Clinical Case of Atypical Botulism with Pseudointernuclear Ophthalmoplegia Syndrome

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Abstract

Botulism is a rare cause of bulbar and oculomotor syndromes. A late diagnosis and, therefore, late initiation of specific therapy may lead to multiple life-threatening complications. Epidemiological history and clinical findings are key to the correct diagnosis, but if these data are not available due to atypical clinical findings, botulism identification is challenging.

In our clinical case, a 31-year-old man was admitted to the hospital with double vision, impaired eye movements, and difficulty swallowing rapidly developing for 2 days. Ocular motility dysfunction included disturbed conjugate eye movements. In young patients, this is most often caused by demyelinating disease with medial (posterior) longitudinal fasciculus damage and symmetrical bilateral ptosis. The patient denied eating foods that could cause botulism and did not have any gastrointestinal symptoms. Differential diagnoses included demyelinating disease onset and Miller–Fisher syndrome. The next morning, completely identical clinical signs appeared in the patient's mother who had eaten canned mushrooms, so botulism was suspected. Over the next few hours, despite the administration of anti-botulinum serum, acute respiratory failure developed, and the patient was placed on a ventilator for 28 days. The patient and his mother were discharged in a satisfactory condition, and their symptoms completely resolved within a few months. The diagnosis of botulism was confirmed by toxicological examination.

Keywords: *botulism; myasthenia gravis; bulbar syndrome; internuclear ophthalmoplegia; ptosis; Miller–Fisher syndrome*

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Клиническое наблюдение ботулизма с атипичным течением и развитием синдрома псевдомежъядерной офтальмоплегии

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Аннотация

Ботулизм является редкой причиной развития бульбарного и глазодвигательного синдромов. Несвоевременно установленный диагноз и, соответственно, поздний старт специфической терапии может привести к многочисленным жизнеугрожающим осложнениям. Ключом к верному диагнозу служат эпидемиологический анамнез и клиническая картина, однако при отсутствии этих данных или атипичности клинических проявлений картины постановка диагноза затруднительна.

В описанном клиническом случае 31-летний мужчина поступил в клинику с острым развитием двоения, нарушением движений глаз и затруднением глотания в течение 2 дней. Дисфункция со стороны глазодвигательных нервов характеризовалась нарушением сочетанных движений глазных яблок, которая чаще всего у молодых пациентов обусловлена демиелинизирующим заболеванием с поражением медиального (заднего) продольного пучка, а также симметричным двусторонним птозом. Пациент отрицал употребление в пищу продуктов, способных вызвать ботулизм, признаки гастроэнтерического синдрома отсутствовали. Дифференциальный диагноз проводился между дебютом демиелинизирующего заболевания и синдромом Миллера–Фишера. Утром следующего дня полностью идентичная клиническая картина возникла у матери пациента, которая употребляла в пищу консервированные грибы, на основании чего был заподозрен ботулизм. В течение последующих нескольких часов, несмотря на введение противоботулинической сыворотки, развилась острая дыхательная недостаточность, ввиду чего пациент был переведён на искусственную вентиляцию лёгких, длительность которой составила 28 сут. Пациент и его мать были выписаны в удовлетворительном состоянии с полным регрессом симптоматики в течение нескольких месяцев. Диагноз «ботулизм» был подтверждён токсикологической экспертизой.

Ключевые слова: *ботулизм; миастения; бульбарный синдром; межъядерная офтальмоплегия; птоз; синдром Миллера–Фишера*

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Источник финансирования. Авторы заявляют об отсутствии внешних источников финансирования при проведении исследования.

Конфликт интересов. Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи.

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Introduction

Botulism is a serious life-threatening disease with neurological symptoms being key ones, and, therefore, patients often visit or are referred to a neurologist. Typical botulism is diagnosed on the basis of epidemiological history and characteristic clinical pattern.

Foodborne botulism accounts for more than 99% of all botulism cases[1]. Typically, patients eat home-cooked canned foods such as mushroom, meat, fish, or vegetables. If having suitable conditions for reproduction in an anaerobic environment, the bacterium called *Clostridium botulinum* produces toxins that

[1] Botulism in children: Clinical guidelines (approved by the Ministry of Health of Russia in 2021). URL: https://cr.minzdrav.gov.ru/schema/697_1 (assessed: 07/28/2024).

cause botulism. The bacteria get into contaminated products due to their poor processing; botulinum toxin is odorless, colorless, and tasteless, making it impossible to identify, especially if the cans with the product are not swollen [1]. There are several types of botulinum toxins. Serotypes A, B, and E are the most pathogenic for humans and the most common; serotype F is less common [1, 2]. There have been rare cases of botulism due to bacterial colonization of a wound (wound botulism) or the intestines (infant botulism and adult intestinal colonization botulism) and botulism due to injection of high-concentration botulinum toxin for cosmetic or therapeutic purposes (iatrogenic botulism) [2].

In typical cases, the clinical picture of botulism includes a combination of gastrointestinal and paralytic symptoms. The incubation period ranges from 2–4 hours to 2–3 days, rarely up to 5 days [2]. Gastrointestinal symptoms often develop early and include nausea, vomiting, abdominal pain and/or bloating with non-profuse diarrhea being less common. Paralytic symptoms include neurological symptoms involving striated and smooth muscles. The following neurological signs of botulism can be identified [2–4]:

- Internal and external ophthalmoplegia, i. e. mydriasis with decreased/no photoreactions, accommodation paralysis with a feeling of "fog before the eyes", ptosis, strabismus, diplopia, and nystagmus;
- Bulbar syndrome: dysphagia, dysarthria, dysphonia, nasolalia, with possible hypoglossal nerve damage;
- Damage to other cranial nerves such as paresis of facial muscles and head drop syndrome due to accessory nerve damage;
- Tetra- or paraparesis, i. e. weakness or fatigue in the limbs, abnormal muscle fatigue syndrome, hypo- or areflexia of tendon reflexes, muscle hypotonia;
- Paresis of the respiratory muscles with acute respiratory failure;
- Autonomic nervous system symptoms such as dry mouth, difficulty urinating, constipation, lack of intestinal motility, heart rhythm disturbances (most often sinus tachycardia), and fluctuations in blood pressure.

Key points to consider when assessing the patient's neurological status:

a) Symmetrical symptoms;
b) The patient is fully conscious with no abnormalities in vital signs;
c) Damage to the skeletal muscles and autonomic parasympathetic system due to acetylcholine transmission blockade.

The diagnosis is confirmed by biological assays on white mice infected with patient's serum, extracts of their feces or suspicious food. Besides detecting botulinum toxin, this assay allows identifying the type of toxin [2, 5].

Of key importance is making prompt decision and starting anti-botulinum therapy as early as possible (before receiving biological assay results), as a delay or wrong diagnosis can cause long-term therapy in the intensive care unit and/or patient's death. Anti-botulinum serum used in Russia contains toxoids against the major toxin types (A, B, E) that cause the disease. Botulism caused by toxin F is extremely rare; there is no toxoid against it in routine Russian sera, and this can negatively affect the patients' prognosis [2]. In another article, we presented a clinical case of severe type F botulism with an unfavorable outcome [6].

Given its rare incidence, diagnosing botulism is challenging, especially in big cities, where industrial production of vegetables and pickles has almost completely replaced home production. However, even with alertness to this disease, doctors may make a mistake when making a diagnosis in the case of atypical botulism. The clinical case presented below fully illustrates this point.

Clinical case

A 31-year-old man was admitted to a hospital by an ambulance with complaints of difficulty swallowing, double vision, unstable gait, and diffuse headache of VAS score 7 to 8. He considered himself ill for 24 hours. The morning before, he started to have headaches, blurred vision, and unstable gait. On the day of admission, his condition got worse (i.e. swallowing disturbance developed, vision and gait unsteadiness worsened), so an ambulance was called. The patient denied abdominal pain, nausea/vomiting, and dry mouth. He was asked many times by different doctors whether he had eaten canned foods, mushrooms, pickles or fish and said he had not eaten any of these products. All his family members were healthy.

Neurological status: completely alert, oriented, adequate. The patient had asthenia and got tired upon examination. Visually, the axis of the eyeballs was not disturbed but the patient noted the "blurriness" of objects in all directions and called this double vision. No restrictions in the range of eye movements were recorded. Disturbed conjugate eye movements with diplopia were seen; when assessing gaze in the horizontal plane, asymmetry and asynchrony of eye movements in both directions were observed: the eye going to the medial corner of the eye (adduction) "lagged behind" the eye going to the lateral corner (abduction). When looking up, he had vertical nystagmus, which "faded" when the gaze was fixed. An assessment of convergence showed slowness and limitation of adductor movements of the eyeballs. No abnormal photoreactions, abnormal accommodation, or mydriasis were observed. Mild bilateral ptosis to the upper edge of the pupil was seen. Bulbar symptoms were also seen such as dysphagia, mild dysphonia and dysarthria; however, palatal and pharyngeal reflexes were of normal vivacity, and the soft palate was symmetrical and mobile. There were abnormalities considered to be cerebellar signs: gait with a wide base of support, instability in the Romberg position, diffuse decrease in muscle tone. In the finger-nose test, tremor was observed,

which persisted in postural position. Tendon reflexes were significantly decreased. All symptoms were symmetrical. No paresis, sensory disorders, meningeal signs, or respiratory failure were seen.

Considering brainstem focal symptoms (especially internuclear ophthalmoplegia [INO] and vertical nystagmus), no epidemiological history or gastrointestinal symptoms, and preserved photoreactions, brain demyelinating disease was suspected. For differential diagnosis, brain magnetic resonance imaging was planned with further decision on the diagnosis the next day. The acute onset of the symptoms, a combination of ocular motility dysfunction, ataxia and a sharp decrease in tendon reflexes also suggested Miller-Fisher syndrome. The patient was transferred to the emergency neurology department.

The next morning, the patient's 49-year-old mother, who had eaten home-canned mushrooms, was admitted to the hospital with similar symptoms. The diagnosis of food botulism became obvious, so the patient with his mother were transferred to the Republican Clinical Infectious Disease Hospital in Kazan. Immediate therapy for botulism was initiated: gastric lavage, cleansing enema, and polyvalent anti-botulinum serum. On the same day, both patients had bulbar disorders worsened with weakness of the neck muscles and acute respiratory failure developed, which required mechanical ventilation. Only at the end of the 3rd day after the disease onset did the patient develop mydriasis, a typical sign of botulism, with no photoreactions (i. e. delayed inhibition of photoreactions). Limitation of eye movements persisted without deterioration.

A biological assay on white mice confirmed botulism and showed type A toxin in both patients. Due to prolonged mechanical ventilation, both patients underwent tracheostomy, and measures were taken to maintain their vital functions. The patient was on a ventilator for 29 days and his mother for 31 days. Both patients were discharged home in satisfactory condition, and all their neurological symptoms resolved over the next few months. The patient's ocular motility dysfunction completely resolved.

Only before discharge to the outpatient stage, the patient said that "he may have tried little mushrooms prepared by his mother".

Discussion

According to Russian Federal Service for Surveillance on Consumer Rights Protection and Human Wellbeing (Rospotrebnadzor), in 2021, 148 people suffered from botulism in Russia; of those, 22 (14.9%) cases were fatal[2]. A significant portion of these deaths might have been associated with late diagnosis. In literature, a high percentage of errors in diagnosing botulism has been highlighted. In a large review of botulism diagnostics in 332 patients in the US, the treating physician provided alternative diagnostic considerations for 83% of cases, most frequently Guillain–Barré syndrome [2]. Botulism may be also misdiagnosed as stroke, Lambert-Eaton syndrome, meningitis, encephalitis, or functional disease [2, 7]. In atypical cases, it becomes even more challenging to suspect botulism, as illustrated by our clinical case.

The primary diagnostic challenge in this case was the absence of a typical epidemiological history for botulism. In the majority of cases, the patient's consumption of canned foods is the primary indicator that prompts the physician to suspect botulism. However, the absence of an epidemiological history does not rule out the possibility of botulism, as the patient may not be aware of eating contaminated food or may forget about it (as our patient). In addition to foodborne botulism, there are wound botulism and infant botulism, in which there is no food history. In 1% to 4% of cases, there are no epidemiology data at all, and patients are diagnosed with "botulism of unknown origin" [8]. Another epidemiological point to consider is that several individuals may get sick simultaneously, which occurred in our clinical case, but our patients fell ill with an interval of about 2 days. The "clustered" distribution of the toxin in food consumed was reported, when the entire amount of toxin can be concentrated in a minimal amount of the food contaminated, so only one person can get sick, although several persons consume the same products [9].

Our patient did not have any gastrointestinal symptoms at disease onset. In foodborne botulism, gastrointestinal symptoms are typical but not obligatory. No gastrointestinal symptoms can be seen in over 50% of cases [2]. For example, vomiting was reported by only 50% of patients, and abdominal pain by only 25% [2]. Vomiting is known to be a protective reaction in case of poisoning with any types of toxins. The absence of vomiting in our patients may have contributed to complete absorption of the toxin from the gastrointestinal tract and more severe disease.

The absence of symptoms from the autonomic nervous system was also unusual. Acetylcholine is the key transmitter not only in neuromuscular transmission but also in postganglionic parasympathetic nerve endings and nerve ganglia. Therefore, botulism can be associated with decreased or no photoreactions, mydriasis, constipation, inhibited intestinal motility, and impaired urination. Mydriasis is the most common of the above symptoms; it is included in the clinical criteria for diagnosis [10]. However, the incidence of autonomic symptoms in the early stages does not exceed 50%, and the prevalence of mydriasis is only 37% [2]. Our clinical case demonstrated that internal ophthalmoplegia can be delayed, as in our patient, who

[2] Federal Service for Supervision of Consumer Rights Protection and Human Welfare. On prevention of botulism.
URL: https://www.rospotrebnadzor.ru/about/info/news/news_details.php?ELEMENT_ID=22031 (assessed: 07/28/2024).

had mydriasis and suppression of photoreactions only 72 hours after the onset of botulism, when he was already on a ventilator.

The most interesting in our clinical case is the nature of ocular motility dysfunction, as well as vertical nystagmus, which, in most cases, is associated with damage to the central brainstem structures. A unilateral or bilateral lesion of the medial longitudinal fasciculus of the brain stem is an anatomical substrate for INO. In patients with INO, adduct of one eye is slowed down, limited, or impossible, while the other abducting eye has nystagmus. In case of unilateral INO, the lesion is on the same side as the eye with the adduction weakness [11]. Ophthalmoparesis in INO is occult and unnoticeable with fast and unfixed gaze, and paresis is detected in the muscles of one eye only with slow tracking eye movements. This is why patients do not have obvious strabismus [12]. Convergence adduction is preserved in most patients. This can help differentiate INO from partial medial rectus palsy. Convergence disturbance in INO can be observed only in cases with a lesion in the medial longitudinal fasciculus at the level of oculomotor nuclei, since the tracts that form the near vision reflex pass here [13–15]. INO is often associated with monoocular nystagmus in the abductor eye. One of the hypotheses explaining abduction nystagmus is associated with an adaptive response to overcome weakness of the contralateral medial rectus muscle (Hering's law). Since the medial longitudinal fasciculus contains tracts involved in the regulation of vertical eye movements, patients with INO often exhibit disturbances in vertical eye movements, including vertical nystagmus [13, 16]. In young people, INO is most often caused by multiple sclerosis [17, 18].

Our patient had disturbed smooth tracking of the eyeballs in the form of slow adduction and asynchrony of eye movements, which is similar to INO in patients with demyelinating disease. However, there were contradictions in the clinical pattern of ocular motility dysfunction: there was no nystagmus in the abductor eye, the ability to converge was impaired, bilateral symmetrical ptosis was seen, and vertical nystagmus was attenuated. These signs suggested peripheral damage and weakness of external eye muscles. Thus, the patient had pseudointernuclear ophthalmoplegia.

Isolated cases of pseudointernuclear ophthalmoplegia in myasthenia gravis and in Miller-Fisher syndrome have been described in literature [19–21]. Nystagmus and disturbed conjugate movements of the eyeballs in patients with botulism can be explained by weakness of the eye muscles due to neuromuscular transmission blockade by botulinum toxin. The "fading" nystagmus was most likely associated with weakness of the superior rectus muscles. The diagnosis must have been slowed down and misled by delayed paralysis of the internal, smooth muscle structures of the eye [21]. Botulism-related pseudointernuclear ophthalmoplegia syndrome has not been described in the literature before.

Conclusion

In patients with acute symmetrical oculomotor and/or bulbar symptoms, differential diagnoses should include botulism. In doubtful cases, immediate administration of anti-botulinum serum is recommended. No epidemiological history, gastrointestinal symptoms or symptoms from the autonomic nervous system does not rule out botulism. In patients with botulism, development of impaired pupillary reactions can be delayed and pseudointernuclear ophthalmoplegia may be observed. However, a thorough assessment of the symptoms allows suspecting peripheral weakness of the ocular muscles such as convergence disorder, attenuated nystagmus, and ptosis together with a limitation of ocular movements.

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