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BRIEF REPORTS

A CASE OF INFANT BOTULISM ASSOCIATED WITH HONEY FEEDING IN ITALY

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A case of infant botulism in a 9 week-old female is described. A strain of *C. botulinum* type B was isolated from the feces of the baby. The epidemiologic study detected in a sample of home canned honey *Clostridium botulinum* spores of the same serotype that was isolated from the patient. The honey had been used only to sweeten the pacifier of the baby. This is the first case of infant botulism in Europe linked conclusively to honey.

Infant botulism results when spores of *Clostridium botulinum* germinate and multiply in the gut of a child less than one year old, producing botulinal toxin *in vivo*.

Since 1976, more than 1000 cases of infant botulism have been reported all over the world, mostly recorded in North America.

Until now, honey is the only identified food associated with the onset of infant botulism cases, although this accounts only for a low number of cases in North America (2, 3) and, recently, in Japan (20). Moreover, various surveys have shown the presence of spores of *C. botulinum* in retail honey samples (12, 14, 15, 16, 22). For this reason, the American Academy of Pediatrics, the Centers for Disease Control and the FDA recommend not giving honey to children less than one year old (1).

In Europe, on the contrary, only a few cases of infant botulism were described and the infection source was never identified. All the surveys (4, 6, 11, 13, 18) from retail honey samples failed to show the presence of botulinal spores. Only a recent Italian survey on honey contamination described the isolation of *C. botulinum* type B spores from two of thirty retail Sicilian samples (9). We are reporting the first European case of infant botulism in which it was possible to identify the source of botulinum spores.

In January 1991, a 9 week-old female was admitted to the emergency room of the Pediatric Hospital in Trieste with a 10-day history that began with constipation and was complicated with difficulty in swallowing, a weak cry and lethargy. The patient, born after a normal pregnancy and delivery, weighed 3,450 g at birth. She was totally breast fed for one month and subsequently a milk formula was introduced. Honey was also used to sweeten the pacifier. On admission she showed respiratory distress, generalized hypotonicity with no head control or trunk extension on ventral suspension. Cranial nerve examination demonstrated bilateral ptosis, sluggishly reactive pupils and an absence of upward gaze and gag reflex. Deep tendon reflexes were reduced. Cerebrospinal fluid was normal and no paroxysmal discharge was found by electroencephalography. Electromyography demonstrated a defect in neuromuscular transmission of the type previously described in infant botulism (19). Infant botulism was suggested from the above clinical findings.

Three days after hospitalization, a first stool sample obtained by enemas was examined only for the

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presence of *C. botulinum* spores because of the inadequate sample size. The diagnosis of botulism was confirmed by the demonstration of *C. botulinum* type B spores (200,000 spores/g of stool). Spore counts of *C. botulinum* were carried out by the three tubes most probable number (MPN) test. The fecal sample was diluted in gelatin phosphate buffer and serial 10-fold dilutions were inoculated into cooked meat medium tubes. The first dilution was treated at 80° C for 10 min. The tubes were incubated in anaerobic conditions at 35° C for seven days. All growth-positive tubes were centrifuged and submitted to the mouse test for toxin research. This test, as well as the isolation of *C. botulinum*, were performed according to standard procedures of the Centers for Disease Control (7). Gas liquid chromatography on the isolated strain, as described by Dowell and Hawkins (10) confirmed the identification of a *C. botulinum* type B proteolytic strain. Seven days later another stool sample was tested and type B toxin and *C. botulinum* spores were found (7,000 spores/g of stool). No serum sample from the patient was obtained.

The patient received neither botulinum antitoxin nor antibiotics. She was treated with lactulose and freezer-dried lactic acid bacteria. Twenty days after admission, the patient was discharged from the hospital with only persistent constipation.

In the epidemiologic investigation of the source of *C. botulinum* spores, we received only three samples of leftover honey, two retail samples and a home canned sample. The home canned sample was positive for *C. botulinum* type B spores (positive culture with a 10 g sample). The method used for spore assay was the MDL-10 reported by Midura et al. (16).

It was not possible to quantify the amount of contaminated honey the baby was fed, however the mother reported having used it to sweeten the pacifier only three or four times ten days before the development of constipation.

The parents obtained the contaminated honey from a local processor. We were not able to recover further samples from the processor because they were sold out.

The wide distribution of botulinal spores is well documented all over the world. In Italy, strains of *C. botulinum* type A and B were isolated from soil and aquatic sediments (5, 21, 8). The presence of botulinal spores in the environment can cause the contamination of honey, particularly during nectar collection when the bees may come in contact with spores on flowers. The possibility of multiplication of *C. botulinum* spores, depending on the suitable conditions of pH and Aw, during honey maturation in the beehive has also been demonstrated (17).

Although a heavily contaminated honey is probably necessary to cause disease (23), and individual susceptibility remains unknown, it seems advisable for pediatritians to suggest, as in USA, that honey should not be fed babies in their first year of life.

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