Primary Pneumonic Plague Contracted from a Mountain Lion Carcass


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Background. Primary pneumonic plague is a rare but often fatal form of *Yersinia pestis* infection that results from direct inhalation of bacteria and is potentially transmissible from person to person. We describe a case of primary pneumonic plague in a wildlife biologist who was found deceased in his residence 1 week after conducting a necropsy on a mountain lion.

Methods. To determine cause of death, a postmortem examination was conducted, and friends and colleagues were interviewed. Physical evidence was reviewed, including specimens from the mountain lion and the biologist’s medical chart, camera, and computer. Human and animal tissues were submitted for testing. Persons in close contact (within 2 meters) to the biologist after he had developed symptoms were identified and offered chemoprophylaxis.

Results. The biologist conducted the necropsy in his garage without the use of personal protective equipment. Three days later, he developed fever and hemoptysis and died ~6 days after exposure. Gross examination showed consolidation and hemorrhagic fluid in the lungs; no buboes were noted. Plague was diagnosed presumptively by polymerase chain reaction and confirmed by culture. Tissues from the mountain lion tested positive for *Y. pestis*, and isolates from the biologist and mountain lion were indistinguishable by pulsed-field gel electrophoresis. Among 49 contacts who received chemoprophylaxis, none developed symptoms consistent with plague.

Conclusions. The biologist likely acquired pneumonic plague through inhalation of aerosols generated during postmortem examination of an infected mountain lion. Enhanced awareness of zoonotic diseases and appropriate use of personal protective equipment are needed for biologists and others who handle wildlife.

Plague is an acute, highly virulent zoonotic disease caused by the bacterium *Yersinia pestis*. The disease was introduced to North America ~1900 [1] and is now considered to be endemic in the western United States. Although plague is primarily an infection of rodents and their fleas, humans and other mammals are also susceptible.

There are 3 principal forms of plague in humans—bubonic, septicemic, and pneumonic. Primary pneumonic plague, acquired by direct inhalation of *Y. pestis*, is the rarest form and is characterized by high mortality and the potential for person-to-person spread. During 1925–2006, only 13 human cases of primary pneumonic plague were identified in the United States; among cases with known exposures, 6 (67%) of 9 were associated with face-to-face contact with infected pets (5 cats and 1 dog) and 3 cases (33%) were acquired in laboratory settings (Centers for Disease Control and Prevention [CDC], unpublished data) [2].

CASE REPORT

On 2 November 2007, a 37-year-old wildlife biologist working for the National Park Service was found deceased in his residence at Grand Canyon National Park
(GCNP), Arizona. Colleagues reported that the biologist had been in excellent health until the night of 29 October 2007 when he became acutely ill with fever, chills, nausea, myalgias, and cough with blood-tinged sputum. The following morning, the biologist sought care at a nearby clinic where he had a temperature of 39.1°C, a pulse of 90 beats per min, a blood pressure of 105/61 mm Hg, and an oxygen saturation of 92% on room air. The biologist’s exposures with wildlife were not documented in the patient history. On physical examination, lungs were clear to auscultation, and no lymphadenopathy was noted. His work-up included a rapid influenza test, which was negative; no chest radiograph or blood tests were performed. The biologist was diagnosed with viral syndrome and given instructions to follow up if symptoms worsened. The biologist worked the next morning; his last known interaction was on the evening of 31 October 2007.

METHODS

To determine cause of death, a postmortem examination was conducted by the Coconino County Medical Examiner in Flagstaff, Arizona; specimens were submitted to the Arizona State Health Laboratory in Phoenix and CDC laboratories in Atlanta, Georgia, and Fort Collins, Colorado. Multiple assays were conducted, including tissue staining, polymerase chain reaction (PCR), direct fluorescent antibody, and immunohistochemistry tests. The immunohistochemistry tests used a multistep indirect immunofluorescent phosphatase technique [3], where the primary antibodies were a rabbit polyclonal anti–Y. pestis antibody and a mouse monoclonal anti–Y. pestis antibody.

To determine the source of infection, interviews were conducted with colleagues and friends, and physical evidence was reviewed, including the biologist’s medical chart, camera, cellular phone, and computer. Archived tissues from an animal the biologist had handled shortly before illness onset were submitted to the CDC and the Colorado State University Veterinary Diagnostic Laboratory. Persons in close contact (within 2 meters) to the biologist after he had developed symptoms were identified and offered chemoprophylaxis for possible exposure to plague [4, 5]. To assess local plague risk, an ecologic investigation was conducted at the South Rim of GCNP.

RESULTS

Postmortem findings. Postmortem examination revealed large amounts of hemorrhagic and frothy fluid in both lungs (right lung, 1.4 kg; left lung, 1.05 kg) and multiple consolidations in the right lung (figure 1A and 1B). No enlarged, necrotic lymph nodes or other gross abnormalities were noted. Testing of lung, liver, and spleen tissues at the Arizona State Health Laboratory yielded positive results for plague by PCR but negative results by direct fluorescent antibody testing.

Testing at the CDC identified the presence of intravascular Y. pestis antigens by immunohistochemistry in multiple tissue samples, including samples of the lung, liver, heart, pharynx, and brain. Confluent plague bacilli admixed with an acute inflammatory infiltrate was identified in the lung; inflammation was conspicuously absent from other infected organs (figure 1C–H). Culture of patient tissue samples (lung and liver) yielded Y. pestis, as confirmed [6] by bacteriophage-lysis testing.

Source of infection. The biologist’s work duties included trapping and collaring mountain lions and removing rodents from buildings. At the time of his death, colleagues reported that the biologist had recently retrieved, skinned, and conducted a postmortem examination on a mountain lion. The deceased animal was a radio-collared female mountain lion that had littered 3 kittens in June 2007 and had been tracked by the biologist for 6 months.

Records indicate that a “mortality signal” (no movement after 6 h) was transmitted from the mountain lion’s collar on 25 October 2007 at 3:13 AM from an uninhabited location in the GCNP. Time-stamped photographs taken by the biologist document that he found the carcass on 26 October 2007 at ~2:00 PM. The carcass was in excellent condition with no signs of scavenging, and blood was present in the animal’s nares and in the soil under its mouth and nose (figure 2A) [7]. The biologist carried the carcass ~1 km to his vehicle and then into his garage, where he performed a necropsy with his bare hands (figure 2B); there is no evidence he wore a mask or other personal protective equipment during the estimated 2.5-h examination. According to his notes, the biologist observed lesions on the animal’s head and chest, which he interpreted as bite and claw marks, and on opening the mountain lion’s thoracic cavity found it filled with blood. From these findings, the biologist concluded that the animal had been attacked by another mountain lion and had died from thoracic hemorrhage. On the basis of interviews, photographs, and cellular phone records, there is no evidence that anyone else was present with the biologist during these activities.

Archived specimens of the mountain lion (hide, 2 paws, skinned head, and liver) and necropsy photographs were examined by a veterinary pathologist who identified no wounds consistent with a fatal penetrating injury, as might be inflicted by another mountain lion. Samples from the mountain lion’s liver and submandibular lymph node tested positive for Y. pestis by PCR. Isolates of Y. pestis cultured from the mountain lion’s tissues were subtyped by pulsed-field gel electrophoresis (PFGE) and found to be indistinguishable from isolates recovered from the biologist (figure 3). Samples of the mountain lion’s tissues were also evaluated by Gram and immunohistochemistry stains for Y. pestis. Abundant gram-negative bacilli were found in the subcapsular sinuses of a submandibular lymph node (figure 2C and 2D) that also stained intensely for Y. pestis. Systemic in-
Figure 1. Gross and histopathologic findings for the biologist. 

A and B, Postmortem sections of the biologist’s right lung with multifocal areas of consolidation and edematous and hyperemic parenchyma. 
C, Abundant bacilli admixed with a neutrophilic infiltrate of the lung; also of note is a partially denuded bronchiole (hematoxylin and eosin stain). 
D, Immunohistochemistry for *Yersinia pestis* on the same field as panel C; red staining indicates large amounts of *Y. pestis* antigens present in the bronchiole and neighboring alveoli. 
E, High-power view of the boxed area in panel D shows invasion of respiratory epithelium by *Y. pestis* by immunohistochemistry. 
F, Acute intra-alveolar inflammatory infiltrate admixed with *Y. pestis* antigens. 
G and H, Brain and heart tissue, respectively; although intravascular plague antigens are present by immunohistochemistry, note that there is no associated inflammation.

Infection was evident by immunohistochemistry detection of plague bacilli in liver and brain samples. 

Contact tracing, prophylaxis, and ecologic investigation. Known contacts of the biologist were notified on 3 November 2007, within 2 h after preliminary postmortem results were available; doxycycline, ciprofloxacin, and cotrimoxazole were dispensed the next morning. Forty-nine persons received chemoprophylaxis, including 11 (22%) emergency responders, 10 (20%) work colleagues, 10 (20%) clinic patients, 8 (16%) clinic personnel, and 3 (6%) residential/social contacts. No contacts developed symptoms consistent with pneumonic plague during the typical 2–4 day incubation period (range, 1–6 days) [5]. A search of the South Rim identified no fleas, rodent die-offs, or other evidence suggestive of a plague epizootic.

DISCUSSION

We conclude that the biologist died of primary pneumonic plague acquired through the inhalation of aerosols generated while handling an infected mountain lion. The presence of heavy intra-alveolar inflammation admixed with confluent plague bacilli—in conjunction with the complete absence of
inflammation in other infected organs—provides strong evidence that the lungs were the primary site of infection and that septicemia occurred secondarily. Other findings consistent with an aerosol exposure include the development of cough and blood-tinged sputum within hours of symptom onset, consolidation of the right lung, and the absence of buboes on clinical and postmortem examination. Matched isolates by PFGE further support the mountain lion as the source of the biologist’s infection.

Of the biologist’s known exposures to the mountain lion, we believe he most likely became infected while conducting the necropsy, during which he had prolonged and unprotected contact with infected tissues. The necropsy involved several procedures likely to generate aerosols, including opening the thoracic cavity and transecting the vertebral column. Power tools were found in the garage where the necropsy was performed and may have been used to open or sever the carcass, further increasing the extent of aerosolization. Alternately, it is also possible that the biologist inhaled bacteria during field transport, particularly if the mountain lion had been carried around the biologist’s neck, facilitating compression of the animal’s chest and potentially expelling bacteria from its lungs as the biologist walked. Our investigation, however, found no evidence that the animal was carried in this way or if it had been wrapped in a tarp or bag which would have contained infectious aerosols. On the basis of the timing of these events, we estimate the time from exposure to symptom onset to be 72–82 h and the time from exposure to death to be 125–144 h. Although acquired through tragedy, these estimates may help efforts to model and prevent the spread of pneumonic plague during natural outbreaks or following the intentional use of \textit{Y. pestis} as a bioweapon [5, 8].

Wild carnivores are exposed to plague through fleabites or...
by consuming infected prey [9, 10] and are potential sources of human infection. In addition to this case, wild carnivores have been implicated as the source of 11 human plague cases in the United States since 1970, including 8 cases for which there is direct laboratory evidence (CDC, unpublished data) [9, 11, 12]. Other wild carnivore–associated cases might have been unreported or prevented by prophylaxis [13]. Of the 11 documented cases, carnivore sources were bobcats (5), coyotes (3), a gray fox which infected 2 family members, and a badger. All cases occurred in patients who spoiled or handled skinned animals, and at least 8 cases presented as axillary bubonic plague, consistent with inoculation of \( Y. pestis \) through cuts in the hand. Though direct contact with infected mammals accounts for nearly 20% of all human plague cases in the United States [14], most exposures involve pets [2] or noncarnivorous wild animals, such as rabbits [15].

Our investigation suggests that the biologist did not seriously consider plague as a possible cause of death in this mountain lion or as a potential etiology for his symptoms. Among carnivores, felids are considered highly susceptible to plague, with fatal cases documented in multiple domestic and wild species [16–21]. From his experience working with felids in endemic areas, including GCNP, the biologist was likely aware of the potential for plague in a mountain lion. Nevertheless, in this case, the biologist attributed the animal’s death to trauma and, perhaps as a result, did not take adequate precautions to protect himself from infection. Furthermore, although a prairie dog epizootic was reported 130 km from GCNP in September 2007 [22], unusual mortality had not been observed among local rodents to alert the biologist to plague activity.

No additional cases of human plague were identified during this investigation, despite the fact that pneumonic plague was not diagnosed until after death. Historical accounts [23] and recent outbreaks in Africa [24, 25] have also demonstrated inefficient transmission of pneumonic plague from patients to close contacts, except during the end stages of disease when hemoptysis is a prominent symptom and large numbers of bacteria are present in respiratory secretions [26]. In this case, the risk for person-to-person transmission was reduced because the biologist primarily worked alone in the field, had limited social contacts, and most notably, was not around other persons at the time of his death. Whether contact prophylaxis might have prevented secondary cases is arguable because, even with prompt action, most potentially exposed persons received antibiotics near the end or outside the expected incubation period. An alternative approach could have been to monitor contacts for fever and other symptoms. Further, some contacts (eg, emergency responders) had no true exposures to the symptomatic biologist, yet still received treatment out of an abundance of caution. These observations highlight the difficulty of providing timely and risk-appropriate chemoprophylaxis for pneumonic plague but also lend further reassurance that \( Y. pestis \) is not readily transmitted via close contact.

Despite the occurrence of 65 primary and secondary cases of pneumonic plague during 1925–2007 (CDC, unpublished data), person-to-person transmission has not been reported in the United States since 1924 [27].

This case report, though associated with an unusual exposure for pneumonic plague, underscores the importance for clinicians to exercise a high index of suspicion for zoonotic diseases, particularly in areas where they are endemic. When considering the diagnosis of plague, clinicians should routinely inquire about occupational and recreational exposures of biologists and other persons who handle wildlife (eg, hunters/trappers, taxidermists). It is also imperative that those at risk are educated on the signs and symptoms of zoonotic diseases in both wildlife and humans, to improve disease recognition in the field and after exposure. In response to this case, the National Park Service has issued Service-wide guidelines for safe handling of wildlife [28], including recommendations on the appropriate use of personal protective equipment as guided by the local prevalence of disease, potential routes of transmission, and the relative risk of species-specific activities. Protocols and policies, including active managerial oversight, for necropsies and other work conducted by biologists are also being developed.

For many activities involving wildlife, simple interventions, such as routinely wearing gloves when handling tissues and exercising vigilant hand hygiene, are probably sufficient [29]; however, there are certain situations (eg, necropsies) where additional safety and prevention measures are prudent [30]. Further attention to these and other measures that can reduce the transmission of zoonotic diseases among persons handling wildlife is warranted.

Acknowledgments

We are indebted to the GCNP law enforcement rangers, special agents, and management team who assisted in all aspects of the public health investigation and provided access to records and other physical evidence. We also extend thanks to the many friends and colleagues of the biologist who shared critical and timely information during the investigation and to the biologist’s family who granted permission to publish photographs of the mountain lion and necropsy. Finally, we pay our deepest respects to the biologist, a highly skilled and widely admired researcher, who was a passionate champion for wild cats and their habitats worldwide.

Potential conflicts of interest. All authors: no conflicts.

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