Journal of Clinical Toxicology
ISSN: 2161-0495

Habibiyan Nejad and Afshari, et al. J Clinic Toxicol 2011, 1:3
DOI: 10.4172/2161-0495.1000115

Research Article Open Access

Foodborne Botulism in Mashhad from 2003 to 2010

Habibiyan Nejad Z[1] and Afshari R[2]*

[1]Medical Toxicology Research Center, Imam Reza University Hospital, Ibne-e-Sina Street, Mashhad, Iran
[2]Pharmaceutical Rasearch center, Medical Toxicology Research Center, Mashhad University of medical Science, Mashhad, Iran

Abstract

Objectives: Foodborne botulism has been an uncommon disease in Iran. It induces symmetrical cranial nerve palsies followed by descending, symmetric flaccid paralysis of voluntary muscles, which may eventually lead to respiratory failure and death. This study aimed to investigate clinical and paraclinical findings, and long onset symptoms of botulism poisoning in Mashhad over an eight - year period.

Materials and methods: All selected files from hospital-referred botulism poisoned patients were investigated which were opted from Imam Reza (p) Mashhad University Hospital, Iran. Patients' clinical and paraclinical findings were noticed. In order to find out the period of time that patients have been free from any symptoms after discharging, all selected patients were contacted in early 2011. Cases were divided into two groups, namely sporadic and epidemic (more than one), and their findings were compared.

Results: Overall, 45 patients were selected. Mean incidence of Botulism in Khorasan-Razavi was 9.8×10^{5}. Among them 49.9% was Male. Main clinical findings were difficult speech (92%), ptosis (91%), diplopia (67%), nausea (58%), general weakness (92%), dizziness (70%) and constipation (25%). Patients were cleared from all symptoms and signs after discharging in 8.3 (9.6, 0-39) weeks. In outbreaks, time elapsed from exposure; headache and dysphagia were significantly different from sporadic cases.

Conclusion: Food borne botulism is rare, but it happens more frequently in middle-income countries such as Iran. The result of this study could be used as a clinical guideline to diagnose botulism in this area.

Keywords: Botulism; Paralysis; Food poisoning

Introduction

Food Borne Botulism is a rare disease which has been reported from Spain, Canada, Taiwan, China and Romania [1-4]. Few cases of food borne botulism are also reported from Iran [5-7].

Botulism was named after the Latin Botulus in 1820, although Clostridium Botulinum (CB) was first cultured by Van Ermengen in 1898 in Belgium [8]. CB is a gram-positive anaerobic bacterium [9]. The species C. botulinum is divided into four physiological groups (I-IV), which produce BoNTs of seven different serotypes (A-G). Physiological group I (proteolytic) includes C. botulinum strains producing toxins of serotype A, B or F and is mainly associated with human cases. Physiological group II (non-proteolytic) consists of strains producing toxins of serotype B, E or F; these strains also cause human botulism [10].

During the disease, nerve tissues collect botulinum toxin from blood which affect muscle nerve junction. As a result, inhibition of neural signaling and flaccid paralysis occur due to acetylcholine release inhibition [8]; however, sensory nerves are not affected [11].

Early symptoms of digestive poisoning with this bacterium include nausea, vomiting, abdominal pain and discomfort that occur between 12 hours to 4 days after food consumption [12]. Initial clinical manifestations include dry mouth, dysphagia, pupil dilatation, Diplopia and ptosis. During the clinical phase, a progressive, symmetric and descending muscle weakness could be observed and may lead to decreased muscle tone, urinary retention, weakness and dyspnea. Nerve palsy usually begins from III and IV central nerves. A decrease in deep tendon reflexes and constipation will then follow [16]. If untreated, respiratory failure could cause death [10].

Definite diagnosis of food botulism is possible via discovering botulinum toxin in serum, stool, gastric and intestinal contents or patient food, or noticing bacteria in the stool, stomach and intestinal contents [13]. It is also possible to inject the obtained substance to mice peritoneum and observe the consequences including paralysis or death [14]. However, these processes are time consuming [12]. In addition, in some outbreaks it is shown that toxin is undetectable in up to a third of cases [14]. Consequently, clinical findings play a crucial role in diagnosis. EMG is also useful for diagnosis [15]. CT scan and Lumbar puncture can exclude brain lesions and infections [10].

This study aimed to find clinical and paraclinical findings of botulism poisoning in Mashhad over an eight - year period.

Materials and Methods

Subjects and design

All hospital-referred botulism poisoned patients' files were screened retrospectively from 21 March 2003 to 20 March 2010 in Imam Reza Mashhad University Hospital in Iran. Data related to patients who were discharged with another diagnosis (six cases) were excluded.

Clinical samples were collected and testing to presence of BoNT toxin from all cases related to clinical symptoms.

***Corresponding author:** Reza Afshari, Associate Profesor of Clinical Toxicology, Pharmaceutical Research center, Medical Toxicology Research Center, Mashhad University of medical Science, Mashhad, Iran, Tel: 0098 511 852 5315; Fax: 0098 511 859 8973; E-mail: AfshariR@mums.ac.ir

Received September 14, 2011; **Accepted** December 18, 2011; **Published** December 22, 2011

Citation: Habibiyan Nejad Z, Afshari R (2011) Foodborne Botulism in Mashhad from 2003 to 2010. J Clinic Toxicol 1:115. doi:10.4172/2161-0495.1000115

In addition, to find the period of time that patients have been free from symptoms after discharging, all selected patients were contacted in early 2011. This study was approved by Ethics Committee, Mashhad University of Medical Sciences.

Spreadsheets were designed for clinical, para-clinical and socio-demographic variables. Patients were divided into two groups, namely sporadic if just one case admitted and outbreak if more than one related case was admitted. Clinical and para-clinical findings' of sporadic and epidemic botulism groups were compared. All patients but two due to hypersensitivity received antitoxin.

Diagnosis

Diagnosis was made based on history, clinical findings, respond to antidote and laboratory test for botulism.

Statistics

To analyze the obtained data t-test and chi-square were used capitalizing upon SPSS 11.5 for the statistical analysis. A probability value of less than 0.05 was considered statistically significant.

Results

Socio-demographic

In total, 45 cases with botulism were investigated. Incidence of Botulism in Khorasan-Razavi province with a population of almost 6 million (16) varied from 1.7×10^5 to 35.3×10^5 from 2003 to 2010 (mean 9.8×10^5). No gender preferences were observed (Male % =49.9). Mean age (SD) was 39.5 (17.0) years with a minimum of 3 and a maximum of 70 years. Among them no cases died under treatment. The mean (SD, Min-Max) lag time between exposure and admission was 40.2 (33.6, 2 to 110) hours. Duration of admission was 5.3 (7.1, 1-46) days. Patients were cleared from all symptoms and signs after discharging in 8.3 (9.6, 0-39) weeks. Frequency was not different in cases living in rural or urban areas. The total number of reported cases in 2006 and 2010 were higher than other years. These cases happened more frequently in autumn and early winter (Figure 1,2). Laboratory evaluation was positive for botulism in 44.4% of food and clinical samples. The found toxin subgroup was A and B.

Clinical findings

On admission, vital signs (pulse, blood pressure, respiratory rate and body temperature) were within normal ranges in 37 cases and 8 (18%) cases that presented with severe clinical manifestation and needed mechanical ventilation.

In sporadic cases, most common clinical symptoms were difficult speech (92%), ptosis (91%), diplopia (67%) and nausea (58%) on admission (Table 1).

General weakness (92%), dizziness (70%) and constipation (25%) were also the most frequent signs in this group on admission (Table 1).

Most clinical symptoms in outbreak group were dysphagia (94%), ptosis (91%), diplopia (87%) and difficult speech (74%) on admission (Table 1).

General weakness (84%), dizziness (80%) and constipation (47%) were also the most frequent signs in outbreak group on admission (Table 1).

There were two outbreaks of botulism in 2003 and 2010. In these cases, lag time between exposure and admission (P=0.026), headache (P=0.026) and dysphagia (0.002) were significantly different from sporadic cases. Other clinical and para-clinical findings were not significantly different between these two groups.

Discussion

Mean incidence was 0.98×10^6. In other reports their incidence was lower. For example, an incidence of 0.03×10^6 has been reported from Georgia, which increased in recent years [17]. This disease is far less common in the United States (0.001× 105) [18]. Reported case fatality rates vary in literature from 0% to 17% [17,19,20]. No deaths reported in this case series were similar to other studies [21,22]. Male to female ratio was rather similar in this study to Georgian, US and Tehran's studies [17,18,23]. Age range of patients was from 3 to 78 years (in Texas) [22] and 1- 66 years (from Tehran) which are similar to our findings [23].

Laboratory confirmation was more common in this study in comparison to Georgian study (17%) [17] and less common in comparison to Tehran study [23] . It should be taken into account that in many cases, laboratory confirmation is available in later stages. As a result, clinical manifestation and response to antitoxin could be more useful tools in diagnosis.

Lag time from exposure to admission in this study was 40.2 (33.6) hours, which are rather similar to other studies [19,24]. Patients were cleared from symptoms in 8.3 (9.6) weeks, which is rather shorter than some other studies [25].

Clinical findings including ptosis, general weakness and diplopia are the most common findings followed by dysphagia, difficult speech, dizziness, blurred vision and dry mouth in this study. This is rather similar to Meyers et al. and Kalluri's studies from USA [21,22]. However, in the present report dry mouth, constipation and mydriasis

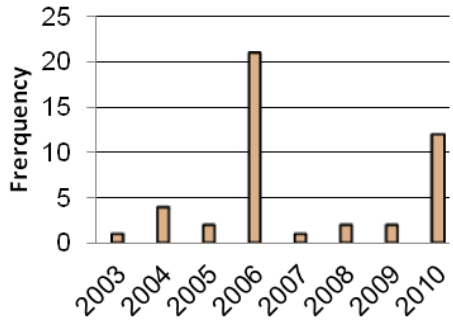

Figure 1: Frequency of cases in the period of study (n=45).

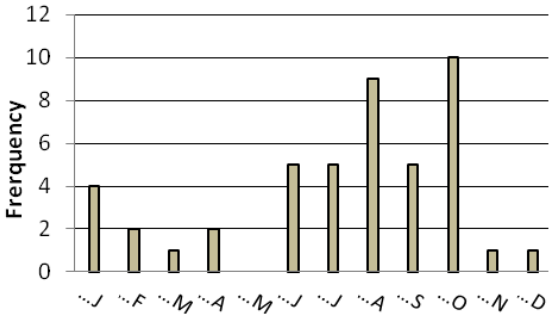

Figure 2: Frequency of cases in different months (n=45).

Signs (%)	Frequency				Vital Signs	Results Mean ± SD			
	All	Sporadic	Outbreak	P Value		All	Sporadic	Outbreak	P Value
General weakness	86	92	84	0.455	Systolic blood pressure (mmHg)	112 (18.4)	107.7 (18.2)	114.2 (18.5)	0.286
Dizziness	78	70	80	0.399	Diastolic blood pressure (mmHg)	71.5 (11.8)	73.3 (11.5)	70.8 (12.1)	0.536
Headache	51	20	62	0.026	Pulse rate (bpm)	88.3 (19)	89.9 (24.0)	87.8 (17.2)	0.748
Constipation	41	25	47	0.166	Respiration rate (bpm)	19 (5.3)	21.4 (6.9)	18.1 (4.4)	0.145
Symptoms (%)					Body temperature (c º)	37 (0.48)	37.1 (0.5)	37.0 (0.5)	0.371
Blurred vision	60	50	63	0.351	Paraclinical findings	Results Mean ± SD			
Dry mouth	71	39	65	0.104					
Nausea	57	58	56	0.588		All	Sporadic	Outbreak	P Value
Ptosis	91	91	91	0.733	Admission period (days)	5.3 (7.1)	5.1 (2.7)	5.4 (8.2)	0.907
Vomiting	25	33	22	0.339					
Diplopia	81	67	87	0.146	Onset symptoms period (weeks)	8.3 (9.6)	3.8 (6.2)	9.1 (10.0)	0.312
Dysphagia	81	46	94	0.002	Elapsed period(hours)	40.2 (33.6)	17.7	47.2	0.026
Difficult speech	79	92	74	0.204	CPK (U/L)	146.9 (122.5)	101.7 (50.0)	159.9 (134.6)	0.314
Inability to keep the neck	33	30	33	0.586	Na (meq/L)	138.3 (4.5)	138.4 (4.8)	138.3 (4.5)	0.894
Mydriasis	14	36	7	0.320	K (meq/L)	4.0 (0.4)	4.2 (0.5)	4.1 (0.5)	0.509
Drooling	11	15	10	0.467	Creatinine (mg/L)	0.85 (0.2)	0.9 (0.3)	0.4 (0.2)	0.582
Hoarseness	0	--	--	--					

Table 1: Frequency of signs and symptoms at the time of administration in all years study.

have been observed less frequently in contrast to Zilker report from Germany [19]. Severity of clinical manifestation may vary in different cases due to botulism [19], making it difficult to compare different reports.

Vital signs on admission in a report from East Coast of the United States were normal. Also clinical laboratory tests included potassium and creatinine were reported to be abnormal in 50% of cases [19]. This is different from our study.

Higher incidence of foodborne botulism in winter has been reported [17], which is different from the present study. As the total number of cases is limited in these studies, this could be a result of outbreaks in certain time of the year.

In this study 18% of cases needed mechanical ventilation on admission. This is proportionate to other reports (6% to 33%) [17,19].

In a comparison between sporadic and outbreak cases, all clinical findings but headache and dysphagia were similar.

Conclusion

In conclusion, foodborne botulism is rare, but it more frequently happens in middle-income countries such as Iran. Based on this study most signs and symptoms were in central nervous system that lead to misdiagnosis. On the other hand if there are more than one case the lag time between exposure and admission would be less due to earlier diagnosis. There is no significant difference between sporadic and epidemic groups to use for conclusion. The result of this study could be used as a clinical guideline for diagnosis of botulism in this area. If antitoxin is applied, foodbonre botulism is not fatal.

Acknowledgement

We would like to acknowledge the kind co-operation of Vice Chancellor for Research, Mashhad university of Medical Sciences.

Conflict of Interest

None to be declared.

References

1. Tejada García M, Guindel Jiménez C (2010) Botulism antitoxin treatment in two cases of food borne botulism. Farm Hosp 34: 47-48.
2. Loutfy MR, Austin JW, Blanchfield B, Fong IW (2003) An outbreak of foodborne botulism in Ontario. Can J Infect Dis 14: 206-209.
3. Zhang S, Wang Y, Qiu S, Dong Y, Xu Y, et al. (2010) Multilocus outbreak of foodborne botulism linked to contaminated sausage in Hebei province, China. Clin Infect Dis 51: 322-325.
4. Neghina AM, Marincu I, Moldovan R, Iacobiciu I, Neghina R (2010) Foodborne botulism in southwest Romania during the post-communism period 1990-2007. Int J Infect Dis 14: 96-101.
5. Afshari R, Shafiei S, Sayyahjezhad M, Dadras-Moghaddam D, Balali-Mood M (2007) A botulism outbreak following a wedding in a rural area in northeast Iran, 2006. Clin Toxicol 45: 388.
6. Vahdani P, Pourshafie MR, Aminzadeh Z (2002) Treatment of two unusual cases of type A and E botulism following consumption of salted fish. Intensive Care Med 28: 1189.
7. Pourshafie MR, Saifie M, Shafiee A, Vahdani P, Aslani M, et al. (1998) An outbreak of food-borne botulism associated with contaminated locally made cheese in Iran. Scand J Infect Dis 30: 92-94.
8. McLauchlin J, Grant KA, Little CL (2006) Food-borne botulism in the United Kingdom. J Public Health 28: 337-342.
9. Collins MD, East AK (1998) Phylogeny and taxonomy of the food-borne pathogen Clostridium botulinum and its neurotoxins. J Appl Microbiol 84: 5-17.
10. Skarin H, Håfström T, Westerberg J, Segerman B (2011) Clostridium botulinum group III: a group with dual identity shaped by plasmids, phages and mobile elements. BMC Genomics 12: 185.
11. Brett MM, Hood J, Brazier JS, Duerden BI, Hahné SJ (2005) Soft tissue infections caused by spore-forming bacteria in injecting drug users in the United Kingdom. Epidemiol Infect 133: 575-582.
12. Terranova W, Palumbo JN, Breman JG (1979) Ocular findings in botulism type B. JAMA 241: 475-477.
13. Cherington M (1998) Clinical spectrum of botulism. Muscle Nerve 21: 701-710.
14. Mac Donald KL, Cohen ML, Blake PA (1986) The changing epidemiology of adult botulism? like illness in chronic drug users. Am J Epidemiol 124: 794-799.
15. MacDonald KL, Cohen ML, Blake PA (1986) The changing epidemiology of adult botulism in the United States. Am J Epidemiol 124: 794-799.
16. Statistical Centre of Iran, 2006 Population and Housing Census, Administrative units of Razavi Khorasan and their populations.
17. Varma JK, Katsitadze G, Moiscrafishvili M, Zardiashvili T, Chikheli M, et al. (2004) Foodborne botulism in the Republic of Georgia. Emerg Infect Dis 10: 1601-1605.

18. Sobel J, Tucker N, Sulka A, McLaughlin J, Maslanka S (2004) Foodborne botulism in the United States, 1990-2000. Emerg Infect Dis 10: 1606-1611.

19. Zilker T (2006) Is Foodborne botulism realy the deadly disease we think 2006. Clin Toxicol 44: 473.

20. Swaddiwudhipong W, Wongwatcharapaiboon P (2000) Foodborne botulism outbreaks following consumption of home-canned bamboo shoots in Northern Thailand. J Med Assoc Thai 83: 1021-1025.

21. Meyers H, Inami G, Rosenberg J, Yuan J (2007) Brief Report: Foodborne Botulism from Home-Prepared Fermented Tofu -- California, 2006. MMWR Morb Mortal Wkly Rep 56: 96-97.

22. Kalluri P, Crowe C, Reller M, Gaul L, Hayslett J, et al. (2003) An outbreak of foodborne botulism associated with food sold at a salvage store in Texas. Clin Infect Dis 37: 1490-1495.

23. Aminzadeh Z, Vahdani P, Mirzaei J (2007) A survey on 80 cases of botulism and its clinical presentations as a public health concern. Iran J of Clin Infect Dis 2: 77-81.

24. Badhey H, Cleri DJ, D'Amato RF, Vernaleo JR, Veinni V, et al. (1986) Two fatal cases of type E adult food-borne botulism with early symptoms and terminal neurologic signs. J Clin Microbiol 23: 616-618.

25. Bacis G, Farina ML (2005) Botulism: It is Still Not Easy to Diagnose at the Beginning of the Twenty First Century. Clin Toxicol 43: 530.