Case Report

First reported case of naturally acquired fatal anthrax from Northeast India

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Abstract

Anthrax is a zoonotic disease and is caused by Bacillus anthracis which is a Gram-positive, nonmotile, spore-forming rod, found in soil. The spores can remain viable for decades. Transmission occurs naturally in humans by direct contact with infected animals or the contaminated animal products. Anthrax is a major health problem in eastern and southern India, with a significant human incidence because the disease is poorly controlled. Here, we discuss such a case of naturally occurring fatal anthrax in North-East India. A 48-year-old man from Assam presented with seizures, hematemesis, and fever. Apart from altered mental status and nonreactive pupils, his cutaneous and systemic examination was unremarkable. Noncontrast computed tomography head showed multiple hemorrhages. Blood and cerebrospinal fluid showed heavy growth of anthrax Bacilli. He was started on specific antibiotics after the reports, but unfortunately, the patient succumbed to infection. Due to high prevalence of anthrax in the endemic regions, a high degree of suspicion is required to clinch the diagnosis. Early initiation of therapy before developing the intracranial hemorrhagic complications might result in a better outcome. Careful history for a possible exposure to animal carcass or a suspected animal death due to anthrax will also help in early diagnosis of the disease and effective therapy.

Keywords: Anthrax, intracranial hemorrhage, meningitis, Northeast India

Introduction

Anthrax is a life-threatening disease. It has a wide variety of clinical manifestations depending on the route of exposure. The spores are infective and also used as a weapon for bio-terrorism. Transmission in humans is due to exposure to infected animals. In this article, we present a case of naturally acquired anthrax. We discuss the pathogenesis and various clinical manifestations. We will also discuss the assessment, laboratory diagnosis and management of anthrax.

Case Report

A 48-year-old man, farmer by occupation, living in a small village near Golaghat, Assam, a state in northeastern part of India, presented to the hospital with multiple episodes of generalized tonic–clonic seizures early morning. There was also history of high-grade fever for 4 days was associated with chills and rigors and a single episode of bloodstained vomitus 3 days back. There was no history of head trauma. He rears animals at his home which included cattle, goats, and pigs. There was a history of one of his cows dying around 3 months back suddenly without any apparent cause. He neither had any history of chronic alcohol intake, smoking, drug abuse, medications, nor any history of diabetes mellitus, hypertension, or pulmonary tuberculosis. On examination, he was unconscious with a Glasgow Coma Scale (GCS) of 4/15 (E1 V1 M2) and both his pupils were around 4 mm in size and nonreacting to light. Neck stiffness was indeterminate. There was frothing from the mouth and a heart rate of 99 beats/min. His blood pressure was 152/100 mmHg. There was no pallor, icterus, or any lymphadenopathy. Cardiovascular system, respiratory system, and abdominal examination

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revealed no abnormalities. Nervous system examination showed normal tone and the deep tendon reflexes in all four limbs. However, the plantar reflex was equivocal on both sides. Cutaneous examination showed no rash or ulcer present anywhere on the body.

Complete hemogram showed hemoglobi*n* = 12.8 g/dL, total leukocyte count = 14,070 cells/mm^3, neutrophils = 89%, lymphocytes = 9%, monocytes = 2%, and platelet count = 150,000 cells/mm^3. Biochemical examination of his blood showed serum creatinine = 0.9 mg/dL, serum sodium (Na+) =134 mmol/L, serum potassium (K+) = 3.3 mmol/L, total bilirubin = 2.4 mg/dL, direct bilirubin 1.1 mg/dL, aspartate aminotransferase = 489 IU/L, alanine aminotransferase = 100 IU/L, alkaline phosphatase = 88 IU/L, serum total protein = 7.7 g/dL, and serum albumin = 4.1 g/Dl. Test for malarial parasite was negative. Viral markers (HIV, HCV, and HBsAg) were all negative. Prothrombin time was 15 s. Blood culture – heavy growth of Bacillus anthracis [Figure 1].

A lumbar puncture was performed and hemorrhagic cerebrospinal fluid (CSF) was obtained which was sent for analysis. CSF routine and microscopy showed a total leukocyte count = 25,960 cells/mm^3, neutrophils = 95%, lymphocytes = 5%, red blood cell count = 1,800,000 cells/mm^3, total protein = 1304.8 mg/dL, sugar = 50 mg/dL, Indian Ink staining = Negative, Gram-stain = Many pus cells, many Gram-positive bacilli with truncated ends resembling box car appearance.

CSF culture showed heavy growth of Bacillus anthracis [Figure 2]. A noncontrast-enhanced computed tomography scan of the brain was also performed, and it showed extensive subarachnoid hemorrhage in the Basal cisterns and bilateral Sylvian fissures. There was also left frontal parietal subdural hemorrhage 6 mm in maximum thickness. Focal small hematoma was also seen in the region of the left Sylvian fissure. Diffuse subdural hemorrhage was seen extending along the tentorium. The mild mass effect was seen on the left with subtle midline shift to the right by 4 mm [Figure 3].

Despite appropriate antibiotic coverage (intravenous ciprofloxacin, intramuscular penicillin, and oral doxycycline) and other life supports, the patient succumbed to the infection.

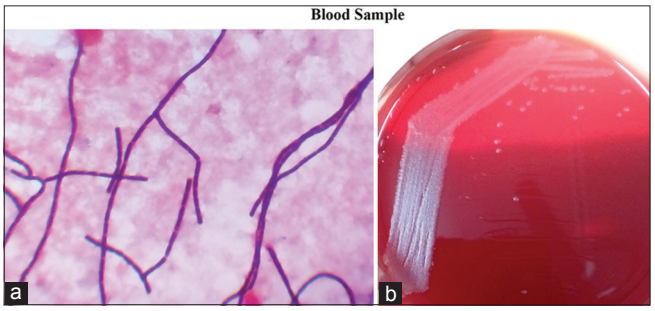

Figure 1: (a) Gram-stain shows Gram-positive bacilli in long chains, giving a bamboo appearance. (b) Blood agar: Colonies are nonhemolytic, opaque, white to gray in color, flat and irregular with a slightly undulated margin

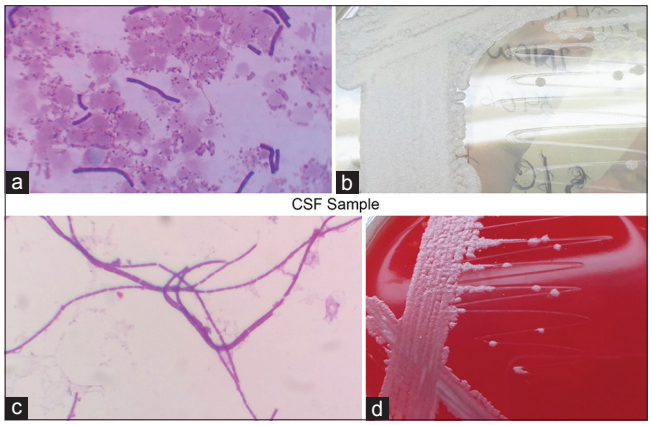

Figure 2: (a) Leishman Stain: Many pus cells and many bacilli with square-ended rods in pairs or short chains. (b) Nutrient agar: Colonies are large, irregular, raised, dull, opaque and greyish-white with "frosted glass" (ground glass) appearance showing curled protrusions "Medusa head appearance". (c) Gram-stain: Shows Gram-positive bacilli in long chains, giving a bamboo appearance. (d) Blood agar: Colonies are nonhemolytic, opaque, white to gray in color flat and irregular, with a slightly undulated margin

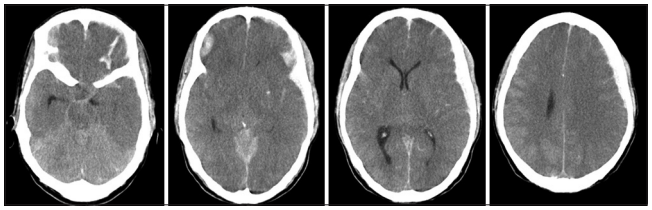

Figure 3: (Computed tomography scan) – Extensive subarachnoid hemorrhage seen in Basel cisterns, bilateral Sylvian fissures. Left frontal parietal subdural hemorrhage 6 mm in maximum thickness. Focal small hematoma seen in the region of left Sylvian fissure. Diffuse subdural hemorrhage seen extending along the tentorium. Mild mass effect seen on the left with subtle midline shift to the right by 4 mm

Discussion

Anthrax is a zoonotic disease which is caused by Bacillus anthracis. It is a Gram-positive, nonmotile, spore-forming rod which is found in soil. It causes disease predominantly in cattle, sheep, and goats. The spores once formed, can remain viable for decades.[1] Reports have suggested that the longest survival for an anthrax spore was 200 ± 50 years' old which was found in bones during archaeological excavations in South Africa and the age of the fossil was estimated using carbon-dating. It also suggested that dryness favors the long survival of the spores.[2]

Transmission occurs naturally in humans by direct contact with infected animals or the contaminated animal products.[3] Anthrax is generally regarded as being noncontagious, however rare instances of human-to-human transmission exists.[4,5] To manifest a clinical disease, generally LD_{50} of 10,000 is considered adequate.

However, in some settings, as few as one to three spores may be adequate.[1] There are four forms of the clinical disease which includes cutaneous, inhalational, gastrointestinal, and meningitis.[3]

The lesion in cutaneous form usually begins as a papule which later evolves into a painless vesicle, eventually forming a coal-black, necrotic eschar. Inhalational form occurs following inhalation of spores which gets deposited in the alveolar spaces. The spores are engulfed by macrophages through phagocytosis and then transported to the regional lymph nodes. The spores germinate in the lymph nodes which lead to active bacterial growth and formation of edema toxin and lethal toxin. The bacteria can spread to other organs through hematogeneous route from here. Gastrointestinal anthrax occurs following ingestion of contaminated meat.[1]

Anthrax is a major health problem in eastern and southern India, with a significant human incidence because the disease is poorly controlled. However, western India is spared due to low soil pH. The incubation period for anthrax after an oral challenge was found to be 3–7 days.[6] To make a clinical diagnosis, a high degree of suspicion should be kept in mind after carefully taking the patient's history and relevant clinical examination. Early symptoms in case of inhalational anthrax are flu-like with mild upper respiratory signs while they resemble food poisoning in case of gastrointestinal anthrax. Meningitis is a serious complication of anthrax which may develop in any form of the disease. It is hemorrhagic,[7] and presents with headache, neck pain, altered sensorium, vomiting, and high-grade fever. The CSF pressure is markedly elevated, and there is rapid disorientation which leads to loss of consciousness, followed by death. There is an extremely poor prognosis with only a handful of survival instances. Sepsis occurs after the spread from the primary lesion which presents as high fever, shock, and toxemia.

The isolation of anthrax bacilli from the body fluids and identification is fairly simple with the colonies showing a Gram-positive, nonmotile, nonhemolytic, aerobic, rod, with central or terminal spores. Various immunological tests to detect capsular antigens and exotoxin components are also available. However, the most reliable indicators are antibody titers to protective antigen and capsular components.[8] Molecular studies using polymerase chain reactions to detect virulence plasmid markers can also be done.

Ciprofloxacin and penicillin are the bactericidal drugs useful for treating anthrax. Linezolid, clindamycin, rifampicin, and doxycycline are protein synthesis inhibitors which can be used to reduce exotoxin production. Intravenous therapy is recommended in case of inhalational, gastrointestinal and meningeal anthrax. However, cutaneous anthrax can be treated with oral drugs which should be taken for a total of 60 days to prevent inhalational anthrax.[9]

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. Since the patient was disoriented, in the form, the patient's son has given his consent for the images and other clinical information to be reported in the journal. The patient's son understands that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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