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Accidental Botulism Poisoning: A Case of Pickled Herring

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INTRODUCTION

Botulism is a rare cause of neuromuscular weakness that presents a diagnostic challenge in the face of respiratory collapse.

Pupil and bulbar paralysis aid prompt recognition and treatment, as clinical confirmation can be time intensive and limited by sample integrity. Early treatment can halt paralysis and prevent ICU and ventilator days.

CASE DESCRIPTION

A 74-year-old male with hypertension and DVT presented with acute weakness and respiratory failure after three days of cough and diarrhea. Upon ICU admission for mechanical ventilation, we discovered sluggish pupils, mild ptosis, and proximal muscle weakness. Symptoms then progressed to unresponsive pupils, complete ptosis, and complete paralysis.

While we suspected a neuromuscular cause, no single cause was identified despite extensive workup and neurology consultation. Our differential was narrowed after electromyogram (EMG) demonstrated a pre-synaptic defect, indicating either Lambert Eaton or Botulism as culprits. Empiric treatment with botulism anti-toxin resulted in clinical improvement of ptosis and proximal weakness.

While ventilated, he wrote of recent pickeled herring ingestion, possibly left unrefrigerated for a week. He ultimately received tracheostomy and transfer to long-term assisted care, and at discharge stool and serum testing for botulism toxin was still pending. While state-run testing finally returned negative, his illness was ultimately attributed to accidental botulism poisoning.

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DATA

Differential and Workup

Myasthenia Gravis
- CT Chest: no mediastinal/thymic mass (thymoma)
- Acetylcholine Receptor Ab: negative
- Striated Muscle and Titin Ab: negative

Lambert Eaton
- Pre-synaptic defect on EMG: low motor amplitudes that increased with sustained exercise
- Voltage Gated Calcium Chanel Ab: negative
- CT Chest: no nodules or masses (paraneoplastic SCLC in 50%)

Demyelinating Polyneuropathy
- LP: Glucose 76, protein 53, WBC 0, RBC 0, lymphocytes 67%
- CSF: Ganglioside GM1 Ab negative

Botulism
- Stool: rejected, poor specimen quality
- Serum: small sample size, Endopep-MS assay negative

Central and Infectious
- MRI brain and C-spine normal

Infectious
- Treponemal Ab negative
- Lyme Ab negative

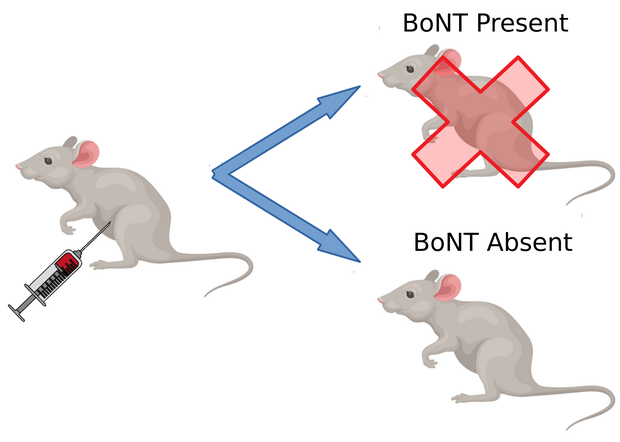
Botulism Key Features

Exam
- Respiratory collapse
- Proximal then distal paralysis, rapid progression
- Preserved sensation, reflexes
- Bulbar Paralysis with pupil involvement, nystagmus

Workup
- Botulism Neurotoxin (BoNT) of fluid, serum samples
- Mouse Bioassay

Treatment
- Botulism equine anti-toxin, early administration can reduce ventilator and ICU days

Sacrificial Mouse Bioassay



Sample suspected for Botulism Neurotoxin (BoNT) injected in peritoneum of mouse, compared to control. After 3-5 days incubation, presence of BoNT confirmed by rapid paralysis and death of mouse.

RESULTS / DISCUSSION

Yearly US incidence of botulism is 100 cases, and early treatment with equine anti-toxin reduces mortality and ventilation time[1]. Timely diagnosis is challenging as respiratory collapse overshadows subtle pupil and bulbar paralysis unique to botulism[2].

Lab diagnosis with Endopep-MS assay is not FDA approved and can be falsely negative with small samples[3] while verified sacrificial mouse bioassays are rarely used[2]. Early suspicion should prompt anti-toxin treatment and discussion with the CDC's botulism consultation service, who will supply anti-toxin and provide assistance with workup in conjunction with state health laboratories.