A case of foodborne botulism type E

Karolien De Leener[1], Bram Depelseneer[2], Jonas Moens[1], Dominique Vandijck[3]
Anne-Marie Descamps[1–3]

[1] Belgian Poison Centre, Brussels, Belgium
[2] ZNA Middelheim, Antwerp, Antwerp, Belgium Ghent, Belgium
[3] Ghent University, Faculty of Medicine and Health Sciences,

Objective

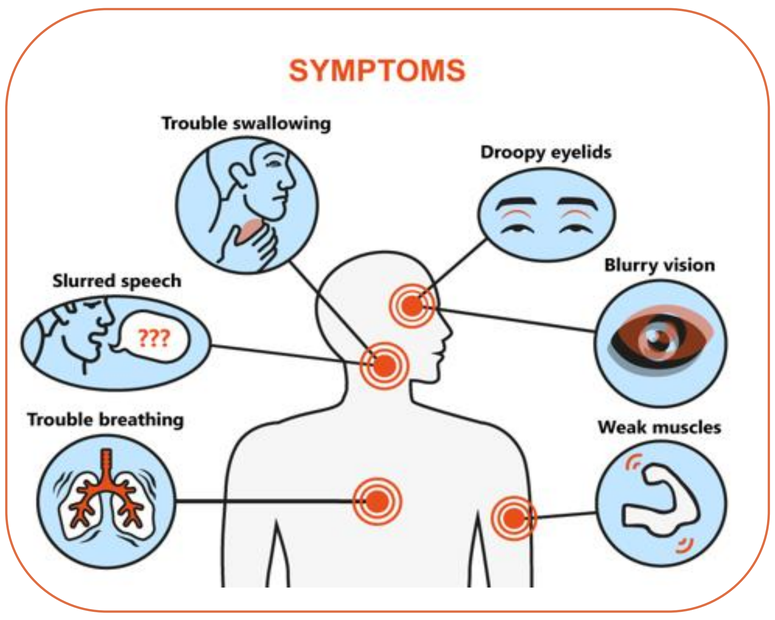
To present a laboratory-confirmed case of foodborne botulism type E. In Belgium botulism type B is predominant in humans.

Case report

A 67-year-old woman, known with arterial hypertension, Hashimoto thyroiditis and cholecystectomy, presented to the emergency department (ED) with sudden progressive weakness. The evening before, a reheated catfish meal (not canned) was consumed.
Physical examination showed the presence of multiple symmetrical cranial nerve palsies with diplopia, dysarthria, bilateral ptosis, hyperreflexia and weakness of the upper and lower limbs. Laboratory showed a slightly decreased renal function. Cerebral CT-scan showed no peculiarities.
Upon Intensive Care Unit (ICU) admission, faeces, gastric fluid and blood were sent to the national reference laboratory for botulinum testing. The Poison Centre was called and the botulism antitoxin was ordered. In the differential diagnosis myasthenic problems were also considered.
Respiratory distress developed with increased oxygen dependence followed by need of intubation and mechanically ventilation. Botulism Antitoxin Heptavalent BAT® was administered within 24-hours since appearance of symptoms.
Electromyography (EMG) suggested axonal motor neuropathy. Additional lumbar puncture was suggestive for Guillain-Barré, despite contradictory clinical presentations. Therefore, immunoglobulins intravenous were administered (day 3 and 4) but were stopped after confirmation for botulinum neurotoxins type E in the gastric fluid and blood. No neurotoxins were detected in the faeces, nor in the residue of the consumed meal.
The patient recovered from day 6 onwards.
On day 8 neurotoxins were no longer detectable in the blood, but still detectable in the faeces.
The patient was extubated on day 9. Because of remaining weakness, non-invasive ventilation was continued for 1 day. Because the patient could not get up nor walk independently, rehabilitation was started (day 13). A new EMG (day 27) showed normalisation of the situation.
Persistent hoarseness was noticed due to intubation, but this recovered completely.
The patient left the hospital fully recovered after 31 days

Conclusion

Early treatment with BAT® is able to neutralise circulating neurotoxins, positively influencing the course of disease. As tests to confirm the presence of neurotoxins in blood or faeces take several days, the decision to start prophylactic treatment should therefore be made based on the anamnesis and clinical presentation upon admission.
Due to the presence of a symmetrical, descending, flaccid paralysis the Poison Centre was called to deliver BAT® and treatment was started. The patient recovered completely.



CONTACT
karolien.de.leener@poisoncentre.be
Belgian Poison Centre
Bruynstraat 1, 1120 Brussels (Belgium)
(+32) 02 264 96 36

antigif centrum centre antipoisons
070 245 245