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Case Report

Type E botulism associated with crude beef: A case report

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Botulism remains infrequent but is widely feared because it can be severe and is potentially lethal. Foodborne botulism results from contaminated foodstuffs in which *Clostridium botulinum* spores have been allowed to germinate in anaerobic conditions. This typically occurs in home-canned food substances and fermented uncooked dishes. We present an outbreak of type E botulism in China. The diagnosis was based on the patient's rapid deterioration and presentation of descending paralysis and was confirmed by laboratory findings. The patient did not received antitoxin administration because of delayed diagnosis. We report this case to alert clinicians to the possibility of major clinical diagnosis, adequate treatment without antitoxin can lead to successful management of mild cases in botulism.

Key words: Foodborne botulism, *Clostridium botulinum*, Type E, crude beef, case report.

INTRODUCTION

Clostridium botulinum which is ubiquitously found in soil and aquatic sediments can produce potent toxins causing botulism, a clinical recognizable syndrome. Eight different strains by their properties and toxins (A, B, C1, C2, D, E, F, G) were identified, but to date, only types A, B and E are involved in human pathology. Botulism remains infrequent but is widely feared because it can be severe and is potentially lethal (Zhang et al., 2010). Foodborne botulism is caused by eating preformed toxin produced in contaminated food. The most frequent source is home-canned foods and foods fermented or preserved with traditional methods in which spores that survive due to a lack of acidifier condition, and inadequate cooking and storing process germinate, reproduce and produce toxin in the anaerobic environment of the food (Peck, 2006). Prompt recognition and treatment of botulism by clinicians remain a critical component of surveillance.

We present here a case of type E botulism in China. The diagnosis was based on the patient's rapid deterioration and presentation of descending paralysis and was confirmed by laboratory isolation of *C. botulinum* from contaminated food.

MATERIALS AND METHODS

In April 2009, a 33-year-old man was admitted to the hospital with a 3-day history of nausea, vomiting and dizziness. The preliminary diagnosis was acute gastritis and symptomatic treatment was given in the form of stomach lavage, and intravenous administration of antibiotics and steroids. At that night, he developed fever, blurred vision, constipation and urinary retention. Computed tomography scan of the brain and the findings of cerebrospinal fluid were normal. The laboratory tests showed a level of alanine transarninase of 60 U/L, aspartate aminotransferase of 50 U/L, total bilirubin of 32.4 µmol/L, conjugated bilirubin of 9 µmol/L, unconjugated bilirubin of 23.4 µmol/L, with increased monocyte percentage (9.3%) and decreased K^+ (3.32 mmol/L), Ca^{2+} (2 mmol/L) and hemoglobin (12.6 g/L). Initially, the patient and his friends all denied having eaten any contaminated food. Finally, they recalled that the patient had eaten dried crude beef at home himself three days earlier.

Considering the eating raw meat habit of local herdsmen in the

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pasturing region of Qinghai-Tibet Plateau including Tibet and Qinghai, in addition to the previous case records, we presumed that the raw beef was contaminated by the spores of *Clostridium botulinum*, which may be responsible for the current case. The serum and stool samples collected on the next day after the appearance of the symptoms were sent for examination. It was not possible to obtain a stool sample due to constipation; therefore, fecal swabs (more than 1 swab for each sampling) were analyzed for toxigenic spores. Food samples were also sent for analysis.

On hospital day 3, the oxygen saturation suddenly dropped from 97 to 82%, and the patient was suffering from tachypnea, hypoxia, cyanotic lips and dysphonia, and was unable to respond to questions. Pupillary diameter was about 5 mm bilaterally with no light reflex. The blood pressure was 130/90 mmHg and the heart rate was 108/min. The laboratory tests revealed an increase of white blood cell (12.6×10^9/L), neutrophil percentage (90.5%), together with toxic granulation in neutrophil cytoplasm under light microscopy. Blood gas analysis showed a severe respiratory acidosis combined with metabolic acidosis. Considering respiratory muscle paralysis, prompt endotracheal intubation with mechanical ventilation was implemented. Sodium bicarbonate was given to correct acidosis. On hospital day 4, the patient became unconscious with incomplete right bundle branch block showing on the electrocardiogram. Hemoperfusion was implemented to improve internal body environment and norvancomycin hydrochloride was given. On hospital day 6, a food sample originally sent to the health department was reported to contain type E *C. botulinum* toxin. There was an improvement in the patient's condition in that he could write to express himself.

The pupillary white light reflex was sluggish. On lung auscultation, rales could be heard from both upper lobes and the breath sound over the right lower lobe disappeared. Chest radiography revealed right lower lobe pneumonia. Dexamethasone was given to reduce inflammatory response of pneumonia. The patient still could not eat and constipated. On hospital day 8, borborygmus could be heard with massive stool passage. Weaning the patient from the ventilator was delayed by the development of aspiration pneumonia but it was finally achieved on day 10. Up to hospital day 20, no recurrence of infection had occurred; the patient recovered and was discharged from the hospital.

DISCUSSION

Foodborne botulism is caused by eating preformed toxin produced in contaminated food. The most frequent source is home-canned foods and foods fermented or preserved with traditional methods in which spores that survive a lack of acidifier condition, inadequate cooking and storing process germinate, reproduce and produce toxin in the anaerobic environment of the food (Peck, 2006). For type E botulism, it was originally thought that was caused by fish or sea products in most countries where the disease was found (Dolman et al., 1963; Horowitz et al., 2010). In the 1930s, a new type of botulism caused by seafood was discovered in the Soviet Union and the United States of America. The cases of botulism were found to be caused by type E *C. botulinum* (Brola et al., 2013). Since then, cases have been reported in other parts of the world, and the implicated foods are primarily fish, fish eggs, and other types of seafood. However, the geographical distribution of *C. botulinum* type E and type E botulism in China are different from that in other part of the world. Cases of type E botulism primarily arise in the Qinghai-Tibet plateau of northwest China rather than in coastal regions. The foods implicated in the incidence of the disease are fermented beans as well as raw meat (Fu et al., 2008).

The diagnosis of foodborne botulism should be based on the patient's initial symptoms and history of food ingestion. The diagnosis may present difficulties, especially if it occurs as a sporadic case or if the relationship between foodstuff ingestion and symptom development is unclear. In the present case report, we describe a case of type E botulism associated with crude beef. The diagnosis was based on the patient's rapid deterioration and presentation of descending paralysis and was confirmed by laboratory isolation of *C. botulinum* from contaminated food. Botulism should be suspected if a patient simultaneously reveals acute onset of autonomic, cranial nerve and gastrointestinal dysfunction. The associated autonomic dysfunction includes urinary retention and dry mouth. Cranial nerve dysfunction includes diplopia, dysphagia and ptosis (Sobel, 2005). The gastrointestinal problems include nausea, vomiting and abdominal cramps (Armada et al., 2003; Lund, 1990). Fever and the abnormality of blood tests are not expected in a patient with botulism unless secondary infection occurs. Confirmatory tests of botulism involve tests for the detection of toxin in samples of food, serum or stool from patients. Associated diagnostic tests include serous toxin type identification by the mouse inoculation test (Aureli et al., 2000; Kalluri et al., 2003).

For years, the treatment of botulism has remained poorly standardized, particularly for the mild forms, which are by far the most common. In the very early stage of botulism, antitoxin therapy is suggested (ideally <24 h after the onset of symptoms). Treatment consists mainly in advanced supportive care with particular attention to respiratory status and administration of a botulinum antitoxin that can neutralize the free toxin molecules in the serum and prevent them from biding to nerve endings. Furthermore, a series of adverse side effects (for example, anaphylaxis, hypersensitivity reactions) are associated with antitoxin administration (Sobel, 2009). Antibiotics targeting the *C. botulinum* bacterium are also not recommended (Hatheway 1995). Early diagnosis and management rely on history and physical examination during botulism outbreaks.

Awaiting laboratory confirmation is a grave error, in particular, some mild cases probably occurred that did not result in a visit to a physician, and diagnoses may not have been made in some severe and even fatal cases (Sobel, 2009). Delay in the early period treatment may allow progression of paralysis, protracted hospitalization and deaths result from complications of long-term mechanical ventilation and ICU care. In conclusion, we present a case of type E botulism to alert clinicians to the possibility of correct clinical diagnosis and adequate treatment without antitoxin which may lead to successful manage-

ment of botulism.

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