BMJ Case Reports

Myth exploded

Infant botulism following honey ingestion

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Summary

An apparently well baby girl born at term was presented with signs and symptoms suggestive of acute onset of generalised floppiness at the age of 3 months. Clinically, the baby had lower motor neuron type of muscle weakness; detailed investigation lead to the diagnosis of neuromuscular junction disorder secondary to botulism toxicity. Further tests confirmed the botulism toxicity secondary to honey ingestion. The baby was treated with specific anticlostridium antibodies; she recovered remarkably, now growing and developing normally.

BACKGROUND

This case illustrates the importance of considering botulism toxicity when an infant present with acute floppy paralysis and taking history of honey ingestion as a cause for botulism toxicity.

This manuscript fulfils the BMJ case reports criteria 'myth exploded'; honey is thought to be having great medicinal value in Asian communities; this report conveys important messages that

(1) giving honey routinely to infants is not safe.
(2) healthcarers need to concentrate on health education 'prevention is better than cure' as well as especially on communities where such customs are prevalent.

CASE PRESENTATION

A previously well 3-month-old girl presented with 10 days history of poor feeding, constipation, oral thrush, cough and floppiness. The baby was born at term by ventouse delivery following an uneventful pregnancy, birth weight 3.3 kg; she is the first child of non-consanguineous parents of Pakistani origin; her developmental milestones were age appropriate.

On examination, she was noted to have generalised hypotonia, decreased deep tendon reflexes, bilateral facial weakness, partial ptosis, weak head control, poor feeding, weak cry and minimal antigravity movements of limbs; she was admitted for further management.

INVESTIGATIONS

Full blood count, serum electrolytes, liver function tests, thyroid function tests, C reactive protein, blood urea, creatine kinase, chest x-ray, electrocardiogram, electroencephalogram and cerebrospinal fluid analysis were all normal.

Colon fully loaded with faeces was seen in the abdominal x-ray; brain MRI was normal; the nerve conduction study showed normal sensory and motor tracings; needle electromyography (EMG) found small, short and polyphasic motor unit action potentials; stimulation single fibre EMG study of the right orbicularis oculi revealed severe abnormality with increased jitter and block in 90% of the recorded potentials (figure 1). Repetitive nerve stimulation, poorly tolerated by the infant was suggestive of decrement in the left abductor digiti minimi but with no facilitation, findings characteristics of neuromuscular junction disorders botulism, or congenital myasthenia.

Diagnosis was confirmed by real-time PCR detection of *Clostridium botulinum* type A neurotoxin genes in faecal specimens from the infant with subsequent isolation of *C botulinum* type A. Two samples of remnant honey from the bottle that had been fed to the infant were examined and both were found to contain *C botulinum* type A spores. Molecular typing, by fluorescent amplified fragment length polymorphism, showed that the *C botulinum* type A isolates from the honey samples were indistinguishable from the *C botulinum* type A strain from the infant but were different from *C botulinum* type A isolated from other UK cases of infant botulism. *C botulinum* was not detected in a sample of the infant's formula milk powder.

TREATMENT

Human-specific botulinum immunoglobulin was administered to this baby following which she made a gradual recovery and was discharged home.

OUTCOME AND FOLLOW-UP

Completely recovered; follow-up assessment showed baby growing and developing normally.

DISCUSSION

Infantile botulism was first described separately in 1976 by Midura and Arnon and by Pickett *et al*[1 2]: the illness is believed to have existed before this time, but was not recognised. The condition has been reported from all continents except Africa, possibly due to difficulty in diagnosing the disease rather than absence of the illness.[3] In USA, the country reporting the highest number of cases, infantile botulism is the most common presentation of acute botulism. Infantile botulism is rare in UK; our patient is

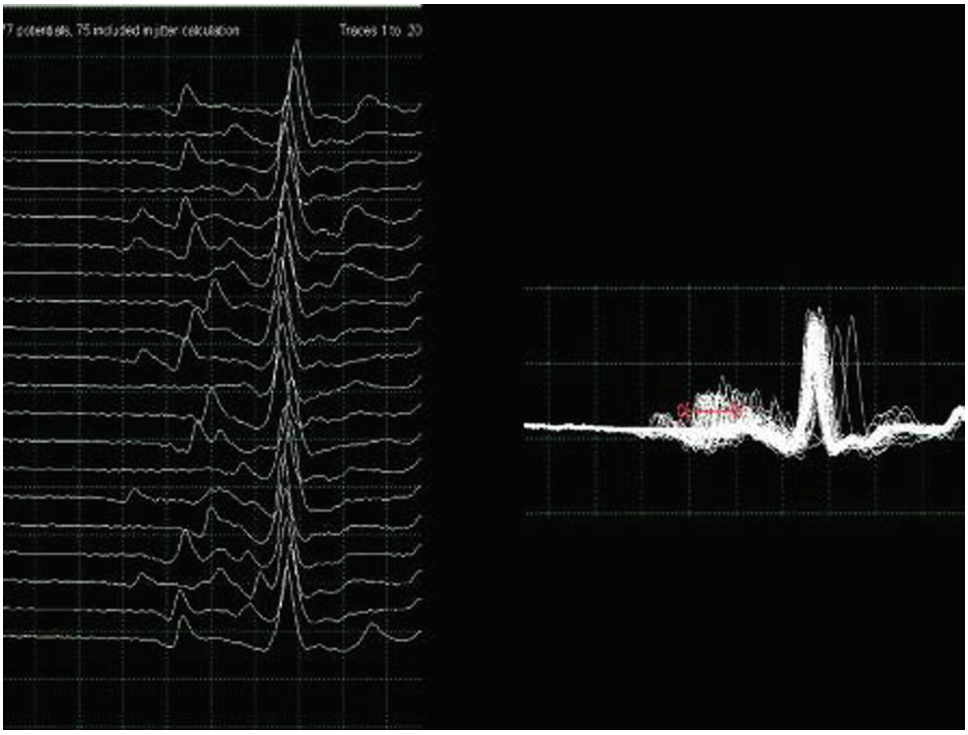

Figure 1 Stimulation single fibre electromyography (Stim SFEMG) study of right orbicularis oculi. The figure on the left shows a faster display of single fibre muscle action potentials and the right shows the superimposed display. The highlighted units (big white arrows) have an increased jitter measurement with mean consecutive difference (MCD) of 145 and 45 μs. The potential on the left is also blocking.

the twelfth confirmed case with eleven cases reported up until June 2011.[4 5] Infantile botulism tends to affect babies between the ages of 2 and 6 months but has been reported in infants as young as 54 h and as late as 1 year. Both *C botulinum* and its neurotoxin can be excreted in the faeces for weeks to months; there were no reports of person-to person transmission.

The clinical presentation can vary from mild hypotonia to severe bulbar paralysis, and to sudden infant death.[6] The typical presenting symptoms include constipation, followed by lethargy, listlessness, poor feeding, ptosis, dysphagia, and loss of head control, hypotonia, visual problems, dry mouth and generalised weakness. Botulism can lead to paralysis lasting for days and weeks, and in some cases to respiratory failure. Fever is usually absent. Once the diagnosis is clinically suspected single- fibre EMG studies helps, it typically reveals increased jitter and block. The compound muscle action potentials (CMAP) are usually small in amplitude with normal latency and velocity. Slow repetitive nerve stimulation at 3 Hz may show a decrement with a significant increment or facilitation following exercise or high frequency stimulation (figure 1). However, in severe botulism, the neuromuscular junction may be so blocked that facilitation or increment in CMAP may not be seen.[7 8]

Diagnosis of infantile botulism can be confirmed by real-time PCR detection of *bont* genes in enrichment cultures from infant faeces or rectal wash out, by isolation and identification of *C botulinum* or more rarely other neurotoxigenic clostridial species, or by detection of botulinum toxin (BoNT) in infant faeces.[5 9]

The spores of BoNT-producing clostridia are widespread in soil, dust and aquatic sediments and infants are considered to be repeatedly exposed. Despite this, infantile botulism is a rare disease and some other factor, such as a disturbance of the infant gut flora, is thought to be providing a window of opportunity for any spores present to germinate, colonise and produce toxin. For most cases of infantile botulism, the source of spores is never identified and it is assumed that they are swallowed from the environment. However, honey is a dietary reservoir of *C botulinum* spores for which there is both microbiological and epidemiological evidence.[10] In order to minimise the risk of infantile botulism, it is recommended not to give honey to less than 1 year old. There is a widespread practice of administering honey or 'ghutti' (an herbal concoction mixed with honey) as a prelacteal feed to newborn babies among Asian families. In a study conducted in Pakistan, 15.6% of babies received honey as prelacteal feeds, often influenced by the elders in the family.[11] A similar study from India reported most of the grandmothers and mothers believed in early feeding of newborn, within 2 h of delivery, by giving prelacteal feeds such as ghutti and honey.[12]

The diagnosis of infantile botulism should be considered in any infant presenting with acute floppiness and history of constipation. The practice of feeding honey should be enquired about, especially in non-Caucasian children. In order to minimise the risk of infantile botulism, public health measures need to be taken such as educating parents, community health visitors, midwives etc against feeding honey to infants.

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Learning points

- Babies presenting with acute floppiness and constipation, infantile botulism should be considered.
- Following approach helps: history of feeding honey, especially in communities with this custom is prevalent, early detailed electrophysiological studies, testing the honey bottle for botulism toxins and educate parents, health visitor, midwives etc regarding feeding honey to infants.

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Competing interests None.

Patient consent Obtained.

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