Caffeine toxicity in a bodybuilder

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Abstract
Substance abuse is well documented among bodybuilders and weight lifters keen to enhance their performance. A case is described of abuse of caffeine to toxic levels by an amateur bodybuilder and is believed to be the first documented case of such recreational abuse.


Keywords: caffeine; bodybuilding; substance abuse

Case report
A 28 year old man was brought to the accident and emergency (A&E) department from the gym where he worked as a fitness instructor. He admitted ingesting a total of 7.5 g of caffeine over the previous three days in the form of nutrition tablets. Each tablet contained 200 mg of caffeine. On the day of admission 3.5 g were taken with three cups of very strong coffee. He took the tablets in the belief they would increase his stamina and performance at the gym. He had empirically increased the dose from the recommended one tablet per day to gain extra effect.

On the morning of admission he had complained of tiredness and nausea and during a strenuous circuit training class he collapsed and was witnessed to have a grand mal seizure lasting 15 minutes.

On arrival at the A&E department he was dizzy, nauseous, and feeling very anxious. He complained of headache, fast pulse, palpitations, insomnia, and aching in both thighs. Systemic inquiry revealed no other problems and there was no significant past medical history. He denied taking any other drugs, prescribed or self administered.

On examination he was well built and muscular. He was anxious and sweating but his temperature was 36.9°C. He had bitten his tongue and his lower lip. His pulse was 90 beats/min and regular, blood pressure was 140/90 mm Hg, and heart sounds were normal. His respiratory rate was 22 breaths/min. There were no other physical findings of note, in particular the neurological examination was normal.

Full blood count, urea and electrolytes, and liver function tests were normal. Random blood sugar was 4.8 mmol/litre. Creatinine kinase was 3270 IU/l. His urine was noted to be dark brown in colour and dip stick test with Multistix was strongly positive for both blood and protein (4+). Urine microscopy, however, revealed no blood cells. An electrocardiogram was normal.

After discussion with the Regional Poisons Centre, the patient was given activated charcoal and admitted under the care of the general physicians. Subsequent urea and electrolytes, liver function tests, full renal profile tests, and ECGs were all normal, but the creatinine kinase continued to rise to 7611 IU/l on day 2 (CK-MB fraction 48.3 or 0.6%). Serum toxicology showed a caffeine concentration of 12.3 mg/l, toxic levels being considered to be 15 mg/l and above. No other drugs were detected. An immunoassay on the urine confirmed the presence of myoglobin.

After three days on the ward and no further fits the patient was discharged with outpatient follow up. He remains seizure-free, has normal renal function, and has stopped taking caffeine tablets.

Discussion
AVAILABILITY
Caffeine (1,3,7-trimethylxanthine) is a naturally occurring alkaloid closely related to theophylline and theobromine. It is found in coffee, tea, cola drinks, and chocolate and at low doses in certain compound analgesics such as Doloxene and Anadin. It is also available in over the counter "wake up" pills at doses of 15-50 mg and is marketed as a performance enhancer, readily available in the form of 200 mg tablets.

PHARMACOLOGY
Caffeine is rapidly absorbed after ingestion, but absorption and peak blood levels are delayed in overdosage. The half life is up to 24 hours and active metabolites may contribute to toxicity.

Physiological changes, largely owing to the inhibition of adenosine synthesis, include smooth muscle relaxation, peripheral vasodilation, cerebral vasoconstriction, and lowering of the fibrillation threshold of ventricular myocardial cells. Of all the methylxanthines, caffeine is the most potent CNS stimulant.1

CLINICAL USES OF CAFFEINE
Caffeine is used as a respiratory stimulant in neonates, and as an adjunct to analgesia in certain compound preparations.

ABUSE OF CAFFEINE
Abuse ranges from dependence on excess tea and coffee, through recreational use by athletes as an “ergogenic aid” to street use of caffeine as an amphetamine-like drug. As a constituent of tea, coffee, and new “stimulant” drinks caffeine is probably the most widely used drug in the world, yet it is generally regarded as safe. However, it can cause serious toxicity and death. Successful suicides and fatal accidental poisonings have been reported.

An “ergogenic aid” is defined as a substance used for the enhancement of performance.2 Studies show that caffeine ingestion increases performance in endurance type sports3-4 but this has been proved not to be dose related.5
Thus our patient’s concept of taking more than the recommended dose to further improve endurance was clearly misguided.

TOXICITY
Poisoning can be a result of chronic oral overdose or acute ingestion of more than 10 mg/kg (or 750 mg in an average person). For comparison, a strong cup of coffee would contain 120 mg caffeine and a cup of tea 40 mg. Serum levels are considered to be abnormal above 5 mg/l, toxic at 15 mg/l, and potentially fatal at 25 mg/l.\(^7\)

Symptoms of toxicity include nausea, vomiting, and abdominal pain. Diarrhoea and gastrointestinal bleeding can occur later. Other symptoms are headache, insomnia, agitation, tremor, hypertonicity, tinnitus, tachyarrhythmias, delirium, seizures, and coma.

Caffeine induces arrhythmias by increasing cardiac conduction velocity which predisposes to re-entry tachycardias through the liberation of catecholamines and by decreasing the fibrillation threshold of the ventricular myocardium. The commonest finding is a sinus tachycardia.\(^1\)

Seizures are usually generalised but can be focal and occur more often in patients with neurological problems (for example, cerebrovascular insufficiency or previous seizures).

Patients may present with hyperventilation, hyperthermia, polyuria, dehydration, gastrointestinal bleeding, seizures, coma, and rhabdomyolysis. Rhabdomyolysis (raised creatinine kinase and myoglobinuria) can occur following caffeine overdose independently of seizures. It would appear to be a direct toxic effect on muscle cells by increasing intracellular movement of calcium, causing cell destruction.\(^8\)

Rhabdomyolysis can also occur secondary to prolonged seizures,\(^9\) or in association with hypoxia, hyperthermia, and heavy physical exertion.

TREATMENT
Treatment begins with advanced life support, as for any acute intoxication. Activated charcoal significantly decreases the absorption of caffeine, as seen with theophylline, and as overdosage delays gastric emptying administration is recommended even in delayed presentation.

Diazepam is the first line drug for caffeine induced seizures. High doses may be needed as there is evidence that caffeine antagonises diazepam.\(^10\) This may need to be followed by a barbiturate such as thiopentone or phenobarbitone, as phenytoin also tends not to be effective.\(^11\)

Treatment of arrhythmias must first include correction of hypoxia, hypokalaemia, and acidosis. Propranolol is the drug of choice for treating supraventricular and ventricular arrhythmias after overdose with caffeine. Reports have confirmed the effectiveness of procainamide.\(^12\)

The most sensitive method of diagnosing rhabdomyolysis is measurement of creatinine kinase levels. The condition is treated by first reversing the cause. Intravenous fluids and regular monitoring of urinary output and renal function are essential, the aim being to prevent the onset of acute renal failure. In severe cases the use of sodium bicarbonate to alkalise the urine has been advocated. Myoglobin in an acidic urine is more toxic to the kidneys. If despite these measures the urine output is still inadequate, mannitol can be given. The next step will be haemodialysis.\(^13\)

CONCLUSION
Bodybuilders have been known to use various drugs to enhance their performance, often with little regard to safety in spite of well publicised side effects. We believe this to be the first documented case of abuse of caffeine as a recreational drug for the purposes of muscle and power enhancement. It highlights the clinical features of a potentially fatal drug.

Caffeine toxicity should be considered in any otherwise fit young person presenting with headache, anxiety, seizures, or rhabdomyolysis, including people who are attempting to improve their physical performance.

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