ASSESSMENT OF THE CLINICAL COURSE FEATURES OF BOTULISM IN CHILDREN

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Abstract. Botulism is a rare but potentially life-threatening neuroparalytic disorder caused by botulinum neurotoxin produced by Clostridium species. In children the disease presents primarily as infant botulism (intestinal colonization) or as foodborne/iatrogenic forms, and its clinical course differs from adults in several important aspects. Pediatric presentations are often subtle early on (constipation, poor feeding, hypotonia, weak cry) and may progress to bulbar palsies, descending symmetric flaccid paralysis and respiratory failure. Rapid recognition and early administration of specific antitoxin (human botulism immune globulin for infants; equine heptavalent antitoxin for older children when indicated), together with meticulous supportive intensive care, significantly improve outcomes.

Keywords: botulism; infant botulism; children; clinical features; antitoxin; diagnosis; prognosis.

Introduction. Botulism is an acute flaccid paralysis syndrome caused by presynaptic blockade of acetylcholine release at neuromuscular junctions due to botulinum neurotoxins (BoNTs). Although rare, pediatric cases—particularly infant botulism—are a unique clinical entity with specific epidemiology, pathogenesis and management considerations. Early clinical suspicion is paramount because supportive measures (notably respiratory support) and specific antitoxin therapy can markedly shorten disease duration and reduce morbidity.

Epidemiology and forms of botulism in children. In children the most commonly encountered form is infant botulism, caused by intestinal colonization with BoNT-producing Clostridium (most often C. Botulinum, less commonly C. Baratii or C. Butyricum). Infant botulism predominantly affects infants aged 2 weeks to 6 months, with peak incidence between 2–3 months, reflecting immaturity of the gut microbiome and clearance mechanisms. Foodborne botulism (ingestion of preformed toxin) is less common in children but has been reported, and iatrogenic/iatrogenic-like exposure (botulinum toxin preparations) is rare but possible in older pediatric patients.

Infant botulism occurs when spores are ingested and germinate in the immature intestinal tract, producing toxin in situ; the absorbed toxin then causes neuromuscular blockade. In contrast, foodborne botulism results from ingestion of preformed toxin. The infant gut’s reduced colonization resistance (immature microbiota) and developing immune response explain susceptibility and the typical age window. The developing neuromuscular and respiratory systems in infants and young children contribute to faster clinical decompensation (feeding difficulties, hypoventilation) and the need for specialized paediatric supportive care.

Clinical presentation — early and progressive features. The hallmark of botulism is a descending, symmetric flaccid paralysis beginning with cranial nerves and progressing to involve respiratory muscles:

Infant-specific early signs (may precede cranial findings): Constipation (often the earliest and most consistent prodromal sign). Poor feeding, weak or altered cry, lethargy and diminished rooting/sucking reflex. Hypotonia ("floppy baby"), diminished spontaneous movements and diminished deep tendon reflexes.

Cranial nerve / bulbar manifestations (frequent in older infants and children): Ptosis, ophthalmoplegia (diplopia), blurred vision (in age-appropriate children), facial weakness, dysphagia, drooling and dysarthria.

Voice changes, weak cry in infants.

Autonomic dysfunction: dry mouth, reduced lacrimation, constipation, urinary retention and sometimes orthostatic changes. Importantly, consciousness is typically preserved and fever is often absent unless secondary infection occurs.

Time course can be variable — from insidious onset over days to rapid deterioration requiring ventilatory support.

Differential diagnosis in children. Early infant presentations may mimic other causes of hypotonia or weak feeding — spinal muscular atrophy (SMA), metabolic disorders, sepsis, neuromuscular junction disorders (congenital myasthenic syndromes), Guillain–Barré syndrome (rare in infants), and encephalitis. Distinguishing features useful for clinicians include: preserved sensation and mentation, descending pattern beginning with bulbar palsy, afebrile course, early prominent constipation, and history of honey or environmental exposure in infants. Electrophysiologic testing (repetitive nerve stimulation showing facilitation with high-frequency stimulation or increment after exercise) may support the diagnosis when available.

Diagnostic approach. Clinical diagnosis remains central and must not be delayed for laboratory confirmation. Because toxin assays and cultures (stool, serum, implicated food) can take days and are available only in specialised laboratories, clinical suspicion should prompt immediate supportive and specific therapy when indicated. The CDC and expert guidelines outline an approach that emphasises early recognition and coordination with public health laboratories for testing and case confirmation. Key diagnostic steps: clinical assessment (bulbar signs, descending paralysis, absence of fever/altered mental status), obtain specimens (stool, serum, gastric contents, implicated foods) for toxin assay/culture before antitoxin if feasible, and perform basic labs and chest imaging to evaluate respiratory compromise. Electrophysiologic studies may be helpful adjuncts.

Management has two parallel arms: (1) specific neutralisation of circulating toxin and (2) aggressive supportive care. Specific antitoxin therapy. Infants (<1 year): Human botulism immune globulin intravenous (BabyBIG; BIG-IV) is the recommended, specific treatment for infant botulism types A and B; randomized and observational data demonstrate shortened hospital stay and improved outcomes. Timely administration is associated with reduced need and duration of mechanical ventilation and shortened recovery. Older children (foodborne, wound or iatrogenic forms): Equine-derived heptavalent botulinum antitoxin (HBAT) is available for treatment of non-infant botulism cases and may be used in severe pediatric presentations; systematic reviews support benefit when administered early, though risk of hypersensitivity must be considered. Respiratory monitoring in a paediatric or neonatal intensive care setting; early elective intubation when signs of hypoventilation or aspiration risk are present. Enteral feeding support (nasogastric tube) and prevention of secondary complications (pressure injuries, thromboembolism). Treatment of secondary infections and physiotherapy for recovery phase. Close monitoring of autonomic dysfunction is necessary.

Conclusion. Botulism in children, though uncommon, demands prompt recognition because early antitoxin therapy and high-quality supportive care substantially alter the clinical course and outcomes. Infant botulism presents with characteristic early features (constipation, feeding difficulties, hypotonia) that should trigger urgent evaluation. Coordination with public health services for antitoxin access and specimen testing, together with vigilant respiratory support, constitute the cornerstone of effective management.

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