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# Selected Topics: Toxicology

## NIACIN TOXICITY RESULTING FROM URINE DRUG TEST EVASION

Anne M. Daul, MD* and Michael C. Beuhler, MD†‡

*Department of Emergency Medicine, Carolinas Medical Center, Charlotte, North Carolina, †Carolinas Poison Center, Charlotte, North Carolina, and ‡Department of Emergency Medicine, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina
*Corresponding Address*: Anne M. Daul, MD, Department of Emergency Medicine, Carolinas Medical Center, 1000 Blythe Blvd., 3rd Floor MEB, Charlotte, NC 28203

□ **Abstract—Background: Niacin, a well-established agent for treating dyslipidemia, has been promoted on the Internet as a method for passing urine drug screening, although there are no data to support its use for this purpose. In a handful of cases, this practice has resulted in serious niacin toxicity. Objectives: The aim of this article is to describe a unique clinical presentation of niacin toxicity. Case Report: A 23-year-old previously healthy man presented to an Emergency Department with altered mental status, fever, acute renal failure, microangiopathic hemolytic anemia, thrombocytopenia, and coagulopathy. It was revealed that he had taken approximately 22.5 g of sustained-release niacin over the preceding 48 h in an attempt to pass a pre-employment urine drug screen. After a complicated hospital course that included mechanical ventilation for respiratory failure and hemodialysis for acute renal failure, the patient made a full recovery and was discharged 10 days after his initial presentation. Conclusion: After a massive niacin overdose, the young man in this case presented with a complex clinical picture that mimicked concurrent thrombotic thrombocytopenic purpura and disseminated intravascular coagulation. Although this patient was fortunate to make a full recovery, the case highlights the potential for multi-system toxicity with niacin overdose, and the potential for harm posed by medical misinformation on the Internet.** © 2011 Elsevier Inc.

□ **Keywords—niacin; overdose; toxicity; thrombotic thrombocytopenic purpura; disseminated intravascular coagulation; urine drug screen**

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### INTRODUCTION

Due to its wide accessibility, the Internet has become an important, but often unreliable, source for medical information and advice. Internet sites may be the first resource that individuals consult in the case of inquiries about medical problems that are felt to be embarrassing or socially discouraged. The scientific integrity of materials posted on the Internet varies dramatically, and misinformation can be outright dangerous. One recent example of such misinformation involves a rash of web postings that promulgate niacin as a means to pass urine drug screening. Over the past several years, there have been a handful of case reports of niacin overdoses after its use for this purpose.

Since the 1950s, niacin has been recognized for its favorable effects on plasma lipoproteins. Today, it remains the least expensive lipid-modulating agent and the single best agent for raising high-density lipoprotein (1). Niacin is sold over the counter in a variety of preparations, including immediate release, sustained release, and extended release; prescription forms include both a regular form (Niacor®; Upsher-Smith Laboratories Inc., Maple Grove, MN) and an extended release form (Niaspan®; Abbott Laboratories, Abbott Park, IL). Many laypersons, who may not realize that a vitamin can be toxic, self-dose niacin for dietary supplementation, dyslipidemia, and other non-medical purposes. The following report describes a novel case with a complex clinical

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picture including features of thrombotic thrombocytopenic purpura (TTP) and disseminated intravascular coagulation (DIC).

## CASE REPORT

A previously healthy 23-year-old man presented to an Emergency Department after the onset of altered mental status, generalized weakness, and vomiting. He was unable to provide history secondary to his mental status. His mother revealed that he had a history of drug abuse, including 3,4-methylenedioxymethamphetamine (MDMA, or Ecstasy). She noted that he had been taking large quantities of niacin over the past 2 days in hopes of passing an upcoming pre-employment urine drug screen.

On examination, the patient was mildly febrile at 38.1°C (100.7°F), with a heart rate of 99 beats/min, a blood pressure of 104/62 mm Hg, and oxygen saturation ($SaO_2$) of 99% on room air. In general, he was a well-developed man who was acutely ill-appearing. He was diaphoretic and jaundiced. The pulmonary examination revealed bilateral basilar crackles and a productive cough. His mental status was notable for confusion and agitation. He had mild right-sided weakness. The remainder of his examination was within normal limits. A fingerstick blood glucose was 142 mg/dL. Approximately 90 min after presentation, the patient had transient hypotension, with a blood pressure of 81/45 mm Hg, which responded to a 1-L bolus of 0.9% saline.

Multiple attempts to obtain blood for laboratory testing failed secondary to hemolysis. The first set of laboratory values and imaging studies from the evening of his presentation were as follows. Arterial blood gas (ABG) revealed pH 7.47, $pCO_2$ 32 mm Hg, $pO_2$ 119 mm Hg, and bicarbonate 20 mmol/L. A complete blood count showed white blood cells (WBC) 21,000/$\mu$L (87% granulocytes), hemoglobin 10.9 g/dL, and platelets 21,000/$\mu$L. Red blood cell studies showed a mean corpuscular volume of 84.7 fL, mean corpuscular hemoglobin (MCH) 30.3 pg, mean corpuscular hemoglobin concentration (MCHC) 35.8 g/dL, and red cell distribution width of 15.7%. Red blood cell morphology was notable for moderate schistocytosis, anisocytosis, and poikilocytosis. A chemistry panel showed sodium 141 mmol/L, potassium 4.4 mmol/L, chloride 105 mmol/L, bicarbonate 23 mmol/L, blood urea nitrogen 20 mg/dL, and creatinine 1.4 mg/dL. The aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were 177 units/L and 49 units/L, respectively. The total bilirubin was 6.95 mg/dL. Prothrombin time (PT) was 16.3 s (normal range 10.5–13.5 s), activated partial thromboplastin time was 37.8 s (normal range 20–34 s), and international normalized ratio (INR) was 1.8. A D-dimer was markedly elevated at 4200 ng/mL (normal range $<$ 400 ng/mL). Creatine phosphokinase (CK) was 349 units/L (normal range 35–230 units/L) with a muscle brain fraction (CK-MB) of 0.08 ng/mL (normal). Urinalysis was notable for a specific gravity of $<$ 1.005, pH of 5.0, 2+ protein, and 4+ blood. Urine microscopy showed 2–4 WBC/high power field (hpf) and red blood cells were too numerous to count per hpf. Salicylate level was 13.3 mg/dL (normal range 2.8–20 mg/dL). Ethanol level was 0 mg/dL and acetaminophen level was 0 $\mu$g/mL. A urine drug screen was negative for amphetamines, barbiturates, benzodiazepines, cocaine, opiates, phencyclidine, and cannabinoids. Computed tomography scan of the head was negative for acute findings. Chest radiography showed diffuse bilateral pulmonary infiltrates.

The patient was empirically started on ticarcillin-clavulanate, imipenem, and metronidazole. He was transferred to the intensive care unit, where, on hospital day 2, he developed generalized seizure activity. He was given diazepam with termination of seizure activity and loaded with 1 g of phenytoin. His hemoglobin and platelets trended down to 9.0 g/dL and 19,000/$\mu$L, respectively, and he subsequently received 3 units packed red blood cells (PRBCs), 4 units fresh frozen plasma (FFP), and a platelet transfusion. The patient's creatinine rose to 1.9 and his urine output was poor. During an episode of agitation, he became acutely hypoxic, and an ABG revealed pH 7.50, $pCO_2$ 29 mm Hg, and $pO_2$ 55 mm Hg. Despite supplemental oxygen, the patient was persistently hypoxic, and endotracheal intubation was performed.

On hospital day 3, the patient's hemoglobin was 10.4 g/dL and platelets were 43,000/$\mu$L. Dialysis was initiated for oliguric renal failure. An electroencephalogram performed at the bedside was consistent with metabolic encephalopathy. On hospital day 4, an Infectious Disease consult was obtained and his antibiotics were adjusted to ticarcillin-clavulanate, ciprofloxacin, and vancomycin. His hemoglobin trended down to a nadir of 8.8 g/dL, prompting a transfusion of 2 more units PRBCs, and his hemoglobin subsequently stabilized around 12 g/dL. On hospital day 5, the patient was extubated and transferred to a general medical floor. Dialysis was terminated secondary to improved renal function. On day 10, the patient was discharged from the hospital to home in good condition.

Additional pertinent laboratory testing and trends revealed the following information. He was persistently hyperglycemic for the first 4 days, with blood sugars running in the mid-200s. Transaminases and CK peaked on days 3 and 4, with peak levels of AST 1400 units/L, ALT 1187 units/L, and CK 3858 units/L. Troponin I peaked on day 2 at 8.62 ng/mL (normal range 0.00–0.10 ng/mL), with a normal CK-MB percentage throughout his course. Bilirubin levels fell precipitously after admis-