

# Chapter 11

## ANTHRAX INFECTION

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### INTRODUCTION

The bacteria *Bacillus anthracis* causes disease, zoonotic infectious disease, transmitted to humans from infected animals or contaminated animal products. The bacteria is non-motile rod, an aerobic or facultatively-anaerobic, gram-positive, encapsulated (1,2).

It produces toxins and a prominent poly-D-glutamic acid capsule which are important for clinical virulence. It is non-hemolytic on blood agar. In culture, the centrally located spores form spores that are highly resistant to external environments, dryness, cold, ultraviolet rays, high and low pH levels and chemical disinfectants. They are inactivated in 30 minutes at 140°C and in 2 minutes at 180°C.

It reproduces by forming large colonies, with irregularly tapered edges at 37°C. When these colonies are examined under the microscope, a wavy filamentous structure extending from the center to the periphery is seen (3).

### EPIDEMIOLOGY

Anthrax appears worldwide, and the World Health Organization (WHO) estimates the annual incidence of between 2000 and 20,000 cases (1). Anthrax is endemic in some Latin American, African and Asian countries. The incidence of anthrax has decreased worldwide, but has not yet been completely eradicated. Anthrax is endemic in Turkey (4,5,8). Infection according to the sources of transmission; it is classified as industrial, agricultural, laboratory, intravenous drug addiction.

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The most common contamination is from agricultural origin and develops as a result of direct contact with infected animals. Skin anthrax by direct contact as a result of slaughtering, skinning, chopping the meat of dead diseased animals or gastrointestinal system anthrax develops by eating infected meat. Anthrax cases seen in our country are generally of agricultural origin. It is formed during processing of industrially sourced spores and animal products such as contaminated goat hair, wool, leather, hides and bones.

## **PATHOGENESIS**

*Bacillus anthracis* has an important toxin in its protein structure, which is responsible for its virulence. The toxin has three components: Protective antigen (PA), edema factor (PF) and lethal factor (LF). The complex of PA and EF, which is called edema toxin, activates calmodulin-dependent adenylate cyclase in the cell and causes edema to occur by increasing cAMP level in the cytoplasm. Protective antigen and LF complex are called lethal toxin and are responsible for necrosis in tissues(6).

## **CLINICAL MANIFESTATION**

In humans according to the place of entry; The disease occurs in three clinical forms as Cutaneous anthrax, Inhalation anthrax, **ingestion** anthrax and Injection anthrax (10). With lymphohematogenous spread of the agent from any of these settlements, severe and lethal clinical pictures such as sepsis and meningitis may develop.

## **CUTANEOUS ANTHRAX**

The form accounts for 95% of human anthrax (11). Anthrax lesions are located on the skin, usually in exposed areas of the body such as the face, neck, hands, and arms (7). It is formed by the inoculation of *Bacillus anthracis* spores into the skin with minor traumas such as trivial trauma. The time between inoculation of spores into the skin and the appearance of a small pruritic papule lesion on the skin is usually two or three days. The incubation period ranges from one to seven days. Typical eschar develops in seven to ten days. The lesion is usually one to three cm in diameter. After necrosis is complete, a black crust forms. With the decrease of edema around it, the crust begins to separate and falls off within two to three weeks (12)

It leaves scar tissue underneath. In pustular form, it sometimes progresses severely, toxemia and sepsis may develop and result in death. In lesions located in the periorbital region, edema is high and tends to spread. Serious cutaneous disease may be marked by extensive edema that spreads to the face, neck and anterior chest wall. It causes respiratory distress by pressing on the trachea. Painful lymphadenitis develops in the neck. .

Toxic shock: Presence of systemic symptoms together with skin lesion (fever, tachycardia, tachypnea, toxemia, altered consciousness, hypotension) may be seen. Changes in blood biochemistry such as kidney dysfunction, leukopenia may be seen(8,9).

### **INHALATION ANTHRAX**

Inhalational anthrax develops as a result of inhalation of its spores. Lung anthrax is characterized by hemorrhagic mediastinal lymphadenitis. The patient may develop toxemia, confusion and coma, resulting in death(13).

This is supposed to be one of the most potent bioterrorism agents because its spores are highly resistant to natural conditions and may survive for several decades in the environment. Anthrax is usually considered the most likely agent for bioterrorism via an aerosol route (14,15).

### **INGESTION ANTHRAX**

Anthrax lesions occur in the gastrointestinal mucosa after ingestion of foods or beverages contaminated with *Bacillus anthracis* spores (16).

The two most common clinical forms of gastrointestinal anthrax are described; oropharyngeal anthrax and intestinal anthrax. In intestinal anthrax, the lesion is most commonly located in the terminal ileum or cecum region of the intestine. The mortality rate is 40-50%. Patients may die by developing severe toxemia, sepsis, and septic shock (17).

### **INJECTION ANTHRAX**

Recently, another form of anthrax infection has been described in heroin-injecting drug users. (18).

## DIAGNOSTICS

Significant to notify laboratory because its may discard as probable contaminants and cases may occurred among workers (17).

According to clinical suspicion, it is diagnosed by the presence of gram-positive, large, encapsulated bacilli in sputum smears, stool, vomit or peritoneal fluid, or by the production of *Bacillus anthracis* in culture.. The material suitable for direct preparation and culture in skin anthrax is vesicle fluid. It is the material taken after removing eschar with forceps. Serologically, demonstrating the increase in antibody titer against PA and LF by ELISA is helpful in diagnosis. In two serum samples taken two to four weeks apart is meaningful to demonstrate an increased antibody titer.

## DIFFERENTIAL DIAGNOSIS

Differential diagnosis includes carbuncle, erysipelas, cellulitis, necrotizing cellulitis, primary syphilis chancre, orf, tularemia tropical ulcer.

## TREATMENT

*B.anthraxis* is sensitive to extensive range of antibiotics. Penicillin is the antibiotic of choice for treatment. Like penicillin, ciprofloxacin and doxycycline can also be used in treatment. Erythromycin, clindamycin, linezolid,rifampin, macrolides, aminoglycosides, cefazolin and other first generation cephalosporins are antibiotics effective against *B. anthracis* in vitro (17).

For the treatment of cutaneous anthrax, penicilin or amoxicillin treatment for mild uncomplicated cutaneous anthrax is recommended for 7-10 days. Penicillin procaine, doxycycline or ciprofloxacin is recommended for penicillin allergy.

In life-threatening cases of pulmonary anthrax, penicillin G, Ciprofloxacin, doxycycline with clarithromycin or clindamycin; In gastrointestinal anthrax, it can be combined with an aminoglycoside.

In anthrax meningitis, rifampicin or vancomycin is recommended together with crystalline penicillin. It can be used together with parenteral quinolone or rifampicin or vancomycin in cases of anthrax meningitis with penicillin allergy. Antibiotic treatment period of 10-14 days is recommended in cases of visceral anthrax (19) .The recommendations for duratin therapy total 60 days for inhalation form .

Corticosteroids can be given in severe-malignant edema, meningitis. In severe forms, human monoclonal antibodies (Raxibacumab) can be combined with anthrax immune globin.

## PROTECTION AND CONTROL

Since *Bacillus anthracis* spores maintain their viability and infectivity in outdoor environments for a long time, the most effective method of protection in areas where anthrax is endemic in agricultural areas is vaccination of animals and people at risk. The anthrax vaccine used in humans today is an inactivated vaccine prepared from a cell-free and protective antigen. Vaccine should be administered at short intervals and boosted (0, 2, 4 weeks). In order to prevent the spread of the disease, dead animals and people should be buried or cremated appropriately. If autopsy is planned, all material used after the procedure should be sterilized in an autoclave (19). Anthrax infected patients may be hospitalized in anormal hospital room with standard universal precautions( 20).

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