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Brain MR Finding of βFluoroethyl Acetate Rodenticide Intoxication: A Case Report[1]

Ji Young Kim, M.D., Cheolkyu Jung, M.D., Seung Ro Lee, M.D., Dong woo Park, M.D.

βfluoroethyl acetate rodenticide intoxication can manifest as several different clinical abnormalities such as respiratory, neurologic, cardiologic and fluid-electrolyte problems. We report here on the MR findings of a case that showed symmetric cytotoxic edema in the white matter of the cerebral hemispheres after the ingestion of β fluoroethyl acetate rodenticide by a woman who was attempting suicide.

Index words : Poisoning
Fluoroacetate
Brain
Magnetic resonance (MR)
Rodenticide

Sodium monofluoroacetate (SMFA) is a highly toxic material that was once used as a rodenticide until 20 years ago. It was banned because its effects were toxic not only to rats, but also to other animals and humans. βfluoroethyl acetate is an ethyl ester form of SMFA, and the former is still available commercially and it is known to have similar toxic effects, though to a lesser degree (1). The toxic effect of fluoroacetate is caused by metabolite fluorocitric acid, which blocks the Krebs cycle and thereby depletes glutamate and ATP, and this increases the ammonia and lactate and causes citrate accumulation (2). It has been frequently hypothesized that the systemic abnormalities could be caused by metabolic acidosis (3, 4).

However, the brain MR findings of central nervous system abnormalities after ingestion of fluoroacetate have never been reported. We report here on a case that showed symmetric signal changes of the white matter in both cerebral hemispheres on the MR images, and this was caused by intentional βfluoroethyl acetate intoxication.

Case Report

A 32-year-old female was transferred to the emergency department with nausea and vomiting after ingesting 4.8 g (30 mL × 8 bottles) of rodenticide in a suicide attempt, and this rodenticide was composed of 2% βfluoroethyl acetate. She had been previously diagnosed with schizophrenia and she had taken psychotropic medication for 2 years. On admission, her vital signs were temperature: 36.2℃, pulse rate: 88/min, respiratory rate: 22/min and blood pressure: 170/80 mmHg. A physical examination did not show any abnormalities, and her mental and neurological examinations were also normal. Echocardiography showed suspicious myocardial ischemia. The myoglobin level of the cardiac enzymes was initially elevated up to 349 ng/mL (normal range: 10.5 - 92.5 ng/mL), but the creatine kinase-MB (CK-MB) was elevated up to 6.1 ng/mL (normal

[1]Department of Radiology, College of Medicine, Hanyang University
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Address reprint requests to : Cheolkyu Jung, M.D., Department of Radiology, Hanyang University Hospital, 17 Haengdang-dong, Seong dong-gu, Seoul 133-792, Korea.
Tel. 82-2-2290-9164 Fax. 82-2-2293-2111
E-mail: ckjung@hanyang.ac.kr

range: 3.6 - 5 ng/mL) two days later. Three days after admission, all the cardiac enzymes were normalized. Echocardiography was not performed initially, but it was normal on the seventh day after admission.

Gastric lavage with charcoal was promptly carried out one hour after she ingested the rodenticide. She was managed with conservative treatment during her hospital stay. Seventy-two hours after ingestion, her mental status changed to a stuporous condition, and motor weakness on the right side had developed. Brain MRI (1.5-T Genesis Signa; General Electric, Milwaukee, WI, U.S.A.) was promptly performed because of her neurological deficits. It showed symmetrical hyperintensities of the bilateral internal capsules, the deep and subcortical white matters and the corpus callosum on the diffusion-weighted images (DWI) (Fig. 1A, B), and decreased apparent diffusion coefficient (ADC) values in the corresponding areas (Fig. 1C). In light of the patient's clinical information, these findings were thought to be the cytotoxic effect of rodenticide intoxication. Four days after the initial neurological symptoms, her mental status completely recovered. The previously noted acute signal changes disappeared on the follow-up DWI that was done 10 days after the initial brain MR (Fig. 1D). Sixteen days after admission, she did not show any significant neurological and cardiological sequelae.

Discussion

The pharmacological properties of fluoroacetate have been extensively studied (4 - 6). The estimated lethal dose (LD_{50}) ranges from 2 to 10 mg/kg with a mean lethal dose of 5 mg/kg for humans (4). Its toxic effect is known to be produced by the metabolite fluorocitrate,

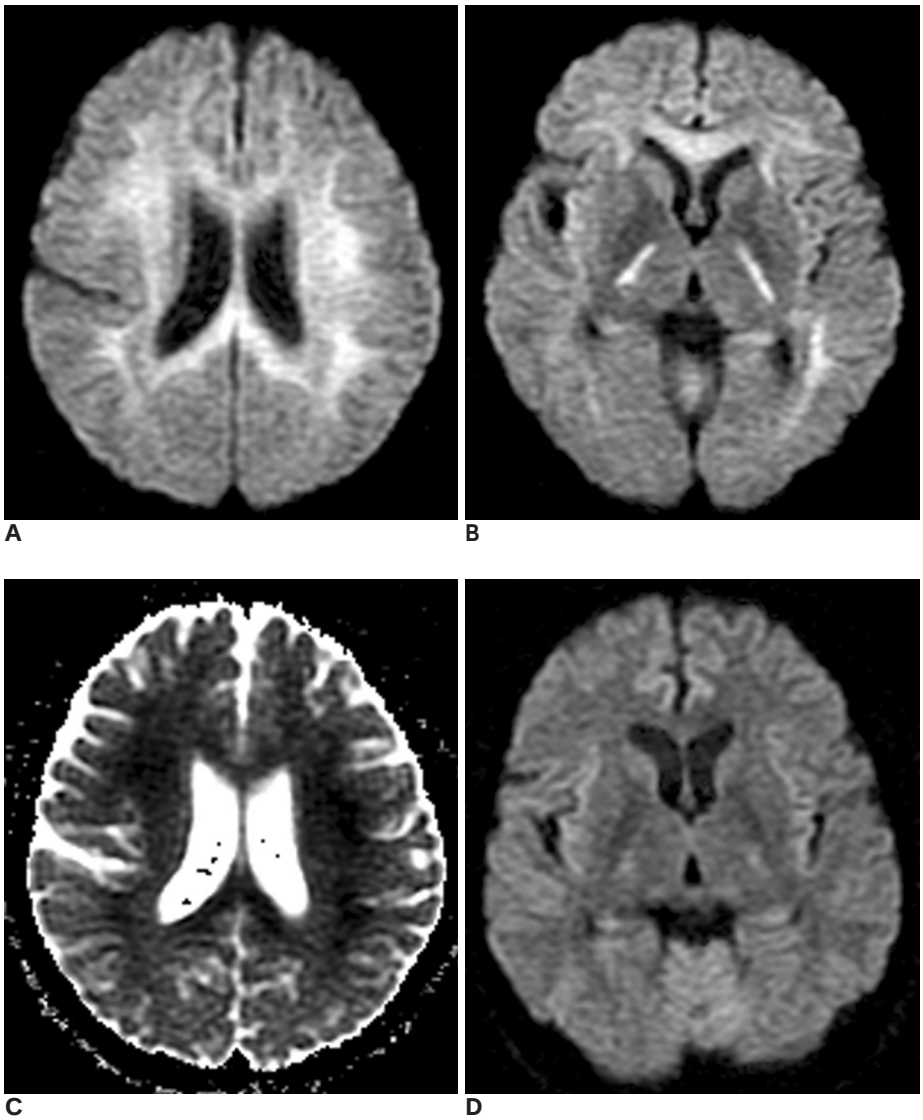

Fig. 1. A 32-year-old female with β Fluoroethyl Acetate intoxication. The initial diffusion-weighted images (**A, B**) and ADC map (**C**) of the brain showed the symmetric hyperintensities in the white matter of both cerebral hemispheres, the corpus callosum and the internal capsules (posterior limb), and a decreased ADC value in the same areas. These hyperintensities disappeared on the follow-up brain diffusion-weighted image (**D**) taken 10 days later.

which blocks aconitase in the Krebs cycle, and this inhibits energy production (5). Although βfluoroethyl acetate can be fatally toxic to humans, it reportedly has one tenth of the toxicity of sodium monofluoroacetate, which is known to be the most toxic fluoroacetate (1). The clinical effects of SMFA usually develop within 30 minutes to 2.5 hours after exposure, but they might be delayed as long as 20 hours (4). In our case, the patient's neurological manifestations were noted 72 hours after exposure, which might be because βfluoroethyl acetate is less toxic than SMFA.

There have been some clinical reports about intoxication with βfluoroethyl acetate (1, 7). However, the radiological manifestations, including the brain MR findings, are not well known. One report described the selective involvement of the cerebellum (1). In that report, seven cases of βfluoroethyl acetate rodenticide intoxication that showed cerebellar atrophy on follow-up brain CT and MRI were retrospectively reviewed. However, that report was published without presenting any radiological images, and the authors could not determine the reason for the selective cerebellar damage caused by β fluoroethyl acetate.

In our case, the initial brain MRI showed symmetrically diffusion restrictions in the deep and subcortical white matter, the corpus callosum and the internal capsules, and the follow-up MRI conducted 10 days later showed complete disappearance of the initial acute signal changes. In some reports, the central nervous system after fluoroacetate intoxication has also been studied (8, 9). These reports suggest that fluroacetate selectively inhibits the Krebs cycle of the glial cells rather than neurons (8). Fluoroacetate is more readily taken up by glial cells through a Na^+ - dependent transport system, the same as for acetate (10). βfluoroethyl acetate is an ethyl ester form of fluoroacetate and it has similar biochemical mechanisms. Therefore, this similarity might explain the selective involvement of the white matter in the brain, as was shown in our case.

From this case, we concluded that βfluoroethyl acetate intoxication seems to predominantly involve the white matter of the brain in the form of reversible cytotoxic edema, and this is probably due to blockage of the Krebs cycle, which manifests clinically as temporary neurologic deterioration.

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β-Fluoroethyl Acetate 쥐약 중독의 뇌 자기공명영상 소견: 증례 보고[1]

[1]한양대학교 의과대학 영상의학과학교실

김지영 · 정철규 · 이승로 · 박동우

쥐약의 성분으로 알려진 β-fluoroethyl acetate에 의한 중독은 호흡기나 순환기, 그리고 신경계통에 이르기까지 임상적으로 다양한 중독증상을 나타낼 수 있다. 저자들은 자살 시도로 β-fluoroethyl acetate를 음독한 환자의 뇌 자기공명영상에서 대뇌반구의 백질에 대칭적으로 일시적인 세포독성 부종을 보였던 증례를 보고하고자 한다.