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КАК НЕ ПРОПУСТИТЬ БОТУЛИЗМ? КЛИНИЧЕСКИЙ СЛУЧАЙ, АНАЛИЗ ОШИБОК И РЕКОМЕНДАЦИИ ДЛЯ ПРАКТИКУЮЩИХ ВРАЧЕЙ

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How Not to Miss Botulism? Clinical Case, Error Analysis, And Recommendations for Practitioners

Резюме

Ботулизм — острое инфекционно-токсическое заболевание, возникающее вследствие блокирования нервно-мышечной передачи из-за действия экзотоксина, продуцируемого вегетативными формами анаэробных грамположительных бактерий *Clostridium botulinum,* характеризующееся развитием парезов и параличей гладких и поперечнополосатых мышц. Актуальность ботулизма обусловлена тяжестью течения, длительными сроками госпитализации, риском летального исхода и трудностями клинической диагностики на начальном этапе заболевания, когда при появлении у пациентов первых признаков болезни они обращаются к врачам различных специальностей — неврологам, офтальмологам, что приводит к диагностическим ошибкам, несвоевременному назначению специфической терапии и ухудшению прогноза заболевания. В статье представлен клинический случай лабораторно подтвержденного ботулизма у пациента, который дважды был доставлен в стационар с подозрением на острое нарушение мозгового кровообращения, и только при повторной госпитализации был заподозрен ботулизм. Относительная редкость патологии и недостаточная информированность врачей в отношении начальных симптомов ботулизма являются причинами трудностей диагностики. Цель публикации и разбора клинического случая — актуализация знаний практикующих врачей по вопросам диагностики и дифференциальной диагностики ботулизма.

Ключевые слова: *ботулизм, Clostridium botulinum, ботулотоксин, дифференциальная диагностика, диагностические ошибки, клинический случай*

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Abstract

Botulism is still an urgent medical problem, leading to fatal outcomes. The article presents a typical clinical case of severe botulinum toxin poisoning, characterized by the maximum severity of all symptoms of the disease with signs of decompensated acute respiratory failure, dysphagia,

ophthalmoplegic and bulbar syndromes. The difficulties and errors of clinical diagnosis that have arisen due to the similarity of botulism with other pathologies are analyzed.

Key words: *botulism, Clostridium botulinum, botulinum toxin, differential diagnosis, diagnostic errors, clinical case*

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Introduction

Botulism is an acute infectious & toxic disease from the saprozoonosis group emerging due to the neuromuscular transmission block caused by the effects of the exotoxin produced by vegetative forms of anaerobic Gram-positive bacteria *Clostridium botulinum*; it manifests with paresis and paralysis of smooth and striated muscles [1, 2].

Botulism is not a common disease, although its severity, prolonged hospitalization, high mortality, and misleading early disease symptoms make it a challenging healthcare issue. Botulism is usually reported as sporadic cases or group (most often family) outbreaks. Massive outbreaks of food-borne botulism are rare, but they are considered public health emergencies that require quick detection of the infection source, determination of the disease outbreak type (wound botulism; infantile botulism; intestinal botulism of adults — food-borne, iatrogenic, or inhalational [3]) to prevent new cases. Botulotoxin does not change the organoleptic features of the food product, while the poisoning can be suspected only when the first symptoms emerge [4, 5].

Thus, in June 2024 the first Russian massive outbreak of food-borne botulism in 30 years was reported simultaneously in Moscow and four regions (Novgorod, Yaroslavl, Kostroma, Tambov Regions; Republic of Tatarstan). Within some days several hundred people got sick, while all of them ate the same product — a salad from the dark kitchen service[1],[2],[3] [6]. The causative agent was detected almost immediately in the canned red beans (main salad ingredients) kept in vacuum packaging[4]. According to the investigation, significant violations of the raw material storage, production technology, periods and temperatures of the finished preservative storage led to the accumulation of botulotoxin in the canned beans[5] [7]. 417 people with the diagnosis of botulism were reported during the 2024 outbreak. Two cases were lethal. For reference: 112 people suffered from botulism in Russia in 2020 (with 7 deaths), 148 people were reported in 2021 (with 22 deaths) [8].

Advanced botulism has signs that enable the differential diagnosis. Several (especially initial) botulism manifestations are similar to those of other diseases, thus patients with the emerging botulism symptoms do not often seek the attention of infectious disease specialists, but rather other physicians. With that, the majority of physicians lacking the experience of clinical botulism diagnosis do not match the clinical signs with this disease, which becomes the source of diagnostic errors [9–11].

The following botulism forms have been described: food-borne botulism, wound botulism, infantile botulism, botulism of uncertain origin [1]. Food-borne botulism occurs in over 90 % of all cases; it is caused by the toxin contained in food products. In the setting of wound botulism the toxin is produced by Clostridia in the infected tissues. Infantile botulism usually occurs in infants under 6 months of age infected by *Cl. botulinum*

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[5] Rospotrebnadzor Has Detected Suppliers and Manufacturers of Food Products that Caused Botulism in Several Regions // Business Newspaper. 2024. June 18. [Electronic source]. URL: https://www.business-gazeta.ru/news/637449 (Date accessed: February 8, 2025)

that produce the toxin in the gastrointestinal tract. The diagnosis of botulism of uncertain origin is established if it is impossible to associate the disease with the food product [5].

Clinical manifestations of various botulism forms are characterized by several syndromes:

- gastrointestinal (nausea, vomiting, liquid stool, abdominal bloating);
- intoxication (short-term subfebrile fever);
- paralytic (myasthenia, respiratory failure, tachypnea, shortness of breath (with patients forcing an inspiration after saying 3–5 words));
- ophthalmoplegic ("mist" in the eyes, blurry object contours, diplopia, limited eyeball motions, pupil dilation, absence of all pupil reactions, nystagmus, diminished corneal and conjunctival reflexes, ophthalmoplegia, ptosis, strabismus (in cases of unequal paresis bilaterally), anisocoria);
- bulbar (voice hoarseness, nasal speech tone, aphonia, dysarthria, flattened nasolabial folds, inability to bare teeth or frown the forehead, dysphagia, dry mouth, glomus sensation in the throat, thirst) [5, 12].

The paralytic syndrome is the leading one in the differential diagnosis. The aforementioned syndromes might not emerge together, and some of those are not specific — thus, patients may refer to different physicians based on the predominant syndrome.

The standard laboratory diagnosis of botulism includes the toxin isolation and identification in biological fluids and food products uding the neutralization test in mice — this requires significant time efforts and special laboratory conditions, thus in the first days the diagnosis is based solely on clinical and epidemiological data [5, 13]. Due to this, the physician should know the main manifestations of this disease and features of the botulism course that will help in the differential diagnosis with several diseases having similar symptoms [14].

Patients with neurological symptoms are referred to the neurologist in order to exclude encephalitis, myasthenia, Guillain-Barre syndrome, acute cerebrovascular accidents (as demonstrated in the clinical case report below).

The clinical case report is aimed at analyzing the typical clinical situation in order to revise the knowledge of practical physicians concerning the clinical diagnosis and differential diagnosis of botulism.

Clinical Case Report

The female patient Sh., 50 years old, was admitted to the emergency department of the infectious hospital on Day 2 of the diseases complaining of diplopia, weakness in extremities, difficulty swallowing and breathing, dry mouth and a sensation of "porridge in the mouth".

History: diplopia, dizziness, blurred vision, weakness in extremities emerged on Day 1 of the disease. The patient was transported by the ambulance to the emergency department of the city hospital due to the suspected acute cerebrovascular accident. She was examined by the general practitioner and neurologist there. The computed tomography and the magnetic resonance imaging of the brain were arranged, and the acute cerebrovascular accident was excluded. The diagnosis of dyscirculatory encephalopathy was established. The patient refused the hospitalization and went home. During the day, diplopia, dizziness, blurred vision persisted, while the weakness in extremities worsened. The body temperature was not elevated, while the patient did not take any medications. During the second day of the disease, while diplopia and weakness in extremities persisted, the patient developed difficulty swallowing and breathing, dry mouth and a sensation of "porridge in the mouth", nausea, with a single liquid stool episode. The patient was transported urgently by the ambulance again to the emergency department of the city hospital due to the suspected acute cerebrovascular accident. The computed tomography of the brain was repeated, and no pathological alterations were detected. The patient was examined by the neurologist. Accounting for neurological signs (diplopia, dizziness, blurred vision, worsening weakness in extremities, difficulty swallowing and breathing, dry mouth and a sensation of "porridge in the mouth"), the neurologist suspected botulism and referred the patient to the infectious hospital.

During the repeated examination, the neurologist clarified the epidemiological history. The patient reported that 3 days before the disease she had eaten home-made canned vegetables (carrots and onions).

Upon the hospitalization to the infectious department, the patient's condition was considered severe based on neurological signs. She was conscious, lying passively in mild stupor; the verbal contact was preserved. She scored 13 points in the Glasgow Coma Scale (opening eyes upon request; confused speech; directed motor response upon request). RASS (Richmond Agitation and Sedation Scale) score was 1 point (drowsy; loss of attention, but the patient did not close eyes for over 10 seconds during the verbal contact). The pupil sizes were normal, symmetric. The photoreaction persisted. The corneal reflex was normal. Bilateral hemiptosis, convergent strabismus, dysphagia, dysphonia. Motions in extremities were preserved. No alterations were detected in other organs and systems. The skin had a normal color and turgor; visible mucous membranes were normal. Respiratory rate: 18 per minute. Oxygen saturation (on room air) 98 %. No dyspnea was reported. Blood pressure 132/94 mm Hg. Pulse 86 beats per minute, with satisfactory filling. Cardiac tones were clear and regular. The tongue was dry and coated with white plaque. The abdomen was soft and non-tender.

The liver and spleen were not enlarged. According to the patient, the urination was not painful (last urination in the morning). Meningeal signs were negative.

Accounting for the epidemiological history (consumption of home-made canned vegetables), clinical signs suggestive of the gastrointestinal (nausea, one-time liquid stool), paralytic (difficulty breathing, weakness in extremities), ophthalmoplegic (diplopia, blurred vision, bilateral hemiptosis, convergent strabismus), bulbar syndromes (difficulty swallowing, dysphonia, confused speech), as well as the dry mouth sensation, the preliminary diagnosis of botulism was established. The patient was hospitalized into the intensive care unit of the infectious hospital. She was administered the polyvalent antitoxic anti-botulism serum (10,000 IU — types A & E; 5,000 IU — type B) intravenously once based on the Instructions for Medical Use.

The following pathogenetic therapy was administered: tube feeding during Days 4–12 of the hospitalization, enterosorbents (activated charcoal for 3 days), infusion therapy (crystalloid solutions with the purpose of detoxification and correction of the water-electrolyte balance), administration of Group B vitamins, oxygen therapy (humidified oxygen, 5 L/min), as well as antibiotics (cephalosporins) for 10 days to suppress *Cl. botulinum* in the gastrointestinal tract and to prevent secondary bacterial infections.

Respiratory disorders, including difficulty breathing, accessory muscles activated for breathing, emerged on Day 2 of the hospitalization, while bulbar disorders (significant dysphagia) worsened — the patient was intubated and connected to the mechanical ventilation device.

See Tables 1–4 for examination results.

Table 1. *Blood analysis*

Parameter	Result	Reference Value	Units of measurement
Complete Blood Count			
Erythrocytes	4.64	3.90 — 4.70	cells x10^{12}/L
Hemoglobin	142.00	120.00 — 140.00	g/L
Leukocytes	5.90	4.00 — 10.00	cells x10^{9}/L
Thrombocytes	91.00	180.00 — 320.00	cells x10^{9}/L
Biochemical blood analysis			
Aspartate aminotransferase	18.50	0.00 — 31.00	U/L
Alanine aminotransferase	8.90	0.00 — 31.00	U/L
Total bilirubin	6.10	5.00 — 20.50	μmol/L
Creatinine	65.00	53.00 — 106.00	μmol/L
Total creatine kinase	113.00	0.00 — 145.00	U/L
Creatine kinase-MB	19.10	0.00 — 25.00	U/L
Total protein	63.10	65.00 — 85.00	g/L
Albumin	37.20	38.00 — 51.00	g/L
α-Amylase	39.00	0.00 — 100.00	U/L
Procalcitonin	<0.50	0.00 — 0.50	ng/mL
C-reactive protein	1.60	0.00 — 5.00	mg/L
Lactate dehydrogenase	363.00	195.00 — 450.00	U/L
Troponin T	negative	negative	ng/L
Gas-electrolyte blood test			
cK+	3.70	3.40 — 4.50	mmol/L
cNa+	139.00	135.00 — 146.00	mmol/L
cCa2+	0.97	1.15 — 1.29	mmol/L
cCL-	110.00	98.00 — 106.00	mmol/L
cGlu	5.40	3.89 — 5.83	mmol/L
cLac	1.10	0.50 — 1.60	mmol/L
pH(t)	7.30	7.350 — 7.450	-
cBase(Ecf), c	-4.40	-3...+3	mmol/L
cHCO3-(P, st), c	20.20	21.00 — 28.00	mmol/L

Table 2. Analysis of cerebrospinal fluid

Parameter	Result	Reference Value	Units of measurement
Protein	0.57	0.15 — 0.45	g/L
Glucose	3.60	2.20 — 3.90	mmol/L
Cell count	0.67	0.00 — 10.00	cells/μL
Lactate	1.50	1.10 — 2.40	mmol/L
Color: colorless, transparent			
Cerebrospinal fluid culture: No microflora growth was detected			

Table 3. Examination of cerebrospinal fluid for the presence of nucleic acids of neuroinfection pathogens by polymerase chain reaction (PCR)

Parameter	Result
Non-polio enteroviruses (*non-polio enteroviruses*)	Negative
Pneumococcus (*Streptococcus pneumoniae*)	Negative
Listeria (*Listeria monocytogenes*)	Negative
Streptococcus (*Streptococcus agalactiae*)	Negative
Meningococcus (*Neisseria meningitidis*)	Negative
Human herpesvirus 6 (*Human herpesvirus 6*)	Negative
Haemophilus influenzae type b (*Haemophilus influenzae b*)	Negative
Herpes simplex virus 1 and 2 (*Human alphaherpesvirus 1, 2*)	Negative
Epstein–Barr virus (*Epstein-Barr virus*)	Negative
Cytomegalovirus (*Human cytomegalovirus*)	Negative
Varicella zoster virus (*Varicella-zoster virus*)	Negative
Borrelia (*Borrelia burgdorferi*)	Negative

Table 4. Other investigations

Investigation	Result
Stool tests	No nucleic acids of viruses or pathogenic bacteria were found in the stool samples
Urine analysis	Without pathological changes
Electrocardiography	Sinus rhythm with a heart rate of 80 beats per 1 min

To confirm the diagnosis of botulism, the patient's blood serum collected before the administration of the polyvalent antitoxic anti-botulism serum was sent to the laboratory of natural focal & hazardous infections and parasitic invasions of FBHI "Center of Hygiene and Epidemiology in the Arkhangelsk Region". The biological test was arranged in mice, as well as the serum was tested for *Cl. botulinum* botulotoxin using the neutralization test. The diagnosis of botulism caused by the A toxin was confirmed on Day 8 of the hospitalization (Day 9 of the disease). On Day 8 of the hospitalization, extubation was attempted with the transfer to mask oxygenation; however, on Day 9 the respiratory failure worsened, with significant confusion (severe stupor), and the patient was again transferred to mechanical ventilation, until spontaneous breathing emerged on Day 12. Paresis regressed by Day 8, although weakness in extremities remained during the whole inpatient period. Dysarthria and dysphagia persisted until Day 15, while dysphonia — until Day 18. Diplopia persisted until Day 9 of the hospitalization. Bilateral ptosis gradually improved and completely resolved by Day 12. Bulbar disorders (convergent strabismus, ophthalmoplegia, difficulty swallowing) gradually improved and regressed by Day 8–9. Salivation completely recovered on Day 10 of the hospitalization.

The follow-up magnetic resonance imaging of the brain was arranged on Day 15 of the hospitalization — no focal brain lesions were detected. On Day 17 of the hospitalization, the patient Sh. was transferred from the intensive care department to the infectious department in the satisfactory condition for further observation and treatment. On Day 18 of the hospitalization, she was examined by the physical therapist, with the rehabilitation cycle recommended.

The patient was discharged on Day 21 of the hospitalization, with severe asthenia and decreased muscle strength in extremities preserved. During the muscle strength examination, the patient could partially overcome the physician's resistance. She completed the finger-nose and heel-knee tests satisfactorily, although she mildly missed during the left-sided heel-knee test. The patient was not stable during the Romberg test, with unsteady gait (the patient required assistance, seeking support with the furniture, walls while walking). Her rehabilitation routing score was 4 points (significantly impaired functions and daily activities). She was discharged in the satisfactory condition, being transferred to Step 2 of the inpatient rehabilitation.

Discussion

A clinical case of severe food-borne botulism (A05.1) (toxin type A) has been presented; this was characterized by the maximum severity of all disease symptoms

with the signs of decompensated acute respiratory failure, dysphagia, ophthalmoplegic and bulbar syndromes. The diagnosis was delayed for 2 days. Due to diplopia, dizziness, blurred vision, weakness in extremities emerging on Day 1 of the disease, the patient was transported by the ambulance to the therapeutic inpatient department with the suspected acute cerebrovascular accident (ACVA). One should account for the fact that neurological signs in ACVA are determined by the locations of brain lesions [15, 16]. The specific feature of the paralytic syndrome in botulism is its symmetry and bilateral signs, which was observed in the case described. Diplopia, blurred vision, weakness in the upper and lower extremities were confirmed bilaterally. Neurological signs in ACVA is more often asymmetric and depends on the blood supply area or the location of the affected vessel, as well as the duration of the disease course. During the first visit, the diagnosis of ACVA was excluded due to no history of cardiovascular diseases typical for ACVA, no unilateral central hemi- and monoparesis, unilateral soft palate, vocal cord and tongue lesions; no alterations were also detected in the brain CT or MRI. With that, after instrumental examinations, despite no data confirming ACVA, botulism was not suspected, although the first complaints in the majority of patients with botulism include visual disorders, i.e. mist in the eyes, unclear object contours, diplopia worsening with the lateral gaze (all observed in the clinical case described). Besides visual disorders, weakness complaints were also underestimated. Weakness is the mandatory complaint in patients even with mild botulism forms. It can be explained both by the intoxication and paralytic toxin effects on the skeletal muscles. These effects mainly manifest in proximal muscle groups, however in general lesions are generalized, which can be explicitly confirmed by the decreased hand strength easily detected during the physical examination [17].

Less than a day from the disease onset passed during the first examination, and clinical signs of botulism were not completely represented. The epidemiological history (consumption of home-made canned vegetables) was not confirmed by the neurologist and the general practitioner during the examination.

Changes in botulism symptoms and the disease severity can be evaluated only following up the patient; in our case the patient refused the hospitalization and went home, despite neurological signs. It is possible that the exclusion of the acute cerebrovascular accident that could require urgent surgical or conservative inpatient treatment and the established diagnosis of dyscirculatory encephalopathy affected the decision to refuse the hospitalization.

Specific botulism signs (mild difficulty breathing, worsened weakness in extremities, diplopia, blurred vision, bilateral hemiptosis, convergent strabismus, dry mouth, sensation of "porridge in the mouth", difficulty swallowing, dysphonia, confused speech) emerged on Day 2 of the disease. Botulism was suspected during the second visit to the general department after the neurologist examination and clarification of the epidemiological history.

Dysphagia and dry mouth caused by the atropine-like effects of botulotoxin on the autonomous nervous system are significant for the diagnosis, being early signs of botulism, which has to be accounted for during the differential diagnosis [17].

Regarding the diagnosis, it is very important to record the time of the emergence of clinical signs from consuming the food "suspicious" of possible botulism. In our case, this period was 3 days, which is typical for the average duration of the incubation period in botulism [1].

Late botulism diagnosis is also associated with its signs being similar to the poisoning with other toxins (i.e., food-borne toxin infections). Nausea and a single liquid stool were observed on Day 2 of the disease in our patient. With the gastrointestinal syndrome, such mistake is possible, as botulism and the food-borne toxin infection are associated with the alimentary factor. Both diseases are characterized by the short incubation period and similar initial symptoms (nausea, vomiting, abdominal pain, stool disorders). However, botulism lacks fever, while food-borne toxin infections are not characterized by the paralysis, visual, speech, and swallowing disorders, which are the main botulism symptoms that determine the severity in this disease [18].

If botulism is suspected, specific laboratory diagnosis is required in all patients to confirm the diagnosis [1]. In this case, detection of the botulotoxin in blood becomes the absolute laboratory confirmation of botulism. The blood serum was collected from the patient before the administration of the botulism antitoxin. The botulotoxin was detected using the biological test and the biological toxin neutralization test in white mice using the diagnostic anti-botulism polyvalent sera [19].

The microbiological culture to detect and identify the causative agent of botulism (*Clostridium botulinum*) and/or determination of its (*Clostridium botulinum*) toxins in vomitus/gastric lavage discharge was not arranged, as the patient was admitted three days after the consumption of canned foot, vomiting was absent, while the products suspicious of botulism were disposed of.

During the first day of hospitalization to the infectious department, the botulism antitoxin (types A, B, E) was administered based on clinical & epidemiological indications, while the result of the biological test confirming the diagnosis of type A botulism was obtained on Day 8 of the hospitalization (Day 9 of the disease). Systemic antibiotics are administered to patients with botulism with the purpose of prevention of complications

emerging due to paresis and paralysis [1], which was also applied in our clinical case, despite no foci of bacterial infections upon the hospital admission. Within 72 hours from the disease onset, patients with botulism undergo gastric lavage and siphon enema to eliminate the toxin [1]. In our case this recommendation was not followed as the patient was hospitalized on the third day from eating the canned food, and botulotoxin already absorbed through the oral and intestinal mucosa [14]. Patients with botulism may remain on nasotracheal intubation for 80 days without any significant adverse effects, allowing to avoid tracheostomy [1]. In our clinical case the patient underwent elective tracheal intubation, which remained for 11 days. It should be noted that the final extubation of patients with botulism is possibly only with the complete recovery of respiration and fluid swallowing [1]. Early extubation with the incompletely restored respiration in our clinical case led to the worsening condition, emergence of the respiratory failure symptoms, depressed consciousness (severe stupor), which required repeated intubation and switching to the mechanical ventilation.

Conclusion

The presented typical clinical case of food-borne metabolism confirmed by the laboratory biological test demonstrated difficulties in the clinical diagnosis of the disease; these were caused, on the one hand, by the relative rarity of the pathology, and on the other hand — by neurological signs during the disease onset (diplopia, dizziness, blurred vision, weakness in extremities) that formed the basis to refer the patient to the inpatient department with the suspected acute cerebrovascular accident.

Thorough collection of the epidemiological history is significant during the botulism diagnosis. Insufficient awareness of physicians of various specialties (general practitioners, neurologists, ophthalmologists) due to the relative rarity of botulism, superficial history collection (including epidemiological history) leads to the fact that even with the timely visit symptoms of typical botulism are underestimated and considered as manifestations of another disease — this requires updating the physician knowledge concerning the issues of clinical botulism diagnosis.

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