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An Asymmetric Onset of Neurological Signs Does Not Rule Out the Botulism

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Abstract

Foodborne Botulism is clinically characterized by a symmetric flaccid paralysis of the cranial nerves with a descending involvement of voluntary and breathing muscles leading to respiratory arrest. Asymmetric neurological signs are unusual and in these cases diagnosis could be delayed or frequently missed. We described a 63-year-old man with a clinical picture related to type A botulism characterized by an asymmetric and left lateralized onset of neurological signs associated with a monolateral parotitis. Physicians must be aware that lateralized onset of neurological signs does not rule out the botulism, and it should be considered even in cases of atypical clinical picture.

Introduction

Foodborne Botulism is a rare disease caused by accidental or intentional exposure to botulinum toxins, characterized by a symmetric flaccid paralysis of the cranial nerves with a descending involvement of voluntary and breathing muscles leading to respiratory arrest and death [1]. Asymmetric clinical signs are unusual for botulism that is typically characterized by a symmetric flaccid paralysis of the cranial nerves. Ptosis, diplopia, blurry vision, and inability to accommodation are very frequent among first symptoms, followed by expressionless facies, dysphagia, lowering of the voice tone and dysarthria, with a descending involvement of voluntary and breathing muscles leading to respiratory arrest and death over hours to days, with a rate apparently proportional to the dose [1]. Therefore, a prompt clinical diagnosis is mandatory to effective treatment [1,2]. In the setting of an outbreak, the diagnosis readily suggests itself. Nevertheless, sporadic cases with atypical clinical features were also reported, and in these cases the diagnosis could be delayed or frequently missed because other causes are usually considered before [1]. Here, we report a new and rare case of foodborne botulism with an atypical clinical presentation.

Case Report

We observed a 63-year-old man who had presented diplopia a few hours before due to left sixth cranial nerve palsy. It was followed by left ptosis after three days and lowering of his own tone of voice and solid food difficult swallowing on the fifth day. On the sixth day, he complained dry mouth, constipation, and pollakiuria. The physical examination documented asymmetric ptosis (left>right), palsies of the left sixth and third cranial nerves, asymmetric mydriasis (left>right) with sluggish bilateral pupillary response to light, and a mild reduction of facial mimicry. On the seventh day, while the neurological signs were remaining almost stable, the patient presented with a swelled and painful left parotid gland that an ultrasound scan defined as an inflammatory process. Serology tests and CSF examination were negative. However, a treatment with a wide spectrum antibiotic and corticosteroid was started. Brain MRI was normal and electromyography did not show significant decrement upon 30 Hz-stimulation. On the eighth day there was an impairment: the ocular palsies became bilateral and almost complete, bilateral facial palsy became evident and constipation became severe (Figure 1). However, the patient never required intubation and mechanical ventilation. His food history was revaluated since the patient had previously denied having eaten contaminated food. In that occasion, the patient remembered eating, about ten days before admission, domestically canned spinach, boiled and then preserved in a jar. Therefore, botulism was actually suspected and immediately it was reported to our poison control center and to the national health authorities using the routine mandatory notification system for the disease. Serum and fecal samples from the patient were analyzed by the national reference laboratory for anaerobic bacteria and botulism at the National Institute of Health in Rome, Italy. Samples recovered from the food were no more available. Subsequently, the diagnosis of botulism (toxin type A) was confirmed by detection of Clostridium botulinum in a fecal sample. Botulinum toxin wasn't found in a blood sample. The patient didn't receive serum botulism antitoxin because too much time (more than 15 days) had passed from the food intake. No other botulism cases associated with this episode were identified in the family because the patient was the only one who had eaten the contaminated food. He was treated with cathartic and gradually recovered: parotid gland deflated and neurological palsies improved. Complete recovery of nearly all symptoms was observed within seven weeks.

Discussion

Atypical cases of botulism were rarely reported. Reviewing through a medline search other similar clinical pictures (incorporating the terms atypical botulism, asymmetrical botulism or botulism), only few cases were found [2-6], and, particularly, an asymmetric clinical presentation has been described in 8-17% of patients who had respectively an asymmetric ptosis or an asymmetric extremely weakness [2]. We reported a new case of type A botulism characterized by an asymmetric and lateralized onset that made botulism diagnosis unlikely. Recently, a similar case characterized by a strictly asymmetric left lateralized onset with slow progression to descending paralysis associated with demyelination of cranial nerves was reported by Filozov et al. [6], but in that case it was due to a type F toxin. Moreover, in our case the onset was not only asymmetrical, but confined to the left side during the first seven

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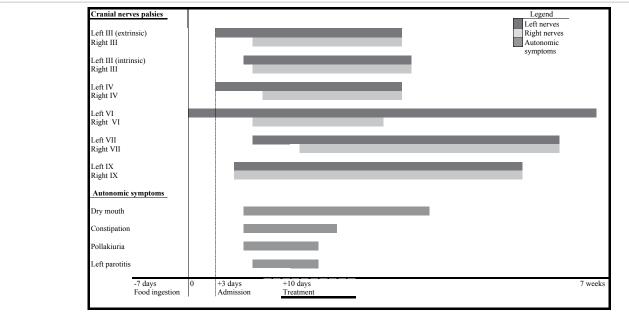


Figure 1: Clinical course of the botulism case.

days, both for the cranial nerves and the secretory palsy of the parotid gland. Parotitis is reported to be a possible, although rare, complication of botulism due to a paralytic secretion of the salivary glands, but in literature only one case of monolateral parotitis has been described [5].

Lastly, in our case symptoms appeared not abruptly but slowly within about 14 days since the contaminated food ingestion. A careful food history is usually fundamental for the diagnosis, but our patient denied having eaten contaminated food and remembered to have eaten the contaminated food only later. On the other hand it is important for the clinician to solicit this information as soon as possible. In fact, in the setting of an outbreak of several people the diagnosis readily suggests itself, even if with some typical features. Conversely, for sporadic cases with atypical clinical features, the diagnosis could be not easy and delayed as other causes are usually considered before (i.e. Guillain Barré Syndrome, myasthenia gravis, stroke syndromes, Eaton-Lambert syndrome, tick paralysis) [1,2,6].

Drawing conclusions, a lateralized onset of neurological signs does not rule out the botulism, and physicians must be aware that it may also occur with a clinical picture atypical and asymmetric.

Disclosure

The authors have no conflict of interest to declare.

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