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CASE REPORT

Clonidine Poisoning in A Child-A case report

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Abstract

Introduction: Clonidine is among the drugs that are in a list of 'one pill can kill' drugs. We are reporting a child with clonidine toxicity with classical manifestations who recovered completely.
Case Report: A 3 year and 2 months old ADHD girl presented with drowsiness for 30 minutes. Her mother gave the history of accidental consumption of 3/4th clonidine 100 mcg tablet 45 minutes prior. On the examination, the child was drowsy with Glasgow Coma Score (GCS) 12/15. Her heart rate was 82 /minute with low volume pulse, respiratory rate 22 /minute, systolic blood pressure was 80 mm of Hg, and diastolic BP was not recordable. Immediately normal saline bolus was administered and her pulse volume improved and BP was 94/60 mm of Hg. After an hour, GCS was 9/15 and the respiration was shallow. After 4 hours, her sensorium improved (GCS 15/15). By 6 hours of ingestion, the child showed complete recovery.
Discussion: Most of the clonidine poisoning children were younger than 6 years according to the NSWPIC database. Among them, 60% developed symptoms, most commonly lethargy (80%), bradycardia (17%), hypotension (15%), and respiratory depression (5%). Symptoms usually develop within 30-60 minutes and resolve within 24-48 hours. In our case, classical symptoms like drowsiness, hypotension, meiosis occurred within 30 minutes of ingestion and her consciousness deteriorated along with shallow respiration by the next 2 hours. By 6 hours of ingestion, the child showed complete recovery.
Conclusion: While prescribing clonidine caution should be exercised regarding the side-effects. When calculating the dose, extra vigilance should be ensured and drug needs to be kept out of the reach of children.

Key Words: Clonidine, Toxicity, Drowsiness, Hypotension, Respiratory Depression.

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INTRODUCTION

Even though clonidine poisoning has been reported increasingly in developed countries, only few reports are available from India (1, 2). Clonidine has become popular for the management of attention deficit hyperactive disorder (ADHD) in children (3). Clonidine is both central and peripherally acting drug with a mixed alpha-1 and alpha-2 adrenoceptor agonist with a predominant alpha-2 action. This inhibits sympathetic outflow (4, 5). It has a narrow therapeutic index, and toxicity can occur with i nadvertent double dosing[3].

The clinical features of clonidine toxicity manifest themselves as central nervous system depression, miosis, respiratory depression, bradycardia, and hypotension (4).In this case study report, we present the case of a child with clonidine toxicity with classical manifestations who recovered completely by supportive management.

CASE REPORT

A 3 year and 2 months old girl (weight=13kgs) was diagnosed with drowsiness for 30 minutes. Her mother gave history of accidental consumption of 3/4th clonidine 100 mcg (0.1mg) tablet about 45 minutes prior to reaching the hospital. The child is a known case of ADHD and has been on tablet clonidine 100 mcg 1/4th tablet in two divided doses since 4 months. The tablets were kept loose in the packet. When being examined, the child was drowsy with Glasgow Coma Score (GCS) 12/15 and her oxygen saturation was 95% at room air. Pupils were constricted and sluggishly reactive to light. Her heart rate was 82 /minute with low volume pulse, respiratory rate 22 /minute, systolic blood pressure was 80 mm of Hg and diastolic BP was not recordable. Immediately normal saline bolus at 10ml/kg over 1 hour was administered, following which her pulse volume improved and BP was 94/60 mm of Hg. After an hour, the child became increasingly drowsy (GCS 9/15) and the respiration was shallow. Her blood counts, liver function, and kidney functions tests were normal. The case was closely monitored with supportive measures. After 4 hours of admission, her sensorium improved (GCS 15/15) and pupils were 3mm and reactive to light. Her pulse rate 84/minute with normal volume, regular respiration and BP was108/60 mm of Hg. By 6 hours of ingestion, the child showed complete recovery.

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DISCUSSION

In the NSWPIC database [2004-2017], there were 802 clonidine exposures which was increasing 4.9% per year. Most of the clonidine exposures were accidental (80.2%). The most common clinical features were drowsiness (44.6%) and bradycardia (12.8%). Two children were intubated due to CNS and respiratory depression and at least eight children were given naloxone and six were given atropine. However, there were no deaths recorded (3). According to American Association of Poison Control Centers Database (1993–1999), there were 10060 clonidine exposures in children. Most of them (57%) were children younger than 6 years and unintentional overdose was the most common cause. Among them 60% developed symptoms, most commonly lethargy (80%), bradycardia (17%), hypotension (15%) and respiratory depression (5%). Management therapies included are naloxone (n=917), atropine (n=160), intubation (n=174), vasopressors (n= 61), and mechanical ventilation (n=105). Twenty three children had respiratory arrest and one expired (6). In a study conducted by Wang et al, out of 27825 clonidine exposures in children, 45% had CNS symptoms, 10% bradycardia, and 9% hypotension (4). Among them 0.4% of the patients were treated with vasopressors, 1.8% with atropine, 8.2% with naloxone, and 2.6% needed intubations. Seven children had cardiac arrests and 3 expired (4). In clonidine intoxication, symptoms usually develop within 30-60 minutes because it is rapidly absorbed and usually resolve within 24-48 hours (5). Peak serum concentrations occur within 60-80 minutes after ingestion (5). In our child classical symptoms like drowsiness, hypotension, meiosis occurred within 30 minutes of ingestion and her consciousness deteriorated along with shallow respiration by the next 2 hours may be because of peak serum concentrations. The therapeutic dose of clonidine is 2–4 mcg/kg and toxicity may occur with doses over 5 mcg/kg. That means, just the double dose can cause toxicity (3). Doses over 10 mcg/kg can cause bradycardia and hypotension. Doses over 20 mcg/kg have resulted in respiratory depression and apnoea (3). In fact, our child had ingested 5.8 mcg/kg of clonidine. Out of 113 patients, all toxic symptoms developed within 4 hours of ingestion in all cases (7). Erickson and Duncan reported 14 cases of clonidine poisoning and the length of hospital stay was less than a day and all had favorable outcome (5). In their seminal study, Wiley et al observed the onset of symptoms within 4 hours in all their 47 patients and concluded that those who remain asymptomatic for 6 hours post-ingestion can be discharged (8). Out of 24 children in a study done by Sinha et al., three needed mechanical ventilation and all were extubated within the first 3 hours of admission (9). Moreover, Fiser et al suggested a relationship between ingested amount and clinical manifestations with clonidine toxicity in their investigation (10). In US, clonidine is the most common exposure associated with endotracheal intubation in less than 6 years of age (3). Management is mainly supportive. Activated charcoal should be given early within 1 hour of ingestion. Children with bradycardia respond well to atropine. Hypotension often responds to crystalloids and few may require vasopressors such as dopamine (8). Our case, responded to one bolus of normal saline infusion. Nalaxone was used to reverse the clonidine toxicity clinical features, but the evidence supporting its use varied (5). Some studies demonstrated some response and some did not confirm the benefit (5). Even though most of the children with clonidine exposures suffered from minimum adverse effects, serious toxic effects and death can occur (6). It has been suggested in the category of 'One Pill Can Kill' list of drugs when children ingest single adult doses unintentionally (4). Our child care takers were using clonidine tablet strength of 100 mcg (0.1 mg). Our child is 13 kg and a therapeutic dose of 2 mcg/kg needs ¼ th (26 mcg) of a 100mcg tablet. But our child had taken ¾ th of a tablet (75 mcg). Similar dosing errors have been reported in children taking a whole tablet instead of a half or quarter-tablet (3).

CONCLUSION

In conclusion, it can be argued that while prescribing clonidine, caution should be exercised regarding the side-effects of the drug even with minimum dosing errors. When calculating the dose, extra vigilance should be ensured and the drug needs to be kept away from children's reach. Likewise, educating care takers regarding clonidine toxicity is very important and should be emphasised. Memorandum should be given to the government for the availability of low strength pediatric formulations and packing

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