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Case Report

Infant dicyclomine overdose: a case report

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ABSTRACT

We report a case of dicyclomine poisoning in a 45-day-old baby given at 15 mg/kg over 6 hours. The patient presented with complaints of decerebrate posturing of all four limbs. At presentation, the GCS was 8/15, with tachycardia (HR: 168 bpm) and tachypnea (RR: 68 bpm) present. During the hospital stay, the patient developed catecholamine-refractory shock, recurrent apnea, intraventricular hemorrhage and eventually succumbed to death.

Keywords: Dicyclomine overdose, Recurrent apnea, Shock, Mortality

INTRODUCTION

Dicyclomine is an anticholinergic medication commonly prescribed to alleviate symptoms associated with gastrointestinal disorders, such as irritable bowel syndrome (IBS), intestinal hypermotility, and dysmenorrhea.[1] Additionally, it is sometimes used to treat morning sickness, motion sickness, and infantile colic.[2] The mechanism of action involves blocking the effects of acetylcholine at parasympathetic sites in smooth muscle, secretory glands, and the central nervous system (CNS). While safe in adults, it is strictly contraindicated in infants under six months due to their immature blood-brain barrier and high risk of CNS toxicity.[1] Reported adverse events in infants include seizures, respiratory collapse, and death.[3] This report describes a fatal dicyclomine overdose in a 45-day-old infant to emphasize the dangers of its use in early infancy.

CASE REPORT

A 45-day-old female born at term via normal vaginal delivery to a primigravida presented to the Pediatric emergency with complaints of tonic posturing of all four limbs for the last 5 hours, followed by unresponsiveness and fever (max. recorded 101.4°F).

At presentation, the child had tachycardia (HR: 168 bpm) and tachypnea (RR: 66/min) with a GCS of 8/15 (E2V2M4).

The infant's father provided a history of administration of 4.5 ml dicyclomine drops over 6 hours, equivalent to a dose of 15mg/kg, following which the infant developed tonic posturing and deviation of the neck to the right side, followed by unresponsiveness to stimuli.

The infant was eventually referred to our center from the District Hospital.

There was no history of any medical or autoimmune, or infective etiology or history of any other concomitant drug intake. The patient was admitted, and gastric lavage was performed, followed by administration of Intravenous fluids. During the course of treatment, the child developed fluid-refractory shock, and Inotropes were started. Eventually, the patient experienced recurrent episodes of the apnea for which she was intubated.

Laboratory workup revealed severe anemia, raised transaminases and coagulopathy. Anemia in conjunction with a bulging anterior fontanelle alluded to the

possibility of intraventricular hemorrhage. Packed red cells and Fresh frozen plasma were transfused. Gradually, the shock started worsening, and inotropes were incrementally increased. However, the child expired during the course of treatment on day 3.

The news was broken to the parents, and they were counselled regarding the necessity of administering prescribed drugs at prescribed doses.

Diagnosis

Diagnosis of Dicyclomine poisoning was made as there was a strong temporal relationship between Dicyclomine overdose and onset of symptoms, and there was no history or laboratory reports suggestive of any metabolic disorder or infection.

Management and outcome

As soon as the infant presented to our center, gastric lavage was performed, and fluid resuscitation was done. Eventually patient developed recurrent apnea and shock. Patient expired on day 3 of hospital stay.

DISCUSSION

Metabolic disorder and infection-associated septic shock were considered unlikely diagnoses given the patient's clinical history and laboratory findings. Furthermore, this patient's condition deteriorated within a very short timeframe and did not respond to antibiotics and other standard therapies. Dicyclomine poisoning was highly suspected because the acute onset of symptoms was observed immediately following massive drug intake.

Dicyclomine is an antispasmodic agent that competitively inhibits muscarinic receptors at parasympathetic sites, resulting in both central and peripheral anticholinergic toxidromes.[1] In young infants, an immature blood-brain barrier significantly increases their susceptibility to CNS toxicity.[1] Central toxicity typically manifests as agitated delirium, lethargy, or confusion, but can rapidly escalate to seizures, coma, and life-threatening respiratory collapse or apnea. Peripheral toxicity presents with tachycardia, tachypnea, flushed and dry skin, hyperthermia, absent bowel sounds, and urinary retention. Our patient presented with classic severe toxidrome features, including a profoundly altered sensorium (GCS 8/15), extreme tachycardia (168 bpm), tachypnea, fever (101.4°F), and eventual fluid-refractory shock and recurrent apnea.

Because of these severe adverse reactions, dicyclomine is strictly contraindicated in infants younger than six months of age. The U.S. Food and Drug Administration (FDA) specifically warn that administering dicyclomine to infants can trigger sudden respiratory symptoms, including breathlessness, asphyxia, pulse rate fluctuations, muscular hypotonia, coma, and death.[4] This correlate directly with the recurrent apnea and tonic posturing seen in our patient.

Our observations are strongly supported by prior literature.[5-7] Garriott et al documented two fatalities in infants following dicyclomine overdose, noting post-mortem blood concentrations up to 10 times the adult therapeutic level.[6] Similarly, Aziz et al reported an occurrence of severe respiratory distress and death in an infant even at significantly lower doses (0.5 to 1 mg).[2] While older pediatric patients and adults with dicyclomine abuse might present with classic "mad as a hatter" symptoms and recover fully with supportive care, young infants are disproportionately vulnerable to rapid central apnea and cardiovascular collapse. The rapid progression to catecholamine-refractory shock and intraventricular hemorrhage in our patient highlights the compounding systemic failures caused by central respiratory depression and subsequent hypoxic-ischemic injury.

CONCLUSION

Dicyclomine poisoning, while relatively rare, poses significant clinical challenges due to its anticholinergic properties. In this case report, we highlighted the critical presentation of a patient who exhibited severe symptoms indicative of anticholinergic toxicity, including altered mental status, tachycardia, shock and recurrent apnea. Rapid recognition and supportive care were vital in managing the condition. Our experience underscores the need for increased awareness among healthcare professionals regarding the potential risks associated with dicyclomine misuse or overdose, as well as the key role of early intervention. Future efforts should focus on educating both healthcare providers and patients about the safe use of dicyclomine and the importance of adhering to prescribed dosages to prevent such toxicological emergencies.

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