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Case Report: Fatal *Burkholderia pseudomallei* Infection Initially Reported as a *Bacillus* Species, Ohio, 2013

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Abstract. A fatal case of melioidosis was diagnosed in Ohio one month after culture results were initially reported as a *Bacillus* species. To identify a source of infection and assess risk in patient contacts, we abstracted patient charts; interviewed physicians and contacts; genetically characterized the isolate; performed a *Burkholderia pseudomallei* antibody indirect hemagglutination assay on household contacts and pets to assess seropositivity; and collected household plant, soil, liquid, and insect samples for culturing and real-time polymerase chain reaction testing. Family members and pets tested were seronegative for *B. pseudomallei*. Environmental samples were negative by real-time polymerase chain reaction and culture. Although the patient never traveled internationally, the isolate genotype was consistent with an isolate that originated in Southeast Asia. This investigation identified the fifth reported locally acquired non-laboratory melioidosis case in the contiguous United States. Physicians and laboratories should be aware of this potentially emerging disease and refer positive cultures to a Laboratory Response Network laboratory.

Melioidosis, which results from infection with the gram-negative bacillus *Burkholderia pseudomallei*, can have a case-fatality rate as high as 40%.[1] The disease is rare in the United States, and most cases are associated with travel to disease-endemic areas, such as Southeast Asia.[2] The organism readily grows in standard media, but it can be difficult to identify accurately and can be dismissed because of its rarity in the United States.[3,4] Acquisition from the environment occurs through percutaneous inoculation, ingestion, or inhalation.[5,6] Melioidosis primarily results from contact with contaminated soil or water; person-to-person transmission is rare.[1] The incubation period ranges from 1 to 21 days (mean = 9 days). However, an incubation period as long as 62 years has been described.[7,8] Patients usually have pneumonia or cutaneous infection.[6] Up to 80% of melioidosis patients have one or more risk factors, including diabetes, excessive alcohol use, and chronic lung disease.[9] Improved survival rates are attributed to early diagnosis and treatment with appropriate antimicrobial drugs and access to intensive care resources.[9] We describe an investigation of a fatal case of melioidosis from Ohio in a patient with no history of ever having traveled outside the United States, of ever having worked in a laboratory, or of ever having served in the military.

In mid-February 2013, a 44-year-old man with a history of poorly controlled diabetes mellitus, hypertension, and chronic back pain came to a small community hospital (hospital A) in Ohio with lethargy, headache, vomiting, body and joint pain, and sore throat. At physical examination, he exhibited hypertension, tachycardia, and fever. He was admitted and given intravenous levofloxacin (750 mg initially, then 500 mg daily) for presumed sepsis associated with an influenza-like illness.

Computed tomography (CT) of the head showed mild bilateral maxillary and ethmoid sinusitis. Chest radiographs and a thoracic CT scan showed normal results. Electrocardiogram results included sinus rhythm, tachycardia, poor R-wave progressions, and nonspecific ST-wave changes on a normal axis. Arterial blood gases at admission found an increased arterial blood gas pH (7.46, reference range = 7.38–7.42) and decreased partial pressure of oxygen (72 mm Hg, reference range 80–100 mm Hg). The total leukocyte cell count was 8,000 cells/μL (reference range = 4,500–10,000 cells/μL) (Table 1). Abnormal laboratory values during his hospital stay included hyperglycemia (glucose = 562 mg/dL, reference fasting range = 70–130 mg/dL), lymphopenia (lymphocytes = 7%, reference range = 20–40%), and monocytosis (monocytes = 13%, reference range = 2–8%) (Tables 1 and 2).

One of four blood cultures was positive for *B. pseudomallei* by Vitek 2 analysis (bioMérieux, Marcy l'Etoile, France) (95% confidence), but the result was reported by the laboratory as *Bacillus* species because of the low suspicion of *B. pseudomallei*. In addition, the colony associated with the single positive culture demonstrated other characteristics typical for *Bacillus* species, such as morphologic characteristics and smell. The patient was afebrile after five days and was discharged with a diagnosis of sepsis and given a 10-day course of oral doxycycline (100 mg every 12 hours).

The patient returned to hospital A on March 31 because of fever and confusion. He was admitted for a presumed urinary tract infection with hyperglycemia (glucose level = 447 mg/dL) and a high serum creatine kinase level (2,734 μg/L, reference range = 52–336 μg/L) and was given intravenous vancomycin (2 grams initially, then 1.75 grams every 12 hours), ceftriaxone (1 gram every 12 hours), and acyclovir (800 mg every 8 hours). Abnormal urinalysis results included hematuria, proteinuria, pyuria, glycosuria, and ketonuria; however, urine cultures were negative.

The patient's condition deteriorated on April 2, and he began to exhibit respiratory distress. He was moved to an intensive care unit, intubated, and afterward transferred to a tertiary care center (hospital B). Initially, clinicians suspected a gram-negative bacterial sepsis from a urinary source. The next day, four of four blood cultures prepared at the time of admission at hospital A had isolates identified as *B. pseudomallei*

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TABLE 1
Hematologic values for the patient with melioidosis at each admission, Ohio*

Laboratory characteristic	February 15, 2013 admission: hospital A	March 31, 2013 admission: hospital A	April 2, 2013 admission: hospital B	Reference range
Hematocrit, %	41.2	ND	**30.4**	40.0–52.0
Leukocyte count, × 10^3 cells/mL	8.0	5.9	**2.6**	4.1–10.9
Differential blood count, %				
Polymorphonuclear cells	79	78	**83.5**	35–80
Lymphocytes	**7**	**14**	**12.7**	20–50
Monocytes	**13**	8	3.8	2–12
Bands	0	0	0	0–10
Platelet count, × 10^3 cells/μL	154	193	**73**	140–450

*ND = not done. Values in bold are outside reference range.

by Vitek 2 analysis (95% confidence), and his antimicrobial therapy was changed to intravenous meropenem (1 gram every 8 hours). On April 5, initial blood culture bottles and additional whole blood samples obtained at hospital B were submitted to the Ohio Department of Health Laboratory, which is part of the Laboratory Response Network. The laboratory conducted real-time polymerase chain reaction (PCR) and biochemical testing and confirmed *B. pseudomallei.* Despite aggressive treatment, the patient's condition continued to deteriorate, and he died on April 8.

The local health department, Ohio Department of Health, and the Centers for Disease Control and Prevention (CDC) investigated the case to identify a source of infection. We abstracted the patient's medical records dating back to September 2007 from hospitals A and B. The patient had sought care at another facility (hospital C), and those medical records were abstracted from July 2011 onward. We also interviewed the patient's physicians and reviewed the autopsy report.

In interviews with the patient's family and close associates, we covered the entire period that they each knew the patient. After obtaining informed consent from all human adult participants and from parents of minors, we collected serum from household members and pets for melioidosis serologic testing by using an indirect hemagglutination assay (IHA).[10] All titers < 1:40 were considered seronegative. The determination was made that the outbreak investigation did not constitute human subjects research and therefore was not subject to institutional review board evaluation. The patient's home was assessed for potential environmental contamination with *B. pseudomallei.* Samples of plants, soil, and liquids were collected for culture and real-time Polymerase Chain Reaction (PCR) testing at CDC.[11] Cockroaches and houseflies were collected and tested at the Ohio Department of Agriculture laboratories.

Review of electronic medical records from hospital A showed that the patient had worsening glucose control starting in early 2012, as shown by glucose levels as high as 564 mg/dL and increasing hemoglobin A1C levels as high as 12.9% (reference range = 4.5–6%). The patient had received lumbar epidural steroid injections during September–November 2012 with medications obtained from U.S. pharmaceutical manufacturers. Three visits for skin-related complaints were identified. The first of these visits, in October 2012, was for a small, indurated shoulder lesion, which resolved after one week of oral trimethoprim/sulfamethoxazole antimicrobial drug therapy and which coincided with an episode in which other family members were treated for "boils". In December 2012, the patient was treated for a nostril pustule, which responded to clindamycin. On January 22, 2013, he reported ear pain. A small erythematous insect-bite was noted. No antimicrobial drugs were prescribed.

Before the most recent visit for ear pain, the patient came to hospital A on January 15, 2013, for evaluation of right lower quadrant abdominal pain. At that visit, two blood cultures were prepared and results were negative. Abdominal radiograph and CT scan results indicated a nonspecific

TABLE 2
Blood chemistry and cardiac enzyme values for the patient with melioidosis at each admission, Ohio*

Laboratory characteristic	February 15, 2013 admission: hospital A	March 31, 2013 admission: hospital A	April 2, 2013 admission: hospital B	Reference range
Sodium, mEq/L	**130**	**125**	**134**	137–145
Potassium, mEq/L	3.8	4.8	3.7	3.6–5.0
Chloride, mEq/L	**95.0**	**89.0**	**113**	98–110
Bicarbonate, mEq/L	**21.0**	**21.0**	**20.0**	22–26
Glucose, mg/dL	**562**	**447**	**41**	65–110
Urea nitrogen, mg/dL	9.0	**23.0**	**37.0**	7–21
Creatinine, mg/dL	0.9	**1.8**	**2.9**	0.5–1.4
Creatine kinase, U/L	141	**2734**	ND	52–336
Alkaline phosphatase, U/L	**145**	84.0	ND	38–126
AST, U/L	25.0	**70.0**	ND	5–35
ALT, U/L	43.0	35.0	ND	7–56
Total bilirubin, mg/dL	**1.8**	0.8	ND	0.2–1.3
Calcium, mg/dL	**7.6**	**8.5**	**3.8**	8.9–10.4
Total protein, g/dL	ND	8.1	ND	6.3–8.2
Albumin, U/L	3.9	**2.9**	**1.8**	3.5–4.8
Troponin, ng/mL	< 0.02	0.15	ND	< 0.4
Lipase, U/L	**65**	**331**	ND	7–60

*Values in bold are outside reference range. ND = not done; AST = aspartate aminotransferase; ALT = alanine aminotransferase.

non-obstructive bowel pattern with no masses or free abdominal fluid. He was discharged the same day and did not report any abdominal pain at his next hospital visit one week later.

Major diagnoses reported at autopsy based upon gross and microscopic evaluation included bilateral severe acute pneumonia and evidence of septic shock. Cause of death was attributed to acute respiratory failure, septic shock, and acute renal failure caused by acute melioidosis.

The patient rarely traveled, spent little time away from home, and had few hobbies. He sometimes traveled across the Ohio River to Kentucky. The patient's only other reported out-of-state travel was one trip to Colorado 22 years ago. His wife noted a significant decline in his overall health beginning in the fall of 2012. By the time of his illness in February 2013, he was mostly bedridden.

The patient and 13 other family members resided in a small town in southern Ohio. Pets kept inside the patient's home included five dogs, three cats, and one locally captured turtle (red-eared slider, *Trachemys scripta elegans*). Cockroach and housefly infestations were readily apparent. Several houseplants were present, including an orchid; several dead houseplants had been discarded in the yard. Because we considered the possibility of a contaminated household, we tested the pets, insects, and houseplants.

Ten of 13 close contacts and 7 of 8 household pets (4 dogs and 3 cats) were tested, and the results were seronegative for *B. pseudomallei*; IHA titers were < 1:10. The patient had a titer of 1:160. Samples of personal care products, tap water, locally purchased loose tobacco, water from the turtle's aquarium, houseplants, soil from each houseplant and a backyard area where the patient had gardened in 2011, and collected insects were tested, and results were negative by culture and by real-time PCR for *B. pseudomallei*.

Molecular subtyping of the isolate at CDC by multilocus sequence typing yielded sequence type 17 (ST17). ST17 is associated with isolates from human cases of melioidosis in Southeast Asia.[12] Isolates from two previous U.S. patients with no travel history to areas endemic for melioidosis were ST426 and ST550. ST17 is not related to these sequence types. The isolate was also characterized by internal transcribed spacer typing and was found to be type CE. Type CE is associated with isolates from human cases of melioidosis in Southeast Asia and Australia.[13]

Despite a thorough investigation, we were unable to determine the source of the patient's infection. The laboratory that reported results as *Bacillus* species in February considered the *B. pseudomallei* Vitek 2 results as too unusual and thus not plausible, especially because only one of four blood cultures were positive. Because the multilocus sequence typing and internal transcribed spacer typing results were consistent with results for isolates from Southeast Asia, and because the patient had not traveled to this region, our hypothesis was that his exposure resulted from a source brought from Southeast Asia to the United States.

Negative IHA results indicated that the patient's family members and pets had no evidence of *B. pseudomallei* infection. However, up to 26% of patients can have negative IHA results at presentation and, on occasion, not show seroconversion.[14] Although environmental samples did not grow *B. pseudomallei*, this bacterium could be viable but in a state that cannot be cultured under standard laboratory conditions, and that could generate false-negative culture results.[15] False-negative results can also occur with direct molecular bacterial detection from soil because PCR inhibitors are often coextracted with DNA.[16] Therefore, it is possible to have undetected *B. pseudomallei* within environmental samples.

The patient's failing health in 2012 might have made him more susceptible to melioidosis. His negative blood culture results in mid-January, followed by his positive culture in February, most likely represents the period during which he first became ill with a *B. pseudomallei* infection. We interviewed only close family contacts and sampled the patient's home, thus limiting the scope of our investigation.

This case is the fifth case of locally acquired melioidosis within the contiguous United States involving someone with no history of working in a laboratory, where occupational exposure to *B. pseudomallei* might occur. The sources of patients' infections were not conclusively determined in any of these cases. The first reported case was in 1950 in a patient who had been in the Army for four months but had never left the continental United States. He had worked in a Chicago stockyard for four years, but the definitive exposure location was never established.[17]

The second case, reported in 1971 and involving a neonate from California, likely resulted from perinatal infection, given the course of illness and findings on pathology. The infant's father had recent military service in Vietnam. However, he had no contact with the infant after delivery and reported no recent illness.[18] Because up to 18% of male melioidosis patients in one study had prostatic abscesses, and sexual transmission has been reported from a patient with melioidosis-related prostatitis, this infection could have been secondary to sexual transmission between parents. Unfortunately, serologic studies for *B. pseudomallei* were not conducted for either parent.[18–20]

The third case involved a patient from California in 2010 who had a history of working with reptiles (CDC, unpublished data, 2010). In 2007 and 2012, *B. pseudomallei* was cultured from two reptiles that had been imported to the United States and purchased at pet shops in California.[21] Because the third patient reported exposure to numerous reptiles daily over several years, exposure could plausibly have occurred from an infected reptile.

The fourth case was reported in 2011 from Arizona; it was extensively investigated, but no exposure source was identified.[22] The patient had a history of diabetes, hypertension, and obesity. When admitted to the hospital to undergo arthrocentesis of his right knee, blood cultures grew *B. pseudomallei.*

These five cases of melioidosis acquired in the United States suggest that melioidosis could be an emerging infection within this country, especially because three of the cases occurred in the past four years. Physicians and laboratories should be aware of this potentially emerging disease and know appropriate steps to accurately and quickly diagnose and treat melioidosis. We recommend that physicians should consider a diagnosis of melioidosis in patients with compatible disease, even patients who have not traveled to known disease-endemic areas. We also recommend that laboratory professionals should not discount results indicating *B. pseudomallei* as false-positive results without considering melioidosis as a possibility. All *B. pseudomallei* isolates should be sent to a Laboratory Response Network laboratory for confirmation.[23,24]

In addition, physicians need to be aware that many antimicrobial drugs commonly used for acute febrile illness are ineffective for melioidosis, and the consequences of infection can

be severe. *Burkholderia pseudomallei* is characteristically resistant to penicillin, ampicillin, first-and second-generation cephalosporins, gentamicin, tobramycin, streptomycin, and polymixin.[1] The recommended treatment of melioidosis consists of 10–14 days of intravenous ceftazidime, meropenem, or imipenem, followed by oral trimethoprim/sulfmethoxazole for 3–6 months.[1] These infections need to be identified quickly and accurately so that patients can receive timely and appropriate medical care, which will increase their chances for survival.

Finally, we recommend that national reporting of melioidosis be required. Mandatory reporting could expedite case recognition and further our understanding of the sources and transmission of this pathogen in the United States.

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