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CASE REPORT

Reversible Toxic-Metabolic Encephalopathy in Fluroacetamide Intoxication: A Case Report and Review of the Literature

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Abstract: Fluoroacetamide, a commonly used convulsant rodenticide, can rapidly damage the nervous, digestive, and cardiovascular systems, potentially leading to fatal outcomes if ingested. This study reports the case of a 62-year-old Chinese woman who presented with symptoms of intoxication, including slurred speech, agitation, and seizure-like episodes, accompanied by gastrointestinal symptoms such as vomiting, skin bruising, and mild liver dysfunction. Toxin analysis revealed the presence of fluoroacetate in her blood and urine, and diffusion weighted imaging (DWI) imaging indicated white matter lesions, leading to the diagnosis of rare fluoroacetamide poisoning. This diagnosis facilitated the administration of treatments including vitamin K, hemodialysis, acetamide, and calcium gluconate. The patient subsequently regained consciousness, with improvements in laboratory results and gradual resolution of toxic-metabolic encephalopathy. This case highlights the importance of considering drug poisoning in patients with challenging neurological symptoms, particularly when there is a potential history of drug ingestion. Accurate diagnosis of such conditions is crucial, as timely and appropriate treatment can significantly improve clinical outcomes.

Keywords: fluoroacetamide, reversible toxic-metabolic encephalopathy, case report

Introduction

Fluoroacetamide is a commonly used convulsant rodenticide that has been banned worldwide due to its high toxicity to both animals and humans. Nevertheless, this substance continues to be a source of both intentional and unintentional poisoning cases in China and other countries.[1,2] Fluoroacetamide exerts its toxicity primarily by inhibiting citrate synthase, a key enzyme in the tricarboxylic acid (TCA) cycle, thereby blocking cellular oxidative metabolism. This enzymatic inhibition disrupts ATP production, leading to the accumulation of citric acid and a cascade of metabolic disturbances, including severe metabolic acidosis, hypocalcemia, and mitochondrial dysfunction.[3] These systemic effects ultimately result in cellular energy failure and multiorgan impairment.

Clinically, fluoroacetamide poisoning can manifest with a wide range of symptoms involving the respiratory, gastrointestinal, cardiovascular, and nervous systems. Gastrointestinal symptoms such as vomiting, nausea, and epigastric discomfort are common, along with cardiac manifestations, including QT interval prolongation, arrhythmias, and myocardial injury. Importantly, central nervous system involvement is frequently observed, with clinical features such as seizures, aphasia, muscle weakness, and coma. These manifestations are believed to arise from toxic-metabolic encephalopathy, reflecting widespread cerebral metabolic derangement and impaired neuronal function, rather than focal or structural brain injury.[4–7]

Despite its clinical significance, reports on fluoroacetamide poisoning remain limited. In many cases, especially when exposure history is unclear, misdiagnosis or delayed diagnosis can occur, potentially leading to adverse outcomes.

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Moreover, due to the the scarcity of clinical toxicology education and limited familiarity with rodenticide-induced neurotoxicity, early-stage recognition of toxic-metabolic brain injury is often missed.

Here, we present a case of fluoroacetamide intoxication manifesting as reversible toxic-metabolic encephalopathy, accompanied by a review of the literature. Our goal is to raise clinical awareness of this rare but potentially reversible condition and to improve early recognition and appropriate management.

Case Presentation

Clinical History

A 62-year-old Chinese woman who lived alone was brought to our emergency department after being found by her family approximately one hour earlier with slurred speech, inappropriate responses, and signs of agitation. Vomitus was observed on the floor at her residence. Upon arrival in the emergency room, the patient was confused. Within 30 minutes of admission, she experienced a witnessed generalized tonic-clonic seizure lasting approximately 2 minutes, characterized by bilateral tonic stiffening followed by rhythmic clonic movements, with no focal or lateralizing signs. The seizure resolved spontaneously, but due to persistent agitation and risk of recurrence, the patient was administered 5 mg intravenous midazolam. No additional seizures were observed during the hospital stay.

The patient had a 10-year history of hypertension, well-controlled with amlodipine. She was unable to provide any history of potential toxic exposure due to her altered mental state, but her family suspected accidental ingestion of rodenticide (possibly mistaken for medication). There was no known family history of neurological or genetic disorders.

Clinical Examination and Diagnosis

The patient was comatose, unresponsive to commands, and silent. Vital signs: Temperature 36.8°C, blood pressure 146/89 mmHg, heart rate 96 bpm. Glasgow Coma Scale (GCS) was 6 (E1V1M4). Her pupils were 3.0 mm with sluggish reaction to light, but brainstem reflexes were intact. Muscle tone was normal. The patient exhibited myoclonic jerks, more prominent in the distal right hand and foot, with no stereotyped movements. Deep tendon reflexes were symmetrical, with positive bilateral Babinski signs. No signs of meningeal irritation, including nuchal rigidity, Kernig's sign, or Brudzinski's sign. The skin and mucous membranes were non-icteric and moist. Scattered bruises were observed, mainly on the upper arms (Figure 1 [A-1, B-1, C-1]), but no petechiae or other signs of bleeding were observed in the conjunctiva, nasal cavity, or oral cavity.

The laboratory test results are as follows: Arterial blood gas analysis (performed on room air, FiO_2 = 21%) showed a partial pressure of oxygen (PO_2) of 85.9 mm Hg and a partial pressure of carbon dioxide (PCO_2) of 44.9 mm Hg. Coagulation function tests revealed a prothrombin time (PT) of 12.3 seconds, activated partial thromboplastin time (APTT) of 28.8 seconds, international normalized ratio (INR) of 1.11, D-dimer of 0.41 mg/L FEL, and thrombin time (TT) of 47.2 seconds. Alanine aminotransferase (ALT) was 54 U/L, aspartate aminotransferase (AST) was 41 U/L, total bilirubin was 30.7 μmol/L, and direct bilirubin was 14.6 μmol/L. Cerebrospinal Fluid (CSF) Analysis: Lumbar puncture revealed an opening pressure of 160 mmH_2O, 2 cells/μL, protein 0.38 g/L, and glucose 3.8 mmol/L (with concurrent blood glucose of 5.2 mmol/L). CSF cultures and PCR testing for bacterial and viral pathogens were negative (Table 1). Other laboratory tests, including complete blood count, creatinine, blood lipids, blood glucose, thyroid function, cardiac enzyme profile, stool routine, urinalysis, erythrocyte sedimentation rate, trace elements, blood ammonia, blood lactate, homocysteine, and electrocardiogram (ECG), were all normal or showed no abnormalities.

Electroencephalogram (EEG) examination revealed multiple sharp waves (Figure 2A). Magnetic resonance imaging of the head showed high signal lesions on diffusion weighted imaging (DWI) in the subcortical white matter of the bilateral frontoparietal lobes, posterior limbs of the bilateral internal capsules, corpus callosum (genu, body, and splenium), and bilateral cerebral peduncles. Corresponding hypointensity on the apparent diffusion coefficient (ADC) map was consistent with cytotoxic edema. FLAIR sequences also showed concordant hyperintensities (Figure 3A). Gadolinium-enhanced imaging or magnetic resonance angiography (MRA) was normal (Figure 3C). Toxicological screening using gas chromatography–mass spectrometry (GC-MS) confirmed the presence of fluoroacetate in both the patient's blood and urine. Based on the clinical, imaging, and toxicological findings, a final diagnosis of rodenticide (fluoroacetamide) poisoning was established.

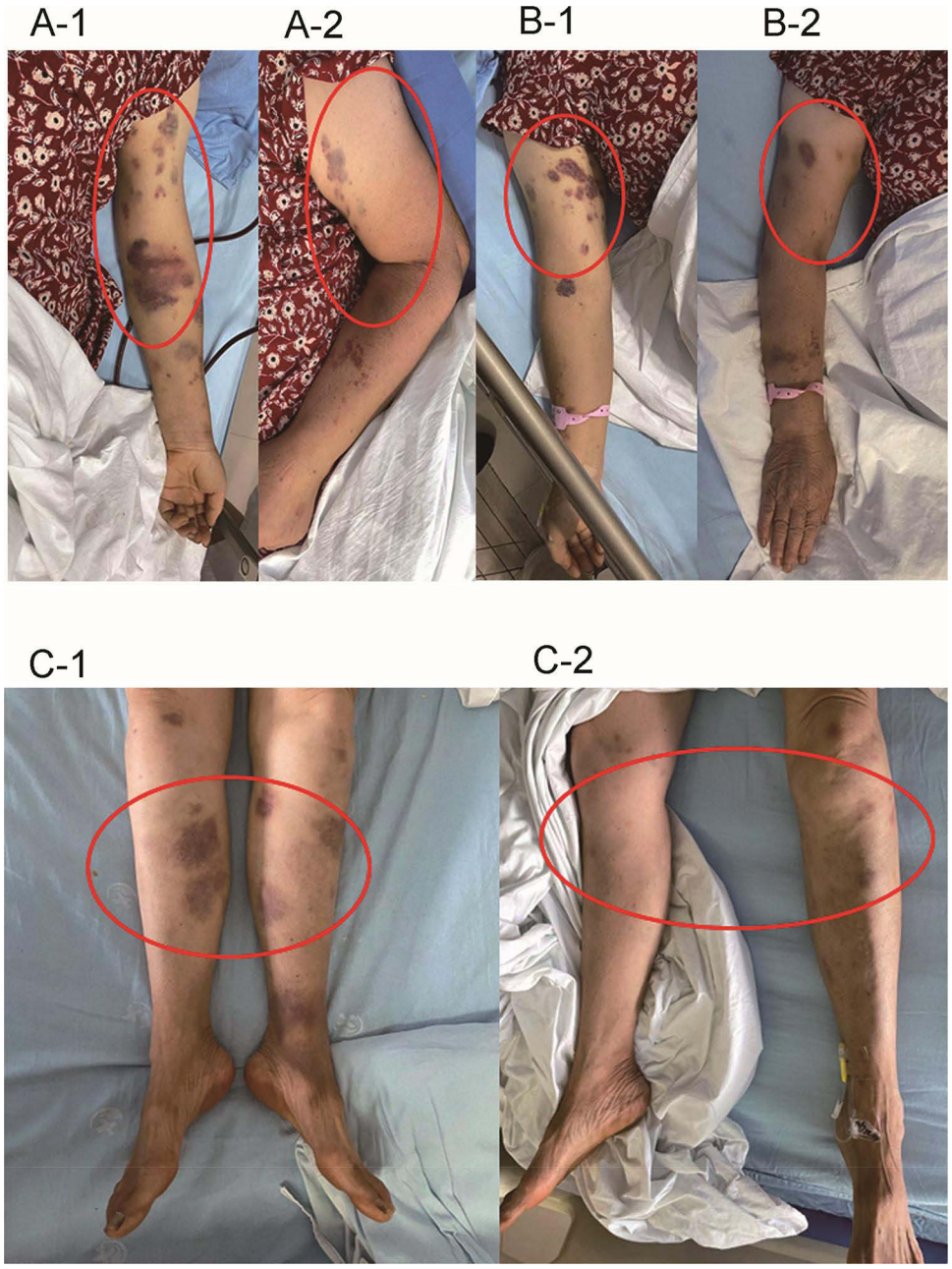

Figure 1 The scattered bruises on her upper limbs (**A, B**) and lower limbs (**C**) are shown in comparison images before (A-1, B-1, C-1) and after treatment (A-2, B-2, C-2). The red circles indicate the corresponding comparison of bruises.

Treatment

After admission, the patient underwent hemoperfusion and was administered intravenous infusions of vitamin K1 10 mg, pantoprazole 40 mg. Oral levetiracetam (500 mg) was initiated to manage seizure activity. The toxin analysis results were available 24 hours after admission, and the patient was immediately given intramuscular injections of acetamide 5 g every 8 hours, along with continuous intravenous infusion. Additionally, intravenous calcium gluconate 2 g per day was administered. Oral levetiracetam was continued at 500 mg daily for a total of 7 days to prevent seizure recurrence.

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Table 1 Summary of Blood Test Results and Reference Ranges

Test	Patient Value	Reference Range
Arterial Blood Gas Analysis		
PO_2 (mm Hg)	85.9	80–100
PCO_2 (mm Hg)	44.9	35–45
Coagulation Profile		
Prothrombin Time (PT, sec)	12.3	10.0–16.0
Activated Partial Thromboplastin Time (APTT, sec)	28.8	22.3–32.5
International Normalized Ratio (INR)	1.11	0.8–1.2
D-dimer (mg/L FEU)	0.41	0.0–0.5
Thrombin Time (TT, sec)	47.2	14.0–21.0
Liver Function Tests		
Alanine Aminotransferase (ALT, U/L)	54	7–40
Aspartate Aminotransferase (AST, U/L)	41	13–35
Total Bilirubin (μmol/L)	30.7	0–21
Direct Bilirubin (μmol/L)	14.6	0–8
Cerebrospinal Fluid (CSF) Analysis		
Opening Pressure (mmH_2O)	160	80–180
WBC Count (cells/μL)	2	0–5
Protein (g/L)	0.38	0.15–0.45
Glucose (mmol/L)	3.8	2.5–4.5*
Concurrent Blood Glucose (mmol/L)	5.2	—
Other Tests		
CSF culture and PCR	Negative	—
Other lab tests (CBC, renal, lipids, etc.)	Normal	—

Notes: *CSF glucose should be interpreted relative to concurrent blood glucose.

Outcome

On the second day after admission, the patient was able to open her eyes spontaneously. A brief cognitive assessment using the Mini-Mental State Examination yielded a score of 22/30, indicating mild cognitive impairment. Her speech, though slightly unclear, was mostly accurate, and her limb strength was normal. By the third day, her speech had fully cleared, and the bruises on her limbs had significantly reduced. On the seventh day, a repeat EEG showed no significant abnormalities (Figure 2B). MRI of the head revealed that the previously noted high-signal lesions on DWI in the subcortical white matter of the bilateral frontoparietal lobes, posterior limbs of the bilateral internal capsules, corpus callosum (genu, body, and splenium), and bilateral cerebral peduncles had all disappeared (Figure 3B). Intracranial spectroscopy indicated normal n-acetylaspartate (NAA)/creatine and choline/creatine ratios (Figure 3D), and the bruises on her limbs had almost completely resolved (Figure 1 [A-2, B-2, C-2]). After discharge, the patient no longer required acetamide, calcium gluconate, or other medications. No seizure recurrence was observed during hospitalization or over the 2-year follow-up period. At follow-up, the patient remained in good condition with no reported cognitive or neurological sequelae.

Discussion

Fluoroacetamide, a highly toxic α-naphthylthiourea rodenticide, is lethal at doses as low as 30 mg.[8] Although globally banned due to its extreme toxicity,[4] it remains widely used as a pesticide in China. Its odorless nature complicates diagnosis, often leading to misdiagnoses or delayed treatment. This paper presents a case of fluoroacetamide poisoning, where the patient's reported ingestion and characteristic clinical and imaging findings facilitated timely diagnosis and effective treatment, resulting in a favorable outcome.

Once absorbed through the respiratory tract, gastrointestinal tract, or skin, fluoroacetamide is metabolized into fluoroacetate, which exerts systemic toxicity. Clinical manifestations typically emerge within hours and involve multi-system injury. Mild intoxication may present with gastrointestinal symptoms, fatigue, and focal neurological signs, while severe cases can rapidly progress to refractory seizures, coma, and multi-organ failure.[5,9,10] Laboratory tests may reveal

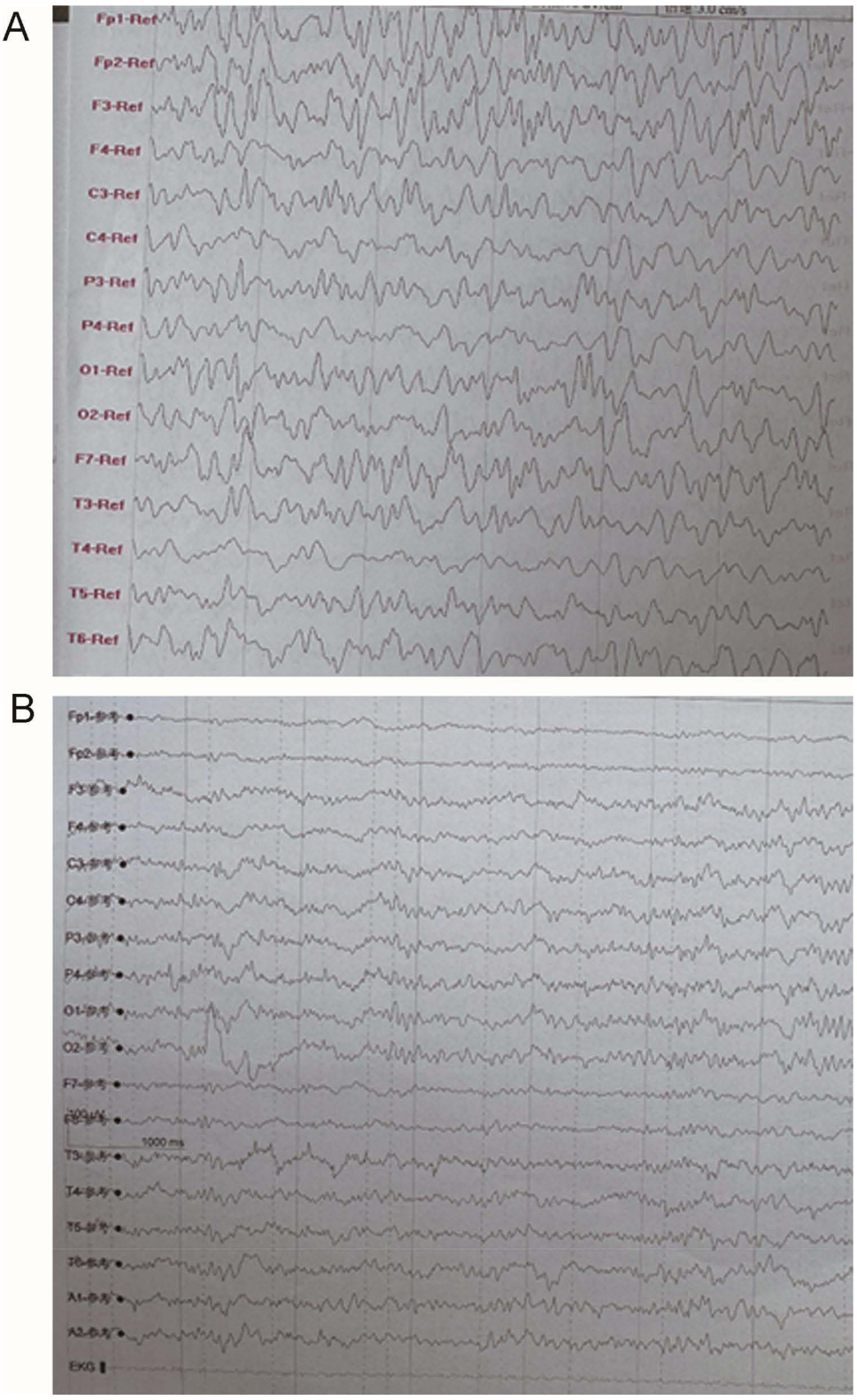

Figure 2 The EEG on admission (**A**) shows occasional single spikes and spike-and-slow wave complexes in the left frontal, central, parietal-occipital, and anterior, middle, and posterior temporal regions. After treatment (**B**), the EEG shows no definite abnormal wave activity across the leads.

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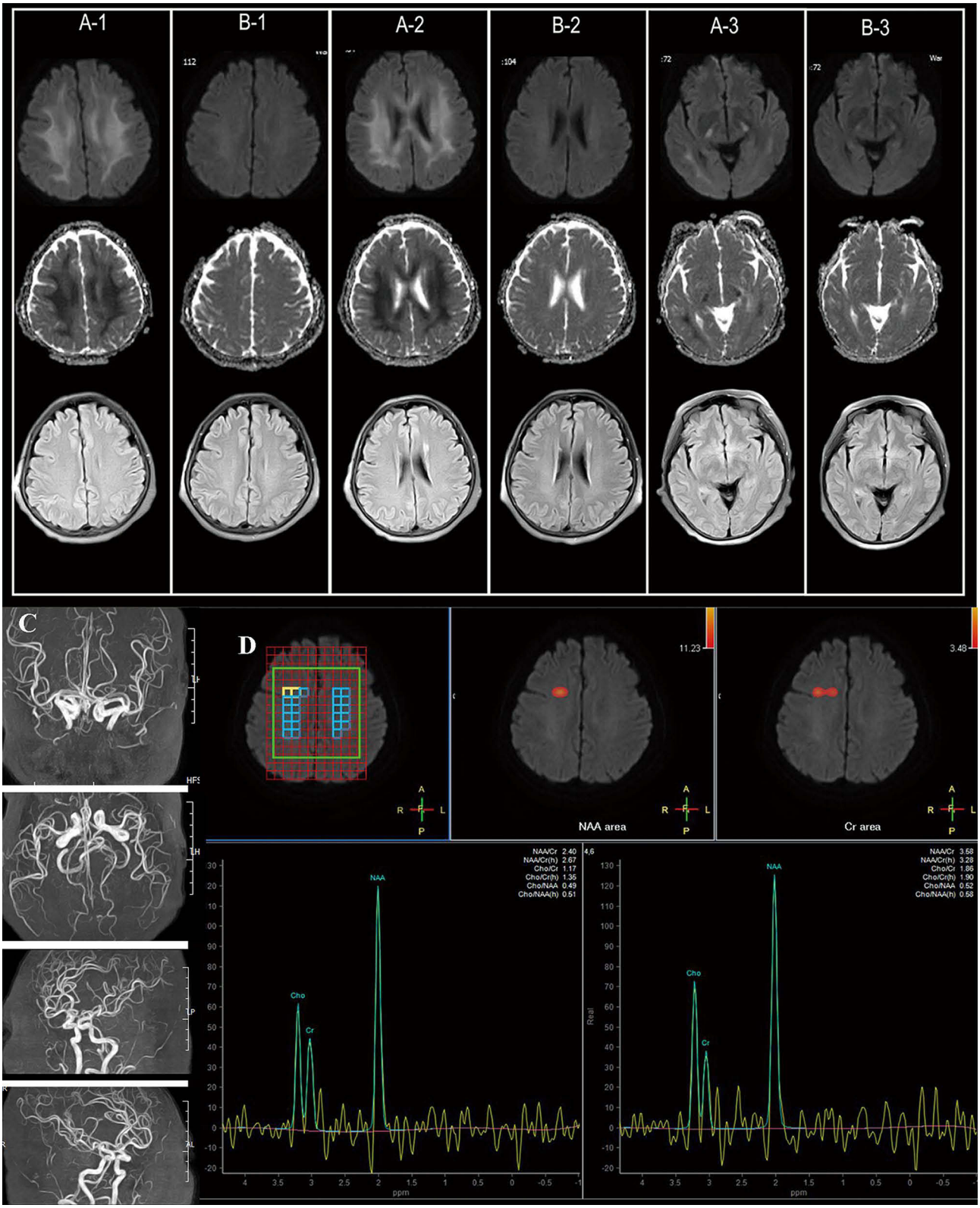

Figure 3 Brain Diffusion Weighted Imaging, ADC and FLAIR. (**A**) On admission; (**B**) 7 days after consciousness recovery. (**C**) Magnetic resonance angiography On admission. (**D**) Magnetic resonancespectroscopy shows normal N-acetylaspartate (NAA)/creatine and choline/creatine ratio.

persistently elevated levels of creatine kinase (CK), lactate dehydrogenase (LDH), ALT, AST, and creatinine. The presence of hematuria suggests cardiac, hepatic, and renal insufficiency, and may be accompanied by atypical changes in ECG or EEG readings.[9–12] Some patients with fluoroacetamide poisoning may develop central nervous system damage, manifesting as dysarthria, difficulty swallowing, and muscle weakness.[4]

Our patient exhibited acute toxic-metabolic encephalopathy, characterized by dysarthria, impaired cognition, agitation, and seizure-like activity. Toxin analysis confirmed fluoroacetate in serum and urine. MRI revealed transient symmetrical lesions in the bilateral basal ganglia and thalamus, which resolved after 7 days. This pattern is consistent with previously reported cases of reversible ischemic-hypoxic injury.[13,14] Several mechanisms may underlie fluoroacetamide-induced neurotoxicity: (1) inhibition of the tricarboxylic acid cycle via fluoroacetyl-CoA formation, leading to citrate accumulation and energy failure;[3] (2) impaired oxidative phosphorylation due to pyruvate metabolism blockade, causing cerebral hypoxia;[15] and (3) calcium ion depletion secondary to fluoride ion release, which exacerbates neuronal excitability and hypoxic injury.[16]

Laboratory tests indicated mild hepatic dysfunction and elevated creatine kinase, suggestive of systemic metabolic stress. Skin ecchymosis may have reflected transient endothelial injury, although coagulation profiles remained within normal limits. Notably, prompt initiation of hemoperfusion likely mitigated systemic toxicity and organ injury.

Additionally, our patient exhibited gastrointestinal symptoms, skin bruising, and mild liver dysfunction. In previous case reports, gastrointestinal symptoms have been the earliest and most common clinical manifestations of drug poisoning.[2] Fluoroacetamide poisoning can also lead to multi-organ failure, with myocardial damage and liver dysfunction being the most frequently reported complications.[5,6,17] The skin bruising may be related to coagulation dysfunction; however, in our patient, coagulation tests showed no significant abnormalities upon admission, and subsequent follow-up coagulation tests also revealed no significant changes. This may be attributed to the prompt initiation of hemoperfusion therapy on the first day of admission, which likely helped to rapidly reduce the toxicity of the substance in the body and minimize damage to the organs.

The diagnosis of fluoroacetamide poisoning primarily relies on a detailed history of exposure, specific clinical manifestations, and toxin detection. Additionally, close monitoring of organ function damage is essential. Management of fluoroacetamide poisoning requires rapid toxin elimination, typically via hemoperfusion and administration of specific antidotes such as acetamide and calcium gluconate.[9,11,12] Vitamin C is also employed to support detoxification and mitigate oxidative damage. Supportive care includes proton pump inhibitors for gastrointestinal protection and agents to support hepatic metabolism, such as reduced glutathione and compound glycyrrhizin, although robust evidence for their efficacy in this context is limited. Regarding anticonvulsant therapy, the patient received oral levetiracetam for 7 days without seizure recurrence. While diazepam remains a first-line agent in toxin-induced seizures, the use of valproic acid in this context should be approached with caution. Valproate's known risks of hepatotoxicity and thrombocytopenia may outweigh its benefits, especially in cases involving hepatic impairment.[4]

Conclusion

In conclusion, this case highlights the diagnostic and clinical challenges of reversible toxic-metabolic encephalopathy caused by fluoroacetamide poisoning. It underscores the critical need for early recognition and rapid intervention in patients presenting with unexplained neurological symptoms, especially in regions where exposure to rodenticides remains a risk. Importantly, this case illustrates the value of incorporating diffusion-weighted and FLAIR MRI sequences to detect dynamic brain changes associated with toxin exposure. Clinicians should maintain a high index of suspicion for toxic etiologies in acute encephalopathy, and consider prompt decontamination and organ-protective strategies to mitigate long-term neurological damage. This report contributes to a deeper understanding of toxic encephalopathies and supports more informed, timely clinical decision-making in similar contexts.

Abbreviations

DWI, diffusion weighted imaging; PO_2, partial pressure of oxygen; PCO_2, partial pressure of carbon dioxide; PT, prothrombin time; APTT, activated partial thromboplastin time; INR, international normalized ratio; TT, thrombin time;

ALT, alanine aminotransferase; AST, aspartate aminotransferase; EEG, electroencephalogram; NAA, N-Acetylaspartate; CK, creatine kinase; LDH, lactate dehydrogenase; TCA, tricarboxylic acid.

Data Sharing Statement

This study complied with the guidelines for human studies and was conducted in accordance with the World Medical Association Declaration of Helsinki. Written informed consent for publication of this case (including the use of images) was obtained from both the patient and their family. Institutional approval was not required for publication of this case report under the policies of Chongzhou People's Hospital, where the study was conducted. The authors confirm that the data supporting the findings of this study are available within the article.

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Disclosure

The authors report no conflicts of interest in this work.

References

1. Chau CM, Leung AKH, Tan IKS. Tetramine poisoning. *Hong Kong Med J*. 2005;11(6):511–514.
2. Deng X, Li G, Mei R, Sun S. Long term effects of tetramine poisoning: an observational study. *Clin Toxicol*. 2012;50(3):172–175. doi:10.3109/15563650.2012.657758
3. Kim JB, Jang J-W, Kim JH. Reversible leukoencephalopathy in sodium monofluoroacetate intoxication. *Neurology*. 2014;82(13):1190–1191. doi:10.1212/WNL.0000000000000262
4. Lin J, Jiang C, Ou J, Xia G. Acute fluoroacetamide poisoning with main damage to the heart. *Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi*. 2002;20(5):344–346.
5. Lu A, Yuan F, Yao Y, et al. Reversible leukoencephalopathy caused by 2 rodenticides bromadiolone and fluoroacetamide: a case report and literature review. *Medicine*. 2021;100(9):e25053. doi:10.1097/MD.0000000000025053
6. Mankad K, Hoey E, Yap KS. Reversible leukoencephalopathy syndrome. *Am J Emerg Med*. 2010;28(3):386.e3–386.e5. doi:10.1016/j.ajem.2009.06.017
7. Poon WT, Chan K, Lo MH, et al. A case of tetramine poisoning: a lethal rodenticide. *Hong Kong Med J*. 2005;11(6):507–509.
8. Proudfoot AT, Bradberry SM, Vale JA. Sodium fluoroacetate poisoning. *Toxicol Rev*. 2006;25(4):213–219. doi:10.2165/00139709-200625040-00002
9. Rajasuriar R, Awang R, Hashim SBH, Rahmat HRBH. Profile of poisoning admissions in Malaysia. *Hum Exp Toxicol*. 2007;26(2):73–81. doi:10.1177/0960327107071857
10. Taitelman U, Roy A, Hoffer E. Fluoroacetamide poisoning in man: the role of ionized calcium. *Arch Toxicol Suppl*. 1983;6:228–231.
11. Tormoehlen LM. Toxic leukoencephalopathies. *Neurol Clin*. 2011;29(3):591–605. doi:10.1016/j.ncl.2011.05.005
12. Tormoehlen LM. Toxic leukoencephalopathies. *Psychiatr Clin North Am*. 2013;36(2):277–292. doi:10.1016/j.psc.2013.02.006
13. Wang R, Zhuo L, Wang Y, et al. Lessons learned from poisoning cases caused by 2 illegal rodenticides: tetramine and fluoroacetamide. *Medicine*. 2016;95(41):e5103. doi:10.1097/MD.0000000000005103
14. Wen W, Gao H, Kang N, et al. Treatment of severe fluoroacetamide poisoning in patient with combined multiple organ dysfunction syndrome by evidence-based integrated Chinese and Western medicines: a case report. *Medicine*. 2017;96(27):e7256. doi:10.1097/MD.0000000000007256
15. Xu X, Song G, Zhu Y, et al. Simultaneous determination of two acute poisoning rodenticides tetramine and fluoroacetamide with a coupled column in poisoning cases. *J Chromatogr B Analyt Technol Biomed Life Sci*. 2008;876(1):103–108. doi:10.1016/j.jchromb.2008.10.027
16. Zhang D, Zhang J, Zuo Z, Liao L. A retrospective analysis of data from toxic substance-related cases in Northeast China (Heilongjiang) between 2000 and 2010. *Forensic Sci Int*. 2013;231(1–3):172–177. doi:10.1016/j.forsciint.2013.05.014
17. Zhang Y, Su M, Tian DP. Tetramine poisoning: a case report and review of the literature. *Forensic Sci Int*. 2011;204(1–3):e24–e27. doi:10.1016/j.forsciint.2010.06.025