Advances in Infectious Diseases, 2012, 2, 67-71
http://dx.doi.org/10.4236/aid.2012.23010 Published Online September 2012 (http://www.SciRP.org/journal/aid)

Gastrointestinal Anthrax: A Case and Review of Literature

Ayhan Akbulut[1*], Handan Akbulut[2], Müge Özgüler[1], Nuran İnci[3], Şinasi Yalçın[4]

[1]Department of Infectious Diseases and Clinical Microbiology, Faculty of Medicine, Firat University, Elazıg, Turkey; [2]Department of Immunology, Faculty of Medicine, Firat University, Elazıg, Turkey; [3]Sehit Kamil State Hospital, Gaziantep, Turkey; [4]Department of Ear, Nose and Throat Disorders, Faculty of Medicine, Firat University, Elazıg, Turkey.
Email: *a_akbulut@yahoo.com

Received June 2nd, 2012; revised July 3rd, 2012; accepted August 4th, 2012

ABSTRACT

Anthrax which is caused by *Bacillus anthracis* is typically a disease of herbivores. Spores existing in the skin, meat, hair or mouth and nose of animals are transmitted to humans through contact with a break in the skin, consumption of infected meat or inhalation of spores [1]. Infected uncooked or insufficiently cooked meats cause oropharyngeal and gastrointestinal system (GIS) anthrax. When this infected materials swallowed anthrax spores may cause lesions from the oral cavity to the caecum. The diagnosis of gastrointestinal system (GIS) anthrax is difficult due to insidious clinical progression of the disease and difficulty in the isolation of agent pathogen. Releated symptoms of GIS anthrax are sore throat, neck swelling, diffuculty swallowing, stomach pain, anoreksia, bloody diarrhea, nause, bloody vomiting and fever. Supportive and antibiotic treatments are required. Benzylpenicillin, rifampicin, clindamycin, chloramphenicol, imipenem/cilastatin, or vancomycin can be use for treatment, ciprofloxacin or doxycycline may be added to this treatment for serious cases. To emphasize the necessity of taking precautions, an oropharyngeal and intestinal anthrax case due to consumption of infected and insufficiently cooked meat is presented below.

Keywords: *Bacillus anthracis*; Oropharyngeal Disease; Gastrointestinal System; Anthrax; Consumption of Infected Meat

1. Case

Forty-two year-old female patient, presenting with swelling which initiated on the right side of the neck and spread all around the neck and chest, difficulty in swallowing and breathing, chills and fever symptoms, referred to the emergency department and hospitalized for evaluation.

Detailed history of the patient revealed that she lived in the countryside, dealed with livestock farming and two days before the swelling appeared, she cooked and ate the meat of an animal which was designated as ill by veterinary surgeon.

In the initial examination, general status is regarded as intermediate, axillary temperature 37.8˚C, heart beat 76/min and arterial blood pressure was measured as 110/60 mmHg. Painless oedema was observed all over the neck, being more prominent on the right side and also on the upper 1/3 region of the chest. During palpation, a number of lymph nodes were detected but the dimension of lymphadenopathy (LAP) could not be determined because of oedema. In the pharyngeal examination, a mass resembling an abscess with darker color in the center and intirely covering the posterior wall of the pharynx was observed (**Figure 1(a)**).

*Corresponding author.

The findings of the initial laboratory evaluation of the patient are as follows: in whole blood cell count, white blood cells (WBC) 13,300/mm^3 with differential count of polimorphonuclear WBC (PNL) 86%, lymphocytes 7.4%, monocytes 6.6%; hemoglobin 11.5 gr/dL, hematocrit 34.9%; platelet count 144.000/mm^3; red blood cell (RBC) sedimantation rate (ESR) 13 mm/hr; C-reactive protein (CRP) 24 mg/L. Transaminase values were detected as AST 32 IU/L and ALT 12 IU/L, urea as 67 mg/dL, creatinine as 1.3 mg/dL; the other biochemical and urine analysis values were found to be within normal limits.

Emergency neck computerized tomography showed a lobulated mass compatible with abscess, with approximate dimensions of 3 × 2.5 × 4 cm according to peripheral and internal septal contrast and constricting the trachea. Multipl LAPs with conglomerated apperance and largest with a dimension of 3.2 cm were detected in the right submandibular, sublingual and cervical regions. On the right side of the neck, subdermal soft tissue with oedematous appearance was observed (**Figure 2(a)**). The details of the radiologic findings of this case were described previously [2].

Two blood cultures and culture from the wound were performed. Bacillus anthracis was grown in the pharyngeal culture of the patient. The patient was pre-diagnosed

as having oropharyngeal anthrax and placed on a daily treatment of intravenous 24 million units penisilin G (6 × 4 million units) and 500 mg levofloxacin. Due to dispnea, dexametason 8 mg *i.v*. and cold steam were added to treatment.

On the second day of hospitalization, oedema on the neck and chest started to recover and ecchymosis developed in the same region (**Figure 1(c)**). Airway passage was evaluated on a daily basis. Passage was observed to be relieved in due course.

On the 4th day of treatment, general status of the patient was good and passage improved considerably. Repeat whole blood cell count results were as follows: white blood cells 9200/mm^3; hemoglobin 11.0 gr/dL, hematocrit 32.6%; platelet count 187.300/mm^3; ESR 12 mm/hr.

Observations of day 6 were significant recovery of the abscess and improvement in respiration; the only symptom observed was dysphagia (**Figure 1(b)**). Unfortunately, even though the patient was adviced not to touch the abscess, her husband informed healthcare personnel that she popped up the abscess with her finger. Upon interview of the patient, she admitted that she popped up the abscess with her finger and swallowed the draining fluid.

On the 8th day of treatment, the patient presented with bloody defecation 4 times in a day; following findings were observed at this stage: axillary temperature 37.0˚C, heart rate 105 /min, arterial blood pressure 130/80 mmHg, pallor of skin and sclera, abdominal distention, diminishing intestinal tones, voluntary resistance and tenderness upon palpation. Melena was observed in rectal touch examination. Following findings were determined in the laboratory evaluation: in whole blood cell count, white blood cell count 23,600/mm^3 (PNL 88%, lymphocyte 12%, monocyte 1.2%); hemoglobin 7.2 gr/dL, hematocrit 20.5%; platelets 115.000/mm^3.

Transaminase values of AST 83 IU/L, ALT 51 IU/L, LDH: 935 IU/L, GGT: 23 IU/L, total bilirubin: 1.3 mg/dL, direct bilirubin: 0.30 mg/dL, total protein: 3.4 mg/dL, albumin: 1.87 mg/dL, urea: 48 mg/dL, creatinine: 1.5 mg/dL, Na: 134 mmol/L and calcium: 6.2 mg/dL were detected. Feces was dark brown, loose, unformed and bloody with no mucus. Microbiological evaluation of the feces revealed a large number of red blood cells and 1 - 2 white blood cells in certain regions. No parasite, parasite egg, cyst or trophozoid was detected. Blood culture was repeated three times at half hour intervals.

Oral nutrition of the patient was discontinued and GIS bleeding protocol was initiated. Supportive therapy and blood transfusion was administered. Emergency abdominal ultrasound (USG) examination showed disseminated and abundant fluid around perihepatic, perisplenic and intestinal loops. Oedematous intestinal loops in the lower abdominal quadrant and fluid in bilateral hemithorax were detected. Upon these USG findings, the patient was evaluated by general surgery department and diagnosed with GIS bleeding. Paracentesis was performed for the fluid in the abdominal cavity; the characteristics of paracentesis fluid was transuda and on microscobic examination, abundant red blood cells (380 hücre/mm^3) were detected.

Endoscopic examination of oesophagus, gastrum and small intestines was not performed due to oropharyngeal abscess. Emergency colonoscopic examination revealed multiple ulcers in the colon. Biopsy was performed from these ulcers. Even though no bleeding focus was observed in the lumen of the colon, the content of colon was bloody. In the computerized tomographic angiography of the patient, contrast material leakage was detected around regions covered by superior mesenteric artery (**Figure 2(b)**). Surgical intervention was deferred on the basis of patient's melena, oedema of small intestine and fluid collection between intestinal loops, presence of ulcers in colon and deterioration of general status of the patient.

Blood transfusion, fresh frozen plasma, platelet suspension and albumin replacement were administered to the patient whose hemoglobin, hematocrit and platelet values decreased continuously. Unfortunately, deterioration of general status persisted. Following bleeding symptoms, a septic state developed with signs of tachycardia, hypotension and tachypnea. *Bacillus anthracis* grew on two concurrent blood cultures (**Fgiure 1(d)**). Patient deceased on the 17th day of penicillin G and levofloxacin treatment.

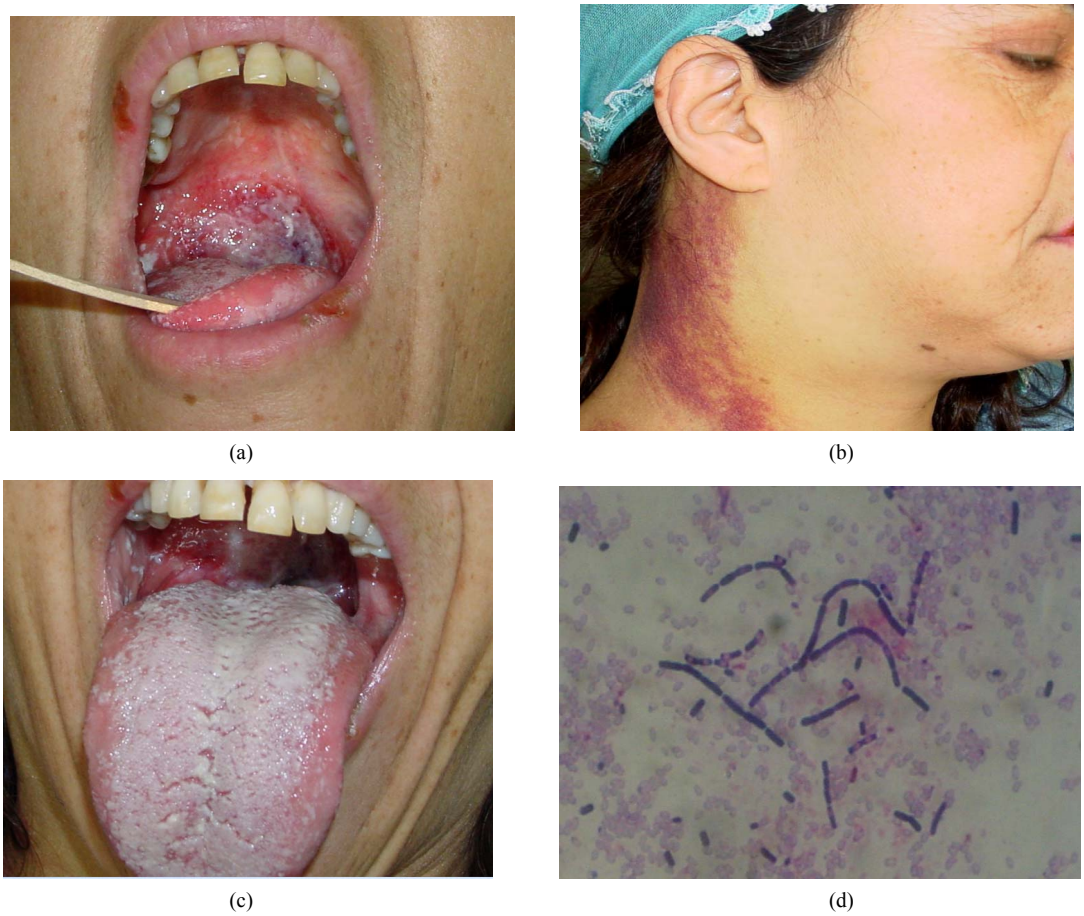
2. Discussion

Bacillus anthracis is a Gram (+), spore-forming, encapsulated and non-motile bacteria. Spores may be located centrally or paracentrally and do not cause dilatation in the bacteria dimentions [1-4].

Around 90% - 95% of anthrax infections are cutaneous infections due to easy transmission. Transmission by inhalation causes pulmonary anthrax and oral transmission leads to gastrointestinal anthrax. Rate of sepsis following internal organ anthrax infections is extremely high. Gastrointestinal anthrax frequently causes multiple and superficial ulcers all along gastrointestinal tract starting from oral cavity to caecum; in some cases, ulcers may also be seen in the colon . Massive and fatal bleeding is seen in serious cases of ulcerative lesions [1-4].

When Bacillus anthracis spores are ingested orally, they attach to the epithelial cells of tonsilla and M cells over the payer plaques of small intestine. Live bacteriae releases a toxin while it reproduces. The disease presents a wide spectrum of states ranging from subclinic infection to death.

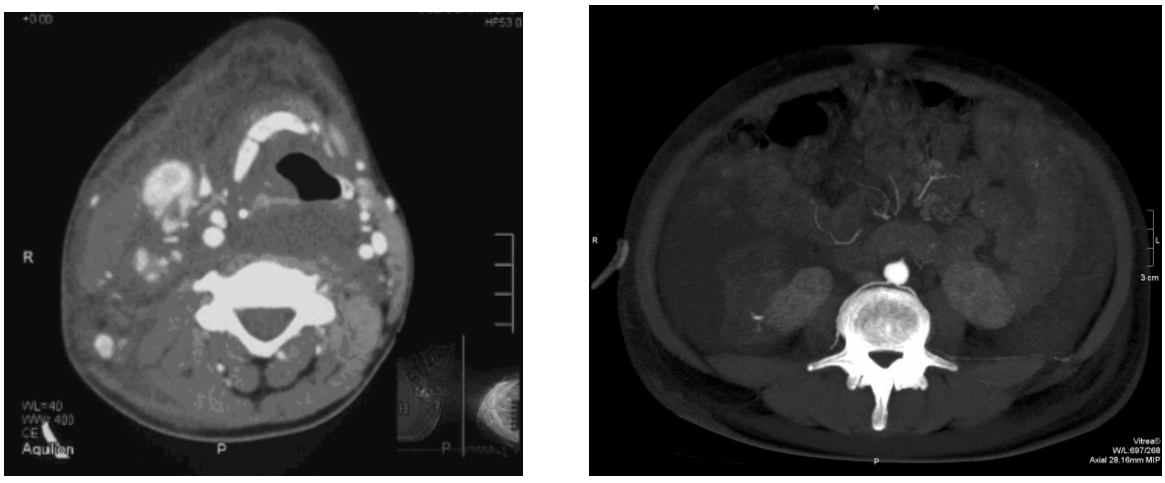
The exact infectious dose in humans is not known but for oral anthrax, the determined minimal infectious dose (Mid50) 50 mg is approximately 10^{11} spores [5].



(a) (b)

(c) (d)

Figure 1. (a) Abscess of anthrax oroferangeal; (b) The observed bleeding after anthrax abscess decrease; (c) Were reduced after drenation abscess oroferangeal of anthrax; (d) Gram stained preparations of the *Bacillus anthracis* in the blood culture



(a) (b)

Figure 2. (a) In contrast CT: cervical region and in multiple necrotic LAPs in that are contrast. Right to be more specific with soft tissue edema in the air passages provided in the plan from the recession is being monitored; (b) The common areas in the region prevertebral cervical fluid (hemorrhagic exudative) increase.

Clinical findings in oropharyngeal anthrax includes fever, inflammatory lesions in the oral cavity or oropharynx, oedema and lymphadenopathy in the neck; the mortality rate of the disease is high [1]. Oropharyngeal anthrax should be differentiated from diphteria, complicated tonsillitis, streptococcal pharyngitis, vincent angina, ludwing

angina, parapharyngeal abscess and deep neck abscess [6].

Ulcerative lesions of gastrointestinal (GIS) anthrax are usually more than one and superficial. Lesions located in gastrum, oesophagus and jejunum may cause fatal massive bleeding. In addition, these lesions may lead to obstruction [7-10], perforation or various combinations of these complications. GIS anthrax may cause ascites. In some cases, dehydration from the vascular compartment may cause shock and fatality [10-15].

Intestinal anthrax presents with three clinical phases:

Phase I: includes fainting, asthenia, mild fever and headache. Patients are rarely treated at this stage because of few symptoms. In a small number of patients, rash on the face or redness of the conjunctiva may be seen.

Phase II: starts in 24 hours and abdominal pain is added to symptoms. Abdominal pain, which is initially mild, intensifies and adopts a continuous course. Mild fever, nausea and vomiting is seen frequently; diarrhea, if present, is mild. On physical examination, mass and abdominal distension is detected in right lower quadrant and periumblical area.

Phase III: there is a persistent and rapidly intensifying abdominal pain and in most cases, GIS bleeding develops. In patients where laparotomy is performed, abundant and viscous, yellow fluid, hypertrophic mesenteric lymph nodes especially in the ileocaekal area (3 - 5 cm) and oedema in some parts of the small intestine, caekum and ascending colon is observed. In case of failure on surgical removal of the diseased part, a transient recovery is seen. In these patients, ascites develops again and most of the patients are lost, due to shock states [16].

During surgery and in autopsy series, small red nodules (0.5 - 1 cm) with surrounded necrotic centers (2 - 3 cm) are seen in hemorrhagic areas surrounded by oedema in the intestinal wall [17].

GIS anthrax should be differentiated from causes leading to early acute abdominal syndrome as well as Clostridium perfiringens which causes necrotizing enteritis and other bacterial or parasite agents which lead to bloody diarrhea.

There are few literature review and case reports. Most of the this case reports and releated articles about GIS anthrax says that, this disease is very dangerous and its mortality rates 25% - 60%. Death due to GIS anthrax is reported to occur in 2 - 5 days [2,7,8,19-21].

In our case, the patient presented with oropharyngeal anthrax. We suspected oropharyngeal anthrax with patient' s epidemiological history. Then we recieved blood and wound cultures and B. anthracis investigated at this cultures. Computerized tomography (CT) used for diagnosis and oropharyngeal abcess detected at CT images. On the 6th day of treatment, her general status improved and the abscess in the pharynx showed considerable decrease. The patient drained the abscess with her own finger and consequently caused GIS anthrax. Since the patient presented with dysphagia, it was not possible to give oral contrast material and therefore, abdominal computerized tomography with contrast material could not be performed; as a result, it could not be confirmed whether she had or did not have GIS anthrax initially. Nevertheless, absence of abdominal pain, diarrhea and vomiting in the beginning, improvement of general status in 7 days and her own statement on draining the abscess with her finger and swallowing the fluid lead us to conclude that GIS anthrax developed later on.

In Turkey, anthrax is reported to show an endemic course with 96.9% of the cases presenting with cutaneous anthrax [18-21]. In an investigation, oropharyngeal anthrax is seen in 7 cases and intestinal anthrax is diagnosed in 1 case [19]. In Turkey, no resistance was observed against penicillin G for *Bacillus anthracis*; therefore, penicillin G is the treatment of choice as an effective agent. It has been reported that aminoglycoside, together with penicillin G is more effective in oropharyngeal or intestinal anthrax [18-21]. In our case, aminoglycoside was not preferred as an adjunctive antimicrobial because creatinine levels of the patient were high (creatinine: 1.3 - 1.5 mg/dL). Even though the usual treatment period is indicated as 10 - 14 days, due to insufficient improvement in our patient, treatment was not terminated.

In conclusion, due to the high mortality rates of GIS antrax must be prevented. In countries which are endemic anthrax areas, carving and eating the meat of sick animals should absolutely be prevented plus people should be informed and warned to take precautions about the disease. Besides, in cases of oropharyngeal abscesses, anthrax should be considered and a careful patient history should be taken, laboratories should be warned under suspicious conditions and necessary evaluations should be performed. We should approach oropharyngeal anthrax cases carefully, taking into account intestinal anthrax cases.

REFERENCES

[1] M. Neguţ and D. M. Caplan, "Epidemiology Studies Regarding Anthrax Epidemic in Romania," *Bacteriologia*, *Virusologia*, *Parazitologia*, *Epidemiologia*, Vol. 47, No. 3-4, 2002, pp. 161-165.

[2] H. Ozdemir, K. Demirdag, T. Ozturk and E. Kocakoc, "Anthrax of the Gastrointestinal Tract and Oropharynx: CT Findings," *Emergency Radiology*, Vol. 17, No. 2, 2010, pp. 161-164. doi:10.1007/s10140-009-0821-y

[3] C. Kunanusont, K. Limpakarnjanarat and J. M. Foy, "Outbreak of Anthrax in Thailand," A*nnals of Tropical Medicine and Parasitology*, Vol. 84, No. 5, 1990, pp. 507-512.

[4] D. P. Perl and J. R. Dooley, "Anthrax," In: C. H. Binford and D. H. Connor, Eds., *Pathology of Tropical and Extraordinary Diseases*, Armed Forces Institute of Pathology, Washington DC, 1976, pp. 118-123.

[5] Z. A. Kanafani, A. Ghossain, A. I. Sharara, J. M. Hatem and S. S. Kanj, "Endemic Gastrointestinal Anthrax in 1960s Lebanon: Clinical Manifestations and Surgical Findings," *Emerging Infectious Diseases*, Vol. 9, No. 5, 2003, pp. 520-525. doi:10.3201/eid0905.020537

[6] WHO, "Antrax in Humans and Animals," 4th Edition, WHO Press, Geneva, 2008.

[7] T. Tantajumroon and K. Panas-Ampol, "Intestinal Anthrax: Report of Two Cases," *Journal of The Medical Association of Thailand*, Vol. 51, 1968, pp. 477-480.

[8] T. Tantachumroon, "Pathologic Studies of İntestinal Anthrax: Report of 2 Cases," *Chiang Mai Medical Bulletin*, Vol. 4, 1966, pp. 135-144.

[9] E. Kohout, A. Sehat and A. M. Ashraf, "Anthrax: A Continuous Problem in Southwest Iran," *The American Journal of the Medical Sciences*, Vol. 247, No. 3, 1964, pp. 565-575. doi:10.1097/00000441-196405000-00006

[10] G. P. Jena, "Intestinal Anthrax in Man: A Case Report," *Central African Journal of Medicine*, Vol. 26, No. 12, 1980, pp. 253-254.

[11] Centers for Disease Control and Prevention (CDC), "Gastrointestinal Anthrax after an Animal-Hide Drumming Event—New Hampshire and Massachusetts, 2009," *Morbidity and Mortality Weekly Report*, Vol. 59, No. 28, 2010, pp. 872-877.

[12] P. Baht, D. N. Mohan and H. Srinivasa, "Intestinal Anthrax with Bacteriological İnvestigations," *The Journal of Infectious Diseases*, Vol. 152, No. 6, 1985, pp. 1357-1358.

[13] W. Dutz, F. Saidi and E. Kohout, "Gastric Anthrax with Massive Ascites," *Gut*, Vol. 11, No. 4, 1970, pp. 352-354. doi:10.1136/gut.11.4.352

[14] A. Alizad, E. M. Ayoub and N. Makki, "Intestinal Anthrax in a Two-Year-Old Child," *The Pediatric Infectious Disease Journal*, Vol. 14, No. 5, 1995, pp. 394-395. doi:10.1097/00006454-199505000-00013

[15] T. Sirisanthana, U. Jesadaporn, "Survival of a Patient with Gastrointestinal Anthrax," *Chiang Mai Medical Bulletin*, Vol. 24, No. 1, 1985,pp. 1-5.

[16] D. R. Nalin, B. Sultana, R. Sahunja, A. K. Islam, M. A. Rahim, M. Islam, *et al.*, "Survival of a Patient with Intestinal Anthrax," *American Journal of Medicine*, Vol. 62, No. 1, 1977, pp. 130-132. doi:10.1016/0002-9343(77)90358-8

[17] A. Ghossain, "Intestinal Anthrax," In: T. S. Harrison, Ed., *Surgery for All*: *A View from the Developing World*, Ferozsons, Lahore, 1992, pp. 144-150.

[18] S. Felek, A. Akbulut and A. Kalkan, "A Case of Antrax Sepsis: Non Fatal Course," *Journal of Infection*, Vol. 38, No. 3, 1999, pp. 201-202. doi:10.1016/S0163-4453(99)90256-6

[19] M. Doganay, A. Almaç and R. Hanağasi, "Primary Throat Anthrax. A Report of Six Cases," *Scandinavian Journal of Infectious Diseases*, Vol. 18, No. 5, 1986, pp. 415-419. doi:10.3109/00365548609032357

[20] M. Doganay and G. Metan, "Human Anthrax in Turkey from 1990 to 2007," *Vector-Borne and Zoonotic Diseases*, Vol. 9, No. 2, 2009, pp. 131-140. doi:10.1089/vbz.2008.0032

[21] M. Doganay, "Ingestional (Oral Route/Enteric) Antrax: Is It Problem in Turkey?" *Flora*, Vol. 14, No. 3, 2009, pp. 97-104.

