

When chewing gum is more than just a bad habit

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In November, 2008 a 13-year-old boy (weight 45 kg, height 160 cm) was taken by his parents to the emergency department. His parents reported that after school the boy had been agitated and aggressive, in contrast with his normal mood. He denied feeling stressed or having used drugs or stimulants. He complained of abdominal discomfort, increased diuresis, dysuria, and prickling sensations in his legs. On examination, he was restless, with sinus tachycardia (147 beats per min [bpm] on electrocardiogram [ECG]), tachypnoea (25 breaths per min), and raised blood pressure (145/90 mm Hg). Chest radiography and laboratory blood test results were normal. Toxicological screen was negative for cocaine, heroin, and amphetamine. Psychiatric consultation was unremarkable. Our patient's condition improved overnight without any treatment and he was discharged the following morning. At discharge he was noted to have sinus bradycardia (40 bpm).

Later that day, his mother returned to the emergency unit with two empty packets of stimulant ("energy") chewing gum that she had found in her son's bag; he had admitted consuming the confectionery at school. The chewing gum contained 160 mg caffeine per packet (0.57% caffeine per gum pellet) and non-specified amounts of guaraná,¹ sweeteners, gum acacia, citric acid, malic acid, glycerol, carruba wax, and food colouring. According to the mother, her son did not usually ingest caffeine-rich beverages or food. A few days later, the teenager attended an outpatient appointment. Although not complaining of any symptoms, he appeared sleepy and sluggish. His mother stated that he had lost interest in any activity and had been absent from school for

3 days. ECG showed sinus bradycardia (45 bpm) and low left-ventricular ejection fraction (55%). His condition improved over the next 5 days and when last seen in December, 2008, the teenager was well and enjoying normal activities.

The transient constellation of signs and symptoms in our patient was most likely secondary to acute intoxication of stimulant chewing gums.² Serum caffeine was not measured, but our patient met the criteria for diagnosis of caffeine intoxication, defined as the consumption of at least 250 mg caffeine followed by the onset of at least five specific features.³ Our patient admitted consuming two packets of the chewing gum containing 320 mg caffeine in 4 h—this is equivalent to about ten cups of tea in a 70 kg adult. Our patient weighed 45 kg, and presumably had high caffeine sensitivity in view of his low habitual caffeine intake, so 320 mg was a substantial amount of caffeine. Caffeine is a methylxanthine, which is rapidly and completely absorbed from chewing gums.⁴ It induces several effects including stimulation of endocrine and exocrine tissues, the central nervous system, cardiac muscle, and skeletal muscle, relaxation of smooth muscle, and diuresis.⁵ These effects result from intracellular calcium redistribution, accumulation of cyclic nucleotides secondary to inhibition of phosphodiesterases, and adenosine receptor blockade.⁵ It was unlikely that guaraná, or other ingredients of the chewing gum played a part in this case, because their doses were low or they would induce different signs and symptoms from those found in our patient.² The use of stimulant chewing gum should be considered in cases of caffeine intoxication. The risk of intoxication is high in children and teenagers in view of general caffeine-naivety, and the unrestricted sale of these substances.

Contributors

FN, CC, GM, LV, LA, AS, MR participated in management of the patient and writing the report. MT, PG, and RC were also involved in writing the report.

References

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Figure: "Energy" chewing gum