

Botulism with bilateral papilledema and venous sinus thrombosis: A case report

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ABSTRACT

Background: Food-born botulism is caused by ingestion of contaminated food with clostridium botulinum neurotoxin.

Case: In food-born botulism, due to acetylcholine release blockade, neuromuscular signs and symptoms are common, but in this case report we present a food-born botulism with papilledema and venous sinus thrombosis.

Conclusion: Papilledema and fever could not rule out the diagnosis of botulism.

Keywords: Botulism, sinus thrombosis

INTRODUCTION

Consumption of foods contaminated with neurotoxin of clostridium botulinum, an anaerobic gram-positive organism, cause serious disease call botulism a rare mostly food-born paralyzing disease. It can be explained as an afebrile, rapidly progressing, symmetrical, and descending paralysis of motor and autonomic nerves beginning with cranial nerves (1). It blocks acetylcholine transmission at all ganglionic synapses and neuromuscular junctions so may lead to respiratory failure and death (2). Diagnosis should be done based on the clinical manifestations and definitive confirmation made by inoculating mice with contaminated serum or stool samples. Treatment includes administration of antitoxin of botulinium and supportive care (3).

CASE REPORT

A 33 year old woman complaining of severe abdominal pain, headache, recurrent vomiting, constipation, and anorexia for 3 days admitted to the surgical ward. She had received serum and intravenous antiemetic drugs because of her complaint in recent 3 days as an outpatient with no improvement. In past medical history, she mentioned to migraine headache for recent 2 years.

At first, the patient's situation was considered as an acute abdomen which ruled out by frequent physical examination and normal abdominal sonography. Dry mouth, severe back pain, fever (T= 38), absent gag reflex, horizontal diplopia with six nerve palsy, blurred vision, bilateral papilledema, weakness, upper limbs paresthesia, and descending paralysis were added to the initial symptoms in second day examination. Serum electrolytes and urine analysis were normal. CRP was 4+ , ESR 1h : 14 , 2h: 21, W.B.C : 18500 /mm³, Hb: 14.9 gr/dl, Htc: 47.3%, Plt: 379000/mm³, Na: 136 mEq/dl, K: 4.4 mEq/dl, ca: 10.1 mEq/dl, and FBS: 217 mg/dl.

The patient pointed to the ingestion of homemade whey and canned fish four days before admission. The clinical situation suggested food-born botulism; so, botulism trivalent antitoxin was administered for her in post admission second day. She was supervised for respiratory function and need for intubation in ICU in next day too. For diagnosis confirmation, serum and stool samples was taken and tested for botulinium toxin types A, B, and E. The results indicated her negative serum sample while the stool sample was suspected to all of them.

Because of unexplained bilateral papilledema, she was consulted with a neurologist and magnetic resonance imaging±

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gadolinium was requested. MRI findings showed no evidence of mass or tumoral lesion, normal patent of ICA (Internal Carotid Arteries), basilar arteries, bilateral mastoiditis, and suspected venous sinus thrombosis and it recommended too MRV (Magnetic Resonance Venography) (Figure 1). In MRV in the posterior aspect of superior sagittal sinus and both transverse sinuses, (RT more than LT) partial patchy thrombosis was seen and some collateral formation around the transverse sinus was detected. Upper and lower limbs EMG and NCV were normal. She received intravenous antibiotic (ceftriaxon 2gr/12h) and heparin (5000 IU/6h) for venous sinus thrombosis. After visit of ophthalmology, ICP measurement was recommended that because of patient's toxic status was not administered. ENT specialist visited her for report of bilateral mastoiditis by MRI; however, no clinical sign or symptom was detected.

The patient was discharged from our hospital eight days after admission with warfarin sodium (2.5 mg/day) and no cranial nerve dysfunction sign or symptom. Her upper limbs strength was 3.5/5 which had disappeared in the follow-up examination 4 weeks after discharge from hospital.

Six months after first symptom, physical examination showed no sign or symptom of intoxication of *C.botulinium* and papiledema; MRI and MRV showed no evidence of venous sinus thrombosis; so, warfarin was



disconnected.

Figure 1 MRV: venous sinus thrombosis

DISCUSSION

Botulism is an acute and rapidly progressing paralytic disease with a descending fashion in an afebrile patient caused by a neurotoxin elaborated by spore-forming bacillus clostridium botulinum (1, 2). It is classified into five categories: classic or food-born botulism, infant botulism, adult infectious botulism, wound botulism, and iatrogenic botulism. The neurotoxin blocks acetylcholine release by binding to neuroexocytosis apparatus which causes irreversible blockade of cholinergic transmission at all postganglionic parasympathetic synapses and neuromuscular junctions that result flaccid paralysis and autonomic nervous system dysfunction(2). Gastrointestinal symptoms (anorexia, constipation, nausea and vomiting, diarrhea, abdominal pain, and paralytic ileuse), genitourinary symptoms (distension of urinary bladder, urinary retention, or incontinency), fatigue, muscle weakness, ophtalmoplegia, ptosis, double or blurred vision, photophobia, fixed or dilated pupils, nystagmus, rapidly descending symmetrical flaccid paralysis, dry mouth, dysphagia, weakness of tongue, slurred speech, impaired gag reflex, and respiratory failure are common in botulism (4).

The diagnosis of botulism must be based on clinical grounds and patient's history. According to these clinical manifestations, before considering a diagnosis of botulism, some neuromuscular diseases (such as Guillain-Barre syndrome, Miller-Fisher syndrome, myasthenia gravis, and Eaton-Lambert syndrome), infectious diseases (such as poliomyelitis and diphtheria), metabolic myopathies (such as periodic paralysis and porphyry), drug intoxications like Mg and atropine intoxication, poliomyelitis and cerebrovascular accidents should be ruled out(5).Definitive diagnosis is made by demonstration of toxin in the serum but it may be negative despite infection and cannot be conducted in all laboratories.

We diagnosed this case based on the neurological examination and patient's history and supported our diagnosis by laboratory tests. She was transferred to ICU for

respiratory function monitoring but intubation was not needed. After administrating botulinium trivalent antitoxin on the second post admission day, clinical improvement occurred.

CONCLUSION

Although botulism is a rare disease but because of its severity, considering a diagnosis of botulism soon after presentation of acute cranial nerve dysfunction is important. Although papiledema, fever, and increase in WBC count can be explained by venous sinus thrombosis, according to our findings, papiledema and fever can not rule out the diagnosis of botulism. In 2005, Penas SC *et al* (6) reported blurred near vision, blurred distance vision, diplopia, and impaired accommodation to be the most common ocular symptoms in botulism. However, we were not able to explain the cause of venous sinus thrombosis occurrence.

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