Energy drinks, hypertension and stroke

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SUMMARY

A man in his 50s, normally fit and well, had an ischaemic thalamic stroke confirmed on MRI, manifesting with left-sided weakness, numbness and ataxia. Admission BP was 254/150 mm Hg. All tests for secondary hypertension were normal. After 72 hours as an inpatient, starting antihypertensives reduced the systolic BP to 170 mm Hg; however, after discharge, his BP rose again and remained persistently high despite up-titration of antihypertensives to five medications. After further questioning, the patient revealed an average daily consumption of eight cans of energy drink, each containing 160 mg caffeine, a habit which had not been specifically asked about during admission. On cessation of this consumption, his BP normalised and antihypertensives were successfully withdrawn. This article explores what we can learn from this case about whether energy drink consumption could be a risk factor for stroke and cardiovascular disease, and therefore the importance of targeted questioning in clinical practice, and greater public awareness.

BACKGROUND

Stroke is said to be the biggest cause of disability in the UK adult population, with a quarter of cases in people of working age; reduction of the risk of stroke is therefore increasingly relevant and important given the social and economic implications of long-term disability on both the NHS and wider society. There is regular publicity about health effects of alcohol and smoking, but little about the increasingly prevalent modifiable lifestyle trend of energy drink (ED) consumption. The year 2018 saw major UK supermarkets implement a voluntary ban on sales of EDs to under sixteens in a drive to tackle obesity, diabetes and tooth decay, but less explored are the possible increased risks of EDs for cardiovascular disease (CVD), including ischaemic and haemorrhagic strokes, particularly in younger demographics otherwise expected to have lower stroke risk.

CASE PRESENTATION

A man in his 50s, previously fit and well, presented with sudden left-sided numbness and unsteadiness. He was a non-smoker, did not drink alcohol and had no history of substance abuse, which were the only lifestyle factors he was asked about in terms of social history. Clinical examination revealed a blood pressure (BP) of 254/150 mm Hg. The general physical examination was otherwise unremarkable. Neurological examination revealed hypoaesthesia over the entire left side of the body and cerebellar signs affecting his left side, with positive finger–nose test and dysdiadochokinesia of his left upper and lower limbs. He scored 4 on the National Institutes of Health Stroke Scale, indicating a mild stroke. There were no cortical features of hemianopia, dysphasia or hemi-neglect, nor any cortical sensory loss. There was a mild drift of his left arm on motor examination with normal tendon reflexes. He received multidisciplinary care with physiotherapy and occupational therapy; secondary prevention was initiated with 3 weeks of dual antiplatelet therapy (aspirin 75 mg and clopidogrel 75 mg once daily) followed by clopidogrel alone to be continued indefinitely, along with atorvastatin; his BP was managed with losartan and amlodipine. His BP at discharge on the third day after admission was 170/80 mm Hg, with instructions to perform daily monitoring at home. He was advised not to drive for 1 month pending further assessment.

INVESTIGATIONS

A CT scan of the head showed no acute infarct or haemorrhage, but a subsequent CT angiogram showed features of focal spasm in the cerebral arteries suggestive of reversible cerebral vasoconstriction syndrome (RCVS). An MRI scan of the head revealed a small infarction in the right thalamus (figure 1a,b). A diagnosis of a right thalamic lacunar stroke was made. Routine blood tests, including a full blood count, erythrocyte sedimentation rate, renal and liver functions, lipids, glucose, thyroid function and HbA1C, were all normal. Bedside ECG showed normal sinus rhythm as did a 24-hour ECG tape. Carotid ultrasound scan, and further specialist blood tests, including antinuclear antibody, antineutrophil cytoplasmic antibody, antiphospholipid antibody and α-glucosidase level, were also unremarkable. There was no evidence of a patent foramen ovale or any valvular or ventricular abnormality on a contrast echocardiogram. As part of outpatient follow-up further investigations for secondary hypertension included abdominal ultrasound and MR angiography of the kidneys and abdomen, which showed no evidence of polycystic kidneys, adrenal masses or renal artery stenosis, but only a few congenital benign cysts in the liver (figure 1c). A 24-hour urinary catecholamine level, plasma aldosterone level and renin/aldosterone ratio were normal.

DIFFERENTIAL DIAGNOSIS

In the context of a lacunar stroke in the right side of the thalamus, differential diagnoses considered were an atheroembolism from a carotid or vertebrobasilar system atheroma, a cardio-embolism due to arrhythmia or heart defect, carotid or vertebral dissection and thrombophilia. Each differential diagnosis was excluded as discussed above. The normal prolonged cardiac monitoring probably ruled out an arrhythmia (although still possible with

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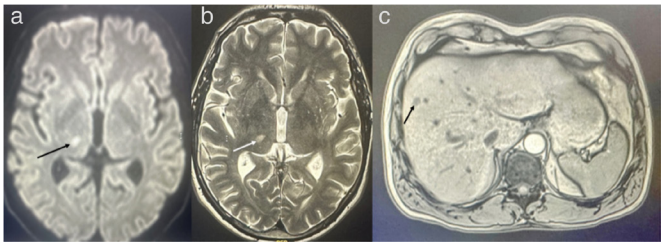

Figure 1 A subacute right thalamic infarct seen on DWI MRA (a) and T2-MRI (b) MR angiography of the abdomen showing incidental hepatic cysts (c).

more prolonged monitoring); the normal carotid ultrasound and carotid angiogram ruled out significant atheromatous disease and dissection of the carotid arteries; an activated protein C test was normal (suggesting factor V Leiden was unlikely); RCVS was therefore the most likely possibility given the severe hypertension and the areas of focal spasm on the CT angiogram.

OUTCOME AND FOLLOW-UP

Over the next 3 months the patient attended multiple follow-up clinic appointments. He appeared to have made an excellent functional recovery, although the sensory symptoms were persistent and deteriorated throughout the day. Worryingly unresolved was his uncontrolled BP, with systolic readings consistently between 190 and 230 mm Hg, on one occasion necessitating a further admission to hospital. Several antihypertensives were introduced sequentially, and after 4 weeks he was taking amlodipine, losartan, indapamide, bisoprolol and doxazosin. A more detailed lifestyle examination disclosed that the patient had an average daily consumption of eight cans of a high-potency ED, each containing 160 mg caffeine per 16 fluid ounces per serving. This equated to 1.2-1.3g of caffeine per day where NICE guidelines suggest a maximum daily intake of 400 mg.

Complete abstention from EDs was advised as the clinician wondered if the high caffeine content was a potential cause of the secondary hypertension. One week after stopping the drinks, his average BP readings showed 120–130 mm Hg systolic and 80–84 mm Hg diastolic. With the reduction of antihypertensives, it remained at healthy levels, and he was able to be completely weaned off all medications after 3 weeks. It was therefore thought to be likely that the patient's consumption of highly potent energy drinks was, at least in part, a contributive factor to his secondary hypertension and in turn his stroke.

Further follow-up at 3 and 6 months showed complete resolution of hypertension and a complete recovery from the stroke with full return to work. Eight years later, his BP was normal; he no longer consumed EDs and apart from residual left-sided sensory disturbances, he has had no recurrence of stroke.

DISCUSSION

Energy drinks (EDs) are defined as non-alcoholic drinks that contain more than 150 mg of caffeine per litre and typically a very high glucose-based sugar content and varying quantities of other chemicals (table 1). The average ED is said to contain around 80 mg of caffeine per 250 mL serving, compared with 30 mg in tea and 90 mg in coffee, but in some cases can contain up to 500 mg in a single serving. This declared amount is the 'pure caffeine', but other ED ingredients contain 'hidden caffeine'— for example, guarana is thought to contain caffeine at twice the concentration of a coffee bean. The hypothesis is that the interaction of these other ingredients, including taurine, guarana, ginseng and glucuronolactone, potentiates the effects of caffeine heightening stroke CVD risk through numerous mechanisms discussed below (box 1). Regarding sugar content, a cross-sectional study of EDs in 2017 found an average of 38.5 g of sugar per 100 mL meaning that the recommended daily sugar intake is exceeded in less than one bottle.[1]

Hypertension

Hypertension is the most important risk factor for both ischaemic and haemorrhagic strokes, frequently cited to explain the increased incidence of stroke with age. In younger individuals, once secondary causes of hypertension are excluded, it is prudent to enquire about ED consumption, as several studies have demonstrated increases in BP following their intake.

In the immediate period after ED consumption, both systolic and diastolic pressures rise measurably.[2 3] Although a single episode may not appear concerning, sharp spikes can trigger catastrophic outcomes; one study attributed a patient's new intracranial haemorrhage to the 'sympathetic surge brought about by the ED'.[4] Surveys indicate ED use beginning from as early as 13 years of age, and in those consuming EDs daily for a week, the day 7 BP rise exceeds that on day 1, suggesting a cumulative hypertensive effect.[5] Hypertension in a 16-year-old

Table 1 Common ingredients in energy drinks and their metabolic effects

Ingredient	Mechanism	Proposed benefit as part of EDs
Caffeine	Adenosine receptor antagonist causing sympathetic nervous system stimulation	Increase HR, BP, alertness
Taurine	An intracellular amino acid that modulates Ca^{2+} contributing to cell membrane stabilisation and osmoregulation	Aid skeletal muscle contraction, thought to potentially increase exercise capacity
Guarana	Plant containing high amounts of caffeine plus some theophylline and theobromine	As per caffeine
Ginseng	Ginsenoside compounds modulate various signalling pathways via effecting DNA: energy/lipid metabolism, apoptosis, neuronal signalling, anti-inflammation	Increase energy, relieve stress, stimulate memory
Glucuronolactone	Precursor to glucuronic acid which binds to metabolic waste products to aid hepatic excretion Involved in synthesis of glycosaminoglycans essential for connective tissue health	Reduce fatigue, increase mental alertness
Ginkgo biloba	Works like antioxidant	Enhance brain function and memory
B vitamins	Water-soluble vitamin coenzymes required for mitochondrial function for energy production	Convert the simple sugars in EDs into usable energy
Antioxidants	Molecules that prevent formation of/neutralise free radicals by donating electrons muscle cell damage from exercise-induced oxidative stress and inflammation	
L-carnitine	Amino acid usually produced by liver, prevents cellular damage and increases maximum oxygen consumption	Aid recovery from exercise-induced stress

ED, energy drink; HR, heart rate.

Box 1 The six main pathophysiological mechanisms thought to contribute to stroke risk as an effect of energy drinks

- Mechanisms of stroke pathophysiology associated with energy drinks
- Hypertension
- Posterior reversible encephalopathy syndrome
- Endothelial dysfunction
- Increased platelet aggregation
- Arrhythmias
- Reversible cerebral vasoconstriction syndrome

boy normalised within 2 weeks of discontinuing EDs,[6] closely mirroring our case.

Caffeine, a methylxanthine adenosine-receptor antagonist (figure 2), blocks adenosine's vasodilatory action and stimulates catecholamine, renin and aldosterone release, increasing cardiac output and peripheral vascular resistance. When combined with inotropic ingredients such as taurine and guarana, this pressor response is amplified; EDs containing caffeine plus taurine produced significantly higher 24-hour mean BP than caffeine alone.[7]

Sustained or recurrent hypertension from ED use may also contribute to posterior reversible encephalopathy syndrome (PRES), characterised by oedema in the posterior lobes on MRI. PRES has been repeatedly linked to hypertension in both its aetiology and reversibility and is associated with haemorrhagic as well as ischaemic strokes.[8]

Endothelial dysfunction

Vascular endothelial cells maintain vessel tone through secretion of vasodilatory and vasoconstrictive mediators. 'Endothelial dysfunction' describes a state of heightened vasoconstriction, inflammation, fibrosis and coagulation caused by reduced vasodilator release.

The high glucose content of many EDs may precipitate this dysfunction, as hyperglycaemia damages endothelial lipids and proteins through reactive oxygen species, generating oxidative stress. Evidence of this mechanism exists for both acute postprandial and chronically elevated glucose.[9–11] Glucuronolactone, a metabolite of glucose, contributes by similar oxidative pathways.[11]

Brachial artery, flow-mediated dilation, a validated indicator of endothelial function, has shown reductions following ED intake in several studies.[3 12] Nevertheless, some ED components such as niacin, pyridoxine and propionyl-L-carnitine possess antioxidant properties that may counteract this effect, promoting vasodilation and improving flow-mediated dilation.[11] Hence, the overall influence of EDs on endothelial function as a stroke risk factor remains inconclusive and probably depends on dose, composition and frequency of consumption.

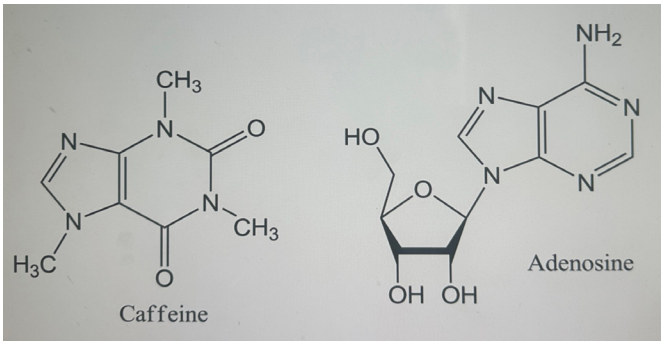

Figure 2 Molecular similarities between adenosine and caffeine.

Enhanced platelet aggregation

Ischaemic strokes comprise about 85% of all strokes, the key mechanism being increased platelet aggregation leading to thrombus or embolus formation.

Hyperglycaemia is known to enhance platelet activity by suppressing nitric oxide, which otherwise inhibits aggregation,[13] but even sugar-free EDs have been shown to increase clotting, implicating other constituents. In one trial, volunteers consuming an ED containing caffeine, taurine and glucuronolactone demonstrated a 13.7% rise in ADP-induced platelet aggregation compared with 0.0% in controls.[14] ADP activates platelets by binding to membrane receptors and initiating the glycoprotein-complex pathway—precisely the mechanism blocked by clopidogrel. Another study found a 6.1% increase in aggregation via the arachidonic acid pathway versus 1.4% in the water control group.[15] Arachidonic acid is metabolised to thromboxane-A_2, a potent platelet activator; inhibition of this pathway is the therapeutic target of aspirin.

The combined stimulatory effects of caffeine and taurine may therefore produce a transient but clinically meaningful hyperaggregable state, potentially predisposing susceptible individuals to ischaemic stroke.

Arrhythmias

Atrial fibrillation (AF) accounts for 20–25% of ischaemic strokes,[16] and numerous studies have reported associations between ED use and arrhythmias, including AF.[2 16–18] Case reports describe otherwise healthy adolescents aged 13–16 presenting with sudden-onset rapid AF following recent ED ingestion.[17 19]

Chronic AF and AF-induced cardiomyopathy were documented in a 58-year-old man consuming one ED daily for 6 months (≈ 4000 mg caffeine per week); both conditions resolved after 6 months of abstinence.[18]

Caffeine's antagonism of adenosine receptors activates cardiac and neurohormonal sympathetic pathways, raising heart rate, BP, norepinephrine release, dopamine sensitivity and intracellular calcium. These combined effects have been shown to dose-dependently increase the likelihood of both supraventricular and ventricular arrhythmias,[16 18] thereby heightening stroke risk, especially in individuals with pre-existing cardiovascular susceptibility.

Reversible cerebral vasoconstriction syndrome

RCVS, first defined in 2007, encompasses disorders characterised by multifocal, yet reversible, narrowing of cerebral arteries, typically resolving within 3 months. It presents with a thunderclap headache and possible focal neurological deficits. Radiological imaging shows alternating segments of constriction and dilation—commonly described as a 'sausage-on-a-string' pattern—which resolves on repeat angiography (figure 3).

Both ischaemic and haemorrhagic strokes occur within the spectrum of RCVS: a systematic review of 139 cases identified ischaemic stroke in 39% and lobar haemorrhage in 20%.[20] The principal precipitating factors are vasoactive substances that transiently disrupt cerebral arterial tone.[8 21]

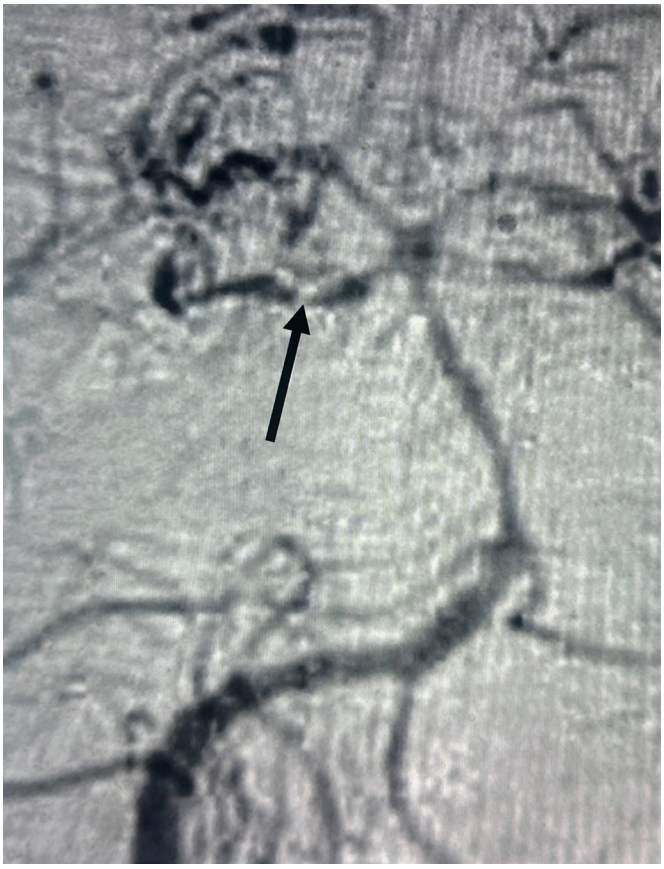

Figure 3 MR angiography showing spasm (black arrow) in the superior cerebellar vessel.

Among ED components, caffeine's potent vasoactivity through adenosine antagonism is the main driver of such dysregulation. Taurine, influencing intracellular and extracellular calcium homeostasis, has been implicated in coronary artery vasospasm and may plausibly exert similar effects in cerebral vessels.[2 21] Repetitive exposure to these agents could therefore create conditions conducive to transient vasospasm, perfusion instability and secondary stroke events.

Conclusion and recommendations

As our case and discussion illustrate, it is possible that both acute and chronic intake of EDs may increase CVD and stroke risk, and importantly, this may be reversible. While the current evidence is not conclusive, given the accumulating literature, the high morbidity and mortality associated with stroke and CVD and the well-documented adverse health effects of high-sugar drinks, we propose that increased regulation of ED sales and advertising campaigns (which are often targeted at younger ages) could be beneficial to the future cerebrovascular and cardiovascular health of our society. Additionally, healthcare professionals should consider specific questioning related to ED consumption in young patients presenting with stroke or unexplained hypertension.

Patient's perspective

"I obviously wasn't aware of the dangers drinking energy drinks were causing to myself, (I) have been left with numbness (in my) left hand side hand and fingers, foot and toes even after 8 years."

Learning points

- In patients with unexplained hypertension, clinicians should enquire about consumption of energy drinks.
- Lifestyle counselling should include a discussion about cardiovascular risk factors of energy drinks.
- Consider reversible cerebral vasoconstriction syndrome in strokes with no identifiable cause and identify what risk factors might cause this.
- Further research is needed in this field as well as education and greater public and health authorities' awareness.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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