Acute poisoning due to massive leaking of methamphetamine in a methamphetamine body packer: A case report

Hossein Motevalli[1], Rezvan Hosseinzadeh[1], Mohammad Barary[1], Terence Sio[2], and Aliasghar Manouchehri[1]

[1]Babol University of Medical Science
[2]Mayo Clinic Arizona

March 18, 2022

Abstract

methamphetamine (MA) has a similar structure to amphetamine which can induce a wide range of symptoms, including alertness, aggressive behavior, chest pain, psychosis, seizures, developed by the psychomotor system stimulation. This case specifically discusses prolonged severe MA intoxication induced by a novel and unique way of the drug's ingestion.

Acute poisoning due to massive leaking of methamphetamine in a methamphetamine body packer: A case report

Hossein Motevalli[1], Rezvan Hosseinzadeh[1], Mohammad Barary[2, 3], Terene T. Sio[4], Aliasghar Manouchehri[5, *]

1. Student Research Committee, Babol University of Medical Sciences, Babol, Iran
2. Student Research Committee, Virtual School of Medical Education and Management, Shahid Beheshti University of Medical Sciences, Tehran, Iran
3. Students' Scientific Research Center (SSRC), Tehran University of Medical Sciences, Tehran, Iran
4. Department of Radiation Oncology, Mayo Clinic, Phoenix, AZ, USA
5. Department of Internal Medicine, Shahid Beheshti Hospital, Babol University of Medical Sciences, Babol, Iran

Correspondence:

Aliasghar Manouchehri, Department of Internal Medicine, Shahid Beheshti Hospital, Babol University of Medical Sciences, Babol, Iran. Email: drmanouchehri@yahoo.com

Acknowledgments

The authors would like to thank the staff of the Medical Records Unit of Shahid Beheshti Hospital for their kind support.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Ethical approval

This study was reviewed and approved by the Ethics Committee of Babol University of Medical Sciences (reference No.: IR.MUBABOL.REC.1400.155).

Consent for publication

The patient's next-of-kin has given a written consent form to publish this study.

Conflict of interest disclosure

TTS reports that he provides strategic and scientific recommendations as a member of the Advisory Board and speaker for Novocure, Inc. and also as a member of the Advisory Board to Galera Therapeutics, which are not in any way associated with the content or disease site as presented in this manuscript. All other authors have no relevant financial interests to be declared.

Authors' Contributions

- **HM:** Data collection and writing the manuscript.
- **RH:** Data collection and helped with manuscript writing.
- **MB:** Data collection, helped with manuscript writing, and contributed substantial revisions to the manuscript's content.
- **TTS:** Contributed substantial revisions to the manuscript's content.
- **AM:** Design of the research study and supervision.

Abstract

methamphetamine (MA) has a similar structure to amphetamine which can induce a wide range of symptoms, including alertness, aggressive behavior, chest pain, psychosis, seizures, developed by the psychomotor system stimulation. This case specifically discusses prolonged severe MA intoxication induced by a novel and unique way of the drug's ingestion.

Keywords: Methamphetamine; Toxicity; Amphetamine; Morbidity

Key Clinical Message

Continuous intoxication after the digestion of the wrapped packs of recreational drugs, such as methamphetamine, requires clinicians to suspect this type of ingestion and act rapidly to avoid lethal outcomes.

Introduction

Amphetamine-type substances and methamphetamine (MA), in particular, are known to be major global health problems due to their increasing consumption [1]. MA (METH; *N* -methyl-1-phenylpropan-2-amine) has a similar structure to amphetamine (AMPH; 1-phenylpropan-2-amine) and, thus, can significantly stimulate the psychomotor system [2]. The potential abuse of MA caused its clinical utility to be limited, though MA can be a choice in the treatment of attention-deficit hyperactivity disorder (ADHD), narcolepsy, and severe obesity [2,3]. Since 2009, there has been an increasing number of MA overdoses because it is cheap and easy to get, whether smuggled into the United States or manufactured in clandestine laboratories [1,4]. According to the 2009 United Nations' World Drug Report, the global prevalence of MA consumption is second among recreational drugs, only behind cannabis, with estimates reporting that in the past 12 months, MA has been used by approximately 51 million individuals[1]. MA is generally consumed intravenously or by inhalation [2]. A new and unusual way of ingesting MA called "parachuting" consists of swallowing MA wrapped in a plastic package, letting it unfold in the gastrointestinal tract, and slowly releasing the drug into the bloodstream [5,6].

The initial symptoms after MA consumption may manifest as arousal, mania, cardiac stimulation, acute improvement in cognitive domains, increased alertness, aggressive behavior, and increased libido. MA overdose symptoms include dilated pupils, tachycardia, hypertension, chest pain, psychosis, seizures, hallucinations, hyperthermia, hyperventilation, and biphasic changes in arterial blood pressure. It can also lead to rhabdomyolysis, coma, acute coronary syndrome (ACS), and cardiopulmonary arrest [3,7,8]. There is no specific antidote or treatment for MA overdose. Most of the therapeutic methods are supportive measures, including airway control for sufficient oxygenation and ventilation, benzodiazepines administration for controlling the

agitation, and whole bowel irrigation to remove any possible interventions unabsorbed drug. Here, a case of the MA overdose caused by the ingestion of MA packages in a MA body packer is described.

Case presentation

A 38-year-old male, serving a two-year sentence in jail, had been authorized to leave for a week and was returning to jail the morning he was exposed to toxicity. The patient ingested a 5 g pack of MA for further consumption in jail. He developed hallucination and delusion approximately after 4 hours and agitation, limb tremor, and foaming at the mouth 1 hour before hospitalization. He received a dose of naloxone while being transported to the hospital, but the symptoms worsened. The patient has no history of prior diseases but mentions a suicide attempt 7 years ago. He also alleged to consume 5 mL of methadone every day and occasional use. Based on the worsening state of the patient. He was admitted into the intensive care unit (ICU).

His initial vital signs were a blood pressure of 120/94 mmHg, a heart rate of 90 beats per minute, a respiratory rate of 23 breaths per minute, a temperature of 39.4@C (103.0@F), and an oxygen saturation of 100%. Except for his mydriatic pupils and tachycardia, his physical examination was unremarkable. The patient's blood and urine were collected for laboratory tests, and a screening urine toxicology test was performed at the hospital, that was positive for amphetamine, methamphetamine, methadone, and morphine. A computed tomography (CT) scan was performed for the patient, in which at least two hyperdense masses containing bubbles were detected in the second part of the duodenum (D2) which were 20 mm and 26 mm in diameter, suggesting a foreign mass. Moreover, bilateral lung consolidation was observed in multiple dependent sites, indicating aspiration pneumonia. Another CT scan was performed on the fifth day of hospitalization, in which the following were reported: a hypodense-centered radio-opaque mass 18 mm in diameter in the superior part of ascending colon next to hepatic flexure, a small radio-opaque mass 5 mm in diameter in the rectum, and moderate bilateral pleural effusion along with passive collapse consolidation of adjacent segment. His brain CT scan was unremarkable. On admission, his laboratory markers were as follow: white blood cell (WBC) count = 6,100/μL (normal range: 4,000-11,000), hemoglobin (Hb) = 10.7 g/dL (normal range: 14-17.5), platelet (Plt) count = 157,000/μL (normal range: 150,000-450,000), blood urea nitrogen (BUN) = 29 mg/dL (normal range: 5-22), creatinine (Cr) = 1.2 mg/dL (normal range: 0.9-1.3), sodium (Na) = 132 mmol/L (normal range: 136-146), potassium (K) = 4.5 mmol/L (normal range: 3.8-5.1), magnesium (Mg) = 2.2 mg/dL (normal range: 1.9-2.5), phosphorus = 2.8 mg/dL (normal range: 2.5-5), troponin I = 0.0 ng/mL (normal range: < 0.04), and creatine kinase MB (CK-MB) = 23 U/L(normal range: < 24) (Table 1). Furthermore, his arterial blood gas (ABG) analysis revealed the following: Ph = 7.234 (normal range: 7.350-7.450), pO_2 = 226 mmHg (normal range: 80-700), pCO2 = 47.2 mmHg (normal range: 35.0-45.0), bicarbonate (HCO_3^-) = 19.6 (normal range: 22.0-28.0), and chloride (Cl^-) = 114 mmol/L (normal range: 65-140), anion gap (AG) = 7.4 mmol/L (normal range: 3.0-10.0), delta AG = -4.6 mmol/L (normal range: 0.0), and delta AG/delta bicarbonate ratio = -1.0, suggesting normal anion gap acidosis.

Intravenous (IV) normal saline, diazepam 5 mg, fentanyl 100 μg, midazolam 5 mg, and one dose of naloxone was administered for the patient. The patient was also intubated to protect his airways. Due to his chest x-ray (CXR) suggesting aspiration pneumonia, IV vancomycin 2 g, amikacin 500 mg, and meropenem 1 g was also administered for the patient. He also received multiple doses of morphine sulfate. Repeated urine toxicology panel on the seventh day of his hospitalization was positive for MA and methadone, indicating the severity of intoxication. On the tenth day, due to his deep vein thrombosis (DVT) manifestations, D-dimer and fibrin degradation products (FDP) was requested for the patient, which was reported to be 2.5 μg/mL (normal range: < 1) and < 20 μg/mL (normal range: < 5), respectively. Therefore, subcutaneous (SC) enoxaparin 40 mg and IV heparin 5,000 U were administered for the patient.

Because of thrombocytopenia (Plt = 29,000/μL) on the eighth day, 8 units of platelet were administered for the patient to increase and maintain the Plt counts. Nevertheless, his Plt counts continue to drop, for which another 26 units of platelets were administered for him on succeeding days. Also, due to his low hemoglobin on day twelve, a transfusion of two units of packed red blood cells (RBC) was ordered for the patient. Moreover, 4 units of fresh frozen plasma were administered for the patient on days eleven and

twelve. Unfortunately, there has been no significant increase in hemoglobin and platelet counts despite multiple transfusions, which may be caused by the poor state of the patients, aggravated by aspiration pneumonia and DVT. After thirteen days of hospitalization, the patient died of respiratory failure.

Discussion

Methamphetamine, a highly addictive stimulator of the central nervous system, has been used for a long time [9,10]. Since the introduction of this drug, there has been a dramatic rise in the prevalence of MA toxicity worldwide, most probably because of its relatively easy accessibility and cheap price [11,12]. In the early 1990s, epidemic abuse of MA in the United States, in particular, Hawaii, began earlier than that in Japan in 1893[13].

Therapy should start with the ABC algorithm (airway, breathing, and circulation), especially if seizures, dysrhythmias, cardiac arrest, and apnea are the initial manifestations. Also, electrocardiography should be performed to detect acute myocardial ischemia due to drug side effects or electrolyte disturbances (especially potassium)[14]. It is critical to control agitation and temperature to lower the morbidity rate. Benzodiazepines and dopamine-antagonist neuroleptics can be used for sedation, but due to dopamine-antagonist neuroleptics' side effects, such as lowering the seizure threshold and temperature dysregulation, benzodiazepines are the drug of choice for sedation in these patients [15]. In order to reduce the patient's temperature, rapid cooling was initiated by placing the ice packs on this patient's axillae and groin, and the patient's temperature dropped from 39.4@C (103.0@F) to 37.6@C (100.0@F). Although the excretion of amphetamine can increase with urinary acidification, it is no longer suggested due to rhabdomyolysis worsening. Rapid IV resuscitation is needed to preserve an alkaline urine to relieve the effects of myoglobin on kidneys. MA is a weak base and alkalinizing the urine leads to an increase in the drug's half-life and reducing the excretion. Retention of the drug can be the result of administering the sodium bicarbonate which alkalized our patient's urine. One liter of 0.9% normal saline solution contains 100 mEq of sodium bicarbonate. This therapy may have caused a prolonged intoxication symptom [11,15]. Elevated patient potassium levels led to the administration of sequential trials of furosemide 20 mg and dextrose 50%. However, high levels of prolonged intoxication prevented potassium levels from returning to normal levels.

The patient discussed in our case report is one of the rare instances in which consuming very high doses of MA in a unique way developed a prolonged severe intoxication leading to excessive agitation that lasted for an extended period. Continuous strong sedation was needed to control his agitation. Oversedation of the patient may have developed aspiration pneumonia, one of the leading causes of the patient's death. Despite aggressive treatment, a lethal outcome could not be avoided.

Conclusion

A case of severe methamphetamine toxicity has been reported following a novel method of ingestion developed by increased focus of law enforcement efforts on drug interdiction, showing the potential for a continuous intoxication after the digestion of the wrapped packs, which requires clinicians to suspect this type of ingestion and act rapidly to avoid lethal outcomes.

References

1. Marshall BD, Werb D. Health outcomes associated with methamphetamine use among young people: a systematic review. *Addiction* . Jun 2010;105(6):991-1002. doi:10.1111/j.1360-0443.2010.02932.x

2. Kitanaka J, Kitanaka N, Hall FS, Uhl GR, Takemura M. Brain Histamine N-Methyltransferase As a Possible Target of Treatment for Methamphetamine Overdose. *Drug Target Insights* . 2016;10:1-7. doi:10.4137/dti.S38342

3. Kiyatkin EA, Sharma HS. Leakage of the blood-brain barrier followed by vasogenic edema as the ultimate cause of death induced by acute methamphetamine overdose. *Int Rev Neurobiol* . 2019;146:189-207. doi:10.1016/bs.irn.2019.06.010

4. Bonk R, Miller RJ, Lanter J, Niblo C, Kemp J, Shelton J. Accidental Overdose Deaths in Oklahoma, 2002–2017: Opioid and Methamphetamine Trends. *Journal of Analytical Toxicology* . 2020;44(7):672-678.

doi:10.1093/jat/bkaa068

5. Hendrickson RG, Horowitz BZ, Norton RL, Notenboom H. "Parachuting" meth: a novel delivery method for methamphetamine and delayed-onset toxicity from "body stuffing". *Clin Toxicol (Phila)* . 2006;44(4):379-82. doi:10.1080/15563650600671746

6. Takekawa K, Ohmori T, Kido A, Oya M. Methamphetamine body packer: acute poisoning death due to massive leaking of methamphetamine. *J Forensic Sci* . Sep 2007;52(5):1219-22. doi:10.1111/j.1556-4029.2007.00518.x

7. Merchant K, Schammel C, Fulcher J. Acute Methamphetamine-Induced Hepatic and Pancreatic Ischemia. *Am J Forensic Med Pathol* . Sep 2019;40(3):285-288. doi:10.1097/paf.0000000000000486

8. Pittman HJ. Methamphetamine overdose. *Nursing* . Apr 2005;35(4):88. doi:10.1097/00152193-200504000-00053

9. Goodchild JH, Donaldson M. Methamphetamine abuse and dentistry: a review of the literature and presentation of a clinical case. *Quintessence Int* . Jul-Aug 2007;38(7):583-90.

10. Naidoo S, Smit D. Methamphetamine abuse: a review of the literature and case report in a young male. *Sadj* . Apr 2011;66(3):124-7.

11. Malay ME. Unintentional methamphetamine intoxication. *J Emerg Nurs* . Feb 2001;27(1):13-6. doi:10.1067/men.2001.111825

12. Qutrio Baloch Z, Hussain M, Agha Abbas S, Perez JL, Ayyaz M. Methamphetamine-Induced Cardiomyopathy (MACM) in a Middle-Aged Man; a Case Report. *Emerg (Tehran)* . 2018;6(1):e9.

13. Karch SB. The unique histology of methamphetamine cardiomyopathy: a case report. *Forensic Sci Int* . Oct 10 2011;212(1-3):e1-4. doi:10.1016/j.forsciint.2011.04.028

14. Callaway CW, Clark RF. Hyperthermia in psychostimulant overdose. *Ann Emerg Med* . Jul 1994;24(1):68-76. doi:10.1016/s0196-0644(94)70165-2

15. Wallace ME, Squires R. Fatal massive amphetamine ingestion associated with hyperpyrexia. *J Am Board Fam Pract* . Jul-Aug 2000;13(4):302-4. doi:10.3122/15572625-13-4-302