

INFANTILE BOTULISM PRESENTING AS ENCEPHALOPATHIC RESPIRATORY FAILURE

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INTRODUCTION

Infant botulism is a rare, life-threatening condition with only 19 cases in the UK reported over the last 50 years. Complete recovery is possible with prompt diagnosis and management. Initial symptoms may be non-specific with fluctuating weakness and thus the diagnosis is often delayed. We present a case of Infantile botulism with an unusual clinical presentation alongside a review of literature.

CASE SUMMARY

A four-month-old girl who had been previously fit and well presented with 3-day history of **lethargy, poor suck and droopy eyelids**. She was treated as a meningoencephalitis with antimicrobials. CSF analysis, MRI brain and EEG were all normal.

Following initial improvement in sensorium, muscle tone and power she had an **acute respiratory deterioration** associated with metapneumovirus infection on day 9 of admission. She was endotracheally intubated, mechanically ventilated and transferred to PICU.

Further clinical assessment and investigations revealed a **flaccid paralysis**. **Clostridium botulinum toxin was detected in her stool sample**, which was sent 20 days following admission. **BabyBIG** was administered within 3 days of diagnosis. She was extubated after 36 days onto non-invasive ventilation and **discharged home after 143 days** when she had made an almost full neurological recovery, requiring only nasogastric feeding and nocturnal respiratory support.

DISCUSSION

The spores of *Clostridium botulinum* are widely present in the environment (such as soil, pet terrapins, vacuum cleaners) and food products including honey, canned food and corn syrup. In our case, the mother gave a history of using **ayurvedic medicine mixed with honey** for augmenting lactation. In infants, the botulinum neurotoxins pass through immature intestine barrier to the circulation. They block acetylcholine release in neuromuscular junction leading to **flaccid descending paralysis**.

The disease affects children below 12 months of age with the majority of cases between 3 and 4 months of age. Species of *Clostridium botulinum* vary from A to F, the ones responsible for infant botulism are most commonly types A and B.

A child presenting with poor feeding or flaccid weakness (“floppy baby syndrome”) should raise the suspicion of Infantile botulism. Other reported **symptoms** include constipation, bulbar palsies, hypotonia, weakness, loss of head control and difficulty in breathing ranging from mild to severe.

The **diagnosis** is confirmed following isolation of *C. botulinum* spores from a stool sample (Fig.1) with conventional microbiological culture or detection of neurotoxin genes in clinical specimens (faeces, serum) with the use of real-time PCR. *C. botulinum* species might also be isolated in food products. Consideration of **differential diagnosis** is particularly important if the cause of deterioration is not found (Table 1).

A human-derived botulism immunoglobulin has been synthesised in California, US and licensed in 2003 as **BabyBIG**. An American study published in 2018 demonstrated reduction in length of stay if administered within the first week of hospitalisation. Importantly, the treatment should not be delayed by waiting for the confirming results.

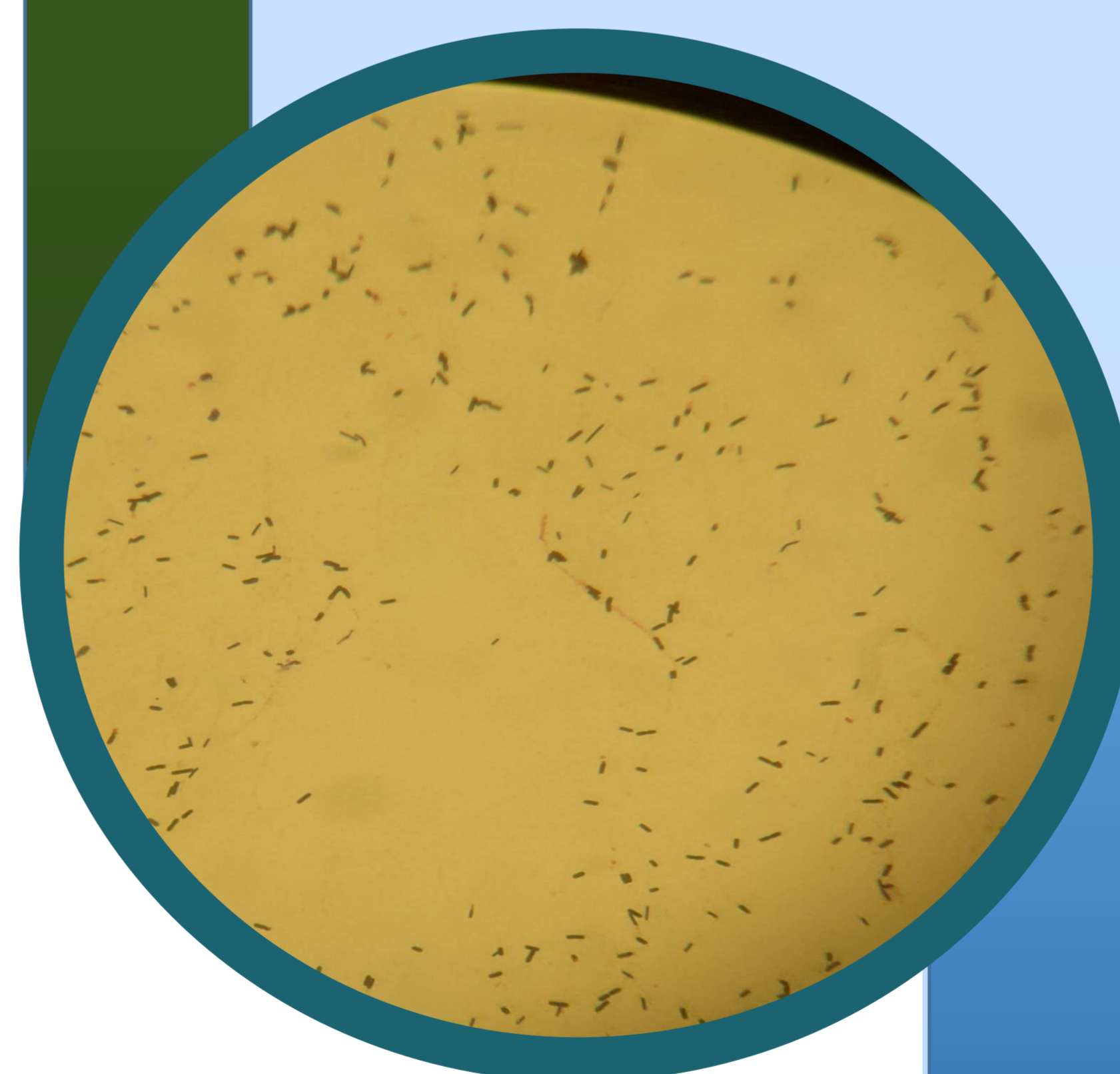


Figure 1. *Clostridium botulinum* isolated from faeces sample.

CONCLUSION

The above case and review of literature demonstrates that the diagnosis of infantile botulism can be a challenging one. Awareness and consideration amongst clinicians of its presentation is key to prompt diagnosis. Appropriate investigations and early botulinum anti-toxin administration may hasten recovery and reduce length of stay.

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| Affected site | Condition |
|------------------------|--|
| Spinal cord | transverse myelitis, cord compression, ischaemic damage |
| Anterior horn cell | SMA, polio, neurotropic viruses (eg. enteroviruses) |
| Neuromuscular junction | transient acquired neonatal myasthenia gravis, congenital myasthenia |
| Peripheral nerve | GBS, trauma, acute toxic neuropathies |
| Muscle | myositis, mitochondrial diseases |
| Systemic disease | sepsis, meningitis/meningoencephalitis, critical illness neuropathy, elevated levels of magnesium or aminoglycosides |

Table 1. Differential diagnosis for flaccid paralysis