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Clinical Medicine Insights: Case Reports

Acute Iodine Toxicity From a Suspected Oral Methamphetamine Ingestion

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ABSTRACT

BACKGROUND: Iodine is a naturally occurring element commercially available alone or in a multitude of products. Iodine crystals and iodine tincture are used in the production of methamphetamine. Although rarely fatal, iodine toxicity from oral ingestion can produce distressing gastrointestinal symptoms and systemic symptoms, such as hypotension and tachycardia, from subsequent hypovolemia.

OBJECTIVE: The objective of this case report is to describe a case of iodine toxicity from suspected oral methamphetamine ingestion.

CASE REPORT: A male in his early 20's presented with gastrointestinal symptoms, chills, fever, tachycardia, and tachypnea after orally ingesting a substance suspected to be methamphetamine. The patient had elevated levels of serum creatinine, liver function tests, and bands on arrival, which returned to within normal limits by day 4 of admission. Based on the patient's narrow anion gap, halogen levels were ordered on day 3 and indicated iodine toxicity. This is thought to be the first documented case of iodine toxicity secondary to suspected oral methamphetamine abuse.

CONCLUSION: Considering that the incidence of methamphetamine abuse is expected to continue to rise, clinicians should be aware of potential iodine toxicity in a patient with a history of methamphetamine abuse.

KEYWORDS: methamphetamine, iodine, toxicity

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Introduction

Iodine is a naturally occurring element discovered in the nineteenth century.[1–3] It is available commercially as a tincture or as crystals and widely found in a variety of products including antiseptics, germicides, water treatment chemicals, contrast media, and pharmacologic compounds.[1–7] Dietary sources are so common that the Recommended Daily Allowance (150 µg/day) is optimized or exceeded in most western countries, where intake may be as high as 930 µg/day.[2,4,5] Human beings appear to have a high tolerance, particularly when ingestion is <2 mg/day acutely, because iodine must be converted to iodide, a generally nontoxic substance, or bound to proteins, starches, or unsaturated fatty acids before absorption from the intestine into the blood.[4,6–8]

Iodine is also used in the production of methamphetamine. Iodine crystals are used to produce hydriodic acid, which reduces pseudoephedrine to D-methamphetamine.[1] Producers unable to obtain iodine crystals may produce them by mixing hydrogen peroxide with iodine tincture, which is more readily available for purchase.[1] The tincture also circumvents the Comprehensive Methamphetamine Control Act of 1996, which requires a detailed record of all iodine crystal sales >400 mg.[1]

Case Report

A male in his early 20's with a history of methamphetamine abuse arrived at our institution after orally ingesting a "spoonful" of a tan, gooey pasty substance without smell or taste found inside a bag on the side of a road that he suspected to be

methamphetamine. Shortly after ingestion, he reported the onset of chills, fever, abdominal pain, nausea, vomiting, diarrhea, and tachycardia. He reported drowsiness but no loss of consciousness. The substance was disposed of by the patient prior to arrival.

Upon arrival, he was tachycardic (110 beats/minute) and tachypnic (24 breaths/minute). His oxygen saturation was 89% on room air, which increased to 99% with oxygen via a non-rebreather mask. His temperature and blood pressure were normal (37.6 °C and 112/56 mmHg, respectively). The patient was oriented and responsive, but drowsy and in mild respiratory distress with diminished breath sounds in bilateral lower lobes. He had an elevated serum creatinine and liver function tests, a narrow anion gap (AG), bandemia, and an increased international normalized ratio (Table 1). His thyroid panel was normal. A urine drug screen was negative. His initial electrocardiogram (EKG) showed sinus rhythm with tachycardia, but the rest of his cardiac examination was normal. Chest radiograph indicated a pulmonary infiltrate in the right lower lobe and a chest computed tomography showed small bilateral pleural effusions with consolidation in the bases of both lungs.

The patient was admitted and placed on levofloxacin for pneumonia. On day 2, his symptoms had resolved, but his white blood count (WBC) increased to 20 with a fall in bands to 37%. By day 4, the WBC had returned to normal limits, repeat EKG was normal, and chest radiograph showed the infiltrate and effusions had resolved. Bromide, lithium, and iodine levels were drawn on day 3 due to the narrow AG. The

Table 1. Laboratory results.

LABORATORY PARAMETER	INITIAL[a]	DAY 1	DAY 2	DAY 4	NORMAL VALUES[b]
pH	NM[c]	7.328	NM	NM	7.35–7.45
PCO_2	NM	37.8 mmHg	NM	NM	35–45 mmHg
PO_2	NM	70 mmHg	NM	NM	80–100 mmHg
Bicarbonate (ABG)	NM	20 mmol/L	NM	NM	22–26 mmol/L
Base excess	NM	–6.0 mmol/L	NM	NM	–2.0–2 mmol/L
O_2 Saturation	NM	93%[d]	97%	95%	94–100%
WBC	3.4	8.6	20.4	7.1	4.8–10.8 x 10^3
Hgb	12.9 g/dL	12.1 g/dL	12.1 g/dL	12.5 g/dL	14.0–18.0 g/dL
Hct	37.4%	35.4%	35.1%	36.1%	42–52%
Platelet	166	142	137	160	150–430 x 10^3
Bands	NM	47%	37%	9%	0–9%
Na	NR[e]	139 mmol/L	138 mmol/L	138 mmol/L	136–145 mmol/L
K	3.3 mmol/L	4.3 mmol/L	3.9 mmol/L	3.8 mmol/L	3.6–5.0 mmol/L
Cl	NR	107 mmol/L	106 mmol/L	106 mmol/L	98–107 mmol/L
HCO_3	NR	25 mmol/L	25 mmol/L	29 mmol/L	22–28 mmol/L
Scr	1.6	1.4 mg/dL	1 mg/dL	0.7 mg/dL	0.6–1.3 mg/dL
BUN	NR	9 mg/dL	9 mg/dL	7 mg/dL	7–18 mg/dL
Glucose	NR	123 mg/dL	96 mg/dL	85 mg/dL	65–110 mg/dL
AST	8 IU/L	330 IU/L	126 IU/L	29 IU/L	10–42 IU/L
ALT	270 IU/L	303 IU/L	212 IU/L	84 IU/L	10–40 IU/L
Albumin	6.3 g/dL	2.9 g/dL	3.0 g/dL	3.2 g/dL	3.5–5 g/dL
Total bilirubin	3.5 mg/dL	0.9 mg/dL	NM	NM	0.2–1 mg/dL
ALP	79 IU/L	39 IU/L	45 IU/L	46 IU/L	32–92 IU/L
CK	NM	36 IU/L	NM	NM	38–174 IU/L
TropI	NM	<0.15 ng/mL	NM	NM	<0.15 ng/mL
Lipase	NM	20 U/L	NM	NM	8–57 U/L
TSH	NM	1.275 uIU/mL	NM	NM	0.35–5.5 uIU/mL
INR	1.4	NM	1.6	NM	1

Notes: [a]Initial laboratory values from outside hospital within the same healthcare system. Normal values remain the same. [b]Normal values are accepted normal range for our institution at time of patient presentation. [c]Laboratory parameter not measured. [d]Oxygen saturation per arterial blood gas measurement. Patient initially presented with an oxygen saturation of 89% on room air per pulse oximetry which increased up to 99% after being placed on oxygen delivered via non-rebreather mask. [e]Laboratory parameter not reported.
Abbreviations: pCO_2, partial pressure of carbon dioxide; pO_2, partial pressure of oxygen; ABG, arterial blood gas; O_2, oxygen; WBC, white blood count; Hgb, hemoglobin; Hct, hematocrit; Na, sodium; K, potassium; Cl, chloride; HCO_3, bicarbonate; SCr, serum creatinine; BUN, blood urea nitrogen; AST, aspart aminotransferase; ALT, alanine aminotransferase; ALP, alkaline phosphatase; CK, creatinine kinase; TSH, thyroid stimulating hormone; INR, international normalized ratio; NM, not measured; NR, not reported.

bromide and lithium levels were undetectable; however, the iodine level was elevated at 325 μg/L indicative of toxicity (normal reference range for our laboratory is 40–95 μg/L). Had an iodine level been obtained at admission, it is suspected the level would have been >1,000 μg/L based on the estimated plasma half-life of 10 hours in an otherwise healthy adult.[9]

The patient was discharged on day 4 with a scheduled outpatient appointment. He did not return for his appointment and was lost to follow-up.

Discussion and Conclusion

To our knowledge, this is the first report of acute iodine toxicity due to suspected oral methamphetamine ingestion. We could not definitively determine the substance to be methamphetamine because it was disposed before arrival. The patient's substance abuse history and product description supports the hypothesis that it was methamphetamine. His negative urine drug screen could not definitively rule out methamphetamine ingestion. Urine drug screens are designed to detect amphetamine; the metabolite of methamphetamine.[10] However, only approximately 4–7% of methamphetamine is excreted as D-amphetamine.[10] Multiple studies have illustrated low rates of detection of methamphetamine ingestion through this method.[9] The exact mechanism is unknown, but it is suspected that the low detection rate may be due to a saturation of amphetamine excretion mechanisms.[10] Furthermore, his clinical presentation was consistent with oral iodine ingestion, which heightens the suspicion of methamphetamine. His narrow AG motivated the order for serum halogen levels, which showed an iodine level congruous with toxicity.

The patient's symptoms were also consistent with oral iodine ingestion. While free iodine is in contact with the gastrointestinal mucosa, even sub-lethal doses are bothersome. He experienced abdominal distress shortly after ingestion. Iodine is extremely irritating to the gastrointestinal tract and often results in gastrointestinal corrosion, abdominal pain, and vomiting.[6–8,11] Subsequent hypovolemia and electrolyte imbalances are thought to be responsible for systemic effects reported in other patients, including hypotension, tachyarrhthmias, cardiovascular collapse, and liver dysfunction.[6,8] Our patient presented with tachycardia and liver dysfunction, which were resolving at discharge, as would be expected with declining iodine levels.

In cases of fatal iodine ingestion, death occurs within 48 hours.[2,5,8] Once one of the most common sources of suicide attempts, iodine's implication in lethal acute toxicity is rare, due in large part to the almost immediate emetic effect iodine induces, and has not been reported since the 1930s.[6–8] The absence of a positive substance identification is a reflection of clinical practice where the understanding of the toxidrome may guide patient care and evaluation. In this case, a blood iodine level was measured. A urine iodide level could also be obtained to help estimate the previous 24-hour average concentration, but this has been studied primarily in patients with more long-term iodine exposure.[7] Thyroid levels for this patient were within normal limits. However, it is important for clinicians to remember to evaluate these biomarkers due to the well-known impact of iodine on thyroid function, which may be particularly evident in a patient with long-term or chronic use.[7]

Methamphetamine use continues to rise and the National Drug Intelligence Center predicts that domestic production will increase over the next few years.[12] It is one of the five most common illicit substances encountered in acute care settings.[9] While this case focuses on a suspected oral ingestion, iodine toxicity could occur with other routes of methamphetamine abuse. Given the findings of this case, clinicians should be aware of the possibility of iodine toxicity in patients with a history of methamphetamine abuse.

Author Contributions

Conceived the concepts: MNB. Analyzed the data: MNB. Wrote the first draft of the manuscript: MNB. Made critical revisions: MNB. The author reviewed and approved of the final manuscript.

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