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# Four Months Old Baby Boy with Descending Paralysis

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**Food-borne infantile botulism is associated with symptoms of cranial nerve palsies and descending paralysis in infants especially in the first six months of age.**

**We report a four months old boy, who presented to our clinic with history of reduced activity, poor feeding and constipation.**

**The serological and microbiological tests were negative for botulinum toxin but the neurophysiological study was highly suggestive of infantile botulism.**

**The clinical condition showed gradual improvement with supportive therapy.**

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Infant botulism is a major cause of neuroparalytic disease caused by neurotoxin released by *Clostridium botulinum* spores[1,2]. The source of neurotoxin found primarily in honey, canned food or soil. Honey is the main reservoir of *C. Botulinum* spores and it is linked to infant botulism. Honey should not be fed to infants less than one year of age. Infant botulism is considered to be one of the rare causes of descending paralysis in infants worldwide.

Infant botulism was first recognized in 1976, and is the most common form of botulism in the United States. Eighty to one hundred cases are diagnosed with infant botulism in the United States each year. Infants are susceptible to infant botulism in the first year of life, more than 90% of cases occurring in infants younger than six months.

Food-borne infantile botulism typically presents with symptoms of reduced activity, poor sucking/feeding and constipation, it is associated with cranial nerve palsies such as ptosis, mydriasis, poor gag reflex and symmetrical descending flaccid paralysis[3].

The diagnosis of infantile botulism should be based on clinical history, physical examination and neurophysiological studies especially incrementation on stimulation of single-fiber electromyography even in the absence of positive serological and microbiological tests[4].

The aim of reporting this case is to stress the importance of the neurophysiological studies in diagnosing infant botulism along with the clinical manifestations and physical examination, even with negative laboratory confirmation.

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## THE CASE

Four months old baby boy delivered by normal spontaneous vaginal delivery presented to our neurology clinic with four weeks history of reduced activity, poor feeding and constipation. There was no history of fever, honey ingestion, vomiting, respiratory symptoms or abnormal movements. Prenatal history shows uneventful pregnancy with normal fetal movements. Family history was unremarkable.

Physical examination showed normal vital signs. Growth parameters were appropriate for his age. The patient was alert, can fix and follow with no dysmorphic features.

Neurological examination revealed no neurocutaneous stigmata and no fasciculation. The pupils were sluggish, reactive to light with weak gag reflex.

The patient was hypotonic and his deep tendon reflexes were depressed. The rest of the examination was unremarkable.

All hematological and biochemical results were normal. The DNA study for spinal muscular atrophy was also normal. All serological and microbiological investigations were negative for botulinum toxin. The electromyography and nerve conduction study showed incremental response with high-rate repetitive nerve stimulation which was highly suggestive of infantile botulism.

The patient was kept on nasogastric tube feeding and started on lactulose with gradual improvement in his clinical condition.

## DISCUSSION

Infant botulism is rare, but a major cause of neuroparalytic disease caused by ingestion and absorption of neurotoxin released by clostridium botulinum. Cox et al stated that the incidence of infant botulism is rare worldwide[1]. McLauchlin et al have reported sixty-two cases of food-borne botulism between 1922 and 2005 caused by ingestion of botulinum neurotoxin[2]. Our patient was the first case of infant botulism to be reported from our hospital, similar to what has been documented by the above authors that infant botulism is a rare cause of neuroparalytic disease.

Food-borne infant botulism most commonly occurs in infant less than twelve months of age, our patient presented to us at the age of four months and according to King et al the average age of clinical presentation of affected infant was hundred and nineteen days[5].

The typical clinical presentations of infantile botulism include reduce activity, weak sucking/feeding and constipation, cranial nerves involvement in the form of mydriasis, ptosis and poor gag reflex, which progress to descending symmetrical flaccid paralysis. Our patient presented with lethargy, poor feeding and constipation along with cranial nerves affection (the third and the ninth) and hypotonia. Hoffmann et al reported that the main clinical manifestations of infant botulism are cranial nerve palsies and descending symmetrical flaccid paralysis[3].

Despite the fact that honey ingestion remains the main source of infant botulism, negative history of honey ingestion does not rule out infantile botulism; our patient had no history of honey ingestion, but the clinical history was strongly suggestive of infantile botulism. Koepke et al suggested that the possibility of infantile botulism should be suspected by physicians based on clinical presentation, even with negative history of honey ingestion[6].

Although the diagnosis of infant botulism depends mainly on identification of botulinum toxins in the patient serum and stool specimens, neurophysiological studies which show incremental response with high rate repetitive nerve stimulation on electromyography can confirm the diagnosis of infantile botulism especially when serological test is negative[7]. Kissani et al described cases of botulism based on neurophysiological studies and had no laboratory confirmation, similar to our case[4].

The management of food-borne infant botulism is mainly supportive therapy; our patient showed gradual recovery with supportive therapy. Gutzwiller et al reported that the primary treatment of botulism is supportive care along with antitoxin therapy and ventilator support if needed[8].

## CONCLUSION

**We were not able to identify the source of botulinum toxins in our patient and despite that serological and microbiological tests were negative, the history, physical examination and neurophysiological studies confirm the diagnosis. Physicians should keep high index of suspicion of infantile botulism based on clinical presentations, physical examination and neurophysiological studies especially in sero negative patients.**

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**Competing interest:** None **Sponsorship:** None

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