Seizure 2003; **12:** 519–521

CASE REPORT

Caffeinated beverages and decreased seizure control☆

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Seizure control is often affected by seizure threshold lowering behaviours. In this case report, the authors address excessive caffeine ingestion from tea with increased seizure frequency. When decaffeinated beverages were substituted for the tea, seizure frequency returned to baseline. Similar findings occurred when the patient was re-challenged. The authors recommend avoidance of excessive caffeine in patients with epilepsy.

Key words: tea; caffeine; theobromine; epilepsy; seizure threshold; methylxanthines.

INTRODUCTION

Lack of seizure control can be related to a series of issues: complexity of seizure disorder, inappropriate anticonvulsant for the particular seizure disorder, and concomitant medical illnesses with fevers[1–5]. With the plethora of recently released anticonvulsants, uncontrolled seizure frequency often leads to a change in prescribed anticonvulsant. What may be overlooked in this rush to use the newer agents are the fundamentals in seizure control—compliance, therapeutic anticonvulsant blood levels, and the avoidance of threshold lowering behaviours[1].

Seizure threshold is decreased by: decreased sleep[4,6]; excessive stress[4,7]; threshold lowering medications[5,8–12]; medications with pharmacokinetic interactions with anticonvulsants[13,14] or threshold lowering substances[15,16]; and caffeine[17–20].

Literature suggests that patients with epilepsy should avoid caffeinated beverages[15,21], but the only report concerning the proconvulsive effect of tea was a study with mice[22]. In this case report, accidental increase in seizure frequency associated with excessive tea ingestion is noted that resolved when decaffeinated beverages were substituted. This finding was duplicated when the patient was re-challenged with the caffeinated beverage.

CASE

A 49-year-old white male with a 36-year history of mixed seizure disorder (grand mal seizures with intermittent auras, absence seizures, atonic seizures, and myoclonic seizures) who had been well controlled on phenytoin 400 mg once daily and primidone 500 mg once daily developed a sharp increase in myoclonic and atonic seizure frequency. Anticonvulsant compliance had been maintained with therapeutic blood levels. A pill count revealed that no anticonvulsants had been missed. There had been no change in sleep patterns or level of stress. Further, the patient had no concurrent illnesses or addition of medications. Change in anticonvulsants to newer agents was contemplated. Prior to doing such, a review of the patient's diet revealed a recent increase in caffeinated beverages. In an effort to avoid caloric beverages, the patient was drinking 4 pints of "Peach diet Snapple

☆Presented at the 55th Annual Meeting of the American Epilepsy Society, Philadelphia, PA, November 30th–December 5th, 2001.

1059–1311/$30.00

Iced Tea" daily. Further, this was typically consumed within 180 minutes. The increased seizure frequency continued over a 2-month time period until the patient's wife commented upon the potential influence of the additional caffeinated beverages. When the "Peach diet Snapple Iced Tea" was changed to the decaffeinated "Kiwi Strawberry Cocktail diet Snapple", the patient's seizure frequency returned to baseline. This simple change in diet obviated the need to change anticonvulsants to control seizure frequency.

The patient thereafter found that he could drink 1 pint of "Peach diet Snapple Iced Tea" without any effect on seizure frequency. When 4 years later he re-challenged himself briefly with 4 pints of "Peach diet Snapple Iced Tea", the blinded wife again commented on the return of myoclonic activity and queried his compliance. Again with immediate reduction to 1 pint or use of decaffeinated beverages, no further myoclonus was noted.

DISCUSSION

Toxicity and seizures associated with over-the-counter stimulants, including caffeine, have been reported[23]; however, this case is unique in that seizures associated solely with excessive tea ingestion have not been previously reported. Nonetheless, it is important to realise that numerous beverages contain caffeine and, as such, it is the cumulative caffeine from all beverages ingested that matters.

As a solitary case, the findings from this case cannot be generalised. Further limitations of this case include the lack of caffeine blood levels and the lack of quantitative myoclonic frequency. However, the fact that the myoclonus returned when the patient was re-challenged is noteworthy and supports the theory that caffeine was the aetiologic factor.

The total time associated with ingestion of caffeinated beverages may be pertinent to the proconvulsive effect of caffeine. In one rat study of caffeine augmentation of electroconvulsive seizures, the proconvulsive effect was dose dependent and lasted for at least 230 minutes[19]. Thus it is important that the patient had ingested the caffeinated beverages within 180 minutes.

Since caffeine is metabolised by cytochrome P450 1A2, saturation of this enzyme (which could occur with high consumptions of caffeine, and may have occurred in this case) or inhibition of this enzyme will result in increased toxicity from caffeine[15]. This is a critical issue for clinicians as selective serotonin reuptake inhibitors, antiarrhythmics, antipsychotics, and bronchodilators all have been reported to inhibit this enzyme[15].

Pharmacokinetic studies have shown that caffeine does not affect standard anticonvulsant blood levels (carbamazepine, valproate, phenobarbitone, and phenytoin) and that protective qualities of these anticonvulsants are progressively reduced with chronic caffeine exposure[18, 21, 24]. In this case, the patient was on phenytoin and primidone (the latter metabolises to phenobarbitone).

Although this case report has focused on caffeine (1,3,7-trimethylxanthine) in tea, it should be noted that tea also includes theobromine (3,7-dimethylxanthine) and theophylline (1,3-dimethylxanthine)[25, 26]. All three methylxanthines, which are ubiquitous in beverage and food preparations, have been shown to have proconvulsive features[27]. As such, the authors speculate that in addition to caffeine, patients with epilepsy should avoid all unnecessary dietary sources of methylxanthines. In fact, the dietary presence of methylxanthines dates to the preclassic Maya civilisation[28]. Finally, in patients wherein dietary issues (excessive methylxanthines) are felt to contribute to the lack of seizure control, levels of these three methylxanthines should be determined[29].

CONCLUSION

The authors have observed that the newer anticonvulsants are important to control epilepsy and in many instances have better side effect profiles than the older agents. Nonetheless, the use of these agents does not permit the practitioner and the patient to forget the fundamentals of epilepsy control, specifically minimisation of seizure threshold lowering behaviours including excessive caffeinated beverages. In this case, the simple elimination of excessive caffeinated beverages obviated the need to change anticonvulsants to improve seizure control.

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