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CASE REPORT

A case of pneumonia caused by *Bacillus anthracis* secondary to gastrointestinal anthrax

Meliha Meric [a,*], **Ayse Willke** [a], **Bahar Muezzinoglu** [b], **Aynur Karadenizli** [c], **Tulay Hosten** [d]

[a] *Kocaeli University, Medical Faculty, Department of Clinical Bacteriology and Infectious Diseases, Kocaeli, Turkey*
[b] *Kocaeli University, Medical Faculty, Department of Pathology, Kocaeli, Turkey*
[c] *Kocaeli University, Medical Faculty, Department of Microbiology, Kocaeli, Turkey*
[d] *Kocaeli University, Medical Faculty, Department of Anesthesiology and Reanimation, Kocaeli, Turkey*

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Abstract We present herein an unusual case of anthrax pneumonia secondary to gastrointestinal infection. In this case, severe abdominal pain occurred during the course of a stent placement procedure. The patient had undergone surgery with the prediagnosis of intestinal ischemia. On the second postoperative day, pneumonia developed and *B. anthracis* grew as the etiologic agent. Pathological examination of small-bowel sections revealed findings in accordance with anthrax.

Introduction

Anthrax is a zoonotic disease caused by *Bacillus anthracis*, an aerobic, non-motile, Gram-positive bacillus with central spores. The spores can persist for years in soil and primarily infect herbivores. Humans can acquire anthrax by exposure to infected animals or their products.[1,2]

The clinical picture of human anthrax varies depending on the mode of entry of the bacteria. The disease can be asymptomatic or might progress to sepsis and even cause death if not treated in a timely fashion. The clinical forms of anthrax are cutaneous, gastrointestinal (GI) and inhalational. About 95% of human anthrax is in cutaneous form. GI and inhalational anthrax are very rare and the mortality rate of these forms is much higher than that of the cutaneous form.[1]

We present herein an unusual case of anthrax pneumonia secondary to GI infection.

Case report

A 59-year-old male patient was admitted to Kocaeli University Hospital with a history of bilateral vision loss for five months. Bilateral severe carotid stenosis was diagnosed and carotid arterial stent placement was planned by the Radiology clinic. He had no history of any known disease, except for diabetes mellitus and hypertension for the past four and eight years, respectively. He was a taxi-driver, but had not worked for one month prior to hospital admission. During this period, he spent his time at home with his wife.

* Corresponding author. Kocaeli University, Medical Faculty, Department of Clinical Bacteriology and Infectious Disease, Umuttepe, Kocaeli, 41380, Turkey. Tel.: +90 262 3037082; fax: +90 262 3038085.
E-mail address: drmelihameric@gmail.com (M. Meric).

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Widespread femoral arterial thrombosis was observed during percutaneous stenting; however, sudden onset of severe abdominal pain, an increase in blood pressure (240/140 mmHg) and rapid deterioration of his clinical condition occurred. The patient was transferred to the intensive care unit (ICU).

On admission to the ICU, physical examination revealed the following: body temperature 39 °C; pulse rate 110/min; blood pressure 180/80 mmHg; respiratory rate 32/min. He was in a toxic condition, with abdominal tenderness, distension and rebound positivity. No skin lesion or skin infection was observed. Laboratory results were as follows: white blood cell (WBC) count 11.4×10^9/l (65% neutrophils, 20% lymphocytes); hemoglobin 12.79 g/dl; platelet (PLT) count 279 000/ul; prothrombin time (PT) 14.8 s; partial thromboplastin time (PTT) 32.6 s; INR 1.15; D-dimer 2.85 μg/ml (normal: 0—0.5); erythrocyte sedimentation rate (ESR) 32/hr; C-reactive protein (CRP) 2.33 mg/dl (normal: 0—0.8 mg/dl); blood creatinine 3.9 mg/dl; urea 120 mg/dl; glucose 327 g/dl. Chest radiographs and abdominal ultrasonography (USG) were within normal limits. Echocardiography revealed an intra-aortic dislodged thrombus.

Despite the supportive treatment, intravenous antimicrobial therapy with ceftriaxone (2×2 g/day) plus metronidazol (3×500 mg/day) and anticoagulant therapy, the medical condition of the patient deteriorated.

Considering his laboratory and clinical status, mesenteric ischemia was suspected and a laparotomy was performed. With the intra-operative finding of three large foci of necrosis in the terminal ileum, a wide resection of the small bowel and primary repairment were performed.

His clinical status and vital signs became stable immediately after surgery. The surgical site was clean. However, on the second postoperative day, respiratory arrest developed with a fever of 39 °C. He had crepitant rales at the right lower pulmonary fields. Chest radiography showed mediastinal widening and pneumonic infiltrate of the right lower part of the lung.

Laboratory studies disclosed the following data: WBC 10.2×10^9/l; hemoglobin 9.9 g/dl; ESR 75 mm/hr; CRP 22.3 mg/dl; blood creatinine 5.0 mg/dl.

Endotracheal intubation was performed and mechanical ventilation was started, in addition to continuous arteriovenous hemofiltration (CAVH). Cultures of blood and tracheal aspirates were obtained. The tracheal aspirate culture yielded *Bacillus anthracis* on the following day. Antimicrobial therapy was switched to intravenous ciprofloxacin (2×200 mg/day) plus crystalline penicillin G (6×4 MU/day), with the diagnosis of anthrax pneumonia.

From his detailed family history, it was learned that, five days before admission, the patient had consumed meatballs prepared with meat that was purchased from sources that were known to be responsible for anthrax infection. Among 12 family members who had eaten the same meat, only the presented patient had severe disease, although his ten-year-old grandchild had a history of mild diarrhea with recovery after treatment with ampicillin. He had no history of possible exposure to inhalational anthrax. Additionally, according to Local Health Office documents, there were no other reported anthrax cases or doubtful deaths in Kocaeli during the same period.

In view of the histological examination of the small bowel and microbiological results, anthrax pneumonia secondary to GI anthrax was suggested. All appropriate precautions for infection control were taken in the ICU and local health authorities were informed.

Despite all supportive and antimicrobial therapy, the patient died on the ninth treatment and thirteenth hospitalization days. Post-mortem studies were not done because his wife refused permission for autopsy.

Microbiological studies

Tracheal aspiration material from the patient was hemorrhagic. Short chains of large Gram-positive rods, erythrocytes and polymorphonuclear leukocytes were seen on microscopy.

Tracheal aspirate culture yielded *B. anthracis* with typical morphologic and microscopic features, motility testing and catalase activity. The confirmation of *B. anthracis* was based on the presence of capsules in defibrinated sheep blood and by animal inoculation (Figure 1). Blood cultures were negative.

Antimicrobial susceptibility testing of *B. anthracis* by disk diffusion showed sensitivity to penicillin, ampicillin, ciprofloxacin, clindamycin, tetracycline, chloramphenicol, cefoperazone and imipenem. It was resistant to ceftriaxone, cefotaxime, ceftazidime and rifampicin.

Animal inoculation method

Overnight culture of the isolated Bacillus strain in Mueller Hinton broth was adjusted to McFarland O.5. A mouse was inoculated intraperitoneally with 1 ml of the suspension. It died 18 hours after inoculation and was autopsied. The abdomen and thorax were opened, and specimens were obtained from cardiac, liver and spleen tissues. All were cultured onto 5% sheep blood agar. Non-hemolytic, irregular, gray colonies were grown overnight after incubation at 37 °C in an incubator. A methylene blue stain of the spleen specimen is presented in Figure 1.

Histopathological findings

On the serosal surface of the small bowel, there were discrete areas that were depressed, yellow-brown and suppurative in appearance. Upon opening the lumen, these areas

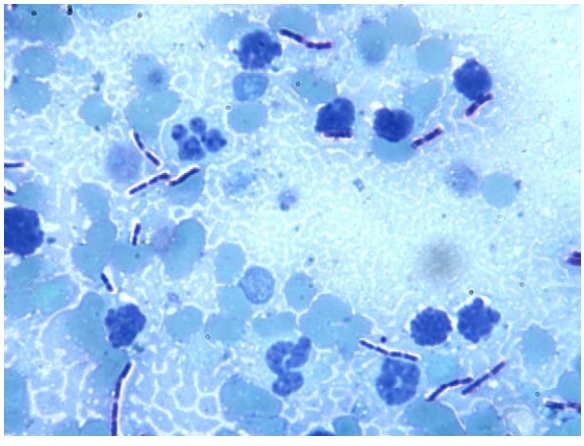

Figure 1 Encapsulated bacilli on the methylene blue stain of the mouse spleen imprint.

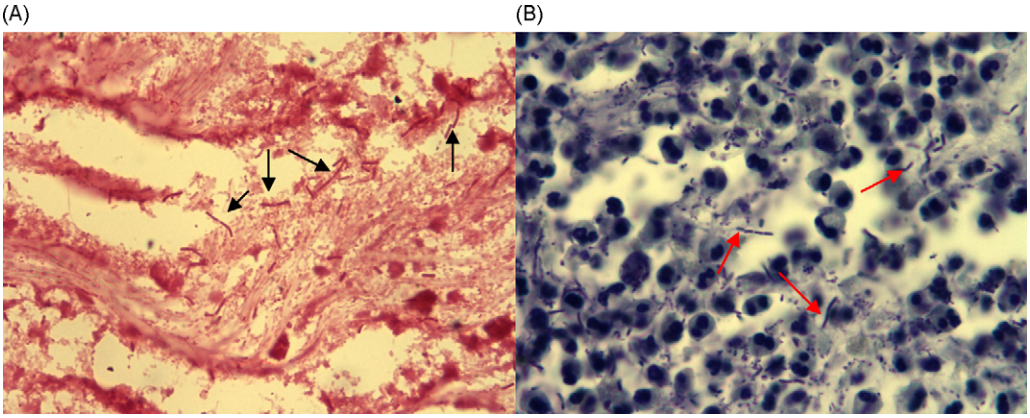

Figure 2 Anthrax bacilli (arrows) shown in (A) Gram and (B) Giemsa stains of a paraffin tissue section from the necrotic ileum.

were ulcerated and flattened, with a bowel wall thickness of 3—4 mm at the underlying mucosa. Microscopically, the mucosa was ulcerated, extending deep into the muscular layer. The entire bowel wall was infiltrated with dense polymorphonuclear leukocytes intermingled with large bacilli. With the preliminary diagnosis of GI anthrax, the same ileal sections were stained with Giemsa and Gram. Encapsulated Gram-positive rods were seen upon pathological examination of excised necrotic ileum parts (Figure 2).

Discussion

Pneumonia caused by *B. anthracis* usually occurs as inhalational anthrax, with the typical presence of hemorrhagic mediastinal adenopathy in chest radiographs.[1] In endemic countries, however, anthrax pneumonia can occur secondary to sepsis originating from cutaneous or GI anthrax.[3] In this case, anthrax pneumonia was diagnosed prior to the primary GI infection. GI anthrax usually occurs 1—7 days after the consumption of raw or undercooked meat from infected animals.[2,4,5] The disease develops with symptoms of nausea, anorexia and fever, followed by abdominal pain and diarrhea.[1—3] In our case, the clinical picture of severe abdominal pain and toxic status appeared during the course of a stent placement procedure. The patient had undergone surgery with the prediagnosis of intestinal ischemia. On the second postoperative day, pneumonia developed and *B. anthracis* grew as the etiologic agent. Pathological examination of small-bowel sections showed findings in accordance with anthrax.

GI anthrax is a very serious form of the disease and is associated with a high mortality rate. Early antimicrobial and surgical treatment might save the life of the patient.[6—8] In this case, the GI infection was diagnosed coincidentally after anthrax pneumonia. Despite the small-bowel resection, appropriate antimicrobial therapy and supportive care, the patient died.

The meat consumed by the patient and his family was brought by a relative who lives in an anthrax endemic region of Turkey. Of the 12 people who had eaten the same meat, only the presented patient had severe disease. This could be related to the use of a proton pump inhibitor by the patient. Unfortunately, we could not investigate the meat that was thought to be responsible as the source of infection.

Because of the high mortality rate of GI anthrax and the similarity of the symptoms to acute abdominal pain, GI anthrax should be considered in the differential diagnosis of patients with acute abdominal pain in endemic areas.

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Conflict of interest: No conflict of interest to declare.

References

1. Lucey D. *Bacillus anthracis* (Anthrax). In: Mandell GL, Bennett JE, Dolin R, editors. *Principles and Practice of Infectious Diseases*. Philadelphia: Elsevier-Churchill Livingstone; 2005. p. 2485—93.
2. Sirisanthana T, Brown AE. Anthrax of the gastrointestinal tract. *Emerg Infect Dis* 2002;**8**:649—51.
3. Doganay M. *Bacillus anthracis* ve diğer bacillus türleri. In: Willke Topcu A, Soyletir G, Doganay M, editors. *Infeksiyon Hastalıkları ve Mikrobiyolojisi*. Istanbul: Nobel Tip Kitabevi; 2002. p. 1533—43.
4. Babamahmoodi F, Aghabarari F, Arjmand A, Ashrafi H. Three rare cases of anthrax arising from the same source. *Infection* 2006;**53**:175—9.
5. Dutz W, Saidi F, Kohout E. Gastric anthrax with massive ascites. *Gut* 1970;**11**:352—4.
6. Kanafani ZA, Ghossain A, Sharara AI, Hatem JM, Kanj SS. Endemic gastrointestinal anthrax in 1960's Lebanon: clinical manifestations and surgical findings. *Emerg Infect Dis* 2003;**9**:520—5.
7. Tekin A, Bulut N, Unal T. Acute abdomen due to anthrax. *Br J Surg* 1997;**84**:813.
8. Kunanusont C, Limpakarnjanarat K, Foy HM. Outbreak of anthrax in Thailand. *Ann Trop Med Parasitol* 1990;**84**:507—12.