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Clinical Letter

Infant Botulism With Asymmetric Cranial Nerve Palsies

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Patient presentation

This for term born, previously healthy, developmentally normal eight-month-old girl presented after four days of progressive episodic symptoms including altered mental status characterized by being less interactive, neck weakness, and weak suck and cry following one week of cough and runny nose. The parents also reported intermittent left ptosis and a right facial droop. There was no history of decreased urine output or weight changes. The frequency of bowel movements decreased slightly in the week before presentation. The patient had been solely breastfed until solid food was introduced at six months of age. The family denied honey exposure. Family history was noncontributory, including no weakness, neuromuscular disease, or neurodegenerative disorders.

Initial examination in the emergency center was notable for weak suck and cry, right nasolabial fold flattening, left ptosis, dilated left pupil with sluggish response and pupillary fatigue, and difficulty sitting unsupported. Extraocular eye movements, gag, and swallow were intact. Skin turgor was normal, and she had moist mucous membranes. Deep tendon reflexes and extremity strength were normal. Cranial computed tomography, done because of her decreased activity level with focal neurological findings, was normal. Results of complete blood count, chemistry, urine analysis and urine drug screen, and brain magnetic resonance imaging were also normal. Stool botulism testing was initiated, and she was admitted for observation.

The patient remained stable with slight improvement in feeding and without signs of respiratory distress or progression of cranial nerve palsies. Parents declined electromyography testing given her overall well appearance, and she was discharged home after 48 hours. After one week the stool sample was reported positive for *Clostridium botulinum* type A toxin. The family was notified of the diagnosis by phone and denied progression of symptoms. The patient continued to make slow but steady clinical improvement and had no signs or symptoms at the time of her follow-up clinical examination four weeks after hospital discharge.

Discussion

Infant botulism is an uncommon illness with an incidence of 1.9 per 100,000 live births per year.[1,2] It is well known to pediatricians for its most severe and potentially life-threatening form in which infants six weeks to six months of age present with constipation, weakness, symmetric pupillary, and bulbar dysfunction, manifesting as poor feeding, a weak cry, and ptosis with dilated pupils followed by a descending paralysis leading to respiratory failure in 75% of cases.[3] This classic severe presentation of infant botulism is easily recognized and therefore infrequently misdiagnosed, with numerous case reports describing such a presentation.[2,4] Honey exposure is the only dietary reservoir definitively linked to infant botulism, although a lack of identified dietary exposure is common and does not exclude the diagnosis.[1] Although milder disease characterized by poor feeding and

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decreased bowel movement frequency occurs, the literature and educational curricula typically focus on the “catastrophic” end of the clinical spectrum.[1,5]

We describe the first child with asymmetric cranial nerve palsies due to infantile botulism among the more than 2943 reported cases worldwide.[6] Our patient's atypical asymmetric cranial nerve palsies and relatively mild clinical course support a wider clinical spectrum for infant botulism than is currently described in the literature. Similarly, the incidence of infant botulism may be higher than now reported because many patients go unrecognized because their mild symptoms result in low clinical suspicion for botulism.[6] The diagnosis of infant botulism should be considered even in an infant older than six months with mild pure motor weakness involving the neck muscles or asymmetric cranial nerve involvement.

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References

1. Arnon SS. Infant Botulism. In: Cherry J, Demmler-Harrison G, Kaplan S, Steinbach W, Hotez P, eds. Feigin and Cherry's Textbook of Pediatric Infectious Diseases. 7 ed. Elsevier Saunders; 2013:1801–1809.
2. Domingo RM, Haller JS, Gruenthal M. Infant botulism: two recent cases and literature review. J Child Neurol. 2008;23:1336–1346.
3. Tseng-Ong L, Mitchell WG. Infant Botulism: 20 years' experience at a single institution. J Child Neurol. 2007;22:1333–1337.
4. Francisco AM, Arnon SS. Clinical mimics of infant botulism. Pediatrics. 2007;119:826–828.
5. Rosow LK, Strober JB. Infant Botulism: review and clinical update. Pediatr Neurol. 2015;52:487–492.
6. Koepke R, Sobel J, Arnon SS. Global Occurrence of Infant Botulism, 1976–2006. Pediatrics. 2008;122:e73–e82.