible to standard drugs.

In March 1992 the patient was admitted to our hospital for treatment of relapsed pulmonary tuberculosis that had caused progressive lung destruction. He was initially given kanamycin, rifampin, isoniazid, ethambutol, and pyrazinamide. Subsequently, a drug susceptibility profile revealed resistance to streptomycin, kanamycin, and rifampin. He was then given isoniazid, ofloxacin, ethambutol, pyrazinamide, and ethionamide. The patient was discharged upon his request a few months later when his clinical condition had improved; however, smears and cultures of sputum were still positive for acid-fast bacilli. He did not return for the recommended outpatient therapy.

In May 1993 the patient was readmitted because his clinical condition had worsened. Radiography showed further deterioration of the lungs. Examination of samples of sputum showed a heavy bacillary load, and the drug susceptibility pattern was similar to that found in 1992. He was given the second-line regimen that he was given in 1992 (isoniazid, ofloxacin, ethambutol, pyrazinamide, and ethionamide). However, after 3 months of hospitalization there was no radiographic or bacteriologic evidence of improvement in his condition.

Although the nursing staff stated that they had directly observed the patient when he took his medication, results of a

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