TOXICOLOGY/ORIGINAL RESEARCH

Emergency Department Identification and Critical Care Management of a Utah Prison Botulism Outbreak

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Study objective: We report botulism poisoning at a state prison after ingestion of homemade wine (pruno).

Methods: This is an observational case series with data collected retrospectively by chart review. All suspected exposures were referred to a single hospital in October 2011.

Results: Twelve prisoners consumed pruno, a homemade alcoholic beverage made from a mixture of ingredients in prison environments. Four drank pruno made without potato and did not develop botulism. Eight drank pruno made with potato, became symptomatic, and were hospitalized. Presenting symptoms included dysphagia, diplopia, dysarthria, and weakness. The median time to symptom onset was 54.5 hours (interquartile range [IQR] 49-88 hours) postingestion. All 8 patients received botulinum antitoxin a median of 12 hours post-emergency department admission (IQR 8.9-18.8 hours). Seven of 8 patients had positive stool samples for type A botulinum toxin. The 3 most severely affected patients had respiratory failure and were intubated 43, 64, and 68 hours postingestion. Their maximal inspiratory force values were -5, -15, and -30 cm H₂O. Their forced vital capacity values were 0.91, 2.1, and 2.2 L, whereas the 5 nonintubated patients had median maximal inspiratory force of -60 cm H₂O (IQR -60 to -55) and forced vital capacity of 4.5 L (IQR 3.7-4.9). Electromyography abnormalities were observed in 1 of the nonintubated and 2 of the intubated patients.

Conclusion: A pruno-associated botulism outbreak resulted in respiratory failure and abnormal pulmonary parameters in the most affected patients. Electromyography abnormalities were observed in the majority of intubated patients. Potato in the pruno recipe was associated with botulism. [Ann Emerg Med. 2013;**E**:1-7.]

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INTRODUCTION

In 2010, there were 112 laboratory-confirmed cases of botulism in the United States. The majority of cases occurred in infants (76%);15% were wound botulism associated with intravenous drug abuse, and only 8% were food borne.¹ The incidence of food-borne botulism in the United States from 1982 to 2002 has been stable, at about 20 cases per year, with occasional outbreaks involving no more than 50 people per year. Of these, one third are caused by toxin type A.¹ Type A botulism toxin is associated with more severe and long-lasting paralysis, with reports of up to two thirds of patients requiring intubation. Before ICUs, the mortality rate was as high as 60% to 70%. However, with intubation and mechanical ventilation, mortality has decreased to 5% to 10%.²

Clostridium botulinum is an anaerobic, Gram-positive, rod-shaped, toxin-producing bacterium responsible for the botulism toxidrome.³ Botulism neurotoxin irreversibly blocks presynaptic terminal release of acetylcholine at the neuromuscular junction, causing flaccid muscle paralysis and eventual respiratory failure. Clinicians should suspect botulism if the patient demonstrates descending peripheral muscle weakness starting with bilateral cranial nerve deficits. Other common presenting symptoms of food-borne botulism include dizziness, blurred vision, dysarthria, and diplopia. Onset of symptoms is usually 12 to 36 hours after ingesting the bacterium, but can also appear within 6 hours to 10 days. Progression of symptoms can be stopped or slowed with early administration of the botulism antitoxin.⁴

Although the illicit manufacture of alcoholic beverages is practiced across the world, the prison setting is particularly challenging. The basic ingredients in brewing alcohol are sugar and yeast. In prison, pure sugar is often unobtainable so it is substituted with ketchup, fresh fruit, juices, and milk. Fruit is mandated to be present in prison cafeterias, making it nearly impossible to ban common pruno ingredients.⁵ Additionally, most prisons require inmates to work in the kitchen, where they have access to these ingredients.⁶

The term for jail-made alcohol varies geographically across the United States. It is known as "hooch" in Sing Sing Correctional Facility and "pruno" in San Quentin State Prison, whereas in other regions it is referred to as "white lightning" or "apple-jack."⁷ In the western United States, it is generally termed "pruno."

The most important step in the production of pruno is the fermentation of sugar into alcohol. This is performed in an anaerobic environment, which is also conducive to *C botulinum*

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Editor's Capsule Summary

What is already known on this topic

Food-borne botulism occurs sporadically throughout the United States. In prisons, illicit wine production is a recurrent cause of botulism outbreaks.

What question this study addressed

One incident in a state prison was investigated when wine ("pruno") ingestion was followed by several cases of botulism.

What this study adds to our knowledge

Prison wine made with potato resulted in 8 cases of botulism, whereas similar recipes made by the same prisoner without potato did not produce botulism.

How this is relevant to clinical practice

The development of cranial nerve deficits and descending paralysis in correctional care facilities should lead to consideration of botulism. The inclusion of potato in the pruno recipe was associated with botulism, but there are too few data to prove causation.

growth. Botulism outbreaks have been associated with pruno production in other prisons when potatoes have been used.⁸ The most recent pruno-associated botulism outbreak occurred in Arizona in 2012 in 2 separate incidents in the same prison.⁹

We report an outbreak of botulism poisoning in prison inmates, associated with homemade wine, who presented to an emergency department (ED).¹⁰ In addition, we explore the unique challenges in diagnosis and treatment of this potentially fatal infection.

MATERIALS AND METHODS

Study Design

This is an observational case series of all patients presenting to the ED who were state prisoners with suspected pruno ingestion during October 2011. A sentinel case was identified in the ED, and data were collected on this patient and all subsequent patients during hospitalization. Data were abstracted retrospectively from the electronic medical records by 2 emergency medicine residents (B.T.W., S.M.S.) and independently verified by an attending physician (E.M.C.). The abstractors were not blinded to the study, and an interrater assessment was not performed. Data collected were the amount ingested and time of ingestion, time to antitoxin administration, symptoms, physical examination results, neurologic examination results, laboratory data, respiratory parameters, electromyogram study results, interventions, and outcomes for each case. Physical examination findings were described as they were documented in the patient's chart. Time of ingestion was determined by patient report. We collected objective data about forced vital capacity, maximal inspiratory force, stool cultures, and electromyogram. All data were retrospectively obtained and clinicians were not blinded to the results of objective measurements. Two-month follow-up on all of the less-affected patients and 6-month follow-up on the most severely affected were obtained through chart review. Microsoft Excel for Mac 2011, version 14.3.8 (Microsoft, Redmond, WA) was used for data analysis.

Approval for this study was obtained from the institutional review board at University of Utah.

Setting

All patients were initially evaluated in the ED at the University of Utah Hospital, an urban 400-bed tertiary teaching hospital. The state prison has an independently functioning infirmary but transfers patients warranting a higher level of care to the ED.

Selection of Participants

A sentinel case was identified in the ED, and 11 subsequent patients were identified by state prison and Utah Department of Health investigations and emergency physician and neurologic critical care physician queries.

RESULTS

The sentinel patient presented complaining of weakness, diplopia, and dysphagia. Physical examination revealed weakness, ptosis, and dysarthria (Table 1, patient #7). This case prompted an immediate investigation by prison officials and the health department, which identified 11 additional potential exposures to pruno, and these patients were referred to the University of Utah Hospital ED for evaluation during the following 48 hours.

A total of 12 inmates presented to the ED with a history of consuming pruno within 4 days of presentation. The amount consumed varied greatly per patient report, with some patients reportedly consuming more than 2 gallons. Four patients consumed a batch that did not contain a potato in the recipe and were either asymptomatic or had symptoms not consistent with acute botulism toxidrome and were discharged. The remaining 8 patients consumed pruno that included potato and presented to the ED at a median time to symptom onset of 54.5 hours from ingestion (range 39 to 89 hours; interquartile range [IQR] 49-88 hours). Both batches of pruno were made by the same prisoner, who confirmed that one batch was made with a potato and one without. All 8 of these patients were admitted and received botulinum antitoxin, with a median time of administration post-ED arrival of 12 hours (range 7 to 23.75 hours; IQR 8.9-18.8 hours) and a range time to administration after ingestion of 72 to 99 hours (median 73.3 hours; IQR 62.6-96.5 hours) approximately 3 days postexposure. The patients were all men, with a median age of 28 years (range 24 to 36 years; IQR 25.5-32.5 years).

Patient symptoms and physical examination findings are presented in Table 1. The most common presenting symptom

	Table 1.	Signs and symptom	is on initial evaluat	ion and follow-up in	patients ingesting pruno
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Patient	Symptoms on Arrival to ED	Initial Physical Examination Findings	Signs and Symptoms at Follow-up
1	Dysphagia	ED examination: no focal neurologic deficits Neurology examination: no focal neurologic deficits	2 month: dysphagia, dysarthria, fatigue, unable to stand in plantar flexion, reduced strength with left eye closure
2	Blurry vision, dizziness, fatigue, nausea, vomiting, diarrhea	ED examination: weak gag Neurology examination: no focal neurologic deficits	2 month: weakness and fatigue
3	Fatigue, blurry vision	ED examination: no focal neurologic deficits Neurology examination: no focal neurologic deficits	2 month: weakness and fatigue
4	Blurry vision	ED examination: No focal neurologic deficits Neurology examination: right lateral rectus weakness causing diplopia	1.5 month: weakness, fatigue, left psoas weakness
5	Dysphagia, dysarthria	ED examination: no focal neurologic deficits Neurology examination: Nystagmus on lateral gaze bilaterally, mild dysarthria, saccades with extraocular movements, decreased Achilles reflex on left	2 month: dysphagia, blurred vision, weakness 5 month: weakness, diplopia, and dysphagia resolved
6	Dysphagia, dysarthria	ED examination: No focal neurologic deficits Neurology examination at 5 AM: no focal neurologic deficits Neurology examination at 5 PM: acute change: bilateral facial weakness, dysphagia, 1+ reflexes, respiratory distress	2 month: dysphagia with liquids, fatigue, constipation, weakness6 month: improved dysphagia and weakness but not back to preingestion status
7	Weakness, diplopia, dysphagia	ED examination: bilateral ptosis, weak in all distributions, dysarthria/slurred speech Neurology examination: bilateral ptosis, multiple weak cranial nerves, dysphagia, severe dysarthria, disconjugate gaze	2 month: weakness, dysphagia, diplopia4 month: weak, dysphagia8 month: persistent dysphagia and weakness; diplopia resolved
8	Diplopia, dysphagia, dysarthria	ED examination: no focal neurologic deficits Neurology examination: nystagmus on lateral gaze, mild dysarthria and dysphagia	 1.5 month: no gag reflex, areflexia, and weakness still needing physical therapy, tracheostomy still in place 5 month: weak, diplopia, dysphagia, 9 month: weak, dysphagia, ptosis 14 month: weakness, dysphagia, and diplopia resolved

was dysphagia (5 of 8 patients). On physical examination, ocular symptoms predominated, with 4 patients (50%) having at least 1 abnormal eye examination finding. At 2-month follow-up from ingestion, the most common symptoms were dysphagia and weakness. Table 2 lists the clinical characteristics of the 3 most severely affected patients.

All 8 patients had stool obtained for culture of *C botulinum*. Stool was placed in standard fashion on gel medium and allowed to grow and was identified as *C botulinum*. Toxin was identified as type A through the mouse inoculation test, which is considered to be the most accurate.¹¹ Seven of 8 patients had a positive stool culture result associated with type A botulism toxin.

Three patients were intubated for impending respiratory failure. They exhibited lower maximal inspiratory force and forced vital capacity values than predicted by their age, height, and sex (Table 3). The median maximal inspiratory force for intubated patients at respiratory distress was $-15 \text{ cm H}_2\text{O}$ (range $-30 \text{ to } -5 \text{ cm H}_2\text{O}$; IQR $-30 \text{ to } -5 \text{ cm H}_2\text{O}$), and the median forced vital capacity was 2.1 L (range 0.91 to 2.21 L; IQR 0.91-2.21 L). The initial values for 2 of the 3 intubated patients were different from the values at respiratory distress, and the 5 patients who were not intubated had normal respiratory parameters (Table 3). The median maximal inspiratory force for nonintubated patients was $-60 \text{ cm H}_2\text{O}$ (IQR $-60 \text{ to } -5 \text{ cm H}_2\text{O}$), and the median forced vital capacity was 4.5 L (IQR 3.75-4.9L).

Electromyogram studies were performed and were assessed for neuromuscular junction abnormalities. Two of the 3 patients with respiratory failure had evidence of neuromuscular dysfunction consistent with acute botulism (Table 3). One patient without respiratory failure demonstrated similar

Table 2.	Clinical	characteristics	of	the	most	severely	affected
patients.							

Patient	Characteristics
6	Intubated on hospital day 0
	Extubated on hospital day 6
	Discharged on hospital day 10
7	Intubated on hospital day 0
	Tracheostomy and PEG on hospital day 9
	Tracheostomy removed on hospital day 19
	Discharged to prison infirmary on hospital day 27
8	Intubated on hospital day 0
	Tracheostomy and PEG; profound ileus on hospital day 4
	Only able to walk 30 ft; still PEG tube fed with tracheostomy on hospital day 31
	Absent reflexes; able to walk 60 ft; still tracheostomy and PEG tube fed on hospital day 49
	Tracheostomy removed; PEG tube feeding continued on hospital day 80
	PEG tube out on hospital day 89

PEG, Percutaneous endoscopic gastrostomy.

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Table 3. Clinical data from patients.

Patient	Days After Exposure Antitoxin Administered	Maximal MIF, cm H ₂ O	Maximal FVC, L	Stool Culture Results	EMG Results
1	4	>-60	4.7	Negative	Normal, no evidence of dysfunction
2	5	-60	3.25	Positive	Normal, no evidence of dysfunction
3	5	-60	5.1	Positive	Normal, no evidence of dysfunction
4	5	-50	4.5	Positive	Evidence of NMJ conduction delay
5	5	>-60	4.25	Positive	Normal, no evidence of dysfunction
6	4	>-60, -30*	3.7, 2.1*	Positive	Normal, no evidence of dysfunction but patient was intubated
7	4	-15	0.91	Positive	Evidence of NMJ conduction delay
8	4	-30, -5*	4.7, 2.2*	Positive	Evidence of NMJ conduction delay
MIF. Maxima	al inspiratory force: FVC. forced vita	I capacity: EMG. electro	mvography: NMJ, neuromu	scular junction.	

MIF, Maximal inspiratory force; FVC, forced vital capacity; EMG, electromyography; NMJ, neuromuscular junction.
*MIF and FVC were repeated because of a change in the patient's clinical status.

electromyogram abnormalities. Table 3 compares clinical data of all 8 patients that was thought to be helpful in identifying factors associated with the need for intubation.

All 8 hospitalized patients, including the patient with negative stool study results, had persistent symptoms on follow-up evaluation 2 months postexposure, and the most severely affected patients had persistent findings up to 14 months postexposure. The most common persisting symptoms were weakness, fatigue, diplopia, and dysphagia, with nearly all patients reporting these symptoms at follow-up (Table 1). The persistence of symptoms appears to correlate with the severity of poisoning, with the 3 intubated patients showing symptoms longer.

LIMITATIONS

Limitations of this article include that none of the patients were able to provide an exact time or quantity of pruno consumption. Also it is difficult to obtain follow-up data on this patient population. The prison policy avoids regularly scheduled appointments to discourage inmates from planning an escape, which makes consistent follow-up difficult. It is impossible to know when some of these inmates were released from prison, which prevented us from obtaining additional follow-up data. This may have provided further information about the duration of symptoms.

Additionally, this is a small case series that is not generalizable. Because of our small sample size, reliable conclusions cannot be made. It is an observational case series, thus limiting access to specific data. The 4 patients who presented with ingestion of the second batch of pruno did not have symptoms consistent with acute botulism and therefore did not have any botulism testing.

1 two-week old baked potato; stored in a closed jar then cleaned with soap and
water
4 skinned grapefruit rinsed before peeling
4 canned oranges rinsed before peeling
4 peeled fresh oranges
4 canned pears
4 canned peaches
4 cups of water
2 12 oz bags of powered juice mix
Store in a plastic sealed bag and let ferment approximately 7 days

Figure. Pruno recipe.

It is conceivable that some patients were exposed without traditional clinical symptoms.

Finally, our article is limited by methodology of chart review. We used trained emergency medicine residents as abstractors, with attending toxicologist oversight and a standard clearly defined spreadsheet for data collection. However, our abstractors were not blinded to the study, and interrater assessment was not performed.¹²

DISCUSSION

This is a case series of 12 prisoners who arrived to the ED and initially withheld the fact that they had recently consumed pruno. The sentinel patient received a diagnosis in the ED, with a chief complaint of weakness. Together with identification of his bulbar findings and revisiting the history, the diagnosis of acute botulism poisoning was made for 8 of the 12 patients. All 8 of these patients drank the batch of pruno that contained a potato.

This incident and a 2012 outbreak in Arizona represent the largest food-borne botulism outbreaks since 2006 and the largest reported botulism outbreaks associated with pruno ingestion. Since 2004, the Centers for Disease Control and Prevention (CDC) has reported 5 separate pruno-associated botulism outbreaks occurring in prisons: 2 in California, 1 in Utah, and 2 in Arizona.^{9,10}

The production of pruno assists in the selection of Cbotulinum as a primary bacterium and increases the concentration of the botulinum toxin. There are several pruno "recipes" available on the Internet. The basic steps are to place a variety of fruit, liquid, and other ingredients into a sealed plastic bag that is allowed to ferment. Pruno is a low-acidity solution that requires a sealed container to create the anaerobic environment necessary to produce alcohol. The brew is left sealed and undisturbed to allow further alcohol fermentation, which concomitantly allows for toxin production. The pruno recipe involved in this outbreak (Figure) was provided by patient 4, who reportedly had cooked this recipe approximately 20 times previously without a potato. The prisoner's rationale behind using a potato was that he thought it would "accelerate fermentation," and he was "experimenting." Potatoes were also used in the contaminated batch in the California report; however, it is unclear whether they were used in the Arizona outbreak pruno recipe, which yielded a

high percentage of intubated patients (7 of 8).⁸⁻¹⁰ A review of the recipe reveals that there are multiple ingredients and factors that would assist in the formation of *C botulinum* toxin. First, the baked potato was stored in a jar for more than 2 weeks at room temperature. It was postulated by Angulo et al¹³ that baking a potato kills competing bacteria but is insufficient to destroy *C botulinum* spores. Room temperature is a viable temperature for botulism spores to germinate and produce toxin.³ The pruno was then placed in a sealed container, which created an anaerobic environment that likely further allowed spore development and bacteria multiplication.¹⁴

The recipe (Figure) contains a high percentage total volume of fruit and sugar, with a paucity of water. A higher sugar and lower water percentage is thought to inhibit botulism toxin formation and spore germination.⁶ It appears that the addition of an old baked potato along with fruit, sugar, and water is responsible for the botulism outbreak in our case series.

Potatoes have been linked to numerous food-borne botulism outbreaks, including a Texas outbreak in 1994 caused by contaminated Skordalia, a potato-based dip, and a crosscontaminated eggplant dip, with 24 confirmed cases.¹⁵ In 1978, 7 cases of type A botulism occurred in persons who had eaten potato salad in a restaurant in Colorado.¹⁶ A 1993 outbreak in Georgia related to contamination of a canned chili cheese sauce was discovered after patients were misdiagnosed with labyrinthitis, transient ischemic attack, allergic reactions, mild glaucoma, and astigmatism. This outbreak was ultimately linked to botulism spores in leftover uncooked potatoes.¹⁷ Angulo et al documented an additional 5 outbreaks linked to potatoes that resulted in more than 50 affected individuals. Potatoes are an excellent source for botulism because they are grown underground, in contact with C botulinum spores. Potato skins are rough and porous, allowing spores to adhere well to their surface; thus, potatoes often contain C botulinum on their peels.¹⁸

We collected objective data measuring the severity of symptoms by comparing forced vital capacity, maximal inspiratory force, stool cultures, and electromyogram results. Both maximal inspiratory force and forced vital capacity are prognostic markers of respiratory function.¹⁹ When the neuromuscular junction is impaired by botulism toxin, the patient will not be able to generate large maximal inspiratory force or forced vital capacity. The data from this outbreak suggest that more severely affected patients had consistent abnormal changes in forced vital capacity and maximal inspiratory force.

The role of electromyogram studies in the evaluation of *C botulism* toxidrome is less clear. Angulo et al¹³ noted similar inconsistent findings with electromyograms, with one asymptomatic patient having an abnormal electromyogram result indicative of botulinum toxin. Electromyogram studies could provide objective data that may help in the diagnosis of the toxidrome, but the sensitivity and specificity of this test remains unknown.

It is unclear why the median onset of symptoms in our patient group was longer than in other documented cases. The 1994 Texas outbreak reported an onset of symptoms at 35 to 40 hours and 25 hours for the individuals requiring intubation.¹⁵ In our series, the median time to onset of symptoms was 54.5 hours. Perhaps the amount of spores ingested was less concentrated in this liquid ingestion compared with the ingestion described in the Texas outbreak. Additionally, there is also uncertainty in the actual time of ingestion as reported by the patients.

The keys for diagnosis of botulism are a high index of suspicion for food-borne botulism poisoning in patients who present with bulbar weakness in association with ingestion of pruno or other foods such as potatoes that have contact with soil and possibly botulism spores. Symptoms may be highly variable and progress rapidly. For example, in our series some patients had nonspecific symptoms such as blurry vision and gastrointestinal complaints and had difficult-to-assess physical findings such as a weak gag reflex or diplopia. The importance of a thorough neurologic examination is of utmost importance in these patients. Because diagnosis by stool study can take up to 4 days, maintaining a high degree of suspicion is paramount to start the process of procuring antitoxin and treating the patient.²⁰ In addition, monitoring the patient's airway is of critical importance because the need for mechanical intubation in patients who are botulism toxic is variable.

Unfortunately, the confirmation of botulism is time intensive and the available tests are often inaccurate. According to the CDC botulism handbook, stool culture results are positive in 51% of patients with botulism, and serum toxin testing is less sensitive, at 37%.⁴

Antitoxin therapy is more effective if administered early in the course of illness because it binds only toxin not yet bound to the neuromuscular junction.^{21,22} The antitoxin does not reverse paralysis but prevents further clinical deterioration. Food-borne botulism is the result of consuming preformed toxin, meaning there is no continual production of toxin after initial ingestion. Therefore, the longer the delay to administration of antitoxin, the longer and more severe the course for the infected individual.

As of March 2010, the bivalent botulism antitoxin and the monovalent antitoxin were replaced with a heptavalent botulism antitoxin. The bivalent antitoxin could neutralize only botulism toxins A and B and the monovalent antitoxin could neutralize toxin E. The heptavalent antitoxin can neutralize all seven *C botulinum* toxins (A through G). The heptavalent botulism antitoxin is relatively new, and there are few data on its half-life or effectiveness. It is equine derived, which carries an inherent risk of anaphylaxis.²³

Of particular interest to the emergency physician is the acquisition process and administration of the *C botulinum* antitoxin to a patient. The CDC is the only source for botulism antitoxin, but there are multiple stockpiles throughout the United States of antitoxin in case of bioterrorism attack.²⁴ To acquire the antitoxin, first call your state health department's on-call provider. They will contact the CDC and report a possible botulism outbreak. The CDC will arrange a clinical consultation to determine whether to release antitoxin. The antitoxin is then released and arrives rather quickly.²⁴ In our case, once the diagnosis was suspected, the admitting attending physician and infectious disease physician from our hospital contacted the Utah Department of Health, which contacted the

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CDC to release the antitoxin. Our infectious disease pharmacist went to the airport to directly obtain the antitoxin after it was flown from Atlanta to Salt Lake City. This entire process from suspicion of diagnosis to administration of the antitoxin to the sentinel patient took approximately 9 hours. The CDC recommends treating first if botulism is suspected rather than waiting for confirmatory testing.

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Editor's Capsule Summary What question this study addressed: One incident in a state prison was investigated when wine ("pruno") ingestion was followed by several cases of botulism. What this study adds to our knowledge: Prison wine made with potato resulted in 8 cases of botulism, whereas similar recipes made by the same prisoner without potato did not produce botulism.