A Rare Case of Caffeine Storm due to Excessive Coca-Cola Consumption

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Caffeine, a methylxanthine, is a mild central nervous system (CNS) stimulant used worldwide. Mild intoxications occur frequently, most often due to consumption of caffeine tablets or energy drinks with high caffeine content, and usually do not require treatment or medical aid. Severe intoxication, a rare phenomenon, may be fatal.

At moderate doses, caffeine inhibits the chronotropic and inotropic activity of the adenosine receptor, both in the brainstem and in the heart. At high doses, caffeine acts as a phosphodiesterase inhibitor, which results in accumulation of intracellular cAMP causing prolonged and intensified sympathetic nervous system activation. It also sensitizes dopamine receptors, increases the intracellular calcium concentration and causes noradrenalin release. Toxic levels may cause a catecholamine storm, affecting the CNS and cardiovascular system and is associated with severe hypokalemia, rhabdomyolysis, renal failure, and lactatemia [1-4]. The CNS effects may range from psychomotor agitation, anxiety and nervousness to altered consciousness, rigidity, and seizures. The cardiovascular effects are cardiac dysrhythmias, including supraventricular and ventricular tachyarrhythmias, hypertension and rarely hypotension; fever may also be present [1-4]. The clinical presentation may simulate diabetic ketoacidosis (DKA) or sepsis and is often a diagnostic challenge.

We present here a rare case of caffeine intoxication due to excessive Coca-Cola consumption.

PATIENT DESCRIPTION

A 27 year old woman with an unremarkable medical history presented to the emergency room of Assaf Harofeh Medical Center, complaining of palpitations, chest pain and shortness of breath of 1 hour duration.

Upon admission, she mentioned having been at a party the night before but denied consumption of alcohol or using any recreational drugs. She also denied previous fever, diarrhea or vomiting. Nevertheless, she said she had consumed extremely excessive amounts of Coca-Cola, approximately 4 L, about 2 hours prior to her admission. She did not report habitual Coca-Cola consumption or previous excessive consumption of caffeine-containing drinks. Physical examination revealed only severe agitation and sinus tachycardia of 160 beats/minute. Her weight was 57 kg; oxygen saturation and body temperature were normal.

Prominent laboratory findings included significant leukocytosis of 25,400 cells/mm^3 (4500–10,500), with 89% neutrophils, hyperglycemia of 289 mg/dl (70–100), hypokalemia of 2.24 mmol/L (3.5–5) and metabolic acidosis with pH of 7.32 (7.35–7.45), pCO_2 35, and HCO_3 level 18 mmol/L (22–26). Severe lactatemia with serum lactate level of 5.94 mmol/L (0.5–2.2) was observed. Urinalysis was positive for leukocytes, erythrocytes, ketones and glucose. Other serum electrolytes including magnesium were within normal range as well as renal and liver function tests. Electrocradiogram showed sinus tachycardia without evidence of acute myocardial ischemia. Plain chest X-ray and abdominal X-ray were normal. Despite supportive treatment including intravenous fluids, potassium supplementations and regular insulin, her hypokalemia and metabolic acidosis worsened, the lactate levels increased and her blood pressure declined. A clinical suspicion of diabetic ketoacidosis most probably due to sepsis was made and she was admitted to the intensive care unit (ICU). Empiric antibiotic regimen with ceftriaxone was begun.

Within the first day in the ICU all her complaints resolved and her vital signs returned to normal with no need for further treatment. All laboratory values – including lactate, potassium, blood gases, complete blood count and glucose – normalized with no need for further insulin treatment. Aerobic and anaerobic blood cultures were negative. Urinary toxicology screen was negative for opiates, amphetamines, methamphetamines and cocaine. Ethanol, acetaminophen, and salicylate blood levels were within normal range.

After a further 24 hours of observation during which she was free of symptoms, with normal vital signs and glucose level, she was discharged home.

COMMENT

This patient presented with transient severe agitation, tachycardia, hyperglycemia, metabolic acidosis, lactatemia and hypokalemia, which occurred approximately 2 hours after excessive consumption of Coca-Cola. This clinical presentation was erroneously diagnosed as DKA and/or sepsis but was consistent with severe caffeine intoxication, which usu-

ally occurs shortly after caffeine ingestion and in this patient resulted from unusual amounts of Coca-Cola drunk within a relatively short time.

Cola and energy drinks are known to contain varying amounts of caffeine. According to the Israel Ministry of Health, a cola drink contains approximately 3–21.2 mg caffeine per 100 ml, and according to online sources "Coca-Cola Classic," which our patient consumed, contains 10–10.5 mg caffeine/100 ml, suggesting that our patient consumed ~400–450 mg of caffeine, which is equal to the amount of caffeine in 4–6 cups of coffee or 5–6 cans of energy drinks. Comparable amounts have been reported in other pediatric and adult cases of caffeine intoxications of different severity [5]. It should be noted that a significant variability exists regarding ingested amounts and their toxic effects. Some reports state that ingestion of as little as 50 mg may result in tachycardia and agitation, though not severe intoxication. Plasma caffeine concentration > 80 μg/ml, or consumption of > 3 g caffeine over a short time, might even cause death [2].

Sporadic cases of metabolic derangements after excessive Cola drink consumption have been reported. Severe hypokalemia and hypokalemic myopathy are usually the hallmark of previously reported clinical presentations rather than the full range of severe caffeine intoxication. It is assumed that caffeine itself is only partially responsible for the cola-induced severe hypokalemia, which is also attributed to hyperglycemia-induced osmotic diuresis and transient hyperinsulinemia causing intracellular shift of potassium [5]. Other ingredients present in significant amounts in Coca-Cola such as phosphoric acid do not cause similar clinical presentation. Severe lactatemia, also found in our patient due to inhibition of pyruvate dehydrogenase, is a less described feature of caffeine intoxication and has been found to correlate directly with severe caffeine intoxication. Thus, the extremely elevated lactate in our patient strongly indicates that her caffeine blood level was very high [3,4]. Unfortunately, due to the diagnostic difficulty the initial toxicology screen in our patient did not include caffeine blood levels. Therefore, the exact amount of ingested caffeine cannot be accurately inferred. Trying to extrapolate the patient's plasma caffeine concentration from her lactate level, as a recent paper implied [4], suggests a caffeine concentration of approximately 50 μg/ml – a level considered to indicate severe toxicity. A review of the literature for other substances that may explain this patient's clinical presentation did not reveal additional substances, other than caffeine, that could account for this clinical presentation. Other drugs or toxins such as methylphenidate (Ritalin®), heroine, cyanide, and papaverine overdose may explain some but not all the characteristics in this patient's clinical presentation.

In conclusion, we describe a rare case of severe caffeine intoxication due to excessive Coca-Cola consumption simulating DKA or sepsis. In view of the increasing consumption of caffeine-containing beverages in particular energy drinks, the clinical awareness of caffeine toxicity should be increased.

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