Anthrax as a Biological Weapon: An Old Disease That Poses a New Threat

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**HISTORICAL PERSPECTIVE**

*Bacillus anthracis* is a soil bacterium that has been present on earth since ancient times. Anthrax spores can persist in the soil for years, particularly in areas with high levels of soil nitrogen, a pH greater than 6.0, and ambient temperatures greater than 15°C (59°F). Heavy rains after prolonged drought also promote high levels of soil contamination. These factors are thought to help maintain the organism in sufficient quantities to promote infection.

Once an obscure disease, anthrax has recently received widespread publicity as a consequence of its use as a biological weapon. Understanding the history, unique characteristics, and pathogenesis of this disease provides insight into threats posed by the organism that causes it. Further, understanding concepts related to the pathogenesis, prevention, diagnosis, and treatment of anthrax can equip critical care nurses with information needed to manage patients with anthrax if the patients require intensive care and to educate the public about this disease.
almost completely eradicated by widespread vaccination of animals. However, outbreaks still occur in Africa and Asia, where vaccination remains spotty. Animal cases also occur occasionally in the United States, where an “anthrax belt” exists across parts of the Midwest.3

Anthrax is a zoonotic disease (acquired by humans from animals). The earliest accounts of human disease thought to be caused by anthrax date from the Fifth Egyptian Plague, 3500 years ago.6 During the Middle Ages, anthrax, then referred to as “The Black Bane,” killed large numbers of humans and animals in Europe.6

In the late 19th century, Robert Koch, a German physician, discovered the source of the disease and linked the spore stage of anthrax with its ability to persist in the environment.7 Using blood samples taken from diseased animals, Koch grew B. anthracis in culture media, showed its ability to form spores, and reproduced the disease by injecting the spores into animals.3 Finally, he isolated and identified the anthrax pathogen from the animals he had infected. Koch’s “experimental requirements,” determined via research on B. anthracis, provided the framework for studying all infectious diseases. Today, we refer to his rigorous technique as Koch’s postulates.8 Unfortunately, bioterrorists use Koch’s postulates to spread anthrax to their victims.

Soon after Koch did his research, it was established that inhaled anthrax spores were the cause of “woolsorters’ disease” (now referred to as inhalational anthrax). This discovery led to the introduction of disinfection procedures and a decrease in the prevalence of anthrax in these workers.9 In 1881, Louis Pasteur prepared and tested the first vaccine to combat anthrax. The success of this vaccine is credited with stimulating the development of immunology as a science.9

By the 20th century, the worldwide prevalence of anthrax had decreased substantially. Nevertheless, several significant outbreaks of anthrax were caused by skin (cutaneous) and aerosolized (inhalational) exposure. A human epidemic of cutaneous anthrax in Zimbabwe late in 1979 affected thousands and resulted in numerous deaths. In 1987, the slaughter of a single infected cow led to 25 cases of cutaneous anthrax in Paraguay.2 In the early 1900s, 89 persons were infected in an outbreak of inhalational anthrax in southwestern Russia. Most of those infected worked with wool in their homes. However, the most chilling human epidemic of inhalational anthrax took place in 1979 in Sverdlovsk (Yekaterinburg today) in the former Soviet Union. Initially ascribed to unsanitary handling of butchered meat, the epidemic was later proved to be the result of an accident at a military microbiology facility. Nearly 100 persons living in a 4-km-wide area downwind from the release point acquired the disease. The disaster resulted in at least 66 deaths. More significantly, it indicated the capability of B. anthracis to be released into the air and bring about multiple casualties.10-12

BIOTERRORISM

Recent reports of dissemination of B. anthracis via the mail are disconcerting and shocking. However, the use of fomites as potential biological weapons is not new (Figure 1). Invading Tartars, attacking the city of Kaffa (now Feodossia, Ukraine) in 1346, experienced an epidemic of plague. In an effort to capitalize on their misfortune, they catapulted bodies of disease-infested corpses into the city, hoping to initiate the plague there. Four hundred years later, the deliberate spread of smallpox was used by the British during the French and Indian War. In this instance, blankets used by smallpox patients were distributed to unsuspecting Native Americans sympathetic to the French.13

Bioterrorism continued into the 20th century with the first recorded use of anthrax for this purpose. During World War I, Germany sought to spread the bacteria to the allied forces via infected livestock and contaminated feed. The Soviet Union, United Kingdom, United States, and Japan all instituted research on biological weapons before and during World War II. The United States and other nations finally denounced biological warfare and halted research on bio weapons 25 years later. In 1972, 103 nations, including the United States, signed a treaty designed to eliminate use of such weapons.13

Nevertheless, many individuals, terrorist groups, and nations continue work to develop B. anthracis as an agent of mass destruction.14-15 The true extent of threat is difficult to evaluate because production of a “weapons-grade” anthrax agent requires advanced biotechnical capabilities.3 Weapons-grade refers to the ability to produce spores that are sufficiently small (<5 µm) to enter the lower part of the airways. In addition, the spores must be treated with a combination of chemicals that eliminate static electricity to maximize dispersion. Further,
8000 to 50 000 spores must be inhaled to induce illness. These requirements make it difficult to cause widespread disease. As an example, members of the Aum Shinrikyo religious cult attempted to infect large numbers of persons in Japan with *B. anthracis* on multiple occasions, but were never successful.

**ANTHRAX IN THE UNITED STATES**

Between 1955 and 1994, a total of 235 cases of anthrax were reported in the United States. All are thought to have resulted from exposure to animals that had the disease. Most cases (95.3%) were cutaneous; the rest (4.7%) were inhalational. Only 20 persons (8.5%) died as a result of the illness.

Before October 2001, only a single confirmed case of cutaneous anthrax had occurred since the early 1990s. The case involved a North Dakota man who contracted the disease in August 2000 after assisting in the disposal of cattle carcasses infected with *B. anthracis*. One case of gastrointestinal illness was reported, but not confirmed, in 2000 in northern Minnesota. Before October 2001, no cases of inhalational anthrax had been reported in the United States since 1976.

Between October 2001 and early January 2002, there were 18 confirmed and 5 suspected cases of anthrax in the United States. Of these cases, 11 were inhalational, contributing to 5 deaths. Direct exposure to anthrax spores sent through the mail was confirmed or likely in all but 2 cases. More recently, in March 2002, a worker in a laboratory processing environmental samples for *B. anthracis* had cutaneous anthrax diagnosed. Exposure most likely occurred when the worker handled vials containing bacterial isolates while not wearing gloves. The Centers for Disease Control and Prevention (CDC) defined this case as suspected rather than confirmed because, contrary to CDC guidelines, the culture specimen was analyzed at the same laboratory where the specimen was obtained.

Figure 1  Anthrax and biological weaponry in the past 3500 years.
Biologists have used sophisticated techniques to decipher the DNA of the anthrax organisms used in the mail attacks. These sophisticated techniques have enabled researchers to determine that the strain is identical to the Ames strain developed by the US Army in 1982 and later shared with other research laboratories. Investigation continues in an attempt to determine the point of origin more specifically.²⁴,²⁵

PATHOGENESIS

*B. anthracis* is an aerobic gram-positive, spore-forming, nonmotile bacillus. It has a “jointed bamboo rod” appearance that makes identification by an experienced microbiologist relatively easy (Figure 2). Anthrax spores require an environment rich in amino acids, nucleosides, and glucose to develop into bacilli. Therefore, animal and human tissue provide an ideal setting for growth.²⁶

Spores are the dormant stage of the microorganism and are an effective means of protection because they are resistant to adverse environmental conditions.² They can survive inside macrophages, germinate into bacteria, and migrate to the lymph nodes. Unfortunately, an antiphagocytic capsule surrounds the cell wall of the bacterium and prevents efficient phagocytosis by macrophages.²⁷ As such, the bacteria freely enter the lymphatic system, multiply, and move to other areas of the body via the bloodstream.

In addition to its antiphagocytic capabilities, other characteristics of *B. anthracis* contribute to its virulence (Figure 3). *B. anthracis* produces 3 proteins (exotoxin components): edema factor (EF), lethal factor (LF), and protective antigen (PA). These 3 components combine to form 2 virulent toxins.

Edema toxin (EF + PA) is associated with increased levels of cyclic adenosine monophosphate, which facilitates detrimental changes in cellular water balance and produces massive edema. This toxin also has an inhibitory effect on neutrophils, which may be the reason patients with anthrax have minimal to no evidence of purulent drainage and secretions.¹,²⁸,²⁹ Lethal toxin (LF + PA) stimulates the production of tumor necrosis factor α and interleukin-1β, which promote widespread hemorrhage and cellular destruction. In addition, the lethal toxin facilitates lysis of macrophages, further impeding activity of the immune system.¹,²⁹ PA, a component of both toxins, provides the mechanism necessary for EF and LF to enter the cytosol of the cell (the matrix surrounding intracellular components) and also contributes to inhibition of neutrophil function.²⁹ Combined, these toxins can rapidly lead to life-threatening manifestations, including respiratory distress, septic shock, meningitis, and ultimately death.³

CLINICAL FEATURES AND DIAGNOSIS OF ANTHRAX

*B. anthracis* can enter the body through the lungs, skin, or gastrointestinal tract. Cutaneous anthrax is the most common type of infec-

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Figure 2 Gram stains of *Bacillus anthracis* show (A) characteristic “jointed bamboo appearance” and (B) release of spores.

Courtesy of Centers for Disease Control and Prevention.
Spores enter the body and reach sites in the  
1. subcutaneous layers of the skin (after penetrating the skin surface)  
2. gastrointestinal mucosa (after being ingested with contaminated meat) or  
3. alveolar spaces (after being inhaled)

Spores are phagocytosed  
They germinate into bacilli inside the macrophages  
Bacilli migrate to the lymph nodes and move throughout the body

Bacilli produce protein exotoxin components  
Edema factor (EF) / lethal factor (LF) / protective antigen (PA)  
These components combine to form 2 virulent toxins

Edema toxin  
(LF + PA)  
Inhibition of phagocytosis by neutrophils  
Stimulation of macrophage lysis

Lethal toxin  
(EF + PA)  
Increased levels of cyclic adenosine monophosphate  
Release of tumor necrosis factor α and interleukin-1β

Altered water intracellular balance  
Potential reason for lack of purulent drainage and secretions

Massive edema  
Potential reason for lack of purulent drainage and secretions

Edema factor (EF)

Figure 3 The pathogenesis of anthrax.

The signs and symptoms of inhalational anthrax follow a characteristic 2-stage pattern: a prodromal phase followed by fulminant disease. Initially, patients typically experience a nonspecific syndrome (Table 1). Typically, these vague signs and symptoms develop within 1 to 7 days, but the incubation period may be as long as 60 days. In addition, the white blood cell count may increase slightly, with an increase in bands.

At this time, the signs and symptoms of inhalational anthrax are impossible to distinguish from flulike illness. However, adults with viral illnesses commonly have a sore throat and rhinorrhea, symptoms rarely observed during the recent outbreak of anthrax. In the setting of risk for exposure, a chest radiograph or a computed tomography scan of the chest should be obtained. Radiological findings consistent with inhalational anthrax include bilateral pleural effusions, mediastinal widening, and mediastinal lymphadenopathy.

After 1 to 3 days, prior reports indicated that some patients experienced a brief period of apparent recovery before disease progression. However, no recovery period was observed in the recently reported series. All cases progressed directly to the fulminant second stage of illness.

The second stage begins when the regional lymph nodes are overwhelmed and anthrax toxin

<table>
<thead>
<tr>
<th>Most frequent</th>
<th>Common</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lethargy/fatigue/malaise</td>
<td>Minimal or nonproductive cough</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>Decreased or abnormal breath sounds</td>
</tr>
<tr>
<td>Fever/chills</td>
<td>Myalgias</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>Chest discomfort or pleuritic pain</td>
</tr>
<tr>
<td>Nausea/vomiting</td>
<td>Diaphoresis</td>
</tr>
<tr>
<td></td>
<td>Confusion</td>
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<td></td>
<td>Headache</td>
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finds its way into the systemic circulation. Clinical features during the fulminant phase of the infection include fever, profound diaphoresis, respiratory distress, hemorrhagic pleural effusions and mediastinitis, septic shock, and coma. Meningeal involvement occurs in about 50% of the cases. Death may occur as quickly as 1 to 2 days after the onset of the fulminant phase. Typically, no pneumonia occurs unless the patient has preexisting pulmonary disease.

Cutaneous Anthrax

The cutaneous form of anthrax occurs when anthrax spores enter the body through a cut or abrasion of the skin. Commonly, lesions occur on the upper extremities or the face. The first evidence of infection is a nondescript papule that, during the next 24 to 48 hours, becomes a vesicle 1 to 2 cm in diameter, with edema around the site. At this point, the organism is easily isolated and is visible on Gram stain. The lesion, which is usually pruritic but not painful, generally ruptures after 1 week, leaving an ulcer that progresses to the characteristic black eschar that gave anthrax its name. 

Consequences of gastrointestinal infection include perforation of the gastrointestinal tract and systemic infection. Recognition of gastrointestinal anthrax is difficult because the disease may mimic other intestinal disorders and has no easily observed lesions.

Gastrointestinal Anthrax

The gastrointestinal form of anthrax occurs 2 to 5 days after the ingestion of undercooked infected meat. Once ingested, B. anthracis penetrates a break in the lining of the gastrointestinal tract and invades the lymph nodes. Manifestations associated with gastrointestinal anthrax can also develop in response to either oropharyngeal or intestinal infections. Oropharyngeal anthrax can lead to the development of sore throat, hoarseness, neck swelling, airway obstruction, and bleeding from the mouth. In more severe cases, edema may extend to the chest wall and the axillae. Signs and symptoms that occur in response to anthrax in the lower part of the gastrointestinal tract include hematemesis, hematochezia, severe abdominal pain, ascites, watery diarrhea, and bacteremia.

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TREATMENT

In a report of the first 10 cases of inhalational anthrax in 2000 and 2001, 8 patients were in the prodromal phase of their illness when they first sought medical attention. Six patients received antibiotics active against B. anthracis that day and survived. Four patients did not begin anthrax-specific antibiotic treatment until they had fulminant disease, and all died. Therefore, effective treatment of any suspected anthrax exposure requires swift recognition of potential exposure and institution of aggressive antibiotic therapy. If exposure is suspected, blood sample should be obtained for culture before antibiotic therapy is started, because cultures may show no growth within 24 hours.

Inhalational Anthrax

In vitro, B. anthracis is sensitive to multiple antimicrobial agents (Table 2). Because of the high mortality associated with anthrax, the CDC recommends polypharmaceutical treatment with ciprofloxacin and 1 or 2 other antimicrobial agents that show in vitro activity against B. anthracis (Table 3). One regimen used during the recent bioterrorism attack included intravenous ciprofloxacin and rifampin, plus either clindamycin or vancomycin. Ciprofloxacin is considered the drug of choice for victims of terrorism, because most likely, no strains of B. anthracis are resistant to it, it has
convenient twice-a-day dosing, and it interferes with an enzyme necessary for the synthesis of DNA.\textsuperscript{2,30} Rifampin provides additional coverage against gram-positive organisms and primarily has an intracellular mechanism of action. However, rifampin stimulates the metabolism of numerous medications, including oral anticoagulants, corticosteroids, digitoxin, oral contraceptives, and \( \beta \)-blockers.\textsuperscript{38} Clindamycin prevents expression of toxin in streptococcal disease and may have a similar activity against anthrax toxins.\textsuperscript{30,33} Penicillin had been the drug of choice for treating anthrax.\textsuperscript{1} However, the CDC did not recommend its use as monotherapy for cases reported in 2001, because evidence suggests that the anthrax organism may become resistant to this antibiotic, owing to an inducible \( \beta \)-lactamase.\textsuperscript{30}

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Centers for Disease Control and Prevention recommendations for treatment of adults with inhalational and cutaneous anthrax</th>
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<tbody>
<tr>
<td><strong>Inhalational anthrax</strong></td>
<td><strong>Initial therapy</strong></td>
</tr>
<tr>
<td></td>
<td>Ciprofloxacin 400 mg intravenously every 12 hours or Doxycycline 100 mg every 12 hours (as primary agents) Plus 1 or 2 other secondary agents with in vitro activity</td>
</tr>
<tr>
<td></td>
<td>Favored regimen used in recent cases</td>
</tr>
<tr>
<td></td>
<td>Ciprofloxacin plus rifampin plus clindamycin or vancomycin</td>
</tr>
<tr>
<td></td>
<td>When clinically appropriate Switch to ciprofloxacin 500 mg by mouth twice a day or doxycycline 100 mg by mouth twice a day</td>
</tr>
<tr>
<td></td>
<td>Duration</td>
</tr>
<tr>
<td></td>
<td>Continue therapy for 60 days after aerosol exposure because of persistence of spores</td>
</tr>
<tr>
<td><strong>Cutaneous anthrax</strong></td>
<td><strong>Initial therapy</strong></td>
</tr>
<tr>
<td></td>
<td>Ciprofloxacin 500 mg by mouth twice a day or doxycycline 100 mg by mouth twice a day</td>
</tr>
<tr>
<td></td>
<td>When clinically appropriate (after improvement and established susceptibility) May switch to amoxicillin 500 mg by mouth 3 times a day</td>
</tr>
<tr>
<td></td>
<td>Duration</td>
</tr>
<tr>
<td></td>
<td>Continue therapy for 60 days because of potential for simultaneous aerosol exposure</td>
</tr>
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</table>

If a patient has considerable improvement with intravenous therapy, the oral route of treatment may be used. Antibiotic treatment does not differ if the patient is pregnant or immunocompromised. Recommended dosage adjustments for children are based on weight and/or age. Finally, treatment recommendations for inhalational anthrax also apply to the gastrointestinal form.\textsuperscript{30,37}

Current recommendations for the treatment of anthrax are based on limited evidence, because no controlled trials have been done in humans and limited work has been done with primates. Although some have advocated other regimens in the past, the success of the CDC’s recommended protocol supports the practice of using ciprofloxacin as the primary agent to combat anthrax.\textsuperscript{1} Although other fluoroquinolones are probably effective, the data to support changing the CDC recommendations to include use of these agents are limited.\textsuperscript{30}

Little information is available about caring for patients with inhalational anthrax in the intensive care unit, and no specific guidelines for management have been published. However, monitoring and supportive care are based on the patient’s signs and symptoms, and detecting changes in health status is critical. Therefore, patients admitted to the hospital with suspected inhalational anthrax in the prodromal phase may be admitted to critical care areas for continuous observation and management.

Swift initiation of antibiotic therapy after blood samples are obtained for culture is of paramount importance. In addition, corticosteroids may be helpful if extensive edema and meningitis occur.\textsuperscript{29} The potential need for invasive hemodynamic monitoring, infusion of multiple medications, and administration of fluids may dictate the placement of arterial catheters and large-bore central venous and/or pulmonary artery catheters.

Vigilant monitoring of pulmonary status is a necessity, because severe respiratory distress develops in all patients with disease progressing to the fulminant phase.\textsuperscript{34} The presence of secretions does not appear to be a significant problem, on the basis of the recent cases. However, recurrent pleural effusions requiring thoracentesis and chest tube placement have been common.\textsuperscript{22,34} Furthermore, marked changes in the lungs and mediastinum due to worsening of the disease may necessitate frequent arterial blood gas monitoring and ventilator adjustments. Patients may require increasingly
higher fractions of inspired oxygen levels via face mask, because of hypoxemia. Patients may later require emergent intubation and mechanical ventilation. Respiratory alkalosis may be present initially, but patients with disease progressing into the fulminant stage may have both respiratory and metabolic acidosis develop. Patients may later require emergent intubation and mechanical ventilation. Respiratory alkalosis may be present initially, but patients with disease progressing into the fulminant stage may have both respiratory and metabolic acidosis develop. High levels of ventilatory support are often needed because of decreased lung compliance.

Hemodynamic status changes quickly as anthrax toxins are released and enter the systemic circulation. If release of toxin occurs, vasopressor therapy is needed to support a failing circulatory system. Antitoxin agents have been investigated in vitro to combat the effects of the circulating toxins. These include angiotensin-converting enzyme inhibitors, calcium channel blockers, and tumor necrosis factor α inhibitors. However, no available data substantiate a benefit from use of these agents in the recent anthrax victims.

Nursing management of patients with anthrax includes coordinating care that involves multiple physicians and other disciplines. Nursing staff are also responsible for instructing patients’ families about the nature of the critical illness and the patients’ care. Finally, nurses may need to provide grief counseling to help a patient’s loved ones deal with the patient’s rapid deterioration and death.

No data support human-to-human transmission of inhalational anthrax. Therefore, it is not contagious. No precautions are indicated in the intensive care unit beyond routine precautions for exposure to blood and body fluids. Prophylaxis is only necessary for contacts at risk of exposure to aerosolized spores. After exposure to aerosolized spores, patients should be decontaminated in the field with soap and water. Potentially contaminated clothing should be removed, carefully handled to avoid dispersion of any spores, and double-bagged in plastic. Potentially contaminated environmental surfaces should be decontaminated with a sporicidal/germicidal agent or a 10% hypochlorite solution (1 part bleach to 9 parts water).

Cutaneous Anthrax
Mild cases of cutaneous anthrax are managed with oral antibiotics. Intravenous therapy should be substituted if any evidence of systemic involvement is present. Cutaneous lesions may become culture-negative within
24 hours, but progression to eschar formation still occurs. Before 2001, guidelines recommended that antibiotic administration be continued for 7 to 10 days. Because of the risk of cutaneous and inhalational exposure in the recent cases, treatment guidelines were changed to recommend 60 days of antibiotic therapy. Direct exposure to draining skin lesions may cause secondary cutaneous infection. Therefore, standard precautions for exposure to blood and body fluids should be used at all times with anyone suspected to have cutaneous anthrax.

Prophylaxis

All persons with suspected exposure to Bacillus anthracis should receive antibiotic prophylaxis for 60 days and possibly longer, for those with heavy exposure. The recommended therapy is ciprofloxacin 500 mg by mouth twice a day or doxycycline 100 mg by mouth twice a day with adjustments for children based on weight and/or age. If susceptibility of the infecting organism to penicillin is confirmed, guidelines recommend that therapy be changed to amoxicillin 500 mg every 8 hours.

MORTALITY

Inhalational Anthrax

Fifty-five percent (6/11) of the recent patients survived inhalational anthrax. Thus, the previously reported mortality of nearly 100% may no longer be accurate. Rapid institution of appropriate, combination antibiotic therapy and aggressive supportive care are the keys to successful management, because they substantially lowered mortality for recent patients. All 6 of this past year’s survivors had anthrax diagnosed quickly after exposure, and treatment was initiated in the prodromal phase of the disease. Sadly, none of those who required intubation and/or whose hemodynamic status was unstable survived. It is important to note that, based on simulated theoretical models and cases in the Soviet Union in 1979, recent experience is not reflective of the ability of anthrax spores to cause disease if there is widespread airborne dispersion.

Cutaneous Anthrax

If untreated, 20% to 30% of the cases of cutaneous anthrax will result in systemic disease and death. Mortality with treatment is less than 1%. Therefore, prompt diagnosis and treatment are particularly important.
Gastrointestinal Anthrax

Less is known about the gastrointestinal form of anthrax, which had never been documented or potentially suspected in the United States until August 2000. The mortality rate among reported cases elsewhere is greater than 25% to 60%. Those who survive typically have signs and symptoms that subside after 10 to 14 days.

ADVERSE EFFECTS OF PROPHYLAXIS

Prophylactic treatment was given to more than 32,000 persons potentially exposed to anthrax in 2001. Postal employees who received antibiotic prophylaxis were surveyed to determine the prevalence of adverse effects, and nearly 4000 provided information. Approximately 3500 persons received ciprofloxacin. Of these, 19% reported having nausea, vomiting, diarrhea, or abdominal pain. In addition, 14% of these persons reported fainting, dizziness, or light-headedness, and 6% had rashes, hives, or pruritis. No anaphylactic reactions were reported. Despite these reports, relatively few (8%) discontinued the prescribed prophylaxis.

Treatments for hysteria are an integral component of prophylaxis during a bioterrorist threat. Media reports of self-dosing with ciprofloxacin and antibiotic hoarding are examples of inappropriate responses to the anthrax scare. Inappropriate use of ciprofloxacin may lead to adverse effects and to increased resistance of other pathogens. Furthermore, mechanisms are in place for distributing medication. In the event of large-scale requirements for antibiotics, the National Pharmaceutical Stockpile will be able to send needed supplies anywhere in the United States within 12 hours.

IMMUNIZATION

Because anthrax has a short incubation period, nonspecific signs and symptoms, and a potentially high mortality, immunization provides the safest and most effective protection against it. The first vaccine given to humans to prevent anthrax was developed in the Soviet Union in 1943 by using live spores. Later, British and American researchers developed cell-free versions. The only anthrax vaccine currently available was licensed to Bioport Corporation (Lansing, Mich) in 1970. Because of production problems, the company had not produced any doses since 1998, but it recently resumed production.

The only persons who have received the vaccine are U.S. military personnel and other selected high-risk groups. Known officially as “Anthrax Vaccine Adsorbed,” the vaccine is produced from a cell-free filtrate of a nonencapsulated, avirulent strain of *B. anthracis* that induces immunity by expression of protective antibodies. In rhesus monkeys, the vaccine was 100% effective at 8 and 38 weeks after immunization and somewhat less effective (88%) after nearly 2 years. Immunization has required 6 subcutaneous injections (initial dose and then injections at 2 and 4 weeks and 6, 12, and 18 months later) followed by yearly boosters to maintain immunity. Local reactions at the injection site occur in 30% (men) to 60% (women) of persons vaccinated. Systemic reactions include muscle and joint aches, headache, fever, loss of appetite, and malaise in 5% to 35% of those immunized.

An alternative approach to immunization is being tested in persons currently receiving antibiotic prophylaxis. They were given 3 options: to stop antibiotic treatment after 60 days, to receive an additional 40 days of treatment with monitoring, or to receive an additional 40 days of treatment with monitoring plus 3 doses of vaccine spread over 4 weeks. The last option is being tested as a means to provide additional protection by quickly stimulating an immune response.

FUTURE DIRECTIONS

Researchers are focusing on a mutated form of the protective anthrax antigen that acts in a negative (inhibitory) fashion and can disarm (dominate) the natural antigen. The goal is to develop a genetically engineered dominant-negative protective antigen. Injected during an infection, the antigen could act as an adjunct to antibiotic therapy by binding to receptors, deactivating the release of the edema toxin and lethal toxin, and prohibiting the toxins from entering and destroying target cells. Potentially, the antigen could also be used to provide protection from a future exposure to anthrax organisms.

Other researchers are studying how lethal toxin binds to cellular proteins. Using x-ray crystallography, they observed that a groove on lethal toxin precisely matches a notch on the target molecule. Potentially, this information could be used to design an antitoxin that could block the action of lethal toxin against macrophages and prevent release of multiple inflammatory agents. In addition, researchers are propos-
ing the use of high-titer immune globulin and antiera from vaccines as possible treatment agents. Until these and other potential treatments for anthrax are available, antibiotics and vaccination are the only pharmaceutical weapons available.

INSTITUTIONAL PREPAREDNESS

Critical care nurses should become involved in institution-wide planning for isolated cases of anthrax and sudden influx of potentially contaminated or infectious casualties. Objectives of institutional plans should include the following:

- ensuring rapid response to community needs,
- maximizing use of available facilities and personnel,
- swiftly informing local and state health departments and law-enforcement agencies of suspected bioterrorism casualties, and
- providing hospital staff with the necessary education and materials to manage casualties safely and effectively.

In addition, communities should begin to develop mechanisms to detect patterns of signs and symptoms that suggest exposure to anthrax and should share such information among hospital emergency departments. One example is the Real-time Outbreak and Disease Surveillance system developed at the University of Pittsburgh to detect patterns of signs and symptoms that suggest outbreaks of disease or other epidemiological events. The system was used in Salt Lake City to link the multiple facilities in the area during the recent Olympic Games.

Finally, nurses must become more adept at acquiring new important information on an ongoing basis during times of rapidly developing crises. A number of excellent Web sites, including the sites of the CDC (http://www.cdc.gov), the United States Army Medical Research Institute of Infectious Diseases (http://www.usamriid.army.mil), and the Association for Professionals in Infection Control and Epidemiology (http://www.apic.org), are available for nurses to learn more about anthrax and keep abreast of new developments related to this health threat as the developments occur.

CONCLUSION

B. anthracis and the disease it causes have an extensive history and infamous reputation. Normally occurring illness due to B. anthracis is under control throughout much of the world, except in developing countries, where comprehensive programs for animal vaccination are lacking. However, the microorganism remains a biological agent with the potential to be used for terrorism. Recent events have indicated the lethal potential of anthrax and its ability to elicit widespread anxiety. As events continue to unfold, it is important for critical care nurses to increase their understanding of concepts related to the pathogenesis, prevention, diagnosis, and treatment of anthrax and to acquire the information necessary to manage patients with anthrax and educate the public about this disease. The recent threat of bioterrorism did not involve a large number of persons, nor did it have a significant effect on critical care. However, the potential for future attacks and greater numbers of patients stricken with anthrax and requiring the care of knowledgeable critical care nurses is very real.

References


CE Test Form

Anthrax as a Biological Weapon: An Old Disease That Poses a New Threat

Objectives:
1. Identify 3 mechanisms by which anthrax can be contracted
2. Describe 3 objectives of institutional plans to deal with anthrax exposure or infection
3. Describe the pathogenesis of anthrax infection

Mark your answers clearly in the appropriate box. There is only 1 correct answer. You may photocopy this form.

1. a   2. a   3. a   4. a   5. a   6. a   7. a   8. a   9. a   10. a   11. a
   b   b   b   b   b   b   b   b   b   b
   c   c   c   c   c   c   c   c   c   c
   d   d   d   d   d   d   d   d   d   d

Program evaluation

Objective 1 was met
Objective 2 was met
Objective 3 was met
The content was appropriate
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This method of CE is effective for this content

The level of difficulty of this test was:
   easy    medium    difficult

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CE Test Questions
Anthrax as a Biological Weapon: An Old Disease That Poses a New Threat

1. Anthrax spores thrive in soil with high levels of which type of nutrient?
   a. Nitrogen
   b. Oxygen
   c. Phosphorus
   d. Potassium

2. In which type of animal does anthrax most commonly occur?
   a. Carnivore
   b. Omnivore
   c. Insectivore
   d. Herbivore

3. Where do anthrax outbreaks tend to occur?
   a. Europe and Australia
   b. Africa and Asia
   c. Europe and Africa
   d. Australia and Asia

4. Who prepared the first vaccine to fight anthrax?
   a. Salk
   b. Pasteur
   c. Sabin
   d. Koch

5. What does the anthrax bacillus resemble under a microscope?
   a. Chains of rods
   b. Corkscrews
   c. A jointed-bamboo rod
   d. A “U-shaped” hook

6. Which is the most lethal type of anthrax infection?
   a. Inhalation
   b. Cutaneous
   c. Gastrointestinal
   d. Neurological

7. Which is the most common type of anthrax infection?
   a. Inhalation
   b. Cutaneous
   c. Gastrointestinal
   d. Neurological

8. Which of the following is among the most frequent signs and symptoms of inhalation anthrax?
   a. Myalgias
   b. Diaphoresis
   c. Tachycardia
   d. Nonproductive cough

9. What is the drug of choice for victims of terrorism?
   a. Clindamycin
   b. Amoxicillin
   c. Rifampin
   d. Ciprofloxacin

10. Individuals with suspected exposure to B anthracis should receive prophylactic antibiotics for at least how many days?
    a. 10 days
    b. 30 days
    c. 60 days
    d. 90 days

11. What is the mortality rate for patients with untreated cutaneous anthrax?
    a. 20% to 30%
    b. 25% to 60%
    c. 75% to 90%
    d. 100%
Anthrax as a Biological Weapon: An Old Disease That Poses a New Threat
Frederick J. Tasota, Richard A. Henker and Leslie A. Hoffman

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