Melioidosis in Tsunami Survivors

To the Editor: A tsunami devastated coastal areas of the Indian Ocean rim in December 2004. Of the affected countries, more than half of the 300,000 deaths occurred in the Aceh Province of Indonesia, close to the epicenter of the earthquake near northern Sumatra. Infrastructure, including medical and laboratory facilities, in this region was severely damaged. Of >1,000,000 survivors, >500,000 likely were injured. Most injuries were from trauma, but a substantial number were caused by aspiration of, or immersion in, saltwater that may have been contaminated by soil, sewage, or other environmental sources.

Melioidosis, caused by the saprophytic gram-negative bacillus Burkholderia pseudomallei, is endemic in Southeast Asia and northern Australia. Most cases have been found in northeastern Thailand, Singapore, and northern Australia. Melioidosis has been reported only sporadically from Indonesia and mainly in returning travelers (1–3).

In the context of acute medical relief efforts to the town of Banda Aceh, we report on 10 patients with pneumonia, including 4 patients with culture-confirmed melioidosis, after their immersion in contaminated saltwater during the tsunami. Clinical and laboratory services were reestablished on January 3, 2005, at Fakinah Hospital and on January 13, 2005, at Zainoel Abidin Hospital by the relief teams. Patients were identified opportunistically; details of treatment and outcome were reviewed retrospectively. Cultures were taken when clinically indicated and when specimens were available. Sputum cultures were plated onto horse blood agar, cystine lactose electrolyte-deficient agar, Haemophilus agar, and colistin/nalidixic acid blood agar incubated in a candle jar at 35°C for ≤3 days. Colonies suspicious for B. pseudomallei were subcultured to B. cepacia-selective media (Oxoid, Adelaide, South Australia, Australia). Blood cultures and screening cultures of throat and rectum specimens were not performed routinely. Isolates of B. pseudomallei and characteristic antimicrobial drug susceptibilities were confirmed with API20NE (bioMérieux, Marcy l’Étoile, France).

From January 3 to January 28, 2005, a total of 10 cases of postimmersion pneumonia were identified. All patients were <18 years of age and previously well; 6 were male. No cases were epidemiologically linked to others. The patients were treated 10–35 days after the tsunami. Eight had bilateral alveolar opacities on chest radiograph; 3 also had empyema. Clinical, radiologic, and microbiologic details are summarized in the online Table (Available from http://www.cdc.gov/ncidod/EID/vol11no10/05-0740.htm#table).

The sputum cultures of 4 patients were positive for B. pseudomallei. Except posttsunami exposure, none had risk factors for melioidosis, including diabetes, renal failure, or thalassemia. Other co-isolated organisms included Pseudomonas aeruginosa and Klebsiella sp.; 2 patients who did not have cultures taken had cavitory lung disease. All patients with melioidosis were treated with meropenem, and all but 1 clinically improved in the hospital.

This is the second report of melioidosis from within Indonesia (1) and the second published report of melioidosis after the tsunami disaster (4). Cases from this event were included in a preliminary communication (5). However, exported cases of melioidosis from Indonesia, as well as the neighboring countries of Singapore and Malaysia, have been reported previously (2,3), a likely indication that this infection is underrecognized in Indonesia. Melioidosis has also been reported in tsunami survivors from Sri Lanka (4) and Thailand.

A striking feature of this event is the lack of predisposing factors and the young age of the patients. This feature likely represents the unique mode of acquisition and magnitude of the infecting inoculum, as well as the vulnerability of children to near drowning after flooding. One third of the patients had empyema, which reflects both the severity of pulmonary disease and delay in receiving medical care. Undoubtedly, a substantial selection bias occurred by including only patients who sought hospital treatment, and this is suggested by the low death rate. Melioidosis, acquired after near drowning, has been associated with a short incubation period and severe disease. However, patients who had melioidosis before medical aid arrived in the region would likely have died. Patients with longer incubation periods also may have acquired melioidosis through contaminated wounds with subsequent hematogeneous dissemination.

Medical services to the region were by no means comprehensive during this time. Many other patients with postimmersion pneumonia and melioidosis may have been overlooked, both during the study (for persons who were not treated or if cultures were not taken) and afterwards; the incubation period of melioidosis may be ≤62 years. A further limitation is the lack of denominator data because no reliable records were kept on hospital admissions, and the exact number of survivors is not yet known. Conflicting data are found on the accuracy of the API20NE test kit used to identify bacteria in this study (6–9), but we believe that the clinical features and microbiologic findings suggest melioidosis.

This report confirms that B. pseudomallei exists in the Aceh Province of Indonesia and that melioidosis and gram-negative pneumonia may complicate saltwater immersion in this region. After near drowning incidents, melioidosis is characterized
by severe pulmonary disease, including pleural effusions. Clinicians worldwide should be mindful that melioidosis in tsunami survivors may appear many years after exposure.

Acknowledgments

We thank Rus Munander; colleagues who participated in acute medical relief efforts in Banda Aceh; Geoff Hogg; Emergency Management Australia; Queensland Health; Queensland Ambulance Service; our colleagues who supported us at home; and Diana Huis in ‘t Velt who translated the Dutch case report.

The Australian Agency for International Development and the Australian Department of Foreign Affairs and Trade provided support for relief efforts.

Eugene Athan, Anthony M. Allworth, Catherine Engler, and Ivan Bastian participated in medical relief efforts, collected data, and contributed to writing this article. Allen C. Cheng analyzed the results and wrote the article.

Eugene Athan,* Anthony M. Allworth,† Catherine Engler,‡ Ivan Bastian,§ and Allen C. Cheng¶

*The Geelong Hospital, Geelong, Victoria, Australia; †Royal Brisbane Hospital, Brisbane, Queensland, Australia; ‡Queensland Health Pathology Services, Townsville, Queensland, Australia; §Institute of Medical and Veterinary Science, Adelaide, South Australia, Australia; and ¶Menzies School of Health Research, Darwin, Northern Territory, Australia

References


Address for correspondence: Eugene Athan, Department of Infectious Diseases, The Geelong Hospital, Barwon Health, Ryrie St, Geelong 3220, Australia; fax: 61-3-5260-3040; email: eugene@barwonhealth.org.au

Chytrid Fungus in Europe

To the Editor: Amphibian species are declining at an alarming rate on a global scale (1). One of the major reasons for these declines is chytridiosis, caused by the chytrid fungus, Batrachochytrium dendrobatidis (1,2). This pathogen of amphibians has recently emerged globally (2,3) and has caused mass die-offs and extensive species declines on 4 continents (1,3); knowledge of its distribution and effects on amphibian populations remains poor. In Europe, little is known about B. dendrobatidis distribution, which is disturbing when one considers that at least 3 European amphibian species are undergoing chytrid-associated die-offs that will likely lead to local extinction (4,5) (J. Bosch et al., unpub. data).

We screened 1,664 current and archived samples of wild amphibians collected in Europe from 1994 to 2004 by researchers using amphibians as study organisms. B. dendrobatidis infects the skin of adult amphibians and the mouthparts of anuran larvae; samples included toe clippings and skin samples from adults and mouthparts of tadpoles. Our sampling was opportunistic, including both caudates and anurans. We screened all samples for chytrid fungus with quantitative real-time polymerase chain reaction (PCR) of the ITS-1/5.8S ribosomal DNA region of B. dendrobatidis (6), including appropriate positive and negative controls. We confirmed real-time PCR positives by amplifying a subset of these positives with a second B. dendrobatidis–specific PCR with a nested reaction developed from the ctsyn1 locus (3). To confirm that detection with real-time PCR indicated a viable chytrid infection, when actual tissue samples were available, we examined a generous subset using histologic features for typical signals of pathogenic B. dendrobatidis infection. Specifically, we found intracellular zoospore-carrying sporangia within the stratum corneum and stratum granulosum of toe and skin samples. We also compared real-time PCR amplification profiles of suspected positives to those generated from samples from animals involved in chytrid-driven die-offs and found these results to be comparable. Furthermore, attempts to isolate the fungus from dead animals were successful when animals were obtained in a suitable condition for this purpose (see below).

Our survey found B. dendrobatidis in amphibians in 5 European countries, Spain, Portugal, Italy, Switzerland, and Great Britain. Previously, chytrid infection has been