Infant Botulism due to Consumption of Contaminated Commercially Prepared Honey

First Report from the Arabian Gulf States

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Key Words
Infant botulism • Honey • Acute flaccid paralysis • Clostridium botulinum

Abstract
Objective: To report the first case of infant botulism in Arabian Gulf States. Clinical Presentation and Intervention: A 6-week-old infant, presenting with signs of sepsis, was intubated and ventilated due to progressive weakness. Infant botulism was suspected with acute flaccid paralysis and a history of honey consumption. An electromyogram showed decreased amplitude of compound muscle action potential in all motor nerves, preserved sensory responses; the motor terminal latencies and motor conduction velocities were normal. Blood, stool and honey samples were sent for culture. Stool and honey cultures showed two identical strains of Clostridium botulinum. Conclusion: This case shows that the infant botulism occurred from the ingested contaminated honey. Hence vigilance should be maintained when a baby is fed honey and shows signs of progressive weakness because the disease can quickly progress to respiratory failure.

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Introduction

Infant botulism can be acquired by ingesting spores of Clostridium botulinum, which are found in soil and honey products [1]. The spores germinate into vegetative forms in the intestinal tract and then synthesize neurotoxins. The toxins bind irreversibly to terminal axons and prevent presynaptic release of acetylcholine [1]. In some cases infant botulism has been associated with other Clostridium species than botulinum, e.g. Clostridium baratii or Clostridium butyricum [2].

Clinical presentation includes constipation, poor sucking, weak cry, progressive weakness (initially bulbar), hypotonia, hyporeflexia and respiratory failure. Spontaneous recovery occurs with the regeneration of new presynaptic motor endplates [1, 3].

Epidemiologically, infant botulism is more commonly seen in the United States than anywhere else in the world. However, some cases have been reported from 13 other countries [1, 4, 5].

We describe a case of infant botulism caused by consumption of commercially produced honey, purchased at a local supermarket. As far as we know this is the first report of infant botulism from the Arabian Gulf States.
Case Report

A 6-week-old Chinese male infant presented to the Pediatric Emergency Department with a 2-day history of decreased activity, weak cry and poor feeding.

Initial vital signs upon presentation were a respiratory rate of 30/min, heart rate of 120/min, blood pressure of 100/50 mm Hg, Glasgow Coma Scale of 10 (E3M3V2) and central temperature of 36°C. Further physical examination was unremarkable except for a prolonged capillary refill time of 5 s.

Initial laboratory investigations showed an arterial blood gas with a pH 7.29, PaCO₂ 6.15 kPa, PaO₂ 28.5 kPa, HCO₃⁻ 21.7 mmol/l, BE −4.7 mmol/l, O₂% 100%, white blood cells (WBC) of 9.5 × 10⁹/l, hemoglobin of 9.8 g/l and a platelet count of 446 × 10⁹/l. Electrolytes, urea and creatinine were within normal ranges. The cerebrospinal fluid (CSF) showed no WBC, and red blood cells (RBC) of 2,035/mm³. Blood, urine and CSF samples were sent for bacterial cultures and virological tests. The chest X-ray was unremarkable.

In the emergency department the patient was given supplemental oxygen by mask, normal saline fluid bolus and intravenous cefotaxime and amikacin. Thereafter the infant was transferred to the Pediatric Intensive Care Unit for further management. A few hours after admission the patient was intubated and ventilated for hypventilation due to progressive weakness.

The past medical history was unremarkable, except that the infant was fed honey daily one teaspoon for 2 weeks prior to admission because of constipation. Unfortunately, the brand and country of origin of the honey were not recorded in the patient’s file.

In the days after admission the infant became increasingly hypotonic, finally showing only movement of fingers and toes, and neurological examination revealed facial diplegia, ophthalmoplegia, ptosis, dilated pupils with minimal response to light, flaccid paralysis of bulbar, axial, limb and respiratory muscles, and depressed deep tendon reflexes. There was no fasciculation of the tongue. At that time the presumptive diagnosis of acute flaccid paralysis (AFP) was made. Additional investigations included complete blood count, cultures (blood, CSF, urine, stool and honey), lumbar puncture, electromyogram (EMG), Tensilon test, thyroid function test and screening tests for metabolic disorders and intoxications were performed. The complete blood count showed WBC of 4.7 × 10⁹/l, hemoglobin of 12.4 g/l and platelet count of 346 × 10⁹/l. The CSF showed no WBC, and RBC <5/mm³, glucose of 3.6 mmol/l with serum glucose of 5.8 mmol/l, lactate 0.5 mmol/l and protein of 577 mg/l (n ≤ 450 mg/l). The Tensilon test was negative as were the routine bacterial cultures, thyroid function test, screening tests for metabolic disorders and intoxications. The EMG showed decreased amplitude of compound muscle action potential (CMAP) in all motor nerves, preserved sensory responses; the motor terminal latencies and motor conduction velocities were normal for age. Repetitive nerve stimulation at low rate of stimulation (3 and 5 Hz) revealed very little increment (2.6–5.2%), but at high rate (30 and 50 Hz) significant increment (29.1–40.8%) of CMAP. Preliminary diagnosis of infant botulism made on day 4 of admission was based on the clinical signs of AFP, history of honey consumption, and EMG findings. On the same day, PCR of stool sample was requested and the result received on day 39 of the admission was positive for Enterobacter aerogenes. The late result of PCR did not delay the diagnosis of infant botulism as the diagnosis was confirmed by cultures of stool and honey samples. Two identical strains of C. botulinum were identified in our Anaerobic Reference Laboratory, Faculty of Medicine, Kuwait and confirmed at the International Anaerobic Reference Laboratory in Cardiff, UK. The patient remained on ventilation for 67 days and was fed by nasogastric tube. The patient experienced two failed trials of extubation due to upper airway obstruction caused by bulbar muscle weakness on day 24 and day 38. In addition he developed hospital-acquired pneumonia.

On discharge 3 months after admission, he was still hypotonic and required nasogastric tube feeding. However, by the end of the 3rd month after discharge the neurological examination was unremarkable. The follow-up has been uneventful.

Discussion

Since 1976 cases of infant botulism have been described worldwide, however 90% of the cases have been reported in the United States [4, 6]. To our knowledge this is the first report on infant botulism in the Gulf States. The recognized sources of botulimum spores are soil and honey; however, Nevas et al. [7] reported vacuum cleaner dust as a source.

Initially we suspected neonatal sepsis/meningitis in our patient and he was treated accordingly. However, the clinical course, progressive hypotonia, and laboratory results were not in agreement with the diagnosis of neonatal sepsis/meningitis but in favor of AFP. Moreover the history revealed that the infant was fed honey prior to the onset of symptoms. Evidence in the literature showed that honey consumption was associated with 15% of the reported cases of infant botulism to the Centers for Disease Control and Prevention (CDC) [6]. Although infant botulism was highly suspected, the minimal increased protein concentration in the CSF was suspicious for Guillain-Barré syndrome, which is a frequent cause of AFP [8]. However Guillain-Barré syndrome is very rare in infancy, even though congenital forms have been described. Based on this differential diagnosis, the patient received immunoglobulins.

The EMG of our patient showed the characteristic features including decreased amplitude of CMAP in at least two muscle groups, tetanic and posttetanic facilitation by an amplitude of >120% of baseline of the CMAP and prolonged posttetanic facilitation of >120 s and absence of posttetanic exhaustion [9]; however, the characteristic features of the EMG have a variable sensitivity, especially in the early stage of the disease, and therefore a stimulation single-fiber EMG should be considered in any suspected case of infant botulism [10].

The California Department of Health Services was consulted for the administration of human botulinum

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immunoglobulin [3]. Human botulinum immunoglobulin, which interrupts neuromuscular blockade, has shown marked reduction in the length of hospital stay and the rate of intubation [1].

At that time, day 10 of admission, the patient was showing some improvement and therefore the decision was made that human botulinum immunoglobulin, only beneficial if given in the early stage, was not indicated at this point any more. Preventive medicine staff were informed; they excluded environmental factors as a cause and investigated the source of the honey thoroughly to avoid further cases.

The diagnosis of acute poliomyelitis was considered after a positive PCR for Enterovirus in the stool was received [8]. As a result appropriate preventive measures were taken according to the protocol of the World Health Organization (WHO). Eventually, the Enterovirus in stool was typed as Sabin-like virus at the regional WHO reference laboratory in the Netherlands. Since the clinical picture, the CSF and EMG findings were not in line with the diagnosis of acute poliomyelitis, the stringent infection control measures were stepped down. The positive PCR for Enterovirus in the stool might have been due to the fact that the patient, who received live attenuated oral polio vaccine after birth, was still shedding the virus [11].

On day 24 an extubation trial was performed since the patient was on pressure support ventilation and showing movements of all four limbs. Reintubation was required due to severe upper airway obstruction as a result of bulbar muscle weakness. On day 38 another trial of extubation was performed, since the patient had clear facial expression and no signs of residual ptosis, but it also failed due to remaining bulbar muscle weakness. On day 67 the patient was successfully extubated. After extubation the patient exhibited an inspiratory stridor, which was successfully managed with dexamethasone nebulization.

Infant botulism is unique in infants, with cases occurring between 6 days and 12 months of age. Our patient was 6 weeks of age at presentation. Risk factors for infant botulism are multifactorial. Consumption of honey is a clear risk factor and this was the case in our patient [3].

In our patient, the diagnosis of infant botulism was confirmed by demonstrating the presence of identical C. botulinum strains in the stool and honey. Hence the consumed honey was recognized as the source of infection. As has been reported, up to 25% of the honey products in the USA contain spores of C. botulinum [1, 12]. This has guided the CDC to advise that honey should not be fed to infants less than 1 year of age, who are susceptible to infant botulism because of the immaturity of the microflora of the intestine.

Conclusion

This case shows that the infant botulism occurred from the ingested contaminated honey. Hence vigilance should be maintained when a baby is fed honey and shows signs of progressive weakness because the disease can quickly progress to respiratory failure.

References