CASE REPORT / PRIKAZ SLUČAJA

RHABDOMYOLYSIS AS A RESULT OF CAFFEINE INTOXICATION IN A SUICIDE ATTEMPT: A CASE REPORT

RABDOMIOLIZA KAO POSLJEDICA INTOKSIKACIJE KOFEINOM U POKUŠAJU SUICIDA: PRIKAZ SLUČAJA

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Abstract

Background: Caffeine intoxication is an increasingly recognized clinical entity due to the widespread availability of caffeine-containing products. Among its broad spectrum of cardiovascular and metabolic manifestations, severe complications such as rhabdomyolysis may also occur.

Case presentation: A 19-year-old male patient was admitted to the emergency department due to intoxication. As reported by the out-of-hospital emergency care physician, the patient ingested 200 tablets, each containing 200 mg of caffeine. Symptoms included sinus tachycardia with a heart rate of 140/min without hemodynamic compromise, profuse vomiting of dark-colored content, and agitation. Initial laboratory tests demonstrated leukocytosis, hypokalemia and mixed acid-base disturbance. The patient was initially treated with symptomatic and supportive therapy. An increasing trend of creatine kinase (500 IU/L to >5000 IU/L) suggested rhabdomyolysis. Therefore, he was admitted to the intensive care unit. Improvement in somatic condition and hemodynamic stabilization were observed. The patient was discharged and referred for further treatment at a psychiatric institution.

Discussion: This case illustrates the variability in the clinical presentation of caffeine intoxication, with rhabdomyolysis but without malignant arrhythmias or acute kidney injury despite ingestion of a potentially lethal dose.

Conclusion: Early supportive therapy, close clinical observation and awareness of less common manifestations are essential for achieving favorable outcomes in patients with caffeine intoxication.

Keywords: caffeine; rhabdomyolysis; tachycardia; toxicology

Sažetak

Uvod: Intoksikacija kofeinom sve se češće pojavljuje kao klinički entitet zbog široke dostupnosti proizvoda koji sadrže kofein. Uz širok spektar kardiovaskularnih i metaboličkih manifestacija mogu se javiti i teške komplikacije, poput rabdomiolize.

Prikaz slučaja: Devetnaestogodišnji muškarac zaprimljen je u hitni bolnički prijam zbog intoksikacije. Prema navodu liječnika izvanbolničke hitne medicinske službe, bolesnik je konzumirao 200 tableta, od kojih je svaka sadržavala 200 mg kofeina. Simptomi su bili sinusna tahikardija frekvencije 140/min bez hemodinamske nestabilnosti, obilno povraćanje tamnog sadržaja i psihomotorni nemir. Početni laboratorijski nalazi pokazali su leukocitozu, hipokalemiju i miješani acidobazni poremećaj. Primijenjeno je simptomatsko i suportivno liječenje. Porast vrijednosti kreatin-kinaze (od 500 IU/L do >5000 IU/L) upućivao je na razvoj rabdomiolize, zbog čega je bolesnik premješten u jedinicu intenzivne medicine. Tijekom hospitalizacije došlo je do poboljšanja somatskog stanja i hemodinamske stabilizacije. Bolesnik je otpušten iz bolnice te upućen na daljnje psihijatrijsko liječenje.

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Rasprava: Ovaj prikaz slučaja pokazuje veliku varijabilnost kliničke slike intoksikacije kofeinom, pri čemu se unatoč unosu potencijalno letalne doze razvila rabdomioliza, ali bez malignih aritmija i akutnog bubrežnog oštećenja.

Zaključak: Rano suportivno liječenje, pomno kliničko praćenje i prepoznavanje rjeđih manifestacija ključni su za postizanje povoljnog ishoda u bolesnika s intoksikacijom kofeinom.

Ključne riječi: kofein; rabdomioliza; tahikardija; toksikologija

Introduction

Caffeine is currently regarded as the most widely used psychostimulant in the world and is available in many foods, beverages and nutritional supplements (1,2). Although generally considered safe at moderate doses, below 400 mg per day, caffeine intake can cause toxicity at much higher, gram quantities (2). Caffeine is absorbed from the gastrointestinal tract within 45 minutes, reaching almost complete bioavailability (1). High caffeine doses induce sympathetic nervous system activity through adenosine antagonism and phosphodiesterase inhibition, resulting in positive chronotropic and inotropic effects. By elevating the intracellular cyclic adenosine monophosphate and calcium levels, cardiac contractility is additionally enhanced. This mechanism may contribute to the development of arrhythmias (3). The clinical presentation of acute caffeine intoxication includes cardiovascular, metabolic, gastrointestinal, neurological/psychological, musculoskeletal and pulmonary symptoms. Although rare, renal failure has also been described secondary to rhabdomyolysis. The cause of this rare clinical feature lies in the induced intracellular calcium release which increases contractile force in the skeletal muscles (2,3). The clinical symptoms are dose-dependent, from mild stimulation to intoxication, which can occur at about 1-2 g of caffeine or serum concentration of >15 mg/L (1). The pharmacokinetic properties of caffeine, such as low plasma protein binding and low volume of distribution, make hemodialysis an effective treatment option in severe intoxication (2).

Caffeine intoxication may cause rhabdomyolysis and acute kidney injury.

Case presentation

A 19-year-old male patient was admitted to the emergency department due to intoxication. As reported by the out-of-hospital emergency care physician, the patient ingested 200 tablets, each containing 200 mg of caffeine, mixed with an energy drink. The medical history revealed suicidal ideation and one previous suicide attempt involving alcohol and diazepam ingestion. Symptoms associated with intoxication included sinus tachycardia with a heart rate of 140/min without hemodynamic compromise, profuse vomiting of dark-colored content, and agitation. Further physical examination revealed no additional abnormalities. The patient also reported a sore throat, upper limb pain, palpitations and nausea. The initial electrocardiogram demonstrated sinus tachycardia without ST-segment or T-wave abnormalities. Initial laboratory tests demonstrated leukocytosis (14.1x109 L), hypokalemia (2.3 mmol/L) and arterial blood gas findings consistent with respiratory alkalosis and concomitant metabolic acidosis (pH 7.473; P_aCO_2 2.18 kPa, HCO_3^- 10 mmol/L; base excess -9 mmol/L). Mildly elevated liver enzymes (AST 65-128 IU/L; ALT 57 IU/L) and D-dimers (746 mg/L) were noted. Creatine kinase (CK) levels were markedly elevated. The patient was initially treated with symptomatic and supportive therapy, including intravenous crystalloid and diazepam administration (10 mg in 500 ml of 0.9% NaCl solution), as well as intravenous potassium replacement (30 mEq KCl). Nasogastric tube placement was attempted to facilitate gastrointestinal decontamination; however, it was unsuccessful due to patient's psychomotor agitation. An increasing trend of CK (500 IU/L to 5317 IU/L) suggested rhabdomyolysis, in the context of intoxication and psychomotor agitation. Therefore, requiring monitoring of renal and cardiac function, he was admitted to the intensive care unit (ICU).

The key events during the patient's clinical course are summarized in Table 1.

Serum creatinine remained within the reference range (74-89 mmol/L) throughout hospitalization, and the patient did not meet KDIGO criteria for acute kidney injury. Initially, the patient was psychotic with expressed suicidal intent. Later during hospitalization, he was cooperative, with correction of metabolic parameters, cessation of vomiting and rhythm stabilization. Continuous cardiac monitoring was performed throughout the three-day ICU stay, and no malignant arrhythmias or conduction abnormalities were documented. Improvement in somatic condition and hemodynamic stabilization were observed. Following regression of relevant laboratory parameters under conservative therapy, the patient was discharged and referred for further treatment at a psychiatric institution. Adequate fluid intake was emphasized among recommendations.

Table 1. Timeline of the clinical course

Time	Event
0 h	Caffeine ingestion
4:30 h	ED presentation
12h	CK increase; ICU admission
96 h	Discharge

Discussion

Clinical presentation of acute caffeine intoxication is characterized by a broad spectrum of symptoms, from sympathomimetic ones to serious cardiovascular and metabolic complications (1,2). The most frequently described early symptoms are severe vomiting and hypokalemia which may serve as diagnostic clues (2). Although our patient presented with tachycardia without hemodynamic compromise, literature suggests more critical presentations (2,4,5). In the case reported by Harsten et al., polymorphic ventricular tachycardia and dangerous acid-base disturbances occurred after 20 g of caffeine ingestion (4). Mitomo et al. described supraventricular tachycardia with a heart rate of more than 240/min following ingestion of 9.8 g of caffeine (5). Delayed absorption, emesis, genetic differences and early treatment may result in heterogeneous clinical outcomes (2). Although the reported ingested dose was approximately 40 g of caffeine, the clinical course was considerably milder than expected. Factors such as repeated emesis with incomplete gastrointestinal absorption, delayed gastric emptying, uncertainty regarding the reported dose and interindividual variability in caffeine pharmacokinetics may explain this discrepancy. Therefore, the reported ingested dose should be interpreted cautiously to avoid underestimating the potentially lethal toxicity of massive caffeine overdose (1,2).

Supportive therapy remains the cornerstone of caffeine intoxication management.

While gastrointestinal decontamination should be considered after potentially life-threatening caffeine ingestion, its application may be limited by agitation and inability to maintain nasogastric access (1,2). Increased muscle activity caused by excessive catecholamine release and psychomotor agitation may lead to muscle injury (2,3). Rhabdomyolysis has been described in about 10% of cases of caffeine intoxication and may lead to acute kidney injury. In this case report, the patient did not develop renal complications, despite CK levels exceeding 5000 IU/L, likely due to prompt initiation of supportive therapy. Serial creatine kinase monitoring should be performed in patients with severe caffeine intoxication, even in the absence of malignant arrhythmias or acute kidney injury, as early recognition of rhabdomyolysis allows timely supportive treatment and may prevent renal complications (1,2,6).

Hemodialysis should be considered in life-threatening caffeine intoxication.

Treatment of acute caffeine intoxication remains supportive and is based on fluid replacement, correction of electrolyte disbalances and cardiac monitoring. In contrast to the favorable outcome of supportive therapy in our patient, several cases required hemodialysis (7,8). In hemodynamically stable patients, conservative therapy may be adequate; however close clinical and laboratory monitoring is essential due to potential life-threatening complications (1,7,8). Recent studies suggest that the largest proportion of severe caffeine intoxication cases resulted from intentional self-poisoning. The second-largest proportion was due to accidental consumption. Ingestion of caffeine with suicidal intent occurs mostly among young adults with underlying psychiatric disorders (1,9).

Reported ingested doses should be interpreted cautiously, as absorption may vary.

Conclusion

Caffeine intoxication may present with a wide range of clinical symptoms, followed by potentially severe systemic complications, such as malignant arrhythmias and acute kidney injury. This case highlights the need for close clinical observation and awareness of less common manifestations, specifically rhabdomyolysis. Early supportive therapy remains crucial when managing intoxicated patients, as well as psychiatric evaluation in cases of intentional overdose.

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