CASE REPORT

Pseudoephedrine-Induced Tachycardia and Hypertension in an Infant Misdiagnosed as Supraventricular Tachycardia: A Case Report

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Abstract

A seven-month-old Hispanic girl was referred to the pediatric emergency department for evaluation of acute onset of supraventricular tachycardia. Physical exam revealed an extremely agitated infant with inconsolable crying, tachycardia, tachypnea and hypertension. Toxicologic studies of urine were positive for pseudoephedrine. The possible mechanisms and the principle of management of pseudoephedrine toxicity are discussed.

key words: Pseudoephedrine, toxicity; symptom; mechanism; management

List of abbreviations: Pseudoephedrine (PE), Emergency Department (ED), Beats per minute (bpm), Central nervous system (CNS)

Introduction

Pseudoephedrine (PE) is a therapeutic constituent of numerous commonly used, over-the-counter cough and cold preparations. Colds, coughs, and upper respiratory infections are common in children. Many times parents will turn to one of dozens of cough and cold preparations for relief. While these products are generally considered to be safe by both health care providers and parents, the potential for significant morbidity and mortality with accidental or intentional overdose does exist. No studies have proven the efficacy of cough and cold preparations in facilitating recovery from these illnesses, and most children will eventually improve on their own (1,2). Although a variety of symptoms of acute PE toxicity have been described in

adults, few descriptions of the toxic effects of this drug in children have been reported (1,3,4). This article describes the presentation and clinical course of an infant exposed to PE for whom the presenting signs and symptoms were tachycardia, hypertension, inconsolable crying, irritability, and fever.

Case Report

A previously healthy Hispanic seven-month-old, 9-kg female infant was transferred to our Emergency Department (ED) from an outside hospital for further evaluation of supraventricular tachycardia (SVT). Earlier in the day, the infant's mother had noticed fever, irritability, and a reluctance to eat. The infant was evaluated by the primary care physician and referred to a local ED because of tachycardia. Initial heart rate was 198 beats/min (bpm) and was noted to be as high as 232 bpm, Blood pressure was 140/70 mmHg, and temperature was 38.5°C. Physical examination was normal except for irritability, tachycardia, and hypertension. She was transferred to our hospital for further evaluation and management.

The infant was transported via pediatrics transport life flight, connected to continuous cardio-respiratory monitoring and heart rate was as high as 230 bpm but otherwise she was stable. A rhythm strip was obtained (Fig.1) that was consistent with sinus tachycardia.

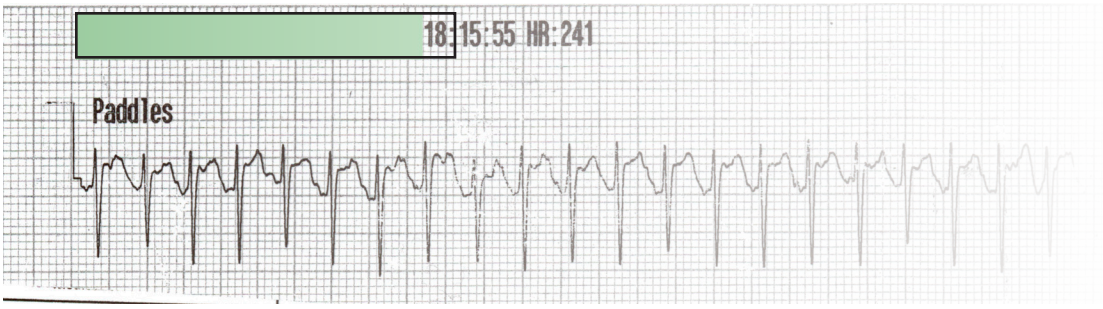

Figure 1. A rhythm strip at paper speed of 25 mm/sec showing sinus tachycardia

Physical examination on admission to our department was remarkable for extreme agitation, crying, and inconsolable irritability, and equal but sluggishly reactive pupils. Vital signs were noted to be temperature 38.6°C, pulse 200 bpm, blood pressure 150/114 mmHg, and respiratory rate 40 breaths/min. The cardiac examination revealed a normal S_1 and S_2 with no gallops or murmurs, and normal capillary refill. There was no hepatomegaly. A bolus of 20 ml/kg of normal saline and a dose of codeine and acetaminophen suspension were administered. Complete blood count and urinalysis were normal. Other laboratory values included the following: sodium 137 mEq/L; potassium 4.2 mEq/L; chloride 111 mEq/L; bicarbonate 13 mEq/L; anion gap 13 mEq/L; glucose 107 mg/dl; blood urea nitrogen 7 mg/dl; creatinine 0.4 mg/dl; calcium 9.1 mg/dl. Urine drug screen was performed with thin-layer chromatography methods, detected amphetamines and opiates.

The patient was admitted to the inpatient service for observation. She became less agitated, the heart rate dropped to 140 bpm and the blood pressure returned to normal value. Child Protective Services were subsequently notified because the initial drug screen was positive for amphetamines. Both parents denied use of this drug. All symptoms resolved within 24 hours of onset, and the patient became relaxed and playful. Additional confirmatory and specific toxicology test demonstrated pseudoephedrine in the urine. During the police investigation, samples from the patient's previously prepared formula bottles were found in the family house and tested positive for amphetamines. Police also found Sudafed tablets in the patient's home. The patient's 7-year-old sister described her mother opening the Sudafed, slicing the tablet in half, crushing the tablet, and then placing the powder in her infant's feeding bottle. The mother denied this action. The patient was discharged to

the parents' care with Child Protective Services to follow-up on an outpatient basis.

Discussion

Pseudoephedrine, a stereoisomer of ephedrine, is a direct nonspecific alpha and beta adrenergic receptor stimulant. It causes the release of stored norepinephrine from neurons, resulting in enhanced sympathetic tone and central nervous system (CNS) stimulation. The release of norepinephrine from sympathetic nerve endings is responsible for the peripheral effects, and the release of dopamine is responsible for the central nervous system response (2-4). The volume of distribution of PE is 2.0 to 3.0 liters, and the average half-life ($t_{1/2}$) is 7 h. It undergoes renal excretion with minor hepatic metabolism (5). The drug is well-tolerated by most adults, with tachycardia and elevated blood pressure generally noted in daily doses > 180 mg.

Children may be especially susceptible to adverse effects of PE. Improper dosing, abuse, or intentional over dosage can result in severe cardiovascular and neurologic adverse effects. Clinical toxicity of PE presents with CNS stimulation, tachycardia, hypertension, dysrhythmias and myocardial infarction. CNS stimulation can manifest as extreme agitation, restlessness, insomnia, seizures, psychosis and intracranial hemorrhage (1,3,4).

These signs and symptoms are also the presenting features of many serious pediatric diseases (e.g., intracranial lesions and sepsis). Therefore, PE toxicity in children can be a difficult diagnosis to make. These patients may undergo extensive tests and expensive procedures before the definitive diagnosis is confirmed. Our case clearly represents PE toxicity: the infant was agitated, tachycardic, and hypertensive. Initial urine drug screen detected amphetamines, but later on, additional confirmatory and specific toxicology tests demonstrated PE in urine. Most sympathomimetics are readily detected as amphetamines in urine by immunoassay (6).

This patient was misdiagnosed as having supraventricular tachycardia by the referring medical center. On arrival to our ED, the patient was in sinus rhythm; the heart rate was variable and as low as 170 bpm when quiet. Over the course of the ED visit, heart rate decreased to150 bpm and blood pressure returned to normal. Dysrhythmias have also been reported due to PE toxicity (7,9). The arrhythmogenic effects of PE could be explained by the sympathetic stimulation and shortening of cardiac refractory periods, which permits the development of re-entrant cardiac arrhythmias (9,10).

Our patient had an unexplained metabolic acidosis, which rapidly resolved. Cocaine, also a sympathomimetic, has been reported to cause metabolic acidosis, partly attributable to its vasoconstrictive effects (11,13). Vasoconstriction may cause anaerobic conditions in the peripheral tissue as has been reported with pheochromocytomas and with epinephrine infusion, or it may inhibit the clearance of lactic acid (12,14). Catecholamines may also induce acidosis by their effect on intermediary metabolism 9150. PE is a sympathomimetic agent and may also cause an acidosis by the same mechanisms. The rise in temperature is caused by vasoconstriction, agitation, and muscle rigidity produced by the overdose.

General supportive care is the main treatment for PE toxicity and there is no antidote. The ABCs (airway, breathing, circulation) should be addressed immediately, and the vital signs need close monitoring. Patients should be well hydrated and should receive appropriate volume replacement; cardiac monitoring for arrhythmias is essential (6). Appropriate laboratory evaluation includes, blood gases, chemistry profiles, urine drug screen, urinalysis, electrocardiography, and complete blood count. Benzodiazepines, butyrophenones, or a combination of these medications have been recommended to treat agitation. Control of agitation helps significantly in cooling a hyperthermic patient, as well as treating PE-induced hypertension. PE is rapidly absorbed, and gastric lavage is unwarranted if there has been more than 60 min since the toxic ingestion.

Kolecki (16) had previously reported a case series of inadvertent methamphetamine poisonings among children. This review revealed that pediatric patients with

methamphetamine toxicity commonly presented with similar signs and symptoms of PE toxicity. *Centruroides sculpturatus* envenomation can cause symptoms such as inconsolable irritability, restlessness, roving eye movements, and increased salivation (16,17).

In conclusion, this case demonstrates that pediatric patients with PE toxicity can present with signs and symptoms very similar to those of cardiovascular or neurologic pediatric emergency. Physicians who encounter such an irritable child should remember to ask the caregivers about the possibility of PE or methamphetamine exposure. With the increasing use of smokable crystal methamphetamine, physicians should be aware of its potential complications. Physicians working in scorpion-infested areas should include infantile scorpion envenomation in the differential diagnosis of sympathomimetic toxicity.

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