

A case of acute suicidality following excessive caffeine intake.

Aleksandra Szpak and Danny Allen

Oxford Health NHS Foundation Trust, High Wycombe, UK

Abstract

Energy and stimulant drinks which contain caffeine provide enlivening effects, reduce fatigue and improve concentration and endurance. However, consumers may also experience physical and psychological side effects as a result of excessive consumption of caffeine within these drinks. Caffeine is known to exacerbate or induce some psychiatric conditions like anxiety, panic attacks, psychosis and mania. Here we discuss a case of acute suicidality following sleep deprivation after the excessive consumption of a popular energy drink. We are concerned that such widely available stimulant drinks should contain health warnings or advice regarding moderating consumption.

Introduction

Energy drinks contain various ingredients to which stimulant properties are attributed. The most obvious ingredient is caffeine. The caffeine content of energy drinks varies from 30 to 500 mg per can/ bottle (250-500ml). (As a comparison- an average cup of brewed coffee contains approximately 100 mg of caffeine, instant coffee - 75 mg and a cup of tea - 50 mg.) It is perceived that caffeine is largely responsible for the spectrum of physiological effects that result from the consumption of these drinks.

The other ingredients of stimulant drinks - taurine and glucuronolactone - have been investigated to determine their side effects. Both occur as natural ingredients in food and are normal human metabolites. However, they are used at much higher levels in energy drinks. EFSA (2003) confirmed a 'No Observed Adverse Effect Level' (NOAEL) of 1,000mg / kg of bodyweight per day for both substances. (The NOAEL was defined by the World Health Organisation in 1994 as being the greatest

concentration or amount of a substance, found by observation or experiment, which causes no detectable adverse effect in the target group concerned).

Thus, the adverse effects observed after acute consumption of energy drinks can mainly be attributed to their high caffeine content.

Dose-dependent effects of caffeine

Neuropharmacological effects of caffeine are mediated through the antagonistic effects on adenosine A1 and A2A receptors, whose suppression increases the activity of dopaminergic neurotransmission and regulates the release of glutamate and acetylcholine (Fredholm et al., 1999; Ferré, 2008).

Caffeine is one of most widely consumed legal stimulants. In moderate amounts it increases alertness and improves endurance and concentration. (moderate amounts have been described as less than 300 mg in some studies, and as much as 500 - 750 mg in others- dependent users; pregnant women are advised not to exceed 300 mg /day).

Higher doses of caffeine take proportionally longer to eliminate from the body. A study showed that clearance was significantly reduced and half-life prolonged at a dose of 500 mg compared to a dose of 250 mg. The lower dose of caffeine produced more pleasant subjective effects (elation, peacefulness, pleasantness), whereas the higher dose produced unpleasant effects (tension, nervousness, anxiety, excitement, irritability, nausea, palpitations, restlessness) (Kaplan et al., 1997)

The authors conclude that most people probably self-regulate the consumption of caffeine at low to intermediate doses so as to experience performance-enhancing stimulant effects and avoid the unfavorable somatic effects and performance disruption that often accompany high doses. People who are not regular caffeine users experience more noticeable stimulant effects, especially with higher doses; however, prolonged daily use can lead to increased tolerance.

Excessive consumption (regarded as 750 mg/ day and above) and acute ingestion have been linked with adverse effects of overstimulation i.e. nervousness, arousal, restlessness, tachycardia, insomnia, nausea, diuresis. Intoxication can lead to panic attacks or, in rare cases, psychoses or mania. Serious physical side effects may progress to arrhythmias.

A condition named caffeinism is described when daily regular intake averages 1500-2000 mg. This condition comprises the cluster of symptoms described above as well as psychomotor agitation and a rambling flow of thought and speech (Greden 1974; Gilliland & Andress 1981). The lethal acute dose of caffeine is approximately 100 mg/ kg body weight.

Caffeine and mental health

Prospective and retrospective studies with large sample sizes report that regular moderate consumption is a preventive factor from developing depressive mood disorders and is linked to lower suicide rates (Kawachi et al., 1996; Tanskanen et al., 2000). However, negative psychological effects of caffeine have been reported in people already diagnosed with psychiatric illnesses.

In one study, patients diagnosed with bipolar affective disorder and with a habitual and relatively moderate consumption of caffeine, exhibited increased rates of suicidal acts and behaviour (Baethge et al., 2009). Furthermore, a high intake of caffeine has been reported to directly induce psychotic and manic symptoms (Hedges et al., 2009) and exacerbate anxiety. Caffeine stimulant tablets in large quantities have been used in planned suicide attempts.

Case Report

Mr R, a 28 year old healthy professional boxer, described himself as physically fit and without medical problems. His personal history was unremarkable with no previous contact with psychiatric services and, no episodes of depression or previous suicidal

thoughts or plans. A brother committed suicide by hanging after the index event following a previous suicide attempt 8 years before and another brother died due to a drug and alcohol overdose (it was unclear whether this was intentional or not). His father became an alcoholic following the death of Mr R's mother when Mr R was 8 years old.

He regarded himself as positive, outgoing and enjoying life. He denied any illicit drug use or smoking and reported that he drinks alcohol only occasionally. He denied any prior excessive use of this energy drink, coffee or highly caffeinated energy drinks. He usually drinks relatively small amounts of a different energy drink with a high sugar content and comparatively low caffeine content (0.012%).

Three days prior to the index incident, knowing that he was scheduled to work as a security guard for the next two nights he drank a total of 14 (250ml) cans of this energy drink throughout the day and evening in order to stay awake and alert. He did not sleep for at least 72 hours and his appetite was poor. He did not feel tired during the day and did not sleep. He denied physical or mental symptoms, although the accuracy of the memory of his emotional state during this period is uncertain.

Before his second shift he had an argument with his partner who reported that he appeared unsettled thereafter; they argued again in the morning. He went to bed at about 6am, was awoken by his children at 8am and returned to bed shortly after. About 20 minutes later he arose, went to the garden shed, locked the door, tied a rope around his neck and jumped off a stool.

When found, he was semi-conscious, in respiratory distress and when the ambulance arrived he was unconscious and had stopped breathing. He was resuscitated, taken to ICU and transferred to a general ward where he was referred to psychiatric services. He had no recollection of what may have triggered the suicide attempt even when re-interviewed a couple of days later. He reported that he did not have any suicidal thoughts and maintained that nothing similar had happened before.

There was no prior indication of suicidal thoughts or planning. He did not leave a note and did not express any suicidal ideation before the incident. His father had been undergoing major surgery just prior to the suicide attempt and he felt relieved after hearing it had been successful.

During inpatient admission neurological examination, together with a CT scan, was normal. He was assessed by a consultant psychiatrist and he did not have any evidence of depression, mania, anxiety or psychotic illness and denied any suicidal ideation. His MMSE was recorded as 30/30. However, he appeared low in mood and upset by his actions. He reported complete memory loss surrounding the incident; his last memory was of going to bed at 6am.

Although without any ongoing suicidal ideation, both he and his partner were worried about the possibility of recurrence and were eager get some insight into what may have caused the incident. Approximately a month after the incident he repeated his concerns about having another impulsive suicide attempt. He reported sleeping poorly but had no suicidal plans or other abnormalities.

He was admitted to our day hospital for observation where he did not exhibit any signs of depressive illness; his mood was bright and his sleep improved greatly. He reported being very much relieved at having discussed his worries with mental health professionals, and with the possible explanation that the high caffeine intake contributed to the unpremeditated and impulsive suicide attempt. He completely ceased using energy drinks.

Discussion

Although there are previous reports of acute caffeine intoxication being linked with fatalities due to physical factors e.g. arrhythmias or cardiac arrest and there are associations with the acute induction of anxiety, panic attacks, psychotic and manic symptoms, this is the first report of a potential fatality from an unpremeditated suicide attempt that is linked to excessive caffeine intake.

DSM-IV-TR defines caffeine intoxication as the consumption of 250mg of caffeine in a short period of time, but most studies refer to an intake of 500mg and above. There are, no doubt, large individual differences in the metabolism of caffeine. Our patient, a mildly dependent user (120 mg / d approx.), drunk 560 mg of caffeine daily for two days (7 cans of this energy drink each day). It therefore appears likely that he experienced caffeine intoxication and that this led to prolonged sleep deprivation. Sleep deprivation is often observed to precede the onset of symptoms of mental illnesses such as depression, psychosis or mania. It is therefore possible that caffeine-induced sleep deprivation led to an altered state of mind (possibly through dopaminergic activation) resulting in the impulsive suicide attempt.

Withdrawal effects from caffeine may offer an alternative explanation for the patient's altered mood. Even cessation of small doses (100 mg) of caffeine can produce mild withdrawal symptoms. Withdrawal effects include anxiety, depressed mood and some physical symptoms e.g. nausea and vomiting, but the most common effects are headache and tiredness (DSM-IV-TR).

In regular users, caffeine withdrawal symptoms usually begin after 12 hours and peak between the first and second day of abstinence. Given the high dose of caffeine taken, the relatively slow clearance rate of high doses and the fact that his last drink of caffeine was 6 to 7 hours prior to his suicide attempt, it is likely that high levels of caffeine were maintained in his system over the 3 day period. Though this would militate against the withdrawal hypothesis, the levels of caffeine in his blood may have reduced by the third day, when he was able to sleep for a short while. Thus, we cannot rule out the possibility that a significant amount of caffeine in his system had been metabolised and therefore that he was subject to the effects of caffeine withdrawal just prior to his suicide attempt.

Mr R had no previously reported mental health issues including suicidal ideation, although, given that there is family history of mental illness and suicidal acts, he may have had an underlying susceptibility to depressive reactions. There is no evidence, however, that he was suffering from major depression around the time of the incident with the argument with his partner and concerns about his father being the only issues around at the time. His sudden suicide attempt was out of character and highly

unexpected and it came as a surprise to both him and his partner (thus indicating a certain loss of self-control).

It therefore seems likely that the state induced by acute caffeine intake, or withdrawal therefrom, was what led to the impulsive attempt to take his own life. His memory loss of the immediate events leading to the suicidal attempt is probably best explained by hypoxia secondary to asphyxiation.

The impulsiveness and seriousness of Mr R's behaviour, not to mention the sleep deprivation which preceded it, should be a potential concern to manufacturers of energy drinks and to persons who use such drinks in excess. We think that there is a good case that such drinks should include advice concerning safe levels of consumption.

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