

Accidental Substance Abuse Poisoning In Children: Experience of the Dammam Poison Control Center

Ahmed R. Ragab^{1*}, Maha K. Al-Mazroua², and Naglaa F. Mahmoud³

¹Department of Forensic Medicine and Clinical Toxicology, Faculty of Medicine, Mansoura University, Mansoura, Egypt

²Dammam Poison Control Center, Dammam, Eastern Region, Ministry of Health, Saudi Arabia

³Department of Forensic Medicine and Clinical Toxicology, Faculty of Medicine, Cairo University, Cairo, Egypt

*Corresponding author: Ahmed R. Ragab, Department of Forensic Medicine and Clinical Toxicology, Faculty of Medicine, Mansoura University, Mansoura, Egypt, Tel: 00966540990033; E-mail: ahmedrefat1973@yahoo.com

Received date: Apr 28, 2014; Accepted date: June 30, 2014 Published date: July 3, 2014

Copyright: © 2014, Ragab AR et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Abstract

Introduction: Cannabis and amphetamines are the most commonly used illegal drugs in adults in Saudi Arabia. Accidental Substance Abuse poisoning is an uncommon form of poisoning in children, but potentially serious.

Objective: To describe the clinical presentation, diagnosis and treatment of children with accidental poisoning from various forms of substances of abuse in a pediatric secondary hospital.

Material and Methods: We report on 14 patients with accidental intoxication by amphetamines, cannabis and opiates.

Results: The clinical presentation was variable deterioration in level of consciousness, somnolence, ataxia, tremor, apnea, hypotonia and seizure. The investigation of toxic urine detected benzphetamine, tetrahydrocannabinol (THC) and morphine in all cases of amphetamine, cannabis and heroine intoxication respectively. In all patients with amphetamine and cannabis intoxication supportive measures were established. In only one case of acute heroine intoxication was naloxone therapy given. All cases recovered well and were discharged within 24-48 hours of admission.

Conclusion: A high index with early priority of suspicion should be maintained for substances of abuse involving "amphetamines, cannabis or morphine" intoxication in previously healthy children with acute onset of neurological symptoms of unknown etiology. Accidental poisoning by various substances of abuse is, in itself, an alarm signal on the attitude of parents in caring for their children. These families deserve special monitoring by social services, since such accidents may be covering up child abuse.

Keywords: Substances of abuse; Amphetamines; Morphine; Coma; Accidental poisoning; Drowsiness; Children

Introduction

Substance abuse is a common problem in families involved with the child welfare system. There is increasing awareness that the abuse of drugs by parents and other caregivers can have a negative impact on the safety, permanence, and well-being of children [1].

Clinical signs of amphetamines toxicity especially in pediatric population include hyperthermia, tachycardia, tachypnea, mydriasis, tremors, and seizures. In addition, amphetamine intoxication has been reported to cause hyperthermia, hypoglycemia and mild thrombocytopenia [2,3]. Diagnosis can be confirmed by detecting amphetamine in stomach contents or vomitus, or mainly by positive results obtained in urine tests for illicit drugs [4].

Regarding cannabis intoxication; the main psychoactive metabolite is delta-9-tetrahydrocannabinol (THC) [5]. It is the most widely consumed psychoactive drug in Spain, especially among adolescents. Despite stabilization in the prevalence of consumption, it was reported

that 11.2% of the population aged 15-64 had ever spent in the last year [6]. In parallel to this high prevalence of consumption there has been an increase in the number of cases of accidental poisoning by this substance in the pediatric population [7].

Opiate poisoning can occur at any time from birth to terminal care. The outcome can range from discomfort such as constipation, to death from respiratory depression. Regarding the epidemiology of acute opiate intoxication in children, it is difficult to get reliable incidence figures [8]. The pediatrics is less liable to poisoning from opiates and less likely to be exposed to them, especially illicit forms of medication. Acute opiate toxicity presents with drowsiness. There may be nausea or vomiting. Respiratory depression may be apparent. Hypotension and tachycardia are possible. There are usually pinpoint pupils but this sign may be absent if other drugs are involved [9].

Because there are so many child cases involving substance abuse, child welfare agencies should begin to use a range of strategies to prevent and treat substance abuse in families, and to improve outcomes for children and families [1].

Objective

The purpose of this study was to shed light on the problem of accidental substance abuse poisoning among children, to determine the factors related to accidental substance of abuse poisoning by the most common abused agents in poisoned patients who were visited a pediatric ER department.

Material and Methods

Study setting

This work was conducted as a cross sectional prospective, (Electronic Medical Review) EMR database review study at Dammam Regional Poison Control Center–Eastern Region, KSA.

Inclusion criteria

Pediatric patients suspected for substances abuse toxicity in two hospitals (Dammam Medical Complex and Qatif Central Hospital) that were participating in two year-long period from the beginning of January, 2011 until the end of December, 2013.

Study population parameters

Investigators noted down important and detailed information of all the patients, like their age, and sex as well as their patient code, in-patient or out-patient admission status and medical service type.

At present, the status of electrolytes, renal and liver function values were evaluated at the same time of screening the substance abuse profile kit. Important laboratory activities and investigations such as blood urea nitrogen, serum Creatinine concentration, both serum ALT and AST levels were also conducted.

Electronic medical records review process

Three reviewers conducted the entire review process – physicians. Taking the help of individual patient records, the individual patient records were accessed by way of medical record number access into the EMR. Predefined data points fed into a standard type Excel worksheet was set up on a share drive that was password protected which was to be used by every single reviewer in order to get the abstraction data information. Then every patient was reviewed on an independent basis to be reviewed for agreement purpose followed by checks carried out by the third reviewer to see if there were still any other discrepancies identified. Data extractors had to have total agreement amongst them. The study was approved by the Medical Ethics Committee of the Dammam Regional Poison Control Center/ Ref No 11/2010.

Results

The description of the clinical case series: Present eight pediatric cases with accidental intoxication by amphetamines. All of them were boys between 14 months and five years old. The clinical presentation was variable (Table 1).

No	Age			Sex	Route of Poisoning	Clinical Presentation	Glasgow Coma Scale	Urine Drug of Abuse Results	Duration of Recovery
	Ys	Ms	Ds						
1	1	11	1	Male	By ingestion	Abnormal behavior, Irritability, Tachycardia	14	Amphetamines metabolites, Benzodiazepines	26 hours
2	2	3	7	Male	By ingestion	Drowsy, Hallucination, Tachypnoea.	14	Amphetamines metabolites	16 hours
3	1	2	5	Male	By ingestion	Abnormal Behavior, Hypertonia, Hyperactivity, Hyperreflexia	13	Amphetamines metabolites	22 hours
4	2	3	19	Male	By ingestion	Abnormal movement in the mouth, Tremors in the tongue	12	Amphetamines metabolites and caffeine.	18 hours
5	3	9	23	Male	By ingestion	Convulsion, Hypertension	14	Amphetamines metabolites	12 hours
6	4	2	4	Male	By ingestion	Seizures, Hyperthermia, Tachycardia, Mydriasis	10	Amphetamines metabolites Caffeine, Benzodiazepines.	18 hours
7	3	1	25	Male	By ingestion	Drowsy, Hallucination, Tachypnoea.	14	Amphetamines metabolites	16 hours
8	1	4	3	Male	By ingestion	Abnormal Behavior,	14	Amphetamines metabolites	24 hours

						Abnormal movement, Irritability.			
9	5	1	3	Male	By ingestion	Drowsy, Convulsions, and Vomiting	15	Cannabinoids	36 hours
10	1	4	2	Male	By inhalation	Comatose, Convulsion, Tachypnoea, Mydriasis	10	Cannabinoids	22 hours
11	3	2	5	Male	By ingestion	Comatose, Tachycardia, Tremors, Flushing	10	Cannabinoids	11 hours
12	2	7	14	Male	By ingestion	Aponea, Drowsy, Irritability, Somnolence, Hallucinations.	10	Cannabinoids	19 hours
13	11	1	1	Male	By sniffing	Vomiting, Drowsy, Depression	13	Morphine and Codeine and 6 MAM	19 hours
14	0	0	1	Male	Transplacental	Meiosis, Irritability, Diarrhea, Hypoglycaemia	9	Morphine and Codeine	37 hours

Table 1: Clinical, diagnostic and resolution time frame table.

Case [1] had a characteristic sudden development of abnormal behavior, irritability and sinus tachycardia. In case [2] we observed a condition characterized by drowsiness, moderate malaise, visual hallucination, and tachypnoea. In patient [3] there was the presence of generalized hyperactivity, hypertonia and hyperreflexia. As regards the fourth case, abnormal movement in the mouth and fine tremors in the tongue were characteristic signs. In patient [5] convulsion and hypertension were the main pathognomonic signs. The sixth patient had characteristic serial attacks of seizures, hyperthermia, tachycardia and mydriasis. In patient [7], hallucination was markedly represented, with drowsiness and tachypnoea. Finally, child number [8] presented at admission a picture characterized by abnormal movement, abnormal behavior and pallor followed by acute episodes of generalized irritability, a few seconds long. Physical examination revealed a significant malaise, mydriasis and peripheral coldness.

Regarding cannabis intoxication we detected four pediatric patients with acute accidental toxicity of cannabis. All of them were males and their ages ranged from 16 months to five years. Patient [9] had a single attack of convulsions, two episodes of vomiting, and presented with marked drowsiness status. In patient [10] we detected a marked disturbed consciousness level with seizures, tachypnoea and mydriasis. Patient [11] presented to the ER department with marked tachycardia, tremors, flushing and coma. The last case of acute accidental toxicity in pediatric patient [12] presented with apnoea, irritability, somnolence and hallucinations.

Accidental opiate toxicity in pediatric patient represent in two cases one had 1 day male newborn with transplacental exposure of morphine from an addicted opiate mother and represented with meiosis, irritability diarrhea and hypoglycemia as a result of withdrawal effects. The second case was an eleven-year-old male boy

who visited the ER department following recurrent attacks of vomiting and drowsiness with depressive mode after a first episode of exposure to heroin.

While follow up the observed interrogation; how all families, except in the cases of patients 8, 10, and 13 omitted information about the possibility of ingesting toxic abuse substance. The parent of patient 8 reported early the accidental ingestion of a "Capatgone tablet" suspected of street medication of his father. The mother of child [10] suspected cannabis poisoning had occurred during his sleep from inhaling cannabis smoke that released from father smoking set. The last case was a child who had sniffed heroin powder, copying one of his friends, and once his condition had deteriorated he told his mother about his toxic exposure.

In all cases, blood count, and biochemical and acid-base status is assessed on admission. Except for the finding of metabolic acidosis in patient 14, the results of these tests were normal. In all patients, consistent support measures were established in regard to ensure the airway, providing oxygen by face mask, administration of activated charcoal, and gastric lavage. In case 11, for the unknown potential toxicity, naloxone and flumazenil were administered intravenously as coma cocktail. After the maneuvers are implemented and necessary for the stabilization treatment of the patients were investigated urine toxicology by semi-quantitative (Bio Rad® TOX / See Drug Screen Test). All patients showed THC above 25 ng/dl values, which was confirmed by GC/MS. In patients [1] and [6] we also identified caffeine and benzodiazepines (diazepam dose received for irritability and seizure control). The outcome was satisfactory in all cases, with resolution of symptoms within 24 h, allowing discharge 24-48 hours after admission.

Discussion

Substance Abuse intoxication in children is a rare form of acute poisoning, but recently an increasing number of cases have been reported to the register.

The primary psychoactive constituents in variable Substance Abuse are THC in cases of cannabis, morphine-3-glucuronide and normorphine in morphine, and benzphetamine in amphetamine. In cases of cannabis the proportion of active constituents varies by consumption in the form of marijuana (3-5%), hashish (5-20%) or hashish oil (16-43%) [8].

The route of intake varies according to the type of Substance Abuse (in cases of amphetamine it is ingestion, in cases of cannabis it is inhalation and in cases of morphine it is injection). In children, poisoning is usually due to accidental ingestion of cannabis material, their effects by ingestion route are slower, durable and variables. Begin to be apparent after 1 h, with a maximum effect at 2-3 h and its action lasts about 5 H [9]. In the present study; only one case by inhalation and the remaining by the ingestion route.

The toxic effects of amphetamine vary widely by age. They include abdominal pain, acne, blurred vision, excessive grinding of the teeth, profuse sweating, dry mouth, loss of appetite, nausea, reduced seizure threshold, tics, and weight loss. The effects of amphetamine on the gastrointestinal tract are unpredictable. Amphetamine may reduce gastrointestinal motility if intestinal activity is high, or increase motility if the smooth muscle of the tract is relaxed [10]. In the current study, abnormal behavior, irritability, tachycardia were the main presented symptoms and signs.

In the present series study the chief characteristic symptom of the studied cases regarding acute cannabis intoxication was disturbed consciousness level. The symptoms of cannabis intoxication include nausea, vomiting, dry mouth, thirst, hyperorexia, pale skin and conjunctival hyperemia. From a neurological point of view, as regards consciousness disorders, abrupt onset hypotonia, ataxia, mydriasis or miosis, decreased reflexes, mood modification, perceptual disturbances, seizures and even coma may be observed. The most common cardiovascular effect is tachycardia, although high doses may cause bradycardia [11].

Typically, families report information about the possibility of accidental ingestion of a toxin from the maternal side. In our series, ten cases omitted accidental ingestion. Sometimes the version offered is not credible, as in patient 11, whose parents reported that the cannabis intoxication had occurred outdoors, through eating "something on the ground".

Clinical suspicion and prompt detection of the drug are the pillars of substance abuse diagnosis. Differential diagnosis should be made with central nervous system infections, head injuries and metabolic disorders. Thinking of Substance Abuse intoxication as a cause of a sudden decreased level of consciousness in previously healthy and afebrile patients, may allow testing save as CT and lumbar puncture, not without complications [12].

Diagnosis is made through urine toxicology research by semi-quantitative (Bio Rad® TOX/See Drug Screen Test) whose detection threshold is (500 ng/dl in amphetamines, 25 ng/dl in cannabis, 200 ng/dl in morphine). The diagnosis is confirmed by gas chromatography/mass spectrometry, the detection threshold (100 ng/dl in amphetamines, 5 ng/dl in cannabis, and morphine 100 ng/dl).

Treatment in Substance Abuse situations consists of supportive measures which vary according to the severity of symptoms. Performing gastric lavage and administration of activated charcoal is recommended. Care must be taken when performing these procedures, given the risk of aspiration in patients with depressed sensorium. This will isolate the airway in cases where Glasgow is less than 8 and no control coal tape nasogastric tube. Isolated cases have been reported in which intravenous naloxone and flumazenil have been used successfully, aiming to reverse depression neurological. In cases of severe agitation and irritability, as in amphetamine toxicity diazepam, has been given intravenously.

In cases of cannabis intoxication, unlike in adults, the evolution of the poisoning in children is variable. The most satisfactory evolution sees the disappearance of symptoms within hours after the establishment of supportive measures. However, there are cases that have presented with seizures, respiratory obstruction or coma, that have required intensive pediatric care [13-15].

Conclusion

The high number of substance abuse users in the current research may be attributed to easy access of pediatric patients to this substance, which explores the increasing number of cases of accidental poisoning by these substances. We cannot rule out this type of poisoning in any home environment, regardless of the inhabitants' socioeconomic status. Monitoring of children is an important responsibility of parents and is the main form of prevention of childhood accidents. Accidental poisoning by various substances of abuse is, in itself, an alarm signal on the attitude of parents in caring for their children. These families deserve special monitoring by social services, since such accidents may be covering up child abuse.

Acknowledgments

No funding or sponsorship was received for this study. Dr. Ahmed Refat Ragab is the guarantor for this article, and takes responsibility for the integrity of the work as a whole.

References

1. Office on Child Abuse and Neglect (2009) Protecting Children in Families Affected by Substance Use Disorders Children's Bureau, ICF International.
2. Lemke TL, Williams DA, Roche VF, Zito W (2013) Foye's Principles of Medicinal Chemistry (7th edn). Wolters Kluwer Health/Lippincott Williams & Wilkins, Philadelphia.
3. Berman SM, Kuczenski R, McCracken JT, London ED (2009) Potential adverse effects of amphetamine treatment on brain and behavior: a review. *Mol Psychiatry* 14: 123-142.
4. Büttesch CM, Davis BC, Sawaki L, Waldvogel D, Classen J, et al. (2002) Modulation of use-dependent plasticity by d-amphetamine. *Ann Neurol* 51: 59-68.
5. Schwartz RH (2002) Marijuana: a decade and a half later, still a crude drug with underappreciated toxicity. *Pediatrics* 109: 284-289.
6. García-Algar O, Gómez A (2010) [Cannabis in paediatric emergencies]. *An Pediatr (Barc)* 72: 375-376.
7. Spadari M, Glaizal M, Tichadou L, Blanc I, Drouet G, et al. (2009) [Accidental cannabis poisoning in children: experience of the Marseille poison center]. *Presse Med* 38: 1563-1567.
8. Drug Misuse and Dependence-Guidelines on Clinical Management (1999) Drug Misuse and Dependence – Guidelines on Clinical Management, Department of Health, University of Cambridge.

-
9. Macnab A, Anderson E, Susak L (1989) Ingestion of cannabis: a cause of coma in children. *Pediatr Emerg Care* 5: 238-239.
 10. Fantegrossi WE, Godlewski T, Karabenick RL, Stephens JM, Ullrich T, et al. (2003) Pharmacological characterization of the effects of 3,4-methylenedioxymethamphetamine ("ecstasy") and its enantiomers on lethality, core temperature, and locomotor activity in singly housed and crowded mice. *Psychopharmacology (Berl)* 166: 202-211.
 11. Heyman RB, Anglin TM, Copperman SM, Joffe A, McDonald CA, et al. (1999) American Academy of Pediatrics. Committee on Substance Abuse. Marijuana: A continuing concern for pediatricians. *Pediatrics* 104: 982-985.
 12. Appelboam A, Oades PJ (2006) Coma due to cannabis toxicity in an infant. *Eur J Emerg Med* 13: 177-179.
 13. Borrego Domínguez R, Arjona Villanueva D, Fernández Barrio B, Huidobro Labarga B, Alonso Martín JA (2007) [Comatose state after cannabis intake]. *An Pediatr (Barc)* 67: 276-278.
 14. Boros CA, Parsons DW, Zoanetti GD, Ketteridge D, Kennedy D (1996) Cannabis cookies: a cause of coma. *J Paediatr Child Health* 32: 194-195.
 15. Bonkowsky JL, Sarco D, Pomeroy SL (2005) Ataxia and shaking in a 2-year-old girl: acute marijuana intoxication presenting as seizure. *Pediatr Emerg Care* 21: 527-528.