Austin Journal of Clinical Case Reports

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Austin Publishing Group

Case Report

Oropharyngeal Anthrax: Case Report

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Received: March 14, 2019; **Accepted:** April 23, 2019; **Published:** April 30, 2019

Abstract

We present here a case of very rare clinical manifestation of Anthrax gastrointestinal form – oropharyngeal anthrax with fatal outcome. A 28-year-old woman was admitted to the hospital in severe condition with respiratory insufficiency, tachycardia, low blood pressure, swelling of lymph nodes of the neck by the 4th day after the disease onset.

Keywords: *Bacillus anthracis*; Oropharyngeal anthrax; Endemic

Background

Anthrax is a bacterial disease caused by the spore forming Bacillus anthracis, a Gram positive, rod shaped bacterium with a remarkable ability to persist in the environment. Infection in humans is primarily from contact with infected animals, typically herbivores, and exposure to contaminated animal products [1]. Found on nearly every continent, the disease is considered a re-emerging zoonotic disease, and despite the development of anthrax vaccines for animals and humans, the disease continues to be endemic in many countries, including Georgia and it is still an essential due to the threat of bioterrorism [2-4]. Clinical manifestations can occur as respiratory, cutaneous, or gastrointestinal depending on route of transmission. The main clinical features for Oropharyngeal anthrax are sore throat, dysphagia, fever, regional lymphadenopathy in the neck and toxemia. Even with treatment, the mortality is about 50% (Doganay et al., 1986). We present here a case of very rare clinical manifestation of gastrointestinal form – oropharyngeal anthrax with fatal outcome.

Case Presentation

A 28-year-old woman presented initial symptoms of hyperthermia (t_{max} 39^{0}C), vomiting, headache, sore throat, difficulty swallowing and swelling of the mandibular glands. Patient was admitted to the hospital in severe condition with respiratory insufficiency, tachycardia, low blood pressure, swelling of lymph nodes of the neck by the 4th day after the disease onset [5,6].

The examination and tests showed low blood pressure (T/A-80/50mm/Hg), tachycardia (P-90, RR-24), signs of respiratory distress, SPO_2-97%, lung auscultation - weak vesicular sound in lover lobes, heart sound decreased in intensity, edema of upper third of the chest, hypertrophy of the right submandibular gland, pain in the neck and occipital area, grayish coating in the pharynx. CBC: WBC-14.2, HGB–135mg/l, RBC-4.84, PLT–164, ESR-7mm/hr. Urinalysis (rapid test): Specific gravity-1020, pH–6.0, Leukocytes-10, protein-0.033%, erythrocytes-0, nitrite-negative, bilirubin-neg, urobilinogen-neg, ketone bodies-pos, Glucose - (-). Coagulation test: PT–16.5, prothrombin activity-79.0, INR-1.31, TT–26.0, fibrinogen-255. Blood gas and electrolytes: pH-7.43, HCO_3–22.8mmol/L, $PaCO_2$-34.0mmHg; PaO_2-30mmHg; Na-135mmol/l, K-3.9mmol/l. Blood chemistry test: Creatinine–58mol/l, UREA–6.8mmol/L, GLUK–7.6MMOL/L [7].

Severity of the condition was caused by infectious-toxic shock, unstable hemodynamics, and acute respiratory distress. The patient was somnolent, consciousness was depressed, edema of the neck and upper third of the chest was evident, big size indurated site in the right submandibular area, specific smell from the mouth, hypertrophy of tonsils and uvula, greyish-whitish coating difficult to remove that bleeds when removed on soft palate and pharynx on the right. The patient was admitted to ICU with provisional diagnosis of acute respiratory distress, infectious-toxic shock, pharyngeal diphtheria [8]. The patient developed generalized seizures, cyanosis as he was moved to bed, because of these the patient was intubated orotracheally without complications and was moved to mechanical ventilation. The patient developed anisocoria, S>D, it rapidly developed into mydriasis. Unstable hemodynamics with increasing doses of inotropic-vasopressoric support [9].

Treatment provided at "Hospital A": SOL.NaCl 0.9%, Metoklopramid 2.0, Dexsamethazon 4mg, Dimedrol 1.0, Analgin 2.0, Penicillin G 1200mg, Fentanyl 2.0, Anti-Diphtheria Serum - 40000 IU.

Prior to given Anti-Diphtheria Serum, had been taken swab samples from nasal and oral cavities for laboratory investigation as well as whole blood for culturing.

Due to the severe condition, patient with preliminary diagnosis pharyngeal diphtheria (A36.0) was transferred to "Hospital B" for additional examination. Diagnostic tests and consultations conducted at "Hospital B" included ultrasound on 5th day of symptoms onset: organs in abdominal cavity were without remarkable changes, pleuritis was revealed on the right. Follow up testing revealed bilateral pleuritic, fluid in the abdomen, around the uterus, signs of ileus. Heart ultrasound – mild dilatation and hypertrophy of cardiac chambers with decrease of systolic function. EF-48%; Ultrasound of skin and soft tissues–lymphadenopathy of neck soft tissues on the right. Due to the extremely severe condition the recommended brain CT could not be performed. Laboratory test results: WBC-47.2, HGB–119mg/l, NEU-78, PLT–103, ESR-8mm/hr, CRP-18, PT–19,7, PT%-64, INR-

Citation: Malania L, Simsive T, Abazashvili N, Tsilosani M, Khmaladze E, Dzotsenidze V, et al. Oropharyngeal Anthrax: Case Report. Austin J Clin Case Rep. 2019; 6(2): 1142.

1,72, aPTT-28.4, PH-7.4, HCO_3-20.1mmol/L, Na-179mmol/K-5, 14mmol/l, Laq-2.8, Crea-634nmol/l, ALT-26, AST-50, GGT-9, ALP-48, TBil-8.9, DBil-0.95, TP-45, ALB-15.

On the 6th day of symptoms onset, a tracheostomy was performed because airways were repeatedly obstructed and active treatment of the oral cavity was needed. A black ulcer developed on the pharyngeal coating, later, satellite ulcers appeared. The patient had increasing edema of the neck and head area, brain edema, infectious-toxic shock, coma, right sided pneumonia, bilateral pleuritic, and fluid in the abdominal cavity.

Despite the provided treatment, the patient's condition was markedly negative, critical in dynamics. On the 6th day after the disease revealed, hypotension developed and monitor showed asystole. Despite complete resuscitation measures, pupils dilated, cardiac function could not be restored and at 20:00 biological death was declared.

Laboratory investigation of swab samples from the oral cavity after 24 hours of culturing on 5% Sheep Blood Agar at 37°C on ambient condition gave growth suspected of *B. anthracis* (non-hemolytic, white-grey sticky colony, with curly tailing at the edges); gram stain showed gram positive rods square ended appearance. Preliminary result from the laboratory was sent to the hospital, where Ciprofloxacin was added to the patient's treatment. The bacterial isolate was confirmed by gamma-phage test, DFA FITC and qPCR (Idaho Technology) as *B. anthracis*.

Epidemiological investigation revealed that 10 days before symptoms developed, the patient had processed ground beef and consumed raw meat (to check the taste of spices before cooking dinner). Meat was bought by a family member at an open market in west Georgia [10].

Based on the patient's medical records, the preliminary diagnosis was acute respiratory distress (J96.0), infectious-toxic shock (R57.8), pharyngeal diphtheria (A36.0). Clinical diagnosis: acute respiratory distress (J96.0), infectious-toxic shock (R57.8). Sepsis caused from Anthrax oropharyngeal form (A.22.7). Sepsis develops after the lymphohematogenous spread of B. anthracis from a primary lesion (in our case - gastrointestinal). Clinical features are high fever, toxemia and shock, with death following in a short time.

Diagnosis of oropharyngeal anthrax requires attention to the patient's history and risk of consumption of contaminated food. Differential diagnosis of oropharyngeal anthrax should consider parapharyngeal abscess, deep tissue infection, Vincent's angina, Ludwig's angina, and streptococcal pharyngitis [2].

The presence of an epithelial lesion would likely increase the risk that spores may gain entry to establish an infection. It is expected that the infectious dose for the gastrointestinal and oropharyngeal epithelium will be similar to the skin [11].

Overt gastrointestinal tract cases are more often fatal, largely because they go unrecognized until it is too late for effective treatment.

Conclusion

The first confirmed case of orthopharyngeal anthrax was reported in the country of Georgia [5]. Medical care sought late and delayed treatment most likely contributed to the lethal outcome. This emphasizes the importance of seeking timely medical care, early diagnostics and timely initiation of treatment [12,13]. We recommend considering anthrax in patients with severe pharyngitis as a part of differential diagnosis.

Acknowledgement

We would like to thank the Defense Thread Reduction Agency for support to the country of Georgia in the strengthening of surveillance of Anthrax by building strong laboratory capacity across the country, by training provided for the medical and laboratory personnel, and for field work and biosafety/biosecurity aspects.

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