CD Summary

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PUTTING THE 'P' IN 'PESTILENCE' — PLAGUE

For centuries, plague has been synonymous with disaster for many populations. Even today, a suspected plague outbreak can cause mass panic, and social and economic disruption. In 1994 in Surat, India, 500,000 fled the city in fear of a plague epidemic. In the U.S., plague is still feared, primarily due to its potential as a biological weapon. In this *CD Summary* we review both natural and unnatural (i.e., bioterrorist-spread) plague.

HISTORY

Biblical references to plague date back to 1300 BC. Three plague pandemics have occurred in recorded history. The first, known as Justinian's plague, occurred between 542 and 546 AD and killed 50 to 60% of the population in Africa, Europe and central/south Asia. The second pandemic, which occurred during the fourteenth century and is known as the "Black Death," killed 50 million. The Third pandemic, which began in China in 1894, spread rapidly throughout the world via steam ship, and killed 13 million in India alone.

It was during this pandemic that plague arrived on U.S. shores, in San Francisco's Chinatown in early 1900. The plague epidemic that followed lasted four years. Anti-rat campaigns were established to rid the cities of the reservoir. San Francisco was struck a second time in 1907–08, after the 1906 earthquake. Co-mingling of homeless rats and homeless people was probably responsible for this second epidemic. By this time, plague had arrived in other areas of the U.S., causing outbreaks in several states.[1,2] Plague has been enzootic (endemic in animals) in the U.S. ever since.

Andre Yersin, a French microbiologist, was the first to identify the etiologic agent of plague in 1894, originally naming it *Pasteurella pestis*, after his teacher. But, giving credit where credit is due, the organism was eventually renamed *Yersinia pestis*. *Y. pestis* is a Gram-negative coccobacillus that exhibits bipolar staining ("safety-pin" appearance) on Wright-Giemsa, Wayson's and Gram's stains. The organism is viable for weeks in a cool, moist environment, and for months to years at near freezing temperatures. However, sunlight and heat readily kill it.

CLINICAL DISEASE

Human plague occurs primarily in three clinical forms, depending on the route of exposure. Most infections are acquired by inoculation (typically via flea bite and sometimes by handling or skinning infected animals), which results in a local infection that spreads to regional lymph nodes. These painfully enlarged nodes are known as buboes. Bubonic plague is the most common form seen in the United States, approximately 80–90% of cases. The case-fatality rate of this clinical form is 50–60%, if untreated.

Primary septicemic plague occurs when there is an overwhelming bacteremia, usually after cutaneous exposure. This form is seen in only about 10% of cases in the U.S. Even with treatment, the case-fatality rate is 50%, due to the development of endotoxic shock, disseminated intravascular coagulation and organ failure. Septicemic plague can develop without an obvious bubonic stage.

Primary pneumonic plague is most deadly form of the disease, and luckily, least common. It follows inhalation of aerosolized droplets containing the organism. Unlike the other clinical presentations, pneumonic plague can be spread from person to person, usually through coughing. A bioterrorism event involving an intentional aerosol release would probably result in pneumonic plague. Other clinical presentations include pharyngitis, meningitis, and endophthalmitis.

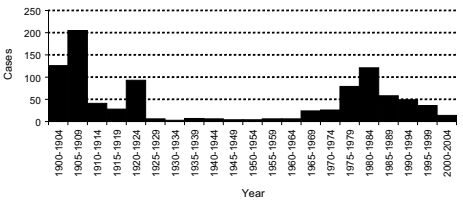
EPIZOOTIOLOGY

Plague is a zoonosis—an animal infection transmissible to man—that occurs worldwide on all continents but Australia. Wild rodents are the reservoir host for plague. The infection is maintained in nature by a rodent-flea-rodent cycle. Reservoir hosts in Oregon include ground squirrels, chipmunks and wood rats.* Humans primarily become infected after being bitten by plague-infected fleas.

PLAGUE IN THE U.S. AND OREGON

Plague is enzootic (a disease common in animals) in the western United States from the Pacific Coast east to Texas and Okalahoma. Since 1974, between 1 and 40 human cases (median, 11) have been reported each year, mostly from New Mexico (53%), Arizona (15%) and Colorado (12%). The number of human plague cases fluctuates as exposure waxes and wanes with changes in rodent and flea population dynamics (figure). These cycles are strongly influenced by climatic conditions, such as temperature and precipitation, and probably explain the increase in cases seen primarily in the Southwestern U.S. during the last 3 decades.[3]

U.S. Plague Cases, 1900–2004



* Sadly, the cuteness of the rodent is no defense.

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In the last 30 years, 52 people have died from plague, the most recent a Colorado resident in late 2004. He was probably exposed while skinning an infected wild rabbit. Twelve human cases (five fatal) have been reported in Oregon since 1934, all but one since 1970. These cases were exposed in Lake (3 cases), Klamath (3), Wallowa, Umatilla, Coos, Jefferson, Deschutes, and Douglas counties (1 each). One case was probably acquired in Wyoming. The most recent case occurred in 1995 in Deschutes County. Most of these infections were acquired by flea bite, but at least two came from handling and skinning rabbits.

DIAGNOSIS

Diagnosis is initially based on clinical suspicion; no rapid diagnostic tests for plague are available. Confirmation is made by identifying the organism in sputum, blood, or lymph node aspirate. *Yersinia pestis* can be cultured within 48 hours. An antibody test for plague is available; however, seroconversion doesn't occur for 1 to 2 weeks after onset of symptoms. Polymerase Chain Reaction (PCR) identification of *Y. pestis* is available at the Oregon State Public Health Laboratory (OSPHL). The OSPHL should be notified prior to submission of clinical samples.[†]

TREATMENT

If plague is suspected, don't wait for lab results, but immediately start appropriate antibiotic therapy. Streptomycin is the antibiotic of choice. Gentamicin, although not FDA approved for the treatment of plague, is a good alternative. Doxycycline and ciprofloxicin are alternatives if the aminoglycocides aren't available, in mass causality settings, or for prophylaxis. The penicillins, cephalosporins and macrolides are ineffective against plague.

PLAGUE AS A BIOLOGICAL WEAPON

The earliest attempt at biological warfare appears to have occurred at the Crimean port city of Caffa in the 14th century. The Tartars fell ill with plague while preparing to attack Genoese sailors in Caffa, so they used their catapults to lob corpses of plague victims over the walls in the hope that the disease would spread throughout the city. The Genoese did become ill with plague. However, the disease was likely spread to the local population by infected rats, not the corpses, since fleas leave the host soon after the corpse cools. The Japanese army successfully infected individuals in Changteh, China with plague by dropping plague-infected fleas over the area during World War II. The United States and the Soviet Union have both developed techniques to aerosolize the organism.[2]

REPORT IT

If plague is suspected, notify your local health department *immediately*, day or night. For more information, call Oregon Health Services at 503/731-4024.

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[†] To protect laboratorians, of course.

2004 CD SUMMARY ISSUE INDEX