CASE REPORT

Food-borne botulism: still actual topic

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SUMMARY

Even though since the mid-1990s the number of food-borne botulism cases has systematically decreased and it now occurs in Poland relatively rarely, it is still a real epidemiological problem. There are about 30 cases of botulism in Poland a year, which ranks Poland the first among the European Union. In most cases the symptomatology of botulism is typical, however it does not always fully coincide with the one described in medical manuals which emphasise the dramatic clinical course of botulism with its frequent fatal consequences. Diagnosis of botulism may be difficult because of its rare prevalence and a variable clinical course, especially in old patients. Authors of this paper describe two cases of botulism and diagnostic problems associated with it.

BACKGROUND

Cases of food-borne botulism are presently uncommon in countries of the European Union; however, in Poland still over 30 cases of food-borne botulism emerge every year. Labour-related emigration to numerous European countries and still persistent tradition of preparing home-made tinned food in polish households may be the cause of emergence of botulism in countries in which it was absent for years. It is worthwhile reminding the symptoms of this dangerous poisoning and difficulties concerning its diagnosis and treatment.

CASE PRESENTATION

Case 1

A 71-year-old woman presented with a 2-day history of stomach ache, nausea and vomiting before diplopia appeared. Upon neurological examination diplopia was found when looking from side to side, with no signs of other cranial nerves involvement or central nervous system damage. Biochemical and haematological investigations were normal apart from mildly lowered level of potassium (2.6 mmol/l). Abdominal ultrasound and CT head scan were normal too. The next day (fourth day since the first symptoms) the patient's conditions suddenly deteriorated. Total bilateral ptosis occurred with ophthalmoparesis to all directions, dilated fixed pupils, hoarseness, dysphagia and mouth dryness. A more detailed interview revealed that on the day before the first symptoms appeared, the patient ate homemade marinated mushrooms. Serology for botulinum toxin was performed. The patient was provided symptomatic treatment to regain water–electrolyte balance. Shoulder paresis and breathing difficulties were observed, but there was no necessity to start mechanical ventilation. Owing to the quickly deteriorating general condition of the patient, a decision was made to immediately administer a polyvalent ABE botulinum antitoxin (150 ml intravenously). To avoid allergic reaction, the patient was also given 70 mg of prednisolone. The patient had no difficulty in breathing, but her general condition did not improve. The next day an additional dose of 80 ml antitoxin was given intramuscularly. The patient was fed through a gastric tube. On day 6 of hospitalisation the patient was stable and neurological symptoms were subsiding. Serological tests confirmed the presence of botulinum toxin type B/E in the blood. The patient was discharged after 21 days in a good general condition with no neurological symptoms.

Case 2

A 29-year-old patient was presented with diplopia, vertigo and gait abnormality for the previous 4 days preceded by nausea and stomach ache. Neurological examination revealed mild divergent strabismus of the left eye, bilateral ptosis, diplopia when looking from side to side, increased deep reflexes in lower limbs, mild left-sided ataxia and a positive Romberg's test. CT head scan was normal. Cerebrospinal fluid analysis revealed protein and glucose levels to be within their normal reference ranges; cellular pleocytosis was 17/μl and absence of oligoclonal bands, IgG and IgM antibodies for *Borrelia burgdorferi* were not detected. Oligoclonal bands were not found. An MRI of the head and chest x-ray were normal. There were no antibodies found for acetylcholine receptors. *Mycobacterium tuberculosis* infection was excluded. On the third day of hospitalisation, the patient's condition exacerbated when difficulty in swallowing solid food was observed. The patient also complained of mouth dryness and persistent constipation. Further, a detailed history revealed that the patient had eaten tinned meat about 24 h before the first gastrointestinal symptoms occurred. Serum for botulinum toxin collected on day 11 of hospitalisation showed type B botulinum toxin. ABE anti botulinum serum was not given because of satisfactory general condition of the patient and the period of time that had already passed from the onset of infection. The patient was treated symptomatically. Fluids were introduced intravenously to preserve proper electrolyte balance. During a few days of hospital treatment, gradual recession of neurological symptoms was observed.

OUTCOME AND FOLLOW-UP

The 'Case 1' patient was discharged after 21 days in a good general condition with no neurological symptoms. During the first year following hospitalisation she complained of fatigability, periodically recurrent mood changes and habitual constipation, but there were no abnormalities found upon neurological examination.

The 'Case 2' patient was provided conservative treatment for the next few days after the final diagnosis and then discharged from hospital in a good general condition. In the course of 1-year outpatient observation there were no signs of neurological symptoms or any further complications.

DISCUSSION

Botulism is caused by neurotoxins produced by anaerobic sporogenic *Clostridium botulinum*, or more rarely by *Clostridium butyricum*, *Clostridium baratii* or *Clostridium argentinese*.[1 2] Eight botulinum toxins were identified on the basis of amino acid compositions and antigenic properties: A, B, Cα, Cβ, D, E, F, G. Botulinum poisoning in humans is usually caused by toxins of A,B,E and sometimes F type.[1–3] Clinical symptoms of botulism occur owing to inhibited release of acetylcholine in neuromuscular connections of motoneurons and synapses of parasympathetic nervous system.[3] Most cases of botulism in Europe result from ingestion of contaminated (type B toxin) homemade tinned meat.[4] Clinical signs of botulism appear after incubation period which lasts several hours—8 days.[2 3] They are usually preceded by nausea, vomiting, stomach ache and loose stools. Clinical botulism includes symmetrical cranial nerves palsy with secondary symmetrical ascending flaccid paralysis, which can lead to respiratory arrest. Symptoms develop in a few hours to a few days and need several weeks or months to subside.[3 5] The most common complications in botulism are aspiration pneumonia and urinary tract infections.[3] Death rate amounts to 7–10%.[1–3]

Classic botulism is nowadays a rare condition. In Europe there are only sporadic cases of it, in USA there are about 110 cases of botulism a year, most of which are cases of infant botulism.[5] In Great Britain, in the past 80 years there have been only 62 cases of food-borne botulism.[6] However, in these countries wound botulism in drug addicts seems to be a growing problem. In 2006, in Ireland the first in 20 years case of food-borne botulism was diagnosed in a man of Polish nationality who got poisoned after ingesting homemade food sent to him from Poland.[6] In a Munich Clinic of Toxicology there were two cases described of such poisoning in 1999 and 2006.[6] In Poland, over the last 10 years, however, there have been about several dozen cases of botulism every year. In mid-1990s there were over 100 cases, and in 2009–2010 about 30.[7] The two cases described in this article were registered in 2010 in two different neurological wards.

Even though recognising botulism on the basis of clinical picture and epidemiological history is relatively easy, it is not always remembered that botulism is still a possibility. Moreover, the course of this disease is sometimes untypical, especially in older people. In the two cases described above, the delay in treatment was owing to several factors: long incubation period, mildly symptomatic course and mistaken preliminary diagnosis (both cases were at first diagnosed as gastroenteritis). The fact that the patient had eaten mushrooms additionally blurred the clinical picture. Literature review shows that food-borne botulism caused by marinated mushrooms is not as rare as it may seem.[8 9]

Even though botulinum intoxication is rare, it should always be taken into account in differential diagnosis, because early diagnosis may turn out to be crucial for a successful treatment. Development of diagnostics extorts performing numerous detailed tests which is both time-consuming and expensive. The cases described in this paper were treated in two different ways and the final result was successful in both of them. The use of antibotulinum serum is still problematic, since it should be administered in the early stages, whereas its availability is limited. On the other hand, if the treatment is not successful, patients or their families may lodge a complaint that the serum was not used, thus it is advised to treat every case individually.

Learning points

- Consider botulism as a cause of patient's cranial nerves palsy and ascending flaccid paralysis.
- Ask the patient of ingesting homemade marinated food—if yes, collect serum for toxin detection.
- Antibotulinum serum treatment is not required in every case of botulism.

Competing interests None.

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