

## Drug Abuse and its Incidence- 3 Different Collective Case- Studies on Acute Iodine Toxicity, Petrol Abuse and Sodium Bicarbonate Abuse

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### ABSTRACT

Substance abuse is the use of a drug or other substance for a non-medical use, with the aim of producing some type of 'mind-altering' effect in the user. This includes both the use of illegally produced substances, and the abuse of legal drugs, in a use for which the substance was not intended. As drug experts, pharmacists can use their knowledge of problems linked to misuse of prescription drugs to better understand comparable problems associated with other legal and illegal drugs. In this article we are concentrating mainly on 3 different case studies in which different substances are abused, First case is about Acute Iodine Toxicity from a Suspected Oral Methamphetamine Ingestion, It is one of the five most common illicit substances encountered in acute care settings. The second case study is about Petrol Abuse in a 10-Year-Old Child with Mental Retardation Inhalant use disorder is being highlighted in this case followed by 3rd of a Forty Years Abuse of Baking Soda, Rhabdomyolysis, Glomerulonephritis, Hypertension leading to Renal Failure. NaHCO<sub>3</sub> abuse is discussed in this in this case.

Conclusion: Educate yourself, Be accessible and open-minded, Be clear, Keep it relaxed, Grab opportunities, Discuss peer pressure, Practice what you preach are some of the points that need to be considered! For combating drug abuse.

**Keywords:** Combating, drug abuse, iodine, petrol

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### INTRODUCTION

Substance abuse is the use of a drug or other substance for a non-medical use, with the aim of producing some type of 'mind-altering' effect in the user. This includes both the use of illegally produced substances, (1,2) and the abuse of legal drugs, in a use for which the substance was not intended. Often this involves use of the substance in excessive quantities to produce pleasure, to alleviate stress, or to alter or avoid reality (or all three) how does Drug Abuse Differ from Addiction? "Compulsively seeking (3) to use a substance, regardless of the potentially negative social, psychological and physical consequences".

#### Incidence of Drug Abuse

Ministry of Social Welfare of the Government of India launched a multi-

centre research programme covering several urban centres including Bombay, Delhi, Hyderabad, Jabalpur, Jaipur, Madras and Varanasi (4). The sample (N =25,000 approximately) covered both male and female students who were pursuing generic as well as professional courses.

All drugs can be divided into seven broad categories (5,6).

- Cannabinoids (e.g., hashish and marijuana)
- Stimulants (e.g., amphetamines and cocaine)
- Depressants (e.g., Xanax and Qualudes)
- Narcotics (aka opioids and morphine derivatives, e.g., heroin, opium, Vicodin)
- Hallucinogens (e.g., LSD and mescaline)
- Dissociative anesthetics (e.g., PCP)
- Other compounds (e.g., steroids and inhalants).

**CASE STUDY 1:**

Acute Iodine Toxicity from a Suspected Oral Methamphetamine Ingestion

**Introduction**

Iodine is a naturally occurring element discovered in the nineteenth century. 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. 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. Iodine is also used in the production of methamphetamine.

**Table 1: Source Khan and Krishna (1982)**

Centre	Non-users (%)	Former users (%)	Current users (%)	Sample size (N)
Bombay	57.8	6.7	35.0	4151
Delhi	52.5	12.9	34.6	3991
Hyderabad	77.8	4.9	17.1	903
Jabalpur	56.4	14.1	29.5	4415
Jaipur	77.6	3.9	18.5	4081
Madras	76.8	3.7	19.5	3580
Varanasi	54.6	11.8	33	3852
Total	62.9	8.9	28.2	24973

**Case Report**

A male in his early 20's with a history of methamphetamine abuse arrived at kims hospital 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) . 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 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**

The patient's substance abuse history and product description supports the hypothesis that it was methamphetamine. 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. Subsequent hypovolemia and electrolyte imbalances are thought to be responsible for systemic effects reported in

other patients, including hypotension, tachyarrhythmia's, cardiovascular collapse, and liver dysfunction. It is one of the five most common illicit substances encountered in acute care settings. 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.

**Table 2: Laboratory Results**

Laboratory Parameter	Initial	Day 1	Day 2	Day 4	Normal Values
pH	NMc	7.328	NM	NM	7.35–7.45
PCO2	NM	37.8 mmHg	NM	NM	35–45 mmHg
PO2	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
O2 Saturation	NM	93%d	97%	95%	94–100%
WBC	3.4	8.6	20.4	7.1	4.8–10.8 x 10 <sup>3</sup>
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 <sup>3</sup>
Bands	NM	47%	37%	9%	0–9%
Na	NRe	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
HCO3	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

## CASE STUDY 2

Petrol Abuse in a 10-Year-Old Child with Mental Retardation

### Introduction

Inhalant use disorder is of increasing interest in the current era, though Indian literature regarding the subject is scant. Inhalants are volatile substances that produce chemical

vapors that can be inhaled to induce a psychoactive or mind-altering effect. These inhalants are grouped into volatile solvents (gasoline, glue, paint thinner, nail polish remover), aerosols (spray paint, hair spray), gases (nitrous oxide, helium), and nitrites (amyl and butyl nitrites). Inhalants most commonly

used by Indian adolescents are glue, shoe polish, toluene, spray paints, petrol, and lighter fluids.

#### **Case report**

A 10-year-old boy, living in a village near the Nalgonda district of Hyderabad in India, presented to the psychiatric outpatient department with his parents.

The parents reported that the child was a full-term normal delivery and had no antepartum/peripartum complication. At the age of one month, he had a single episode of febrile generalized tonic-clonic seizure. Later, the child had delayed developmental milestones. He started speaking at the age of three years in monosyllables such as mama and papa. At five years of age, he started speaking two to three words with no further development until presentation at our department at age 10 years. His speech was not clear and was difficult to comprehend even by his parents. His social judgment and skills were also not appropriate for his chronological age. His motor milestones were appropriate for his age.

At the age of approximately 5 years, the parents noted the child being increasingly restless, running the whole day, playing, and having difficulty keeping calm and being seated. At the age of approximately eight years, one of the neighbors reported that the child was seen huffing petrol from the tank of a vehicle. He was observed huffing petrol from the tanks of vehicles regularly two to three times a day. After huffing petrol, there would be remarkable changes in the child's behavior; his restlessness and hyperactivity would reduce for two to three hours. After this, the child would again grow restless and become hyperactive. His IQ was initially assessed by a clinical psychologist using Seguin Form Board (SFB) Test. The score was in the range of 40–45, categorized as moderate mental retardation (moderate intellectual disability as per Diagnostic and Statistical manual 5th edition (DSM 5). Considering that petrol has alcohol-like CNS depressant properties and carbamazepine had property of reducing impulsive behavior, the child was prescribed clonazepam (a benzodiazepine) 0.5 mg thrice daily and carbamazepine 100 mg thrice daily. Parents reported gradual reduction of petrol use, and the child was fully abstinent after three months. Conners ADHD parent version revised short form questionnaire was again filled after three months, and the score was 60. Thus, there was minimal improvement in ADHD features.

#### **Discussion and conclusion**

A calming effect with the petrol use was reported by the parents. Gasoline acts as a CNS depressant, which calms the hyperactivity and

gives the child a pleasurable effect. Inhalant dependence is now increasingly reported as a substance use disorder among adolescents, who are always curious about experiencing new things. Withdrawal features such as restlessness, irritability, inattention, sleep disturbance, and craving (difficulty in preventing child to remain away from substance use) were observed in this child.

#### **CASE STUDY 3**

Forty Years Abuse of Baking Soda, Rhabdomyolysis, Glomerulonephritis, Hypertension leading to Renal Failure.

#### **Introduction**

In the 1800 century baking soda (BS; NaHCO<sub>3</sub>) was used to put air into breads and cakes, and it was a common ingredient in many baking goods to cause batter and dough to rise. BS was also a common home remedy to treat dyspepsia. Intake of large amounts of BS could cause serious adverse effects with hypokalemic hypochloremia, metabolic alkalosis, hypernatremia, and hypocalcaemia. Prolonged administration of NaHCO<sub>3</sub> (NaBic) did not produce renal failure (RF) unless volume depletion was present.

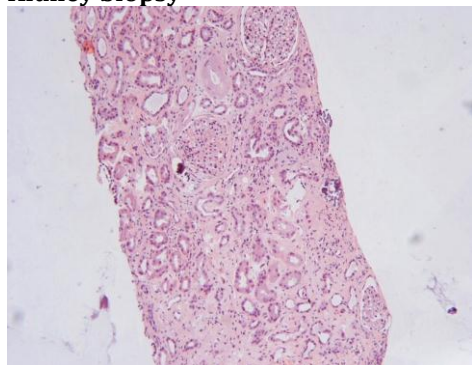
#### **Case Study**

A sixty-four year old man, with no previous medication and no previous visit to any doctor or nurse, was sent to our emergency department due acute epileptiform convulsions. At arrival to the hospital he was unconscious and he appeared hypovolemic and dehydrated. Pupils, thus moving around had normal reactions to light and no parietic regions were registered. Neurological reflexes were normal and symmetric, and muscles of the extremities were slightly rigid.

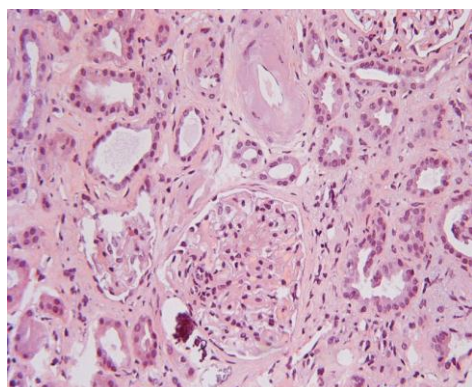
He had a history of many years of alcohol abuse which included short episodes of extreme drinking. At arrival to the hospital he tested negative for alcohol, barbiturates and benzodiazepine. Initially, the blood pressure (BP) had been measured to 195/148 mmHg and had fallen to 130/104 mmHg at arrival. Heart rate was 100 beats/min, regular, with normal heart and lung status. Electrocardiogram showed slightly descending ST levels in lateral leads. Chest x-ray was normal. Fever was absent. Plasma creatinine (p-creat) and urea nitrogen (p-urea) concentration was 780 μmol/l and 33 mmol/l, respectively. Serum creatinine kinase (CK) concentration was 2980 U/L (ref. 40–280 U/L) decreasing to 309 U/L during the next 4 days. The pro-brain natriuretic peptide (pro-BNP) concentration was 28100 ng/l (ref. 194 ng/l). Blood glycosylated hemoglobin fraction was 5.2% (ref. 4–6%). Central venous pressure

was 0–1 mm Hg. BP and tachycardia was controlled with multiple i.v. doses (2.5 to 5 mg) of metoprolol tartrate. After correction of the metabolic alkalosis, the patient regained consciousness and could give an exact history of his alcohol and BS abuse. He had eaten BS during 40 years due to dyspepsia caused by alcohol consumption. Initially he took 10–15 grams daily, slowly increasing the amount to about 30–35 grams daily, and during the last year he ingested more than 50 gram of BS every day (about 1.5–2.0 kg/month).

#### **Kidney biopsy**



**Figure 1: Overview of the renal biopsy showing slight increase of mesangial material and interstitial calcium deposits (HE stain, magnification 125x)**



**Figure 2: Glomeruli with increased mesangial material and calcification outside glomerular capsule (HE stain, magnification 250x)**

#### **Discussion and Conclusion**

The metabolic derangements due to chronic bicarbonate ingestion may cause sustained hypokalemia, hypernatremia, and hypoxia, with variable clinical presentations including seizures. Beside chronic alcohol abuse and its abrupt withdrawal, seizures, hypoglycemia, and hypokalemia, may all together have contributed to rhabdomyolysis. We assume that the sequence of events was as follows; the presence of a long-standing untreated hypertension and a previous not diagnosed mesangial proliferative

glomerulo-nephritis which caused the initial renal failure. Then long-term alcohol and BS abuse provoked convulsions and subsequent rhabdomyolysis associated with severe volume contraction aggravating the renal failure. Concomitantly he had developed a grave metabolic alkalosis with massive disarrangements of the electrolytes contributing to the manifest renal failure. We suggest that the renal failure was not brought about by BS alone in our patient, but it might have participated or provoked its development.

#### **CONCLUSION**

Educate yourself – Find out about the issues. Check with local schools, agencies and information services for the resources you will need. Find books at the local library. The more informed you are, the easier it will be to discuss the issues.

- Be accessible and open-minded – The idea is to open a dialogue. Listen to what your teens have to say. Ask questions and do not judge.
  - Be clear – Your main message should be clearly stated: “don't use drugs” should be the core theme of your discussions.
  - Keep it relaxed – Avoid the “We have to talk” approach. Relax and talk about it over supper or when you're driving to the mall. If you are casual, it will help your children to be more honest and willing to talk.
  - Grab opportunities – Use teachable moments. If you have just seen a TV show or poster that discusses the issue, use this to allow the discussion to come up naturally.
  - Discuss peer pressure – Talk about ways to say no and how to deal with the pressures to conform and fit in.
  - Practice what you preach – Kids imitate adults. If you abuse drugs yourself, no matter what you tell your teens, your actions speak louder than words. Avoid being a hypocrite and perhaps it is time that you examine your own problem first.
- Educate, be accessible and open-minded, be clear, keep it relaxed, grab opportunities, discuss peer pressure, and practice what you preach.

#### **ACKNOWLEDGEMENT**

I would like to acknowledge the people who mean world to me, my parents, & my sister. I extend my respect to my parents and all elders to me in the family. I don't imagine a life without their love and blessings. Thank you mom, dad, for showing faith in me and giving me liberty to choose what I desired. I consider myself the luckiest in the world to have such a supportive family, standing behind me with their love and support.

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