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TOXICOLOGY LETTER

COMPRISING THE NEW YORK CITY AND UPSTATE NEW YORK POISON CENTERS

Fearless Fungi Foraging

Contributed by: Nicholas Nacca, MD, Medical Toxicology Fellow, Michael Hodgman, MD Medical Toxicologist SUNY Upstate Medical University, Upstate NY Poison Center. Syracuse, NY Poison Center, Syracuse NY

"There are bold mushroom gatherers and there are old gatherers, but there are no old bold mushroom gatherers." - *unknown origin*

Case:

A 65-year-old gentleman presented to an emergency department (ED) stating that he foraged mushrooms from his back yard in Upstate NY. He reports having cooked and eaten the mushrooms at 9 AM the day prior to presentation to the ED. Fourteen hours after ingestion, the patient began to experience nausea, vomiting, and diarrhea. The symptoms persisted for 12 hours, after which emergency care was sought.

In 2009, there were 6818 cases of exposure to mushrooms reported to poison control centers, the vast majority of which were non lethal (2011 AAPCC report). These exposures ranged from minor GI upset to critical illness. The clinical manifestations will depend on the type of mushroom consumed and the quantity ingested.

What toxins are found in mushrooms?

For the practice of Primary, Emergency and Intensive Care, it is necessary to understand that mushrooms can be classified by the toxin they contain *(see table 1)*.

Table 1. Common mushroom toxins and the associated target organ

Gastrointestinal	Muscarin (Peripheral Cholinergic toxicity) Cyclopeptides (Hepatotoxicity) Coprine (Disulfram-like reaction with Alcohol) Other irritants (most common)
Central Nervous System	Gyromitrin (Seizures) Muscimol/Ibotenic aid (Sedation/Delerium/Seizures) Psilocybin (Hallucinations)
Renal	Orelline (Delayed renal failure) Allenic norleucine (Delayed renal failure)

How can one distinguish hepatotoxic from gastrointestinal irritant ingestions?

In New York State the majority of poisonous mushrooms ingested are GI irritants, however cyclopeptide containing species that cause hepatotoxicity are indigenous as well. In the majority of cases the initial clinical presentation will make it difficult to distinguish cyclopeptied containing mushrooms from the more benign GI irritant mushrooms. Most patients will present with nausea, vomiting and diarrhea. These presentations may

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Program Announcements

UNY: Combined Medical Examiner/Toxicology Case Conference, Center for Forensic Science 10/10 1:30-3:30pm

Please mark your calendars for the **Seventeenth Annual Toxicology Teaching Day** 11/6/13 in Syracuse. Email *morer@upstate.edu* for more information.

NYC: Consultants Case Conference • The first Thursday of the Month from 2-4pm

Please call administrative telephone numbers for more information and to attend remotely.

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represent the early stages of a disease progressing to fulminent heptic failure and death. A critical piece of clinical information is the time from ingestion to the onset of symptoms. A patient with onset of symptoms within 3 hours of ingestion suggests a GI irritant mushroom. Alternatively, delayed onset of GI toxicity, especially if greater than 12 hours, is more worrisome for ingestion of a cyclopeptide containing mushroom. The cyclopeptide containing mushrooms are found in the Amanita, Galerina and Lepiota genera. The toxic cyclopeptide here, amatoxin, is a hepatotoxin. If leftover mushrooms are available, a physical specimen can be sent to a mycologist for identification. A less reliable method involves pictures sent to the poison control center to be forwarded to a mycologist for visual identification. One should be mindful that once cooked, it is unlikely mushroom particulates can be visually identified.

What is the initial approach to management of a mushroom poisoned patient?

With any and all poisoned patients, the initial approach involves assessment of airway breathing and circulation followed by assessment of vital signs and intravenous access. A blood glucose measurement should be immediately obtained. Laboratory tests including LFT's, renal function, and coagulation factors must be sent. The clinician should be aware that a normal initial laboratory workup does not rule out a potentially toxic ingestion. The poison center should be contacted. They may be able to enlist the aid of a mycologist in the identification of the mushroom. The patient will likely require antiemetic and crystalloid therapy. Activated charcoal may be useful in select cases to limit absorption of toxin but administration is often limited by symptoms of nausea and vomiting.

What is the management of confirmed cyclopeptide containing mushroom ingestion?

If suspicion for cyclopeptide containing species ingestion is high based on time of ingestion and onset of symptoms, or if the mushroom has been identified as such species by a mycologist, the patient warrants aggressive management with multiple medical interventions. Treatment options for a suspected or confirmed amanita exposure include the following:

N-Acetylcysteine - Though administration of intravenous N-Acetylcysteine lacks the support of large scale well designed clinical data in the setting of poisoning with hepatotoxic mushrooms, its activity as a free radical scavenger and safe adverse reaction profile make it a reasonable therapeutic option (Ward et al).

Penicillin - In vitro studies suggest that penicillin may inhibit human hepatocyte uptake of cyclopeptides. When used in ingestion of cyclopeptide containing mushrooms, the dose is significantly higher than is typically used for alternative disease states (Letschert et al).

Silibinin - Though not FDA approved, and currently in clinical trials, Silymarin is an herbal preparation containing Silibinin which may be a life saving intervention in the mushroom poisoned patient. It is available over the counter in the nutritional supplement milk thistle extract. In addition to free radical scavenging properties, it is thought to inhibit transport of cyclopeptide across hepatocyte membranes *(Enjalbert et al)*. Legalon® is the brand name of Silibinin-C-2', 3-dihydrogen succinate disodium salt. This drug is currently being evaluated in the US as part of an FDA approved open label study. The drug can be obtained via the investigator's 24 hotline (866-520-4412).

Case Conclusion:

The above described patient was given antiemetics, IV fluids, activated charcoal, and IV N-Acetylcysteine on presentation. Pictures of the uncooked mushrooms were available and sent to a mycologist who identified the mushroom as a cyclopeptide containing mushroom of the Amanita species known as "Destroying Angel." The patient's laboratory profile was consistent with acute hepatic failure with hepatic enzymes peaking at AST 4597 and ALT of 3393. Additionally his INR peaked at 1.9 and total bilirubin at 5.1. The patient was started on over the counter available milk thistle while Legolon® SIL was drop-shipped to the hospital. Amatoxins undergo enterohepatic recirculation. Although the possible benefit is as yet well defined this patient also was treated with percutaneous biliary drainage. Over the course of one week's treatment the patient's hepatic injury resolved. On hospital day 7 the patient was discharged from the hospital.

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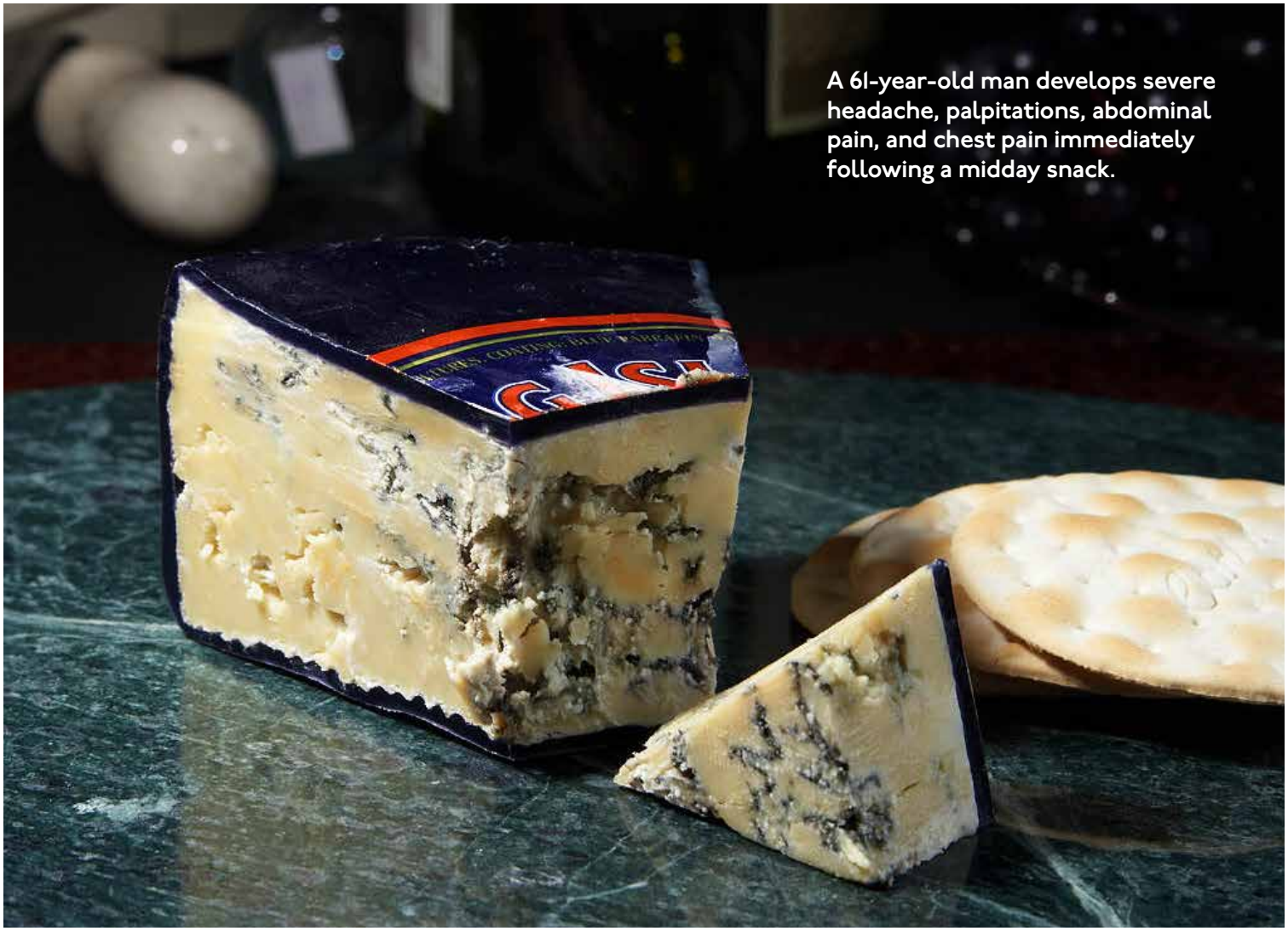
Moment on Your Lips…

Rana Biary, MD, Lewis S. Nelson, MD, and Brenna Farmer, MD



A 61-year-old man develops severe headache, palpitations, abdominal pain, and chest pain immediately following a midday snack.

Case

A 61-year-old man with a history of coronary artery disease, hypertension, hyperlipidemia, and depression presents to the ED complaining of severe headache, palpitations, abdominal pain, and chest pain, which he states developed immediately following a midday snack. He denies use of alcohol or illicit drugs and has not had any recent changes to his medication regimen. His vital signs are: blood pressure (BP), 228/114 mm Hg; heart rate, 56 beats/min; respiratory rate, 16 breaths/min; temperature, afebrile. Oxygen saturation is 97% on room air. The patient's physical examination is within normal limits. A bedside abdominal sonogram shows a normal abdominal aorta of 2.5 cm in diameter.

The patient receives metoclopramide and morphine for his headache. A computed tomography of the head is unremarkable. The patient's initial serum troponin is negative, with an electrocardiogram (ECG) showing sinus bradycardia in the range of 40 beats/min, with no acute ST-T wave changes.

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Moment on Your Lips…

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What are some of the pharmacotherapeutic causes of hypertension?

Many therapeutic drugs, particularly in overdose, can elevate BP. For example, amphetamine causes central nervous system (CNS) sympathetic overactivity by increasing the release of catecholamines and decreasing their reuptake from the synapses.[1] This leads to both peripheral vasoconstriction (α_1-adrenergic receptor-induced) and increased inotropy ($ß_1$- and $ß_2$-adrenergic receptor-induced), which results in elevated BP, tachycardia, diaphoresis, and dilated pupils, all consistent with the sympathomimetic toxidrome.[2]

In cases of overdose, certain medications will initially cause hypertension, followed by hypotension. For instance, clonidine, guanabenz, and guanfacine are all structurally similar and act centrally as α_2-adrenergic receptor agonists.[2] At initial presentation of an overdose, however, the peripheral α_2-adrenergic effects may predominate, causing pronounced hypertension.[3] Excessive doses of centrally acting α_2-adrenergic receptor agonists typically lead to depressed mental status and respiratory rate.

In contrast to clonidine, yohimbine, an alternative sexual enhancement agent, is a centrally acting α_2-adrenergic receptor antagonist. In overdose, hypotension may occur initially due to vasodilation resulting from the blockade of peripheral α_2-adrenergic receptors. Central effects eventually prevail and cause enhanced sympathetic output and sympathomimetic effects.[2]

Case continuation

The patient's spouse arrived at bedside an hour after presentation and provided additional history—notably, that the patient has been taking tranylcypromine (a monoamine oxidase inhibitor [MAOI]) 70 mg daily for longer than 20 years for depression. She further stated that he had consumed a snack consisting of aged cheese approximately 20 minutes before onset of symptoms.

Why are monoamine oxidase inhibitors of concern?

MAOIs, such as norepinephrine, serotonin, and dopamine, are neurotransmitters that are deaminated by MAO in the presynaptic terminal following reuptake. MAO exists in two forms, each with distinct anatomic and physiologic characteristics. MAO-A, though most prominent in the intestines and liver, is responsible for metabolizing serotonin and norepinephrine in the CNS. When MAO-A is inhibited by an MAOI, the elevated synaptic concentration of serotonin produces an antidepressant effect. The second type, MAO-B, is preferentially located in the CNS and metabolizes dopamine. MAO-B inhibitors are used to treat Parkinson disease.[2]

Tranylcypromine and phenelzine, the antidepressant MAOIs available in the United States, irreversibly inhibit both MAO-A and MAO-B.[4] Tyramine, like amphetamines, induces the release of norepinephrine at the synaptic terminal.[4] Although present in many foods, tyramine is metabolized by MAO-A in the intestinal wall and liver and is not systemically bioavailable.[2] In patients taking a nonselective MAOI (which inhibits MAO-A), ingestion of greater than 6 mg of tyramine can cause a hyperadrenergic crisis. Foods with a high content of tyramine include aged cheese, soy sauce, sauerkraut, certain wines and beers, and chicken liver.[5] Approximately 20% of patients on tranylcypromine will develop a hyperadrenergic crisis; in one case series, 6 of 27 crises were precipitated by consumption of cheese.[6] Of note, tranylcypromine is an amphetamine derivative and can lead to a hyperadrenergic crisis in overdose—independent of tyramine.[7]

Other causes of hyperadrenergic crisis in patients on MAOIs include amphetamine-like decongestants in cough and cold preparations (eg, pseudoephedrine). While the hyperadrenergic crisis is related to serotonin toxicity, it is a clinically distinct entity. Serotonin toxicity occurs in patients taking MAOIs in which triggers of presynaptic serotonin release, such as meperidine or dextromethorphan, produce muscle rigidity, hyperthermia, delirium, and tremor.

How should drug-induced hypertension be managed?

The decision to urgently lower a patient's BP is based on the presence of end-organ damage and not solely on numerical BP. A severe headache, for example, may not simply be due to hypertension but rather represents a hypertensive emergency resulting from cerebral edema or hemorrhage. If treatment for drug-induced sympathomimetic toxidrome is required, the sole use of a ß-adrenergic receptor antagonist (ß-blocker) to normalize vital signs is contraindicated. Because ß-blockers block both $ß_1$- and $ß_2$-adrenergic receptors, the peripheral ß-adrenergic receptors are unopposed, allowing enhanced peripheral and coronary artery vasoconstriction.[2,8,9]

Phentolamine, a short-acting α-adrenergic antagonist, lowers BP through vasodilation and is the preferred treatment agent.[2] Often, patients taking an MAOI are given a prescription for an immediate-release dihydropyridine calcium channel blocker (CCB), which may be utilized in the pre-hospital setting. CCBs, such as nifedipine, can also be administered in the ED.[10] Because the elevated BP and resulting headache are due to excessive CNS stimulation, benzodiazepines also have a therapeutic role.

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Mail Order Madness

Nicholas Connors, MD, and Lewis S. Nelson, MD

A 14-year-old boy has a seizure after smoking a substance he and a friend purchased through the Internet.

Case

A 14-year-old boy suffered a seizure shortly after he and a friend smoked a substance they ordered from an herbal products Web site. His friend called EMS before fleeing from the scene. Upon presentation to the ED, vital signs were: blood pressure, 137/86 mm Hg; heart rate, 143 beats/min; respiratory rate, 20 breaths/min; temperature, 100.0˚F. Oxygen saturation was 99% on room air. On physical examination, he was awake, though laughing and making nonsensical word associations. He did not appear to be hallucinating, but did not respond appropriately to questions. Abrasions were noted on his forehead, and the pupils were dilated and reactive. His skin was not flushed, diaphoretic, or dry. With the exception of tachycardia, cardiac, pulmonary, and abdominal examinations were normal. He had no focal motor or sensory deficits or tremor, and motor tone was normal. Initial laboratory values were: finger-stick glucose, 210 mg/dL; sodium, 140 mEq/L; anion gap, 20 mEq/L. An electro-cardiogram showed sinus tachycardia at 135 beats/min, with a QRS duration of 80 ms and QTc interval of 453 ms.

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Mail Order Madness

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What could these boys have taken?

Abused substances that cause agitation, confusion, and central nervous system (CNS) and cardiovascular excitability are numerous and varied. Users of cocaine, amphetamines, and methylxanthines present with symptoms and signs characteristic of the sympathomimetic toxidrome, such as agitation, tachycardia, and hypertension. Additional sequelae of their use include hyperthermia, seizure, dysrhythmia, and ischemia.

Although this patient was somewhat agitated, postictal, and tachycardic, his mental status was suggestive of a dissociative agent such as phencyclidine (PCP), ketamine, or dextromethorphan. PCP use may result in extreme agitation and, at times, violent behavior; ketamine and dextromethorphan produce similar, but more moderate, effects. Dextromethorphan is found in cough and cold preparations and is commonly abused by teenagers due to its accessibility as a nonprescription medication. Herbal products like jimsonweed *(Datura stramonium)*, yohimbine *(Pausinystalia yohimbe)*, and *Salvia divinorum* are readily available over the internet but do not generally cause the combination of hyperadrenergic vital-sign abnormalities and CNS effects seen in this case.

Synthetic cannabinoids such as "Spice" (also known as "K2" or "potpourri") and synthetic cathinones ("bath salts") are also available through the Internet because of their uncertain legal status as products labeled, "not for human consumption." In addition to these agents, which have garnered significant media and regulatory attention over the last several years, substituted phenylethylamine derivatives also have potent psychoactive sympathomimetic effects.

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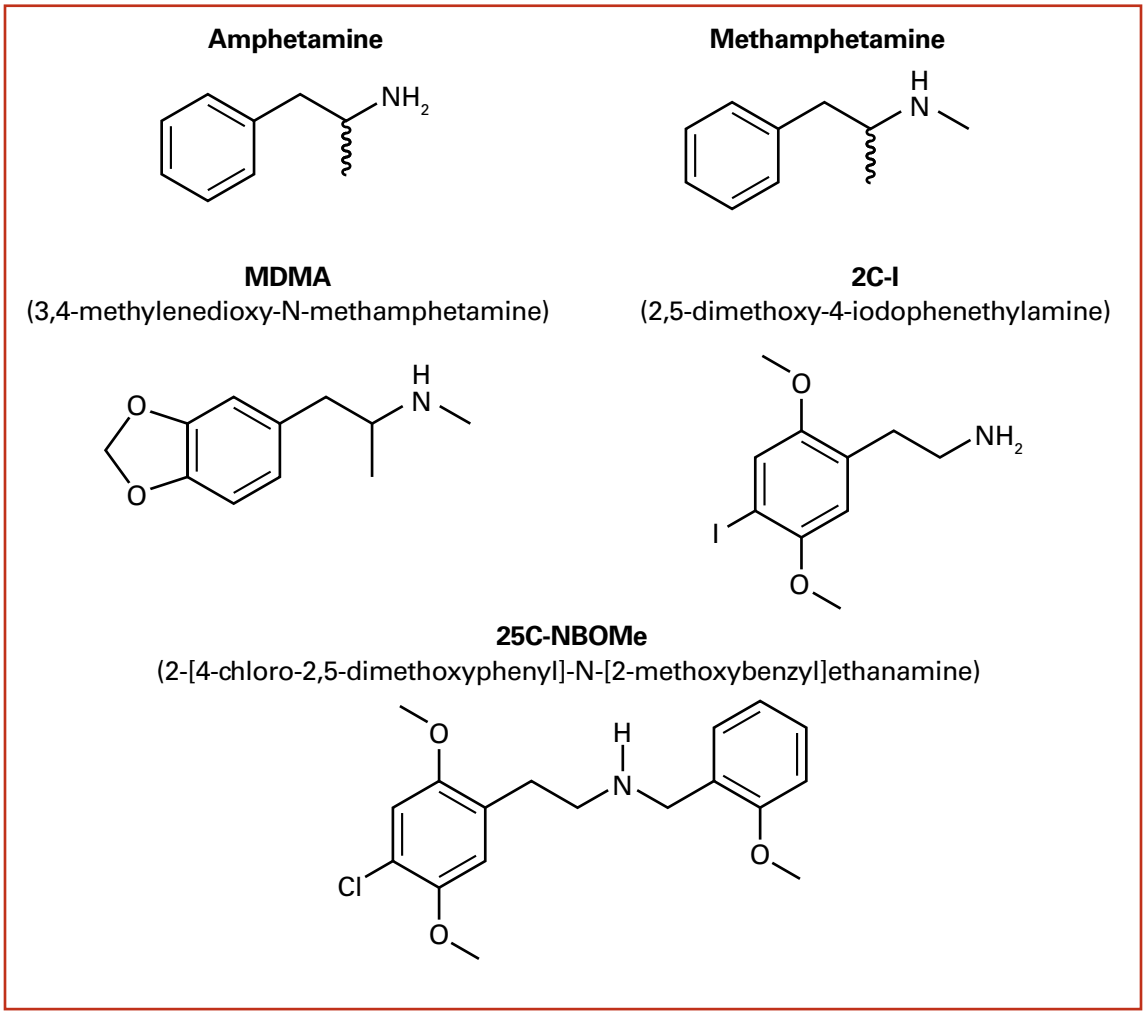

FIGURE Molecular structure of substituted phenylethylamines.

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Mail Order Madness

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Case continuation

When the patient's parents arrived at the ED, they brought a small sheet of blotter paper, thought to be the source of the drug. Few drugs are sufficiently potent enough to be distributed on blotter paper, which requires doses in the microgram range. Examples include LSD, certain tryptamines, and substituted phenylethylamines (4-bromo-2,5-dimethoxyamphetamine; DOB). In this case, the label on the blotter paper suggested the nature of the agent.

What are substituted phenylethylamines?

Phenylethylamines, also generally known as amphetamines, are organic compounds that function as CNS neuromodulators. They have a clinical role as stimulants, psychedelics, antidepressants, decongestants, bronchodilators, and anorectics. The addition of functional groups to the backbone of the phenylethylamine molecule may alter the clinical effects (both psychoactive and sympathomimetic) and pharmacokinetics of these drugs. (See the Figure on page 18 for the chemical structure of commonly abused substituted phenylethylamines.) Substitutions along the ethylamine chain tend to enhance the sympathomimetic effects by stimulating the release of catecholamines such as dopamine and norepinephrine from presynaptic neurons. These substitutions make the agent more "speedy." In addition, substitutions on the ring structure improve the effects mediated by the serotonin receptors, making the substance more psychoactive or "trippy." With the substitution of halogens along the ring structure, the agent typically becomes more potent, and only microgram doses are needed to achieve the same "speedy" or "trippy" effects, or a combination of these. Phenylethylamines can be taken orally, intravenously (IV), or through insufflation or inhalation.

The 2C series agents fall into the phenylethymamine class and generally have the core structure of 3,4-methylenedioxy-N-methamphetamine (MDMA, "Ecstasy") with a substituted halogen on the six-carbon ring and methoxy side chains at the 2 and 5 positions on the ring structure. As noted, a halogen typically increases the substance's potency, and the methoxy groups on the ring enhance serotonergic activity, resulting in greater psychoactive affects. Of late, there has been a proliferation of synthetic products that vary slightly in their chemical structure, with resulting unique clinical effects.

The 2,5-dimethoxy-N-(2-methoxybenzyl)phenylethylamine (NBOMe) series of substances are structurally similar to the 2C series but were not widely used before 2010. The mechanism of action involves partial agonism at the $5HT_{2A}$ receptor, resulting in its enhanced "trippy" effect. NBOMe is generally insufflated or applied to blotter paper for buccal absorption. In one cohort, 57% purchased the substance through the internet, and 83% used it in conjunction with other illicit drugs.[1] The most common subjective effects were changes in tactile, visual, and auditory perception. Trembling, sweating, and blurry vision were the most common adverse effects.[1] Case reports of intoxication with a 2C agent describe recurrent seizures and serotonin toxicity,[2] and fatal toxic leukoencephalopathy[3] noted on magnetic resonance imaging. Effects last about 4 to 8 hours.[4]

What is the legal status of phenylethylamines?

The Controlled Substances Act of 1970 established the Drug Enforcement Agency's (DEA) five "schedules" of substances based on abuse potential, indications for medical use, and safety profile. Schedule I substances have a high potential for abuse and are deemed unsafe even under medical supervision.[5] Drugs such as heroin, lysergic acid diethylamide (LSD), mescaline, and marijuana are on the list of Schedule I substances. Additions to this list include MDMA in 1985, gamma-hydroxybutyrate (GHB) in 2000, and specific synthetic cannabinoids and synthetic cathinones in 2012.

On January 4, 2013, certain substances within the class of 2C drugs were designated Schedule I by the DEA.[6] However, a major difficulty with synthetic agents is the efforts of "street chemists'" to be "one step ahead" of legal authorities. As each specific substance is banned, a derivative or analog is synthesized, avoiding legal prosecution. Furthermore, it is difficult to ban an entire class of agents, and research into potential benefits of substances can be stifled without significant justification.

The Federal Analog Act of 1986 is an addition to the Controlled Substances Act and addresses issues related to "designer drugs."[7] The wording in this act is vague— perhaps intentionally—with regard to what constitutes an analog, and there has been mixed experience in case law. For example, a federal district court in Colorado ruled that the law was "unconstitutionally vague," making successful prosecution of those who possess or use these substances difficult—especially since these compounds are typically labeled as "not for human consumption," suggesting, almost tongue-in-cheek, that they are not to be abused.[8]

How is phenylethylamine toxicity managed?

After attending to the patient's vital systems, sympathomimetic effects should be treated with rapid-onset benzodiazepines such as diazepam or midazolam. If seizures are refractory to benzodiazepines, propofol, paralysis with a neuromuscular blocker, and intubation should be considered. A core body temperature above 105°F necessitates rapid cooling. For patients with altered mental status and only mild agitation, reducing stimulation as much as possible can be sufficient.

Evaluation should include assessing the patient's electrolytes to screen for hyponatremia (common with certain ring-substituted amphetamines) and other causes of seizure. Measuring creatine kinase to assess for rhabdomyolysis should be considered if there a history of prolonged immobilization or severe hyperthermia. Noncontrast computed tomography (CT)

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Moment on Your Lips…

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of the head can be performed in cases of altered mental status, particularly if there was head trauma or concerns for subarachnoid bleeding. Patients should be observed until symptoms abate and then discharged home, as appropriate, after evaluation for suicidality along with counseling on the dangers of drug use and possible referral for drug treatment.

Case conclusion

The patient received multiple doses of IV diazepam for sedation. Upon waking the next day, he stated that he used 25C-NBOMe (2-[4-chloro-2,5-dimethoxyphenyl]- N-[2-methoxybenzyl]ethanamine; also called "Pandora," "Dime," "Vortex," "Cimbi-82") on blotter paper for the first time on the day of presentation. He added that he and his friend had ordered the drug from a syntheticproduct website that advertised the substance as "legal LSD."

The patient was admitted to the hospital for further testing. He had a normal noncontrast CT of the head, and anion gap narrowed to within normal parameters. He was observed overnight in the pediatric intensive care unit where his vital signs normalized. He was discharged the next day without report of sequelae.

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CASE STUDIES IN TOXICOLOGY **Series Editor:** Lewis S. Nelson, MD

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Case conclusion

The patient was treated with diazepam, which lowered the BP and relieved his other symptoms; phentolamine was *not* required. He was then admitted to the hospital for observation and had serial troponin measurements that peaked at 0.46 ng/mL, without associated ECG changes. The inpatient team discussed changing the patient's antidepressant regimen, but he declined to do so, stating that tranylcypromine was the only medication that could successfully treat his depression. Upon discharge, patient was counseled to avoid consuming aged cheese, wine, and other tyramine-containing foods and decongestants.

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