A Foodborne botulism Occurrence in Mashhad: Clostridium botulinum in local cheese

Mohammad Hossein Kamaloddini¹, Hamid Reza Kheradmand²

¹Department of Toxicology, Imam Reza Hospital, Mashhad University of Medical Sciences, Mashhad, Iran
²Medical School, Mashhad University of Medical Sciences, Mashhad, Iran

Introduction

Foodborne botulism is caused by the anaerobic bacterial agent – Clostridium botulinum – that is a gram-positive bacteria (1) and one of the most common life-threatening agents in the United States, Europe and Iran (2-4). In 2011, 140 cases of botulism were reported to the US Centers for Disease Control and Prevention which 14% of cases were due to food-borne botulism (5). Seven types of clostridium named A-G are studied (6) in which A, B, E, and F are the main potent poisoning types in human that produce botulinum neurotoxin (BoNT). These types of clostridium get absorbed in GI after ingestion and may lead to morbidity such as paralysis and mortality, although their toxins are sensitive to heat (4,7). Mostly, foodborne botulism is associated with consuming canned tuna fish, local dairy products and home-preserved or commercial food. People use a good deal of local dairy products such as cheese and it exposes a variety of population to botulinum intoxication. It is indispensable for the physician to identify major features of foodborne botulism in order to differentiate it from other illnesses and early initiation of intensive care for intoxicated patients. To prevent the incidence of dairy product derived botulinum, it is firmly suggested to have quality control on local dairy products. Therefore, clinical findings are the principal key to the early diagnosis of botulism (2).

Case Presentation

A 48-year-old Caucasian woman referred to the emergency department complaining about acute dysphonia accompanied with symmetric hypotonia and bilateral ptosis. She had a history of consuming local dairy cheese. Based on clinical findings, the patient was admitted with the impression of botulinum intoxication. Botulism anti-toxin was administered. One vial of tetravalent botulism antitoxin was given intravenously as a 1:10 vol/vol dilution in 0.9% sodium chloride TDS. Since the most common cause of mortality is respiratory failure (17), supportive care was prepared and O₂ therapy, cardiac monitoring, pulse oximetry, and rapid sequence intubation equipment were considered and prepared in case of necessity. To rule out other probable causes, a neurology consult was requested. Blood and feces sample were taken and the probable occurrence of botulism was informed to the Health Center. The patient gradually became better. On the fifth day, the patient still had dysphonia and perioral paresthesia. She was hospitalized...
for 11 days and discharged after recovering and stable conditions. Tables 1 and 2 show laboratory data.

**Discussion**

In our case, early diagnosis and treatment were based on clinical findings obtained by history taking and physical exam. Data on history revealed the consumption of a suspicious dairy product and findings in the physical exam identified neuromuscular presentations such as the decreased force of limbs, dilated pupils and ptosis. Botulinum intoxicated patients most often do not present paresthesia (18) but in our case, the patient suffered from facial paresthesia. This finding is in line with other cases reported in the United States, San Francisco (19) and Atlanta, Georgia (20) especially in patients who were poisoned with BoNT type A or B.

It is important to differentiate botulism and other central nervous system deficits which may mimic botulism such as Guillain-Barre syndrome, Myasthenia Gravis and Lambert–Eaton myasthenic syndrome (3,9). These patients usually have fever, ataxia and elevated cerebrospinal fluid (CSF) protein, but they often have normal pupils (18,21). Central neural damages with asymmetric weakness should be considered in these patients. Ruling out central neurological damages can be performed by an appropriate module of radiologic imaging or electromyography/nerve conduction study. It is recommended to take samples from serum, stool and contaminated substances (22), so it would be possible to confirm the diagnosis.

**Conclusion**

It is essential to consider other possible etiologies with similar symptoms such as thallium, CO intoxication, and electrolyte abnormalities, foremost hypokalemia, and hyperkalemia which may present with dyspnea, palpitation, vomiting, and myalgia. Laboratory tests and imaging may help to exclude other diagnoses, but it is indispensable for the physician to identify major features of foodborne botulism in order to differentiate it from other illnesses, therefore, it would be possible to initiate treatment and supportive cares. Also, it is firmly suggested to have quality control on dairy products to prevent the incidence of botulinum intoxication.

**Authors’ contributions**

MHK conceived and planned the experiment and carried it out. Data acquisition and obtaining findings were done by HRK. HRK wrote the manuscript with support from MHK. All authors read and approved the final manuscript.

**Ethical Issues**

Ethical issues have been completely observed by authors.

**References**

8. Anniballi F, Chironna E, Astegiano S, Fiore A, Auricchio B,


