Case Report

Central Anticholinergic Syndrome due to Hypoxia-Induced Bradycardia in a Child with Difficult Intubation Undergoing Complete Dental Restoration: A Case Report

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Received: 28 February 2014
Accepted: 19 June 2014

Abstract

Central anticholinergic syndrome (CAS) following general anesthesia (GA) is a well known syndrome in children and adults. Many cases of CAS have been previously reported in the literature. However, there are only two reports of post resuscitation CAS after administration of small doses of atropine. Hereby, we report a case of CAS in a child undergoing complete dental restoration under GA after receiving a small dose of atropine to reverse hypoxia induced bradycardia.

Intraoperative events such as hypoxia or cardiac arrest may play a role as triggers for CAS. However, we cannot establish a causal relationship between the occurrence of CAS and such critical events.

Key words: Central anticholinergic syndrome; Hypoxia; Bradycardia; Atropine, Pediatric

Journal of Dentistry, Tehran University of Medical Sciences, Tehran, Iran (2014; Vol. 11, No. 5)

INTRODUCTION

Central anticholinergic syndrome (CAS) is caused by decreased inhibitory activity of acetylcholine in the brain. When central cholinergic sites are occupied by drugs with anticholinergic activity, acetylcholine is inadequately released, which in turn leads to development of symptoms of CAS [1, 2].

The prevalence of CAS is reported to be 8-12% after GA and 4% after regional anesthesia with sedation [3]. Clinical manifestation of CAS is secondary to central nervous system effects, peripheral nervous system effects, or both. Common symptoms include flushing, fever, altered mental status, dry skin and mucous membranes, myoclonic jerking, tremulousness, and mydriasis with cycloplegia.

Historically, this syndrome has been reported after administration of anticholinergic medications such as scopolamine and atropine. However, more recently, cases of CAS after administration of benzodiazepines, opioids, nitrous oxide, volatile anesthetics, ketamine, propofol, and etomidate have been reported [4, 5].

Due to non-specific symptoms associated with CAS, the diagnosis can be challenging and requires a high index of suspicion. It is generally a diagnosis of exclusion after ruling out

other factors such as electrolyte and acid-base disorders, hypoglycemia, anesthetic overdose, hypovolemia, postoperative neurological injuries, and embolic events. The definite diagnosis is made by complete resolution of symptoms following administration of physostigmine infusion [4]. Physostigmine is a central acting cholinesterase inhibitor with minor side effects such as nausea, vomiting, and bradycardia, which can be avoided if infused slowly [3,6].

CASE REPORT

The patient was a four-year-old girl, who presented for elective complete dental restoration under GA in the operation room. Preoperatively, the patient was calm and did not require premedication. Difficult intubation on preoperative examinations was not suspected. General anesthesia was induced with 3-8% sevoflurane and oxygen in presence of her parents.

Direct laryngoscopy was performed for nasal intubation after administration of 2μg/kg of fentanyl and 0.6 mg/kg of atracurium. Endotracheal intubation was proven to be difficult, with only the epiglottis visible during direct laryngoscopy (Cormack and Lehane grade 3). The patient experienced a brief period of hypoxia. The patient experienced a brief period of hypoxia lasting 38 seconds with resultant bradycardia 52/minute.

The patient received 0.02 mg/kg of atropine and mask ventilation was performed. Subsequently, endotracheal intubation was performed using a GlideScope video laryngoscope (have to insert the manufacturer's information here). General anesthesia was maintained with propofol infusion (100 μg/kg/min) and nitrous oxide (60%) and O2 (40%), and was supplemented with repeated doses of fentanyl for analgesia and atracurium to maintain muscle relaxation. The surgery lasted for three and a half hours. Reversal of neuromuscular blockade was achieved with 50 μg/kg of neostigmine and 0.02mg/kg of atropine.

The patient emerged from GA after 30 minutes. Since the patient fulfilled all the discharge criteria from the post anesthesia care unit, she was discharged home on the same day. The following day her parents reported unusual behaviors by the child, including agitation, crying, and unreasonable fear of her surroundings. A detailed neurological examination did not reveal any deficits and all electrolytes were within normal limits. The child was discharged home with parental reassurance. After 48 hours, the parents returned the child with chief complaint of hallucinations. The child reported seeing beetles crawling on the walls. Central anticholinergic syndrome was suspected and the patient received 0.03 mg intravenous physostigmine while under electrocardiogram monitoring, followed by a repeat dose in 30 minutes. All symptoms resolved promptly and a follow up visit a week later was unremarkable.

DISCUSSION

Most reports of CAS are in adults and occur during the recovery period after GA. Reports of CAS in pediatric patients following hypoxia and severe bradycardia are rare. The first report of this disease in children was by Kulkal et al, who presented a case of CAS in infants less than one-year-old with signs of deep coma [7]. Holland et al. diagnosed CAS in a nine-year-old boy with hallucinations and urinary incontinence 48 hours after administration of scopolamine patch [8].

Similarly, in our patient, the diagnosis was delayed due to non-specific symptoms. Central anticholinergic syndrome has also been reported in a one-day-old infant after cardiac arrest [9]. Similar to our case, in this patient CAS occurred after resuscitation from hypoxia-induced bradycardia with a small dose of atropine. It seems hypoxia acted as atrigger factor to produce CAS after administrating low dose atropine. However, we cannot establish a causal relationship between the occurrence of CAS and hypoxia.

Schaltz et al. reported a case of an eight-year-old boy who experienced deep sedation following circumcision and was not responsive to stimulations. His symptoms improved immediately after adiminstration of physostigmine [10]. Cohen et al. also reported two cases of CAS after intravenous sedation for screening colonoscopy resulting in delayed emergence from general anesthesia. Both patients were treated successfully with intravenous physostigmine [11]. In a randomized control trial of 211 children one- to five-years-old, signs of severe agitation, which were attributed to CAS, were successfully treated with 30μg/kg of intravenous physostigmine. In this study, it was recommended to infuse physostigmine slowly in order to prevent development of the associated side effects [6].

CONCLUSION

Transient intraoperative events such as hypoxia-induced bradycardia may be risk factors for development of CAS even after small doses of atropine.

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