

Influence of coffee drinking on epilepsy control

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Abstract: Caffeine is a global stimulant the increased intake of which leads to a lowering of the seizure threshold, which may result in reduced control of epilepsy. Two cases are reported: a male with well-controlled epilepsy, and a female with drug-resistant, moderately-controlled epilepsy. In both patients, temporary excessive coffee consumption led to increased seizure frequency, and withdrawal of coffee from the diet resulted in a return to the previous status.

Key words: epilepsy, caffeine, seizure frequency, coffee

INTRODUCTION

Appropriate pharmacologic treatment provides satisfactory seizure control in about 70% of patients with epilepsy. It is unclear why one third of epileptic patients, in spite of state-of-art medication with antiepileptic drugs (AEDs), do not achieve optimal control of seizures. Lack of epilepsy control may be related to factors lowering the seizure threshold, for instance, in the case of excessive stress, concomitant illness with fever, sleep deficit or medications interfering with AEDs [1]. Coffee and tea contain considerable quantities of caffeine, which is also contained in many soft drinks. Experimental studies performed to date in experimental models of epilepsy indicate that chronic caffeine exposure may progressively reduce the protective potential of AEDs [2]. Isolated clinical data has also provided evidence that epileptic patients should avoid caffeinated beverages [3-5].

In this study, two case reports are presented. Both patients, in spite of different treatment regimens, seizure control and complexity of seizures, demonstrated increased seizure frequency during a period of excessive coffee consumption. In both cases, withdrawal of coffee from the diet resulted in a return to the previous status of epilepsy control.

CASE REPORTS

1. A 29-year-old man with a 3-year history of treatment for partial complex seizures with temporary secondary generalization. MRI image showed a focus of hypodensity in the deep brain structures, and an EEG recording displayed paroxysmal discharges originating from the left temporal region. He received 300 mg of valproate twice daily which resulted in excellent epilepsy control – the patient became seizure-free. During one month (September 2005), the patient – during preparation of his doctoral dissertation – consumed 5-6 cups of coffee daily, which was associated with 2 episodes of seizures within 2 weeks, as reported by his father. Withdrawal of coffee without changing the existing

medication resulted in rapid improvement – the patient became seizure-free again.

2. A 47-year-old woman had been receiving anti-epileptic treatment since the age of 13, with many AEDs and with various combinations of 2-3 AEDs. Additionally, she did not tolerate administration of lamotrigine and topiramate. She was hospitalized on several occasions when clustering seizures occurred. CT, angio-CT and MRI investigations revealed mild vessel malformation in the left hemisphere, but neurosurgical procedure was decided against. From September 2004, she was treated with a combination of carbamazepine 2×400 mg, phenytoin 3×100 mg, tiagabine 5 mg-0-10 mg, phenobarbital 50 mg-0-100 mg, which limited the number of seizures to 2 per month. During November and December 2004, the frequency of seizures increased to several a week. According to the report from the patient's husband, the worsened epilepsy control was associated with an increased intake of coffee – she was consuming at least 4 cups of coffee every day. After strict coffee limitation, and without changing the doses of AEDs, she returned to the previous status of epilepsy control.

DISCUSSION

Caffeine (1,3,7-trimethylxanthine), and other methylxanthines – theophylline (1,3-dimethylxanthine) or theobromine (3,7-dimethylxanthine), which are present in coffee, tea and numerous soft drinks and beverages, have been shown to possess proconvulsive and convulsive activity [7, 8]. There are several presumable mechanisms by which caffeine acts on the central nervous system. They include antagonism of adenosine receptors, inhibition of phosphodiesterases and mobilization of calcium ions from intracellular stores [8], which may negatively modify the protective effects of AEDs. Animal data have provided evidence of caffeine-induced reduction of the protective activity of AEDs against experimental seizures [2, 9, 10]. There are clinical reports on this issue. Specifically, Bonilha and Li [4] have observed hazardous effects of heavy coffee drinking on epilepsy control. Kaufmann and Sachdeo [3] have reported that a large intake of caffeinated beverages dramatically decreased seizure control.

The presented cases of 2 quite different epileptic patients, in terms of duration of illness or seizure patterns, point to

the fact that the result of excessive coffee intake was identical – deterioration of epilepsy control. Apart from the evident pharmacodynamic nature of the observed interaction between caffeine and antiepileptic drugs – pharmacokinetic events are also likely to be contributing factors, in the case of some AEDs at least. For instance, caffeine has been shown to cause significant reduction in both the bioavailability of carbamazepine and in its plasma concentration. Such effects have not been found for valproate [11]. On the other hand, carbamazepine and valproate do not seem to affect the caffeine metabolism, although phenytoin has been reported to increase the clearance of methylxanthine [12].

CONCLUSION

The experimental data point to the hazardous effects of caffeine on the protective potential of AEDs have been confirmed in epileptic patients. Deterioration of epilepsy control by caffeine seems pharmacodynamic in nature, although pharmacokinetic events may also contribute in the case of some AEDs (carbamazepine). Epileptic patients should be advised not to consume caffeine-rich beverages.

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