Cranial MR Imaging Findings of Potassium Chlorate Intoxication

Hakan Mutlu, Emir Silit, Zekai Pekkafali, C. Cinar Basekim, Esref Kizilkaya, Hakan Ay and A. Fevzi Karsli

AJNR Am J Neuroradiol 2003, 24 (7) 1396-1398
http://www.ajnr.org/content/24/7/1396

AJNR Am J Neuroradiol 24:1396–1398, August 2003

Case Report

Cranial MR Imaging Findings of Potassium Chlorate Intoxication

Hakan Mutlu, Emir Silit, Zekai Pekkafali, C. Cinar Basekim, Esref Kizilkaya, Hakan Ay, and A. Fevzi Karsli

Summary: We present the case of a patient who attempted suicide by ingesting matchstick heads (55% potassium chlorate). The patient presented to the emergency room with loss of consciousness, and MR imaging revealed symmetric hyperintense signal within the deep gray matter and medial temporal lobes. The patient improved after undergoing conventional treatment and hyperbaric oxygen.

Potassium chlorate is a highly reactive oxidating agent that is used in the production of matchstick heads, fireworks, explosives, mouthwashes, and weed killers (1). Matchstick heads consist of 55% potassium chlorate. Potassium chlorate intoxication is very rare. We present the cranial MR imaging findings of a patient who tried to commit suicide by ingesting matchstick heads.

Case Report

A 21-year-old man was referred to our hospital with a decreased level of consciousness 24 hours after swallowing three boxes of matches (40 matchsticks within a box) in an apparent suicide attempt. At admission, his blood pressure was 108/70 mm Hg; heart rate, 100 beats per minute; temperature, 36.6°C; and respiration rate, 34 breaths per minute. Neurologic examination revealed obtundation, bilateral abducens palsy, and hypoactive deep tendon reflexes. Analysis of blood chemistry showed potassium at 7.45 mmol/L (normal range, 3.50–5.30 mmol/L), urea at 21 mmol/L (normal range, 5.3–15.7 mmol/L), aspartate aminotransferase at 58 U/L (normal range, 5–40 U/L), creatine kinase at 209 U/L (normal range, 0–190 U/L), and bicarbonate at 4 mmol/L (normal range, 24–28 mmol/L). ECG reflected signs of hyperkalemia, with T-wave peaks of increased amplitude. The patient then underwent toxicology screening for methanol, ethanol, barbiturates, and benzodiazepines. The patient underwent gastric lavage and hemodialysis.

Coronal and axial T2-weighted (TR/TE, 4500/99) turbo spin-echo images, sagittal and axial short TR/short TE (TR/TE, 690/14) spin-echo images, and axial fluid-attenuated inversion recovery (FLAIR) (TR/TE/TI, 9000/110/2500) images were obtained. T2-weighted and FLAIR images revealed symmetric abnormal signal intensity within the deep gray matter (Fig 1A and B) and medial temporal lobes (Fig 1C and D). MR findings were consistent with potassium chlorate intoxication. The patient underwent hyperbaric oxygen treatment at 2.2 absolute atmosphere pressures in three sessions (90 minutes/session). The patient responded well to hyperbaric oxygen treatment and was discharged from hospital with complete recovery in a week. Cranial MR imaging findings 2 months later were normal (Fig 2A–C).

Received October 21, 2002; accepted after revision, November 21.

From the Departments of Radiology (H.M., Z.P., C.C.B., E.K., A.F.K.) and Undersea and Hyperbaric Medicine (H.A.), Gata Haydarpasa Teaching Hospital, Istanbul, Turkey.

Address correspondence to Hakan Mutlu, M.D., Gata Haydarpasa Egt. Hst. Radyoloji Böl., 81327 Uskudar/Istanbul, Turkey.

Discussion

Potassium chlorate is highly reactive and toxic agent that is used in matchstick heads. Chlorates are toxic substances when ingested or inhaled. Ingestion of potassium chlorate results in the rapid oxidative destruction of red blood cells, which is possibly followed or preceded by increased methemoglobin, cyanosis, and progressive kidney failure (1). The toxic dose of potassium chlorate in humans is 5 g, and the lethal dose is 15–35 g (2). Death in the early stage of chlorate poisoning is attributable to anoxia from methemoglobinemia or disseminated intravascular coagulopathy. Death is generally a result of renal failure. Our patient ingested nearly 2 g of potassium chlorate by ingesting a total of 120 matchstick heads.

Basal ganglia are at risk for many pathologic processes because of high metabolic rate and border zones of vascular supply. Gray matter structures have a higher cellular activity and a higher oxygen requirement than do white matter structures and thus are more vulnerable to oxygen deprivation. The damage that results from oxygen deprivation is actually mediated by toxic products causing irreversible neuronal damage and death. The selective vulnerability of gray matter structures to energy depletion is also reflected in the preferential affliction of gray matter in different types of intoxications (3).

In our case, T2-weighted and FLAIR images showed symmetric abnormal signal intensity within the deep gray matter and medial temporal lobes. These cranial MR imaging findings were consistent with hypoxia or anoxia caused by potassium chlorate intoxication. Although one may suggest that the changes described in this report are secondary to potassium chlorate intoxication, the other toxic substances commonly used to make matchstick heads (eg, potassium dichromate, carboxymethylcellulose, zinc oxide) might also have played a role in these imaging changes. Similar cranial MR imaging findings can be found in several conditions. Carbon monoxide intoxication is the prototype of toxic hypoxic brain injury. It predominantly involves the globus pallidus and has hyperintense signal on T2-weighted images. Methanol intoxication more often involves putamen and is associated with hemorrhagic necrosis (4). Both show toxic effects with selective necrosis of basal

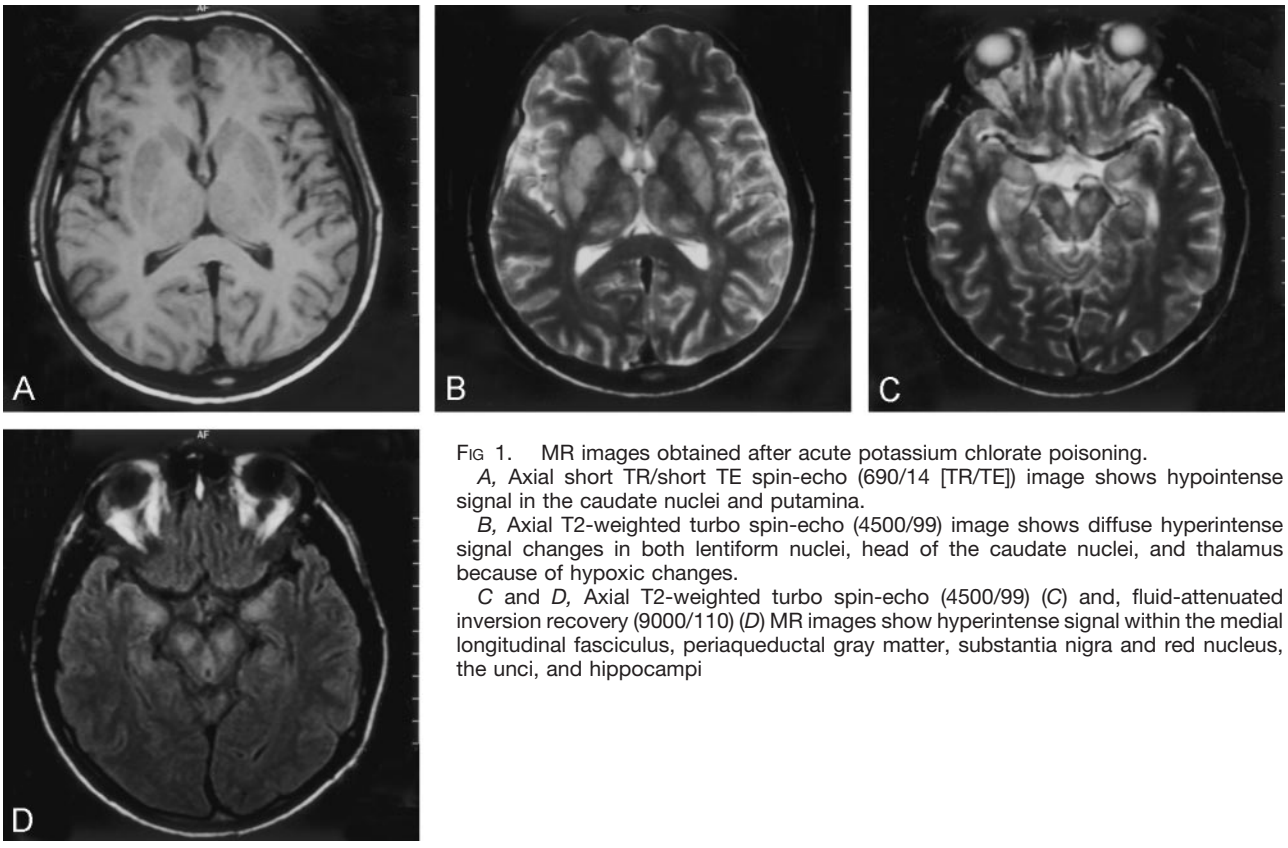

FIG 1. MR images obtained after acute potassium chlorate poisoning.

A, Axial short TR/short TE spin-echo (690/14 [TR/TE]) image shows hypointense signal in the caudate nuclei and putamina.

B, Axial T2-weighted turbo spin-echo (4500/99) image shows diffuse hyperintense signal changes in both lentiform nuclei, head of the caudate nuclei, and thalamus because of hypoxic changes.

C and *D,* Axial T2-weighted turbo spin-echo (4500/99) (*C*) and, fluid-attenuated inversion recovery (9000/110) (*D*) MR images show hyperintense signal within the medial longitudinal fasciculus, periaqueductal gray matter, substantia nigra and red nucleus, the unci, and hippocampi

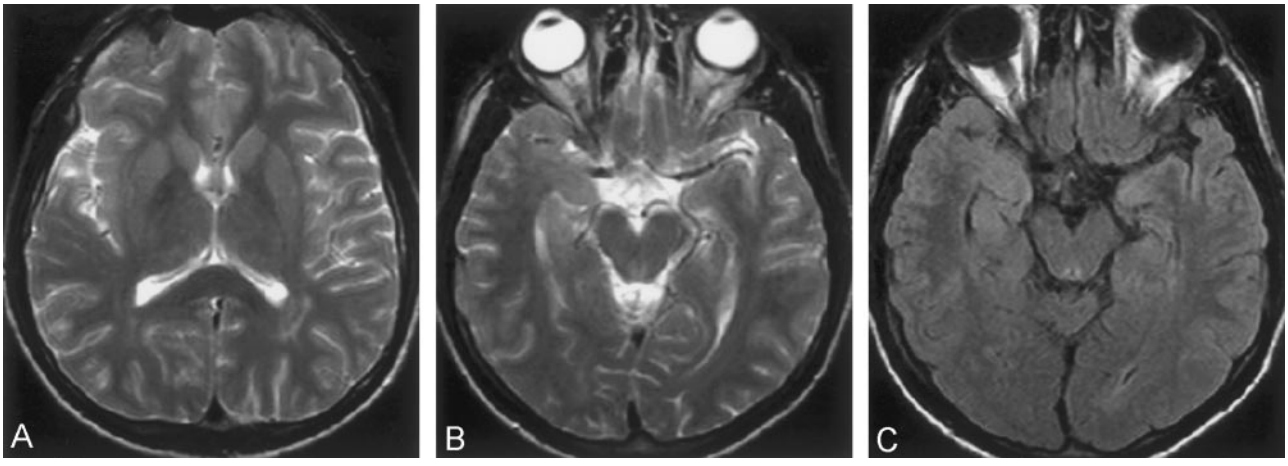

FIG 2. Follow-up MR images obtained 2 months after potassium chlorate poisoning. These images show normal in the lentiform nuclei, head of caudate nuclei, thalamus, and midbrain.

A and *B*, Axial T2-weighted turbo spin-echo (4500/99) images.

C, Fluid-attenuated inversion recovery (9000/110) image.

ganglia bilaterally (5). Hypoxia in cases of near drowning involves putamen and caudate nucleus (4). Wernicke encephalopathy, a toxic encephalopathy caused by thiamine deficiency in alcoholics, shows atrophy of the mamillary bodies, periventricular thalamus, periaqueductal gray, and hypothalamus (6). Central pontine myelinosis refers to a toxic form of demyelinization that primarily involves central pons, which is hypointense on T1-weighted images and hyperintense on T2-weighted images when this abnormality is present (7). Our search of the literature disclosed a few cases of potassium chlorate intoxication, but corresponding MR findings were not reported.

The recommended treatment for ingestion of toxic or potentially life-threatening doses of potassium chlorate is gastric lavage; administration of activated charcoal, sodium thiosulfate, alkaline diuresis, or methylene blue; and an exchange transfusion with possible hemodialysis in severe cases (1). Our patient had an exchange transfusion with hemodialysis, and the use of hyperbaric oxygen may

have contributed to his improvement. The patient responded well to hyperbaric oxygen treatment and was discharged from hospital with total recovery in a week. Cranial MR imaging showed normal findings 2 months later. We anticipated atrophic changes in those areas affected and advised long-term clinical and MR follow-up.

Conclusion

In the rare case that a patient presents with potassium chlorate intoxication, cranial MR imaging should be performed as soon as possible. Cranial MR imaging of potassium chlorate intoxication shows symmetric, abnormal signal intensity within the deep gray matter and medial temporal lobes on T2-weighted and FLAIR images, which to our knowledge, has not been previously reported.

Acknowledgments

We would like to thank Orhan Konez, M.D., for his helpful comments when reviewing the manuscript.

References

1. Dreisbach RH, Robertson WO. **General considerations.** In: *Handbook of Poisoning: Prevention, Diagnosis, and Treatment.* 12th ed. Norwalk, Conn: Appleton and Lange; 1987:23–56
2. Flume MZ. **Final report on the safety assessment of potassium chlorate.** *J Am Coll Toxicol* 1995;14:221–230
3. Hantson P, Duprez T, Mahieu P. **Neurotoxicity to the basal ganglia shown by MR imaging following poisoning by methanol and other substances.** *J Toxicol Clin Toxicol* 1997;35:151–161
4. Van der Knapp MS, Valk J. *Magnetic Resonance of Myelin, Myelination, and Myelin Disorders* 2nd ed. Berlin: Springer-Verlag; 1995:23–28, 351
5. Burgener FA, Meyers SP, Tan RK, Zaunberger W. *Differential Diagnosis in Magnetic Resonance Imaging.* Stuttgart: Thieme; 2002:88
6. Schoene WC. **Degenerative diseases of the central nervous system.** In: Davis RL, Robertson DM, eds. *Textbook of Neuropathology.* Baltimore: Williams & Wilkins;1985:788–823
7. Korogi Y, Takahashi M, Shinzato J, et al. **MR findings in two presumed cases of mild central pontine myelinosis.** *AJNR Am J Neuroradiol* 1993;14:651–654