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Case Report

Acute Toxicity Related to 25G-NBOMe Use: An Internet High

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The NBOMe series is an emerging class of synthetic hallucinogens with limited data available on their use and effects. Whilst toxicity to related substances exist in the literature, no such cases exist for 25G-NBOMe. This case describes a 17-year old male who presented to the Emergency Department with seizures having ingested 25G-NBOMe that had been purchased over the Internet. He was tachycardic, hypotensive and hyperthermic on arrival and required admission to the Intensive Care Unit (ICU) due a persistently low Glasgow Coma Scale (GCS) and profound metabolic derangement. His inpatient stay was prolonged by a persistently high creatine kinase with associated transient acute kidney injury. In contrast, an accompanying friend who had ingested the same drug developed no adverse effects. Our patient's clinical presentation was consistent with reports of adverse outcomes associated with other drugs in the series and demonstrates that acute toxicity can also be seen with 25G-NBOMe with potentially life threatening outcomes.

Key words: *acute kidney injury, drug misuse, NBOMe*

Introduction

This case report highlights the difficulties and dangers associated with an emerging class of synthetic hallucinogens easily available via the Internet. There is limited data available on the use and effects of the NBOMe series and currently no easily accessible rapid screening test to enable analytical confirmation. Unfortunately their high potency, as compared to other psychoactive substances, means users are at high risk of acute toxicity due to inadvertent overdosing. In addition to this, as illustrated in this case, individuals may show wide variation in response to the drug, which further complicates identifying those at risk of toxicity.

Case Report

A 17-year-old male presented to the Emergency Department (ED) having ingested 25G-NBOMe. He was fitting on arrival and received 4 mg IV lorazepam to terminate the seizures. His subsequent Glasgow Coma Scale (GCS) fell from 9 (E2, V2, M5) to 6 (E1, V1, M4) out of 15 and initial arterial blood gas (ABG) analysis revealed a severe respiratory and metabolic acidaemia as is presented in Table 1. Initial observations demonstrated a tachycardia, HR 145; mild hyperthermia, T 38.0°C; hypotension, BP 97/76; and bradypnoea, RR 10; SpO_2 100% (on 15 l). Pupils were equal and reactive, 4mm in diameter. An ECG demonstrated a sinus tachycardia but normal QRS and QT intervals. He was intubated shortly after arrival and transferred for a CT head en-route to the Intensive Care Unit (ICU). This demonstrated no acute bony injury, intracranial haemorrhage, cerebral contusion, oedema or evidence of raised intracranial pressure. Sequential blood gas analyses are presented in Table 1 along with subsequent blood tests in Table 2. Paracetamol and salicylate levels were normal. Un-

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Table 1. Blood gas analysis on arrival, 75-mins and prior to discharge from ICU

	Initial ABG (15 l/min)	Plus 75-mins (FiO_2 0.6)	Final ABG prior to discharge from ICU (FiO_2 0.21)
pH	< 6.80	7.34	7.36
pCO_2 (kPa)	15.7	5.0	5.4
PO_2 (kPa)	48.5	31.4	13.8
HCO_3^- (mmol/L)	Incalculable	20.0	22.6
BE (mmol/L)	Incalculable	-5.2	-2.7
Lactate (mmol/L)	> 20.0	4.0	0.9
BM (mmol/L)	12.6	9.3	5.1
Na^+ (mmol/L)	143	135	141
K^+ (mmol/L)	3.8	3.7	4.3
Cl^- (mmol/L)	97	104	108
Ca^{2+} (mmol/L)	1.44	1.2	1.26

Table 2. Creatine Kinase and renal function assays during inpatient stay

	Day 0	Day 1	Day 2	Day 4	Day 6
CK [40-235] (mmol/L)	394	468	7,281	24,253	17,066
Na^+ [136-145] (mmol/L)	151	137	137	140	137
K^+ [3.6-5.0] (mmol/L)	4.4	3.7	3.6	4.3	4.0
Ur [2.0-7.8] (mmol/L)	6.4	5.8	5.4	3.6	4.2
Cr [71-122] (umol/L)	126	92	144	96	74

fortunately, no screening test was available to enable analytical confirmation of the drug.

Collateral history came from the ambulance crew who had labelled drug packaging and his accompanying friend who also admitted to purchase and consumption of 25G-NBOMe. Each individual had reportedly inhaled 1mg of the substance. After inhalation the index patient described feeling generally unwell and then fell hitting the back of his head, but without loss of consciousness. The ambulance crew found him surrounded by vomit with a GCS of 15/15. A tonic-clonic seizure was then observed, self-terminating after approximately 1-minute. Two further seizures occurred during transfer and a fourth on arrival in the ED.

Treatment was directed at symptoms and metabolic derangements and guided by TOXBASE -- a toxicology database available to clinicians from the U.K. National Poisons Information Service. The patient was fluid resuscitated in ED and ICU with close monitoring of biochemistry and acid base balance. He was extubated without complication the following day with near-resolution of metabolic derangement following fluids and ventilatory support and stepped down from critical care. Creatine kinase (CK) climbed from 394 mmmol/L at presentation to 24,253 mmmol/L on day four and was 17,066 mmol/L on discharge. This was accompanied by a transient derangement in renal function (as demonstrated in Table 2) and mild generalised myalgia. He was discharged on day six after medical clearance and psychiatric review. A further test three-days later revealed a CK of 1,974. In comparison his friend who had consumed the same volume of the substance demonstrated no toxic features but described only mild euphoria and heightened sensory perception. His serial CK levels were normal and he was discharged following an overnight stay in hospital for observation.

The patient made a full recovery with no apparent neurological, renal or hepatic sequelae. He had an open referral to a local walk-in drugs-and-alcohol service and was due to be followed up by his general practitioner.

Discussion

The NBOMe series represent a novel class of synthetic hallucinogens. Their use and reports of acute toxicity have been increasingly documented over the last 5-years.[1,2] However, current literature reports involve toxicity following use of 25I-, 25B- and 25C-NBOMe toxicity and to the author's knowledge there are no reports of acute toxicity associated with 25G-NBOMe.

NBOMes are N-benzylmethoxy derivatives of the 2C family of hallucinogens.[3] Initially discovered in 2003 for research purposes, they act as potent agonists of the 5-HT2A receptor with resulting effects of both mental and physical stimulation, and visual and auditory hallucinations.[4] Euphoria, increased alertness and self-esteem and heightened emotions are commonly reported. The class includes the most commonly used 25I-NBOMe, as well as 25B-NBOMe, 25C-NBOMe, 25D-NBOMe, 25E-NBOMe, 25G-NBOMe (as used in our case), 25H-NBOMe, 25N-NBOMe and 25iP-NBOMe.[2]

Awareness and use of this drug class remains limited when compared with other psychoactive substances as demonstrated by the 2013 global drugs survey where only 2.6% of the 22, 289 responders had used an NBOMe,[5] These substances are increasingly available over the Internet with numerous online forums providing anecdotal analysis of the experience taking any of the 25-NBOMe series. They are sold as "smiles," "solaris," "n-bombs" or by their shortened names such as "25I," "25C," "25G," etc.[3,6] They most commonly appear in the form of "blotters," small pieces of paper infused with the drug and often decorated with colourful designs, but are also available in powder or liquid form.[2] As of June 2014, the NBOMe series have become a Class A controlled drug having previously been described as a "legal high."[7]

Due to the high potency of the NBOMe drugs, typical doses to achieve the desired effects are lower than for other psychoactive substances such as mephedrone and LSD.[6] A high risk of acute toxicity due to inadvertent overdosing is therefore possible. A recent review found 29 published cases describing acute toxicity secondary to NBOMe ingestion, 23 of which relate to 25I-NBOMe.[2] Clinical features of acute toxicity in order of incidence include tachycardia, hypertension, agitation/aggression, seizures, hyperthermia and rhabdomyolosis associated with acute kidney injury.[2,6,8] These features reflect the findings in our case, although our patient was hypotensive on arrival which is also sometimes seen. The cases highlight that a high proportion of those with acute toxicity require a prolonged inpatient stay and often intensive care input.[2] Treatment consists of supportive care with aggressive fluid resuscitation, benzodiazepines for agitation and convulsions, ventilator support and haemofiltration in some cases. Additionally, 25I-NBOMe has been detected in post mortem analysis of eight deaths however the significance of this is uncertain.[2,3]

There is limited data on the use and effects of NBOMe ingestion. It remains difficult to predict which users will develop severe symptoms,[3] as demonstrated by the two patients admitted in our case. In addition, the NBOMe class are not part of routine drug screening available in hospitals, with no current rapid screening test easily available.[3] Liquid chromatography-tandem mass spectrometry methods have been used to identify subtypes within the NBOMe class but these are not available at all centres.[9] Further research is required to evaluate the effects of NBOMe abuse and clinicians should be aware of the potential dangers of this emerging class of synthetic hallucinogens.

Conclusion

The N-BOMe series is an emerging class of synthetic hallucinogens that may increasingly present doctors with a challenge in the acute setting: They are easily available via the Internet with limited data available on their use and the effects of the various subtypes. They are not currently routinely tested for in drug screens and their high potency means users are at high risk of acute toxicity which appears to show inter-individual susceptibility. Our case report adds to the cadre of information regarding these drugs and suggests that 25G-NBOME shares many of the features of toxicity associated with other drugs in it's class. Treatment is currently largely supportive focussing on fluid resuscitation and treatment of complications including seizures, rhabdomyolysis, hyperthermia, metabolic acidosis, agitation and delirium.

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