

症 例 短 報

A rare case of coffee-induced acute caffeine poisoning

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Introduction

Caffeine (1,3,7-trimethylxanthine) is a xanthine derivative that is abundant in coffee. Although moderate caffeine consumption can improve health, excessive consumption may be harmful¹⁾, leading to symptoms such as tachycardia, frequent urination, flushing, nausea, anxiety, headaches, sleep disorders, nervousness, tremors, indigestion, and dizziness. Indeed, caffeine poisoning has become a problem in recent years, and fatal cases have been reported²⁾. Although energy drinks and caffeine tablets have been implicated in such overdoses³⁾, caffeine poisoning from the excessive intake of coffee is rare. Herein, we report the case of a 25-year-old Japanese man who was diagnosed with caffeine poisoning after drinking large amounts of coffee.

Case presentation

A 25-year-old Japanese man with no significant medical and family history presented to our emergency department with restlessness; he had palpitations and nausea before arriving emergency depart-

ment. To concentrate on the studying for the test, the patient had consumed 10 tablespoons of powdered coffee (approximately 2 g of caffeine) and 5 cans of coffee (approximately 1 g of caffeine) about 11–17 hours before arrival to the emergency department. He drank only coffee. His initial vital signs were as follows: respiratory rate 23 breaths/min, blood pressure 142/97 mmHg, heart rate 103 beats/min, temperature 36.8 °C, peripheral capillary oxygen saturation 100 % in room air. The patient's Glasgow Coma Scale (GCS) score was E4V4M6, however, he was restless. Physical examination and a coagulation study revealed no abnormalities. He mentioned that he does not usually drink coffee and is on no medication. The laboratory test results were as follows: white blood cell count 9,300/ μ L, red blood cell count 490×10^4 / μ L, hemoglobin 14.5 g/dL, platelets 30.4×10^4 / μ L, sodium 137 mEq/L, potassium 3.0 mEq/L, chlorine 99 mEq/L, urea nitrogen 6.0 mg/dL, creatinine 0.80 mg/dL, aspartate aminotransferase 97 IU/L, alanine aminotransferase 49 IU/L, and creatinine kinase 6,288 IU/L. Arterial blood gas results were as follows: pH 7.50, partial pressure of carbon dioxide 30.3 mmHg, partial pressure of oxygen 149 mmHg, bicarbonate 23.1 mmol/L, base excess 1.1 mmol/L, glucose 117 mg/dL and lactate 4.8 mmol/L. Electrocardiogram showed a corrected QT interval of 468

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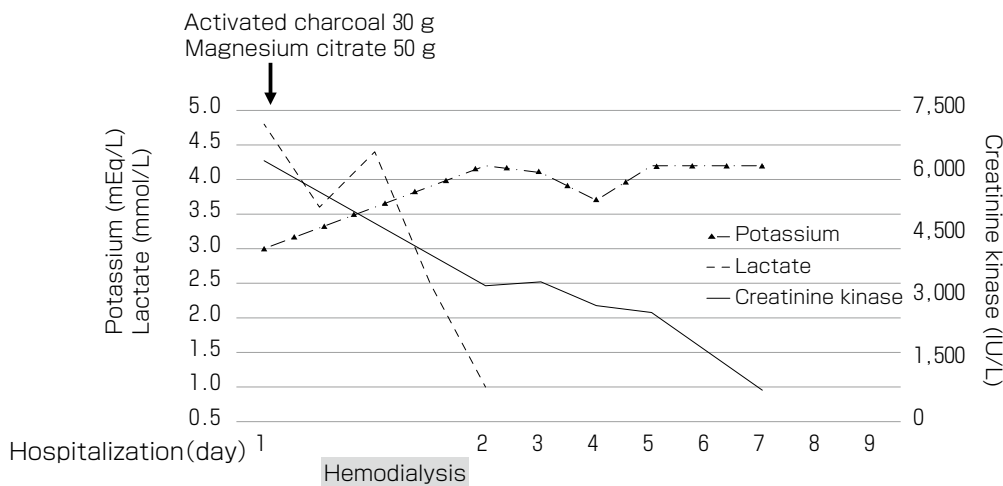


Fig. 1 Patient's clinical course

ms. Based on the above results, we diagnosed the patient with acute caffeine poisoning. He was administered activated charcoal 30 g and magnesium citrate 50 g. However, 3 hours after administration, the patient's GCS score declined to E1V1M1. For that reason, he underwent 4 hours of hemodialysis, after which the patient's state of consciousness dramatically improved (GCS : E4V5M6). Thereafter, his clinical course was uneventful, and the patient was discharged on Day 9 without complications. An outline of his clinical course is shown in **Fig.1**.

Discussion

Caffeine has a molecular weight of 212. The toxic dose is 1-3 g and the lethal dose is 5-50 g. Caffeine is rapidly and completely absorbed orally, with a time to peak blood level of 20-60 minutes. Its distribution volume is 0.5 L/kg, its protein binding rate is 35 %, and its elimination half-life varies between 2 and 10 hours. However, in patients with liver disease, caffeine's half-life increases by up to 50-160 hours⁴⁾. We administered activated charcoal because the patient had mild liver damage and thought that the half-life of caffeine was prolonged. When caffeine is consumed beyond a certain amount, metabolic enzyme activity is saturated⁵⁾. This may have affected the severity. Caffeine is metabolized by the cytochrome

P450 enzyme system (CYP1A2) ; therefore, genetic polymorphisms in CYP1A2 can cause individual differences in the rate of caffeine metabolism⁶⁾. Although we could not analyze CYP1A2 polymorphism in our patient, we cannot deny the possibility that he was a poor CYP1A2 metabolizer.

Several reports have been published on the usefulness of hemodialysis in caffeine poisoning^{7) 8)}, including the present study. More importantly, acute coffee-induced caffeine poisoning is extremely rare and only few case reports exist⁹⁾. The case serves as a reminder that caffeine poisoning can be caused by excessive intake of coffee.

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要旨

これまでに、カフェインの錠剤やエナジードリンクの過剰摂取による急性カフェイン中毒は報告されているが、コーヒーの過剰摂取による急性カフェイン中毒はほとんど報告されていない。当院にて、コーヒーの過剰摂取による急性カフェイン中毒に対して血液透析を実施した結果、有効であった1例を経験したため報告する。症例は25歳男性で、大量のコーヒーを摂取した後(推定カフェイン摂取量3g)、落ち着きがなく動悸が続いたため、当院に救急

搬送となった。入院時、頻脈・頻呼吸を認めており、採血結果ではクレアチニンキナーゼと乳酸値の上昇、および低K血症が認められた。心電図ではQTの延長が認められた。カフェインの排泄を促進するため、活性炭と下剤を投与したが、症状が改善せず意識障害が認められた。そのため、血液透析を4時間行ったところ、意識障害は改善し、神経症状は認められず第9病日退院となった。