Confusion in Deglutition: A Case of Botulinum Toxin Ingestion*

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ABSTRACT

Purpose: To report on a case of dysphagia secondary to botulinum toxin ingestion.

Method: Case report

Result: A 30-year-old female with no comorbidities, presented with dysphagia associated with ptosis and diplopia 12 hours after ingestion of double dead pork. Probable diagnoses of a neurologic, esophageal problem or a neoplasm were initially considered. Ancillary procedures to support diagnoses were performed including esophagogastroduodenoscopy, cranial CT scan and Facial and Extremity Nerve Conduction Velocity which all revealed unremarkable results. A possible polyneuropathy specifically foodborne toxicity was considered given a history of dysphagia, ptosis and diplopia with consumption of double dead pork. Confirmatory stool culture studies revealed *Clostridium botulinum*, hence appropriate antibiotics and supportive therapy were provided which led to the patient's recovery.

Conclusion: Early diagnosis and a high index of suspicion is important in cases with unfamiliar presentations, therefore a careful history and physical examination is warranted. Foodborne botulinum toxicity is a public health matter that should be addressed. Proper food handling and storage must always be practiced.

Key words: botulism, foodborne botulism, deglutition disorders, dysphagia, public health

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INTRODUCTION

Dysphagia refers to difficulty in swallowing that may be caused by neurologic, congenital and developmental conditions, obstruction, muscular problems, psychologic causes or a combination of these. Foodborne botulism also presents with dysphagia and is clinically difficult to diagnose because of its extensive differentials. This report aims to remind physicians of a potentially fatal condition presenting with a nonspecific symptom as well as to promote safety and awareness of cases that are of public health concern that must be addressed immediately.

Case Report

A 30-year-old female had a two-day history of progressive dysphagia to both solids and liquids twelve hours after ingestion of pork. The pork was bought from a public market, noted to have an unusual foul stench but was still adequately refrigerated and thoroughly cooked after it was purchased. Associated symptoms include pooling of saliva and consequently, poor oral intake.

One day prior to consult, there was persistence of dysphagia with accompanying ptosis, diplopia, occasional difficulty of breathing and no bowel movement. There were no episodes of fever, headache, weakness, paralysis, numbness or change in sensorium. Persistence of symptoms prompted consult at the emergency room and was initially assessed by the otorhinolaryngology service.

On the day of admission, the patient was conscious, coherent and oriented, with no alteration in sensorium. Oral cavity examination showed no hyperemia, swelling or exudates. Initially, a probable retropharyngeal abscess or epiglottitis was considered as a cause of dysphagia. However, there was only pooling of saliva with no masses or swelling and fully mobile vocal cords. There was also absence of soft tissue swelling and widening of neck spaces on neck soft tissue lateral. Bilateral cranial nerve VI palsy as well as poor gag were noted on further examination. Manual Muscle Test showed 4/5 on all extremities and the patient had no sensory deficits and signs of meningeal irritation. Hence, the working impression was revised to Dysphagia secondary to 1) neurologic causes (cerebrovascular accident versus an intracranial mass) or 2) esophageal disorder.

On Cranial CT scan, there were no masses, hydrocephalus, acute infarct or hemorrhage. Esophagogastroduodenoscopy was essentially normal showing a distensible esophagus with good peristalsis.

Based on the aforementioned findings, a peripheral neuropathy was then considered, however, whether it was due to an immunologic (Miller-Fischer variant Guillain-Barre Syndrome, Myasthenia Gravis) versus a toxic (foodborne toxicity) cause was still in question. Findings on Nerve Conduction Velocity were inconclusive. It revealed normal results for both upper and lower extremities, orbicularis oculi showed low amplitudes and nasalis and frontalis muscles were nonreactive. On review of history, the patient experienced dysphagia after eating pork, with characteristics similar to those of double-dead meat. Physical examination findings such as bilateral nerve palsy and ptosis and ancillary tests which were negative for the aforementioned initial diagnoses supported the impression of a possible foodborne toxicity, particularly Botulism. Diagnosis was confirmed when stool culture revealed *Clostridium botulinum*, Final assessment was Dysphagia secondary to Acute Toxic Polyneuropathy (Foodborne Botulism); cerebrovascular disease, Myasthenia Gravis, Guillain Barre Syndrome ruled out. Benzathine Penicillin and Metronidazole were given to address the toxicity leading to gradual resolution of symptoms.

DISCUSSION

Foodborne botulism is a potentially life threatening condition although it is fairly rare[11]. Between 1993 and 2008, fifty-eight foodborne botulism outbreaks were reported to the Centers for Disease Control and Prevention. Four hundred sixteen foodborne botulism cases were documented, two hundred five (49%) were associated with outbreaks[12]. Last 2011, smoked seafood imported from the Philippines were recalled for potential contamination and following laboratory tests, products were eventually proven to be positive for botulinum toxin[15].

Ingestion of a preformed toxin produced in food by Clostridium botulinum causes foodborne botulism. A highly poisonous toxin is produced when food is contaminated by Clostridium botulinum before preservation. Incubation period for foodborne botulism can range from 2 hours to eight days after ingestion. Sources of the toxin include smoked meat, canned and preserved food. Severity of symptoms depend on the dose of bacteria or toxin ingested[3].

One source of foodborne botulinum toxicity is "botcha" or double-dead meat. It is meat taken from an animal who died from a disease then sold to consumers[13]. Among the illnesses that one can get from consumption of double dead meat are diarrhea, sepsis and even death.

The clinical picture of botulism toxicity shows an acute, afebrile, symmetrical and descending paralysis with no alteration in the level of consciousness, intact sensory functions and electrolyte values. More often, symptoms of diplopia, dysphagia, dizziness, blurred vision, dry mouth and constipation are present. In severe cases, botulinum toxicity may lead to respiratory failure from paralysis of muscles of respiration.

Differential diagnoses for foodborne Botulinum toxicity include: 1) Guillain-Barre Syndrome (GBS), specifically the Miller-Fischer variant which presents with ascending paralysis, loss of sensation, pain and triad of ophthalmoplegia, ataxia and areflexia[7]. GBS is diagnosed by an elevated CSF protein and abnormal nerve conduction and EMG studies, 2) Myasthenia gravis presents with fatigable muscle weakness and fluctuating proximal weakness[7]. Diagnosis is confirmed by Tensilon test and abnormal EMG and nerve conduction velocity test[6].

Detection of botulinum toxin in serum, stool or vomitus helps confirm the diagnosis of botulinum toxicity. Although findings are nonspecific, electrophysiologic tests such as nerve conduction studies and electromyelography may add to the diagnosis[4]. In foodborne botulism, nerve conduction studies yield normal results.

Primarily, supportive care, respiratory support and antitoxin administration is the current treatment for botulinum toxicity. Early application of botulinum antitoxin will not reverse symptoms but it can only limit the extent of paralysis. According to Swenson, JM, et al. Clostridium botulinum were tested by agar dilution for susceptibility to tetracycline, metronidazole,erythromycin, penicillin, rifampin, chloramphenicol, clindamycin, cephalothin, cefoxitin, vancomycin,sulfamethoxazole-trimethoprim, nalidixic acid, and gentamicin. At least 90% of the C. botulinum strains tested were susceptible to all drugs except sulfamethoxazole-trimethoprim, nalidixic acid, and gentamicin[5].

Consumers should be proactive in identifying double dead meat. Characteristics of pork that the consumers have to watch out for are the following: 1) meat is pale or taking on a bluish or greenish tinge, 2) meat smells bad with a stench stronger than the ordinary slaughtered meat, 3) not properly cleaned hair or skin, 4) meat easily crumbles, 5) sticky consistency[13].

Prevention of foodborne botulism can be achieved through good practice in food preparation. WHO recommends *5 Keys to Safer Food*: 1) keep clean, 2) cook thoroughly, 3) separate raw and cooked, 4) keep food at safe temperatures, and 5) use safe water and raw materials[9]. Intensive surveillance and response to foodborne botulism is warranted. It is the role of the general public to report cases of suspicious botulinum toxicity in order for the food item to be identified and removed from distribution and circulation without delay.

CONCLUSION

Foodborne botulinum toxicity is a serious condition that may often be missed as it presents with nonspecific symptoms. A careful history and physical examination as well as appropriate diagnostics are essential especially in cases with unfamiliar presentations. It is important to always keep botulism in mind when presented with patient with dysphagia. An early diagnosis and a high index of suspicion can be lifesaving.

Foodborne botulinum toxicity is a public health matter that should be addressed. Rapid recognition of source may prevent an outbreak from occurring. Strict regulations and screening of products that may be sources of the toxin should be vigilantly implemented. Prevention may be achieved through appropriate practices in food preparation and preservation which should be instilled to the public by information dissemination and education.

REFERENCES

1. Dembek, ZF, et al. (2007). *Botulism: cause, effects, diagnosis, clinical and laboratory identification, and treatment modalities*. Disaster Med Public Health Prep. 2007 Nov [cited 2 February 2017];1(2):122-34. Accessible from: https://www.ncbi.nlm.nih.gov/pubmed/18388640

2. Sobel, J. (2005). *Botulism.* Clin Infect Dis. 2005 Oct 15 [cited 2 February 2017];41(8):1167-73. Accessible from: https://www.ncbi.nlm.nih.gov/pubmed/16163636

3. Cardoso, T. et al. (2004) *Food-borne botulism: review of five cases*. Acta Med Port. 2004 Jan-Feb [cited 1 February 2017];17(1):54-8. Accessible from: https://www.ncbi.nlm.nih.gov/pubmed/15636728.

4. Anlar O. et al. (2003). *Food-borne botulism cases in Van region in eastern Turkey: importance of electromyography in the diagnosis*. Electromyogr Clin Neurophysiol. 2003 Sep [cited 2 February 2017];43(6):373-6. Accessible from: https://www.ncbi.nlm.nih.gov/pubmed/14535050

5. Swenson, JM. (1988). *Susceptibility of Clostridium botulinum to thirteen antimicrobial agents*. Antimicrob Agents Chemother. 1980 Jul [cited 2 February 2017];18(1):13-9. Accessible from: https://www.ncbi.nlm.nih.gov/pubmed/6998374

6. Lindstrom, M. et al. (2006*). Laboratory Diagnostics of Botulism*. Clin Microbiol Rev. 2006 Apr [cited 2 February 2017]; 19(2): 298–314. Accessible from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1471988/

7. Wenham, T. and Cohen, A. (2008). *Botulism*. Contin Educ Anaesth Crit Care Pain [Internet]. 2008 [cited 2 February 2017] 8 (1): 21-25. Accessible from: https://academic.oup.com/bjaed/article/8/1/21/277773/Botulism

8. Torii Y, Tokumaru Y, et al. *Production and immunogenic efficacy of botulinum tetravalent (A,B,E,F) toxoid*. Vaccine [Internet] 2002 [cited 2 February 2017].20(19-20):2556-61.

9. World Health Organization. *Botulism*. 2016 [cited 2 February 2017]. Available from: http://www.who.int/mediacentre/factsheets/fs270/en

10. Johns Hopkins University Bloomberg School of Public Health. *Botulinum Toxin* [Internet]. 2011 [cited 2 February 2017] Accessible from: http://www.jhsph.edu/research/centers-and-institutes/johns- hopkins-center-for-public-health- preparedness/tips/topics/Biologic_Weapons/botulinum.htm

11. World Health Organization [Internet]. Botulism [cited December 2016]. Available from: https://www.nlm.nih.gov/bsd/uniform_requirements.html

12. Newkerk, RW and Hedberg, CW. Rapid detection of foodborne botulism outbreaks facilitated by epidemiological linking of cases: implications for food defense and public health response. Foodborne Pathog Dis [Internet]. 2012 Feb [cited 2017 March 16]. Available from: https://www.ncbi.nlm.nih.gov/pubmed/22315952

13. Department of Health. Philippine Health Advisories. 2012 [cited 16 March 2017]. 117. Available from: http://www.doh.gov.ph/sites/default/files/publications/PhilippineHealthAdvisories2012.compressed.pdf

14. Berger, S. Infectious diseases of the Philippines-2017 edition. Gideon Informatics Inc. 2017 [cited 16 March 2017]: 36. Accessible from: https://books.google.com.ph/books?id=aj0WDgAAQBAJ&pg=PA36&lpg=PA36&dq=botulism+philippines+statistics&source=bl&ots=PsXYwecWDZ&sig=hEZmaJVml_koXpVv4wDGo PK5mw&hl=en&sa=X&ved= 0ahUKEwjBwreQ6NnSAhUBl5QKHeILDNI Q6AEIOzAF#v=onepage&q=botulism%20philippines%20statistics&f=false

15. promedmail.org [Internet].Oregon Botulism, Smoked Fish – USA: Risk, Recall, ex Philippines; c 2011 [cited 16 March 2017]. Accessible from: https://www.promedmail.org/post/20111105.3300