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Case Report

Neuroimaging and Clinical Course of Methanol-Induced Encephalopathy: Insights from a Recent Mass-Poisoning Event

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ABSTRACT

Methanol poisoning remains a significant public-health concern, especially in regions where restrictive alcohol policies foster illicit and unregulated alcohol production. The Kuwait methanol outbreak of August 2025 resulted in numerous fatalities and neurological injuries. This case report describes a survivor who initially received dialysis in Kuwait but returned to India several days later with persistent cognitive dysfunction. At presentation, he exhibited slowed mentation but had remarkably good visual recovery. MRI revealed classical features of methanol-induced neurotoxicity, including bilateral putaminal hyperintensities and deep white matter involvement. The patient was managed at the Department of Neurology, Fortis Hospital, Jalandhar. Although fomepizole was not administered during the initial Kuwait-based treatment, supportive care and neuro-rehabilitation resulted in gradual cognitive improvement. He was eventually discharged in stable condition with only minor residual neural complications. This case highlights the characteristic imaging findings of methanol toxicity, demonstrates the potential for partial neurological recovery even after delayed presentation, and emphasizes the need for improved regulatory and clinical preparedness to mitigate the consequences of toxic alcohol outbreaks.

Keywords: Methanol poisoning, methanol-induced encephalopathy, putaminal necrosis, toxic leukoencephalopathy.

INTRODUCTION

Methanol poisoning remains a life-threatening toxicological emergency and a persistent public-health problem worldwide, particularly in regions where restrictive alcohol policies, socioeconomic vulnerability, and illicit alcohol production coexist. Despite increased awareness and established treatment protocols, periodic outbreaks continue to result in significant morbidity and mortality [1]. Methanol itself is relatively non-toxic; however, its metabolism to formaldehyde and formic acid leads to severe high–anion gap metabolic acidosis and selective central nervous system injury through inhibition of mitochondrial cytochrome c oxidase, resulting in cellular hypoxia and energy failure. Clinically, this manifests as the characteristic triad of metabolic acidosis, visual impairment, and altered mental status [2].

Neuroimaging, especially magnetic resonance imaging (MRI), plays a crucial role in the diagnosis and prognostication of methanol-induced encephalopathy. Bilateral putaminal necrosis, diffusion restriction, and variable white matter involvement are well-recognized radiological hallmarks and reflect the selective vulnerability of metabolically active deep gray nuclei and myelinated tracts[3]. While these imaging patterns are well described, the temporal evolution of neurological injury and recovery following partial or delayed treatment, particularly when antidotal therapy is unavailable, remains incompletely characterized[4].

In August 2025, a large methanol poisoning outbreak in Kuwait resulted in multiple fatalities and survivors with neurological sequelae, predominantly affecting expatriate laborers [5]. The present case is noteworthy for several reasons: early life-saving haemodialysis was administered during the acute phase in an outbreak setting, but specific alcohol dehydrogenase inhibition with fomepizole or ethanol was not available; neuroimaging was performed only after delayed presentation; and there was a notable dissociation between substantial visual recovery and persistent cognitive impairment. This report therefore provides insight into the delayed neuroimaging correlates, clinical–radiological dissociation, and recovery trajectory of methanol-induced encephalopathy following early dialysis alone. By documenting these features, the case adds clinically relevant information to existing literature and highlights important considerations for diagnosis, prognosis, and follow-up in survivors of methanol poisoning, particularly in resource-limited or outbreak scenarios.

CASE PRESENTATION

A 35-year-old South Asian male with no prior comorbidities consumed illicit alcohol during the methanol poisoning outbreak in Kuwait in August 2025. Multiple individuals who ingested the same substance reportedly developed acute visual symptoms, rapid neurological deterioration, and death. The patient presented to a local healthcare facility in Kuwait approximately 18–24 hours after ingestion with altered sensorium and severe metabolic acidosis. Initial arterial blood gas analysis demonstrated a pH of 7.08, bicarbonate level of 8 mmol/L, and an anion gap of 32 mmol/L. Serum creatinine was 2.1 mg/dL, and calculated serum osmolal gap was markedly elevated (38 mOsm/kg). Serum methanol and formate levels were not available due to resource constraints during the outbreak. Emergency intermittent haemodialysis was initiated within hours of presentation, resulting in gradual metabolic stabilization over the next 48 hours. Fomepizole or ethanol-based antidotal therapy could not be administered due to limited availability. No neuroimaging was performed during the acute phase. After resolution of acidosis (pH 7.36; bicarbonate 22 mmol/L) and improvement in renal parameters, the patient was discharged.

Over the subsequent five to six days, he developed progressive cognitive slowing, impaired concentration, and intermittent confusion, prompting evaluation in India. On admission to the Department of Neurology, Fortis Hospital, Jalandhar, he was conscious, cooperative, and fully oriented, with a Glasgow Coma Scale score of 15/15. Cranial nerve examination revealed preserved visual acuity with mild photophobia. Motor, sensory, and cerebellar examinations were normal. Cognitive screening revealed a Montreal Cognitive Assessment (MoCA) score of 22/30, with deficits predominantly in attention, processing speed, and executive function.

Repeat laboratory investigations showed normal acid–base status, serum creatinine of 1.1 mg/dL, and no electrolyte

abnormalities. Brain MRI demonstrated bilateral symmetrical putaminal T2/FLAIR hyperintensities with corresponding diffusion restriction and low apparent diffusion coefficient values, consistent with cytotoxic injury. Patchy deep periventricular and subcortical white matter hyperintensities were noted, suggestive of toxic leukoencephalopathy. Susceptibility-weighted imaging showed no hemorrhagic transformation. The optic nerves appeared structurally intact.

The patient was managed with supportive neurological care, hydration, high-dose thiamine supplementation, and structured cognitive rehabilitation strategies. Over the hospital course, gradual improvement in attention and psychomotor speed was observed. At discharge, mild residual cognitive slowing persisted (MoCA 25/30). He was discharged in stable condition with advice for continued outpatient neurological follow-up and cognitive rehabilitation.

DISCUSSION

Methanol poisoning remains a significant global health problem, characterized by its high potential for morbidity and mortality despite being fully preventable. Outbreaks such as the Kuwait incident in 2025 reflect systemic vulnerabilities driven by social constraints, economic disparities and limited public oversight of alcohol production. This case exemplifies the classical clinical and radiological features of methanol-induced neurotoxicity while also demonstrating a less commonly reported pattern of delayed presentation with partial neurological recovery.

Methanol's toxicity results from its metabolism to formic acid, a potent inhibitor of mitochondrial cytochrome c oxidase. The consequent disruption of oxidative phosphorylation leads to cellular hypoxia, lactic acidosis and selective neuronal damage. Deep gray nuclei—particularly the putamen—are especially vulnerable due to their high metabolic demand [1,2]. The patient's MRI findings of bilateral putaminal hyperintensities and diffusion restriction align with extensively documented imaging patterns in methanol poisoning, including those described by Blanco et al (2006) [3]. White matter changes, reflecting toxic leukoencephalopathy, further corroborate severe methanol-related CNS injury [4].

Unlike many reported cases, this patient underwent dialysis early in Kuwait, which likely contributed to his relatively favorable systemic recovery despite the absence of fomepizole therapy. In this patient, early intermittent haemodialysis initiated during the acute phase of poisoning likely played a pivotal role in systemic stabilization and visual recovery, despite the absence of alcohol dehydrogenase inhibition with fomepizole or ethanol Studies, including those by Zakharov et al (2012), have shown that early haemodialysis is a crucial determinant of survival even when neurological injury has already begun [6]. However, because dialysis alone does not inhibit methanol metabolism, residual formate generation may continue in the absence of fomepizole or ethanol. This could explain the persistence of the patient's cognitive slowing, despite systemic stabilization and good visual recovery.

The patient's visual improvement is notable, as optic neuropathy is often irreversible. Early dialysis likely mitigated prolonged exposure of retinal ganglion cells to formate, preventing irreversible optic nerve injury, as described in prior literature [7]. This contrasts with typical outcomes of methanol poisoning, where blindness is frequently permanent. The case also parallels epidemiological findings from other methanol poisoning disasters, including the Hooch Tragedy in Majha Region of Punjab, described by Singh et al (2021) [8]. Their analysis highlights that

methanol disasters disproportionately affect marginalized populations who rely on illicit alcohol due to economic constraints or legal restrictions. The Kuwait outbreak similarly affected vulnerable expatriate laborers, demonstrating that methanol poisoning events are not isolated incidents but recurrent consequences of systemic socio-economic vulnerability [8].

The delayed presentation in India highlights the evolving neurological course after methanol toxicity. Methanol-related cognitive impairment may persist even after acidosis correction and dialysis, as structural CNS injury continues to evolve. The patient's MRI findings confirm that basal ganglia damage persisted despite clinical improvement. The study by Bhatia et al (2008) emphasizes that putaminal necrosis is a reliable predictor of persistent neurological sequelae [9]. Nevertheless, the patient's eventual discharge with only minor neural complications demonstrates that meaningful recovery is achievable, especially when early lifesaving measures such as dialysis are administered. Overall, this case reinforces the importance of rapid recognition, early dialysis and the indispensable diagnostic role of MRI, while also highlighting the broader public-health implications and preventability of methanol poisoning incidents.

CONCLUSION

This case from the 2025 Kuwait methanol outbreak demonstrates the classical radiological features of methanol neurotoxicity, including bilateral putaminal necrosis and toxic leukoencephalopathy. Despite delayed MRI evaluation and absence of fomepizole therapy during the initial treatment in Kuwait, early dialysis likely contributed to the patient's good visual recovery and overall systemic stabilization. On presentation in India, he exhibited mild but significant cognitive slowing, which gradually improved with supportive neurological care. He was ultimately discharged in a healthy and stable condition with only minor neural complications. This case highlights the importance of early intervention, the diagnostic value of MRI in delayed presentations, and the need for robust public-health oversight to prevent methanol-related tragedies.

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