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ABBREVIATION
ED—energy drink

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abstract

We report here the case of a 17-year-old boy who suffered acute renal failure after consuming 3 L of energy drink (ED) in combination with 1 L of vodka amounting to 4600 mg of taurine and 780 mg of caffeine mixed with 380 g of alcohol. The consumption of this mixture is extremely popular in adolescents, because the joint effects of caffeine and taurine reduce the effect of alcohol. Although there have been case reports of deaths linked to the consumption of EDs with and without alcohol, awareness of the possible dangers is still low. The fact that athletes and major sports events are sponsored by ED manufacturers implies that they may even be healthy and performance-enhancing. *Pediatrics* 2011;128:e000

The consumption of energy drinks (EDs) is popular in the adolescent community. In the United States alone the use of these drinks constituted a $5.4 billion market in 2006.[1] In a survey conducted in Milwaukee, Wisconsin, 6% of the respondents to a telephone survey admitted to using alcohol plus EDs; respondents were more likely to be white and younger.[2] These drinks, which contain caffeine, taurine, inosite, and glucoronolactone in varying concentrations, are commonly mixed with strong alcoholic beverages such as vodka ("vodka energy") or whiskey ("whiskey energy").[3,4] Although many countries have imposed regulations on the distribution of those drinks, and several "premixed" alcoholic EDs are prohibited for sale in the United States,[1] scientific data on their influence are scarce to nonexistent.

Most of the research has focused on the effect of EDs on the central nervous system.[3–11] When combining EDs with alcohol, the caffeine functions as a stimulant and the depressant effects of alcohol are reduced.[6,9,11] Another observation with regards to EDs and the central nervous system is the occurrence of seizures after ED consumption.[12,13]

Apart from their effects on the central nervous system, there has been a report linking the consumption of EDs mixed with alcohol with the death of 3 patients.[12] All 3 patients died within 24 hours after consumption: 1 died of hemorrhagic pulmonary edema; 1 died in ventricular fibrillation; and 1 showed extensive cerebral and comprehensive pulmonary edema.

The effects of EDs on the kidneys were also presented in the case of an active football referee who had consumed 750 mL of ED before running a 3000-m race.[12] The examination revealed rhabdomyolysis and acute kidney failure with tubular necrosis, probably caused by taurine consumption.[12] In patients with chronic renal insufficiency, taurine levels are low[14] and thought to be of relevance for the muscle weakness, cardiac insufficiency, and neurologic symptoms in this group of patients.[15,16] A study that investigated the effects of the administration of taurine in patients with chronic hemodialysis had to be abandoned before the planned expiry date because the patients suffered from severe dizziness caused by the taurine.[17]

TABLE 1 Blood and Urine Chemistry Values at Admission, Before Hemodialysis, and at Discharge

	Value at Admission	Before Hemodialysis	Value at Discharge
Blood alcohol level, ‰	0.4	—	—
C-reactive protein, SI	0.1	—	—
Serum urea, mg/dL	25	64	33
Serum creatinine, mg/dL	1.0	6.9	0.7
α_1-Microglobulin, mg/dL	1.0	1.4	Below detectable limits
Protein excretion in 24 h, mg	—	448	98
Creatinine clearance in 24 h, mL/min	—	7.4	113 (25 d after hemodialysis)
Sodium concentration in urine, mmol/L	—	33	—
Urine osmolality, mosmol/kg	—	263	—
Measured serum osmolality, mosmol/kg	—	303	—
Fractional sodium excretion, %	—	3.87	—
Fractional urea excretion, %	—	60	—
Calculated serum osmolality, mosmol/kg	—	290.39	288.21

CASE REPORT

A 17-year-old male patient was admitted by ambulance because of vomiting and dizziness after having run two 100-m races at school. On arrival in the hospital he was hyperventilating and slightly tachycardic (heart rate: 110 beats per minute). After breathing back into a plastic bag, the tachycardia and hyperventilation receded. He presented with bloodshot eyes and was exceptionally tired but awakable at any point in time. His weight was 65.5 kg, his blood pressure was 123/68 mm Hg, and his rectal temperature was 36.8°C.

A clinical examination, electrocardiography, electroencephalography, and chest radiography revealed no pathologic findings. Results of differential blood examination and measurement of total blood count, clotting parameters, electrolytes, liver enzymes, and the parameters for acid-base metabolism remained within normal ranges over the whole course of the treatment. There was no sign of rhabdomyolysis; his creatine kinase, troponin, and lactate dehydrogenase levels were normal. His blood alcohol and C-reactive protein levels are listed in Table 1.

The course of the creatinine level was 1.0 mg/dL (Fig 1). On the second day the creatinine levels went up to 3.5 mg/dL (Fig 1). During that first day the patient developed capsular pain over both kidneys. An ultrasound examination revealed no pathologic findings and normal kidney size on both sides. Over the next 24 hours his creatinine level shot up to 6.9 mg/dL (Fig 1), and hemodialysis became necessary. The course of his urea levels is listed in Table 1. The daily urine samples always tested negative for leukocytes, nitrite, ketone, and bilirubin and revealed normal levels for glucose and urobilinogen. Electrophoresis of the urine revealed elevated parameters for albumin (0.311 SI), but those levels returned to normal before discharge. Although α_1-microglobulin, a parameter that suggests tubular injury, was always below pathologic values of 2 mg/dL, the level showed a distinctive rise above detectable limits (Table 1) before decreasing on the fourth day. The urine sediment revealed acute tubular necrosis.

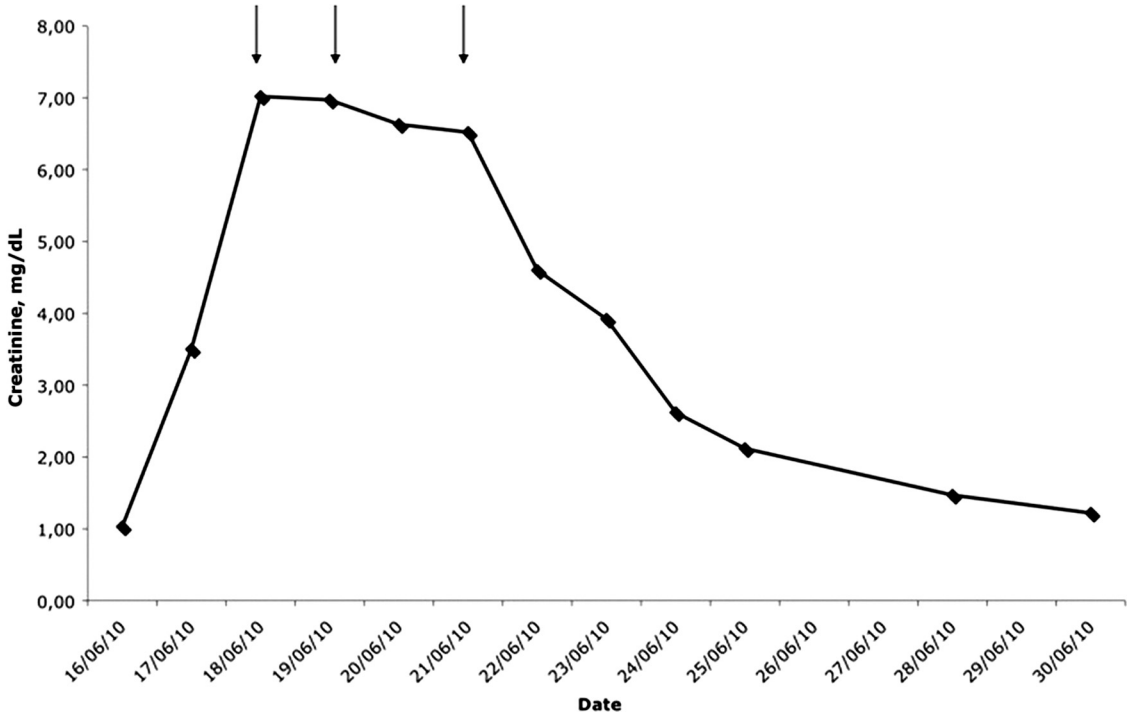

FIGURE 1
Creatinine levels over time. The arrows indicate the administration of hemodialysis.

The following antibodies were investigated to rule out an autoimmune disease as a cause of the rapid renal failure: antinuclear antibodies; p- and c-anti–neutrophil cytoplasm antibody; anti–glomerular basement membrane; anti-streptolysin antibodies; and serum complements C3 and C4. To rule out infection as a cause we performed hantavirus serology with polymerase chain reaction for hantavirus DNA, a screening for HIV antibodies, and hepatitis serology (A, B, and C). Results for all these parameters were negative.

The sodium concentration in the urine, the lower urine osmolality compared with the measured serum osmolality, the fractional sodium excretion (>1), and the fractional urea excretion (<35%) allowed us to rule out a prerenal cause for the acute renal failure. The calculated serum osmolality ($[Na^+] + 1.38\ [K^+] + 1.03$ [urea] + 1.08 [glucose] + 7.45) was at no point higher than the measured serum osmolality, which indicated that the kidney failure was not a result of osmotically active substances.

As the patient realized the graveness of the situation, he confessed to having drunk a combination of EDs with vodka. Noteworthy, however, was the dosage. He drank 3 L of ED together with 1 L of vodka, which amounted to a taurine consumption of 4600 mg and a caffeine consumption of 780 mg (Fig 2) over the course of an evening. Because his creatinine levels dropped back to normal levels on their own and no residuals could be detected, no biopsy of the kidney was necessary. The patient

FIGURE 2
Depiction of 2 of the bottles of ED consumed by the adolescent.

was discharged after 10 days in the hospital. Transient hypertension was treated with enalapril for 3 days. Outpatient follow-up was without pathologic finding 10 days and 3 weeks after discharge.

DISCUSSION

The worrying aspect of this case was the fact that the adolescent had bought the EDs not in cans of 200 mL, which is the normal distribution size in most European countries, but in bottles of 1 L, which amounted to taurine levels of 4000 mg and caffeine levels of 120 mg/L. Because the size of the bottles suggests that there is no harm when consuming large amounts of it, adolescents might do so. The patient later stated that he was thinking of doing something good for himself trying to improve the outcome of the races planned for the next day. There was no warning label on the bottles.

Several studies have pointed to the potential dangers of the consumption of EDs, especially in combination with alcohol with respect to the effects on the central nervous system.[3,5–9,11,18] This combination leads to impaired judgment regarding motor skills and reaction-time loss caused by the alcohol and leads to drinking high volumes of alcohol per drinking session by keeping the person awake longer,[1] but it also might cause seizures in patients with known epilepsy[12,13] and in patients without a previous history of epilepsy.[13] Furthermore, the mixing of EDs with alcohol might confer a risk of alcohol dependence.[19] Another dangerous implication of EDs is a higher heart rate as a consequence of the stimulating effects of caffeine and taurine, possibly causing dangerous arrhythmias and sudden cardiac deaths.[12] This pathology is even more dangerous considering that the manufacturers of EDs sponsor major sport events and famous athletes, which indicates that the consumption of EDs is both beneficial for health and performance-enhancing.

To the best of our knowledge, the fact that EDs may cause renal failure in healthy adolescents has never been described before. However, there has been 1 case of acute kidney failure with tubular necrosis in a 31-year-old football referee that resulted from rhabdomyolysis,[12] and the only agent that could have caused the kidney failure was found to be an ED (the patient had consumed 750 mL). When taurine was administered to patients with chronic hemodialysis, it led to dizziness that ceased after the administration was discontinued, so the study had to be abandoned.[17]

CONCLUSIONS

This brief report is meant to elevate awareness of possible dangers of EDs and warn about the possible effects, especially for adolescents, in whom the mixture of EDs with strong alcoholic beverages is extremely popular and drunk in large amounts. Taurine is probably important in the maintenance of cell volume.[20] Its transport in vivo seems to be regulated precisely by the kidneys and is mimicked in vitro in a variety of renal systems, including uptake into renal slices, renal cells in culture, isolated renal tubules, and isolated brush border membrane vesicles regulated at both the level of messenger RNA transcription and protein synthesis.[20] The amount of consumed taurine, therefore, is reflected in the transport through the kidneys. The fact that liter bottles can be bought in supermarkets reduces the inhibition threshold. Our patient would have had to drink 15 cans of ED before achieving the same amount of taurine as that in the 3 liter-sized bottles. More information and awareness of the dangers of EDs are needed for protecting youth from the possible dangers. The sale sizes should not exceed 200 mL, and a warning, similar to those in advertisements for drugs and cigarettes, should be added to ED advertisements.

REFERENCES

1. Arria AM, O'Brien MC. The "high" risk of energy drinks. *JAMA*. 2011;305(6):600–601
2. Berger LK, Fendrich M, Chen HY, Arria AM, Cisler RA. Sociodemographic correlates of energy drink consumption with and without alcohol: results of a community survey. *Addict Behav*. 2011;36(5):516–519
3. Ferreira SE, de Mello MT, Formigoni ML. Can energy drinks affect the effects of alcoholic beverages? A study with users [in Portuguese]. *Rev Assoc Med Bras*. 2004;50(1):48–51
4. Oteri A, Salvo F, Caputi AP, Calapai G. Intake of energy drinks in association with alcoholic beverages in a cohort of students of the School of Medicine of the University of Messina. *Alcohol Clin Exp Res*. 2007;31(10):1677–1680
5. Aragon CM, Trudeau LE, Amit Z. Effect of taurine on ethanol-induced changes in open-field locomotor activity. *Psychopharmacology*. 1992;107(2–3):337–340
6. Azcona O, Barbanoj MJ, Torrent J, Jane F. Evaluation of the central effects of alcohol and caffeine interaction. *Br J Clin Pharmacol*. 1995;40(4):393–400
7. Ferreira SE, de Mello MT, Pompeia S, de Souza-Formigoni ML. Effects of energy drink ingestion on alcohol intoxication. *Alcohol Clin Exp Res*. 2006;30(4):598–605
8. Fillmore MT, Roach EL, Rice JT. Does caffeine counteract alcohol-induced impairment? The ironic effects of expectancy. *J Stud Alcohol*. 2002;63(6):745–754
9. Fudin R, Nicastro R. Can caffeine antagonize alcohol-induced performance decrements in humans? *Percept Mot Skills*. 1988;67(2):375–391
10. Marczinski CA, Fillmore MT. Clubgoers and their trendy cocktails: implications of mixing caffeine into alcohol on information processing and subjective reports of intoxication. *Exp Clin Psychopharmacol*. 2006;14(4):450–458
11. Oborne DJ, Rogers Y. Interactions of alcohol and caffeine on human reaction time. *Aviat Space Environ Med*. 1983;54(6):528–534
12. Lehtihet M, Sundh UB, Andersson DE. Energy drinks: dangerous or not? Cases with severe symptoms with possible connection to energy drinks: more case reports wanted

[in Swedish]. *Lakartidningen*. 2006;103(38):2738–2741

13. Iyadurai SJ, Chung SS. New-onset seizures in adults: possible association with consumption of popular energy drinks. *Epilepsy Behav*. 2007;10(3):504–508
14. Bergström J, Alvestrand A, Furst P, Lindholm B. Sulphur amino acids in plasma and muscle in patients with chronic renal failure: evidence for taurine depletion. *J Intern Med*. 1989;226(3):189–194
15. Azuma J, Hasegawa H, Sawamura A, et al. Taurine for treatment of congestive heart failure. *Int J Cardiol*. 1982;2(2):303–304
16. Huxtable RJ. Physiological actions of taurine. *Physiol Rev*. 1992;72(1):101–163
17. Suliman ME, Barany P, Filho JC, Lindholm B, Bergstrom J. Accumulation of taurine in patients with renal failure. *Nephrol Dial Transplant*. 2002;17(3):528–529
18. Kuribara H. Enhancement of the behavioral toxicity induced by combined administration of ethanol with methylxanthines: evaluation by discrete avoidance in mice. *J Toxicol Sci*. 1993;18(2):95–101
19. Arria AM, Caldeira KM, Kasperski SJ, Vincent KB, Griffiths RR, O'Grady KE. Energy drink consumption and increased risk for alcohol dependence. *Alcohol Clin Exp Res*. 2011;35(2):365–375
20. Chesney RW, Han X, Patters AB. Taurine and the renal system. *J Biomed Sci*. 2010;17(suppl 1):S4