Treatment of infant botulism

Thompson et al. review their experience with 39 infants with botulism. Treatment with botulism immune globulin shortened the hospital stay by an average of $2^1/_2$ weeks.

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Infant botulism: Epidemiology and treatment with BIG

Commentary by John T. Sladky, MD

Infant botulism is one of three toxin-mediated disorders caused by *Clostridium botulinum*. The disease results from the introduction of *C botulinum* spores into the infantile gastrointestinal tract, which then germinate into the vegetative state with subsequent elaboration and absorption of the toxin. The spores are ubiquitously found in soil or may be encountered in agricultural materials such as fresh produce or honey. Like the clostridial spores, the disease occurs worldwide although it has a higher prevalence in discrete geographic locations: California, Pennsylvania, and Utah. It affects infants within the first days of life and up to 1 year but has a peak incidence at around 14 weeks of age.

Infant botulism was first identified in patients from California where it is more common than in most other regions of North America. Because of its stereotypical clinical presentation, the disorder was recognized as a distinct entity earlier among infants in eastern Pennsylvania,[1] another area with an increased prevalence of infant botulism. The causal relationship to *Clostridium* botulism awaited the article by Pickett et al. in 1976.[2] Thompson et al. describe their experience with infant botulism over a 17-year period in Utah, the third recognized hotbed of infant botulism in the United States. They present evidence that the antitoxin botulism immune globulin (BIG) can reduce the duration of hospitalization for these infants compared to the supportive care that was standard prior to the addition of BIG to the therapeutic armamentarium.

Clostridial species are classified based on the nature of elaborated toxin, with infant botulism reported after infection with types A, B, E, and F.[3] The vast majority of cases are related to infection with types A and B. Type A predominates west of the Mississippi river valley while type B is most common in the East. Both toxins act on distal axons at cholinergic nerve terminals to block synaptic transmission. Although similar, these toxins have distinct structures and mechanisms of action which, in part, accounts for different clinical characteristics among affected infants infected with type A or B (including the absence of papillary involvement and preservation of deep tendon reflexes noted in the Utah cohort in comparison to infants from eastern Pennsylvania). The development of BIG appears to provide a useful modality to reduce morbidity in infant botulism.

References

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