Bladder Paralysis Due to Foodborne Botulinum Toxin Type B

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INTRODUCTION

Foodborne botulism is a rare toxi-infection in France (20 to 30 cases per year).[(1)] Incubation time is comprised between few hours to 8 days. *Clostridium botulinum* is an anaerobic gram-positive organism which is ubiquitously found in soil and aquatic sediments in the spore form. Several forms of botulism exist. The foodborne form is the most frequent in the human botulism. *Clostridium botulinum* produces 7 different toxins of type A, B, C, D, E, F, and G. Toxin type A is the most frequent and is found in the home-canned. Toxin type B is found in the cooked pork meats. All forms of botulism produce the same clinical symptoms: symmetrical cranial nerve palsies followed by descending, flaccid paralysis of voluntary muscles, which may progress to respiratory arrest. Prominent autonomic symptoms include accommodative paralysis with mydriasis, anhydrosis with severe dry mouth and throat, and orthostatic hypotension. Constipation and bladder paralysis are rarely reported.[(2)] All toxins exert their action on the cholinergic system at the presynaptic motor-neuron terminal by blocking acetylcholine transmission across the neuromuscular junction. It causes neuromuscular blockade, resulting in a flaccid paralysis.[(3)]

This article describes a case of a 43-year-old man who presented with severe botulism manifestations. In addition to the severe cranial motor nerves paralysis, a complete bladder paralysis was observed.

CASE REPORT

A 43-year-old man, without a previous medical disorder presented to the neurological unit with progressive cranial motor nerves impairment, including bilateral and complete intra-ocular and extra-ocular muscles paralysis, swallowing deficit, and dysphonia. A few days before, he had experienced a painful abdominal syndrome with diarrhea and vomiting. The patient did not display any motor or sensory limb deficit. Vigilance and cognition were not affected. The patient, required hospitalization in intensive care unit due to respiratory failure 24 hours following the first neurological signs. Moreover, he showed a complete bladder paralysis with preservation of bladder-filling sensation, imposing indwelling urethral catheterization. Dysautonomic symptoms were also noted with bilateral mydriasis (without reaction to light), constipation due to a paralytic ileus, orthostatic hypotension, and oral dryness.

Brain tomodensitometry and lumbar puncture were normal. Electroneuromyography examination revealed dysfunction at the presynaptic neuromuscular junction which was suggestive of botulism. Botulism toxin type B was present in blood. A diagnostic of botulism was confirmed by mouse inoculation few days after the first symptoms. A complete sanitary investigation was made and no contaminated food was founded. There was no other similar case in the family.

The spontaneous recovery of different neurological symptoms was assessed by clinical examination and urodynamic investigation. Two periods of recovery could be distinguished: a first period from day 30 to day 45 postonset, and a second later period from day 120 to day 150 postonset. During the first period, improvement was seen in ptosis, swallowing deficit, dysautonomic symptoms (constipation and orthostatic hypotension), and external ophtalmoplegia. At this stage, the patient kept accommodative deficit. A first urodynamic investigation at day 30 revealed a normal bladder-filling sensation and normal compliance with detrusor acontractility. Increased urethral closure pressure was observed. A vesicosphincter dyssynergia was present. Self intermittent catheterization was decided.

During the second period, the patient recovered from spontaneous micturition, and self catheterization was progressively stopped from day 120 to day 150. Moreover, at the same time, the patient regained normal accommodative function. On day 120, urodynamic investigation revealed a hyposensitive and hypocompliant bladder. Detrusor contractility was reduced. Micturition during urodynamic test was impossible despite a good external sphincter relaxation and a transient falling of urethral pressure. The pressure flow analysis revealed a dysuric urination without residual urine in the bladder. All the urinary disorders disappeared 3 months later.

DISCUSSION

The only reported case of a bladder paralysis due to botulism was a wound-botulism due to toxin type A in a context of an intravenous drug use.[4] The patient also had a severe limbs paralysis and respiratory failure which led to intubation and tracheotomy. He presented bladder paralysis resulting in self intermittent catheterization. Three months later, the patient was able to void spontaneously and correctly.[4] We report a similar bladder paralysis due to a foodborne botulism (toxin type B). Our patient presented with bladder paralysis due to botulism toxi-infection with cranial motor nerves paralysis and several dysautonomic disorders.

The Table compares characteristics of the two reported bladder paralysis cases due to botulism. The clinical manifestations are similar: diplopia, dysarthia, dysphagia, and internal ophtalmoplegia. Our patient did not show limb

Comparison of 2 Cases of Bladder Paralysis Due to Botulism*

Characteristic	Sautter et al[4]	Present Report
Cause of botulism	Wound	Foodborne
Type of toxin	A	B
Clinic data		
Ophtalmoplegia	+	+
Dysarthria	+	+
Facial paresis	+	+
Dysphagia	+	+
Limb paralysis	+	-
Respiratory failure	+	+
Oral dryness	…	+
Constipation paralytic ileus	…	+
Bladder paralysis	+	+
Orthostatic hypotension	…	+
Paraclinic data		
Electroneurographical examination	+	+
Lumbar puncture	Normal	Normal
Presence of the toxin in the blood	…	+
Mouse inoculation	+	+
First Urodynamic investigation		
Bladder filing sensation	Normal	Normal
Compliance	…	Reduced
Detrusor activity	Acontractility	Acontractility
Vesicosphincter dyssynergia	…	+
Final pressure flow analysis		
Micturition volume, mL	600	596
Micturition time, s	…	80
Peak flow, mL/s	23.1	12.0
Residual urine, mL	0	0

*Plus sign indicates the presence of the condition; minus sign, the absence of the condition; and ellipses, no data available.

paralysis or respiratory failure, but he displayed several dysautonomic symptoms (orthostatic hypotension, oral dryness, constipation, and bladder paralysis). The diagnosis was established in the presence of the toxin in the blood and the positive mouse inoculation. Urodynamic investigations are not really comparable because details of the compliance and the dyssynergia were not provided in the other published case.[4] The final pressure flow analysis shows complete micturations while detrusor hypocontractility is still present. Recovery period was longer in our case, particularly for the bladder paralysis that lasted for 150 days.

Therapeutic approach for botulism is first intensive care (mechanical ventilation). Antitoxin therapy could be used in serious cases, which can stop progression of the paralysis. Antitoxin should be given early, ideally at the first 24 hours from the onset of the first symptoms. Antitoxin therapy is associated with adverse effects (anaphylaxis). Skin testing must be made to test sensitivity. In our patient, the diagnosis was made a week after the first symptoms, so it was too late to use antitoxin.[2]

Lastly, the botulinum toxin has been largely used in therapy for several years. In particular, it is an important treatment for dystonia, selective spasticity, and detrusor overactivity.[3, 5-7] For the detrusor hyperactivity, the toxin type A is predominantly used.[8-10] Toxin type B is also used, but duration of its therapeutic action is shorter than type A. This is somewhat contrasting with the delayed recovery due to foodborne botulism toxin type B in our patient.

CONFLICT OF INTEREST

None declared.

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