Neurological Manifestations Following Ingestion of Hand Sanitizer: An Unfortunate Morbidity Tale during COVID-19 Pandemic

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Abstract

Introduction: COVID-19 was declared a pandemic in January 2020. To stop the spread of the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) virus, the use of hand disinfectants increased significantly, even in household areas. These alcohol-based solutions may be the cause of suicides and homicides by both intentional and accidental exposures. Cases reported with magnetic resonance imaging (MRI) features of central nervous system involvement in toxic alcohol-based hand sanitizers are limited.

Case description: A 26-year-old patient with schizophrenia was brought to the emergency department (ED) following self-ingestion of alcohol-based hand sanitizer. An initial non-contrast computed tomography (NCCT) brain was normal. During weaning from ventilatory support, we noticed a motor deficit in all four limbs with difficulty in weaning. Subsequent MRI brain showed features suggestive of toxic leukoencephalopathy. Nerve conduction studies (NCSs) revealed motor polyneuropathy in all four limbs.

Conclusion: Alcohol-based hand sanitizer ingestion may produce extensive brain involvement with polyneuropathy, which can be fatal. **Keywords:** Coronavirus disease of 2019, Glasgow Coma Scale, Hand sanitizer, Leukoencephalopathy, Polyneuropathy, Schizophrenia. *Indian Journal of Critical Care Case Report* (2023): 10.5005/jp-journals-11006-0035

INTRODUCTION

After the declaration of COVID-19 as a pandemic in January 2020, the use of hand disinfectants increased significantly to stop the spread of the SARS-CoV-2 virus. Currently, available hand sanitizer products in the market are mainly alcohol-based formulations and solutions with alcohol content between 60 and 95% in volume, being the most prevalent and effective. Such concentrations of alcohol are able to desaturate the proteins of microbes and inactivate viruses.¹ The toxic alcohols which are constituents of hand sanitizers include ethanol, methanol, ethylene glycol, isopropyl alcohol, diethylene glycol, and propylene glycol. These alcohols may be the cause of suicides and homicides by both intentional and accidental exposures. Usual presentations of lethal poisonings due to high concentrations of ethanol-based sanitizers are coma, metabolic acidosis, respiratory depression, hypothermia, cardiac dysrhythmias, or arrest.² Despite the best supportive medical therapy, it can have a lethal outcome, even in young patients.

We present the clinical status, MRI features, and outcome of poisoning by intentional suicidal ingestion of 80% ethanol-based disinfectant in a young lady suffering from schizophrenia. Written informed consent was taken from the surrogate decision maker. We present the following article in accordance with the case reports (CARE) guidelines.

CASE DESCRIPTION

A 26-year-old young female was brought to our ED with a history of multiple episodes of vomiting and one episode of seizure 2 days after an alleged history of ingestion of approximately 250 mL of 80% alcohol-content hand sanitizer (brand name—Go-Safe, marketed by Clarichem Industries Private Limited, Pune, Maharashtra, India, with ^{1,2,5,6}Department of Critical Care Medicine, Institute of Medical Sciences & SUM Hospital, Siksha 'O' Anusandhan (Deemed to be University), Bhubaneswar, Odisha, India

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a composition of ethanol 96% IP 83.33% volume per volume (v/v), glycerol 98% IP 1.45% (v/v), hydrogen peroxide 3% IP 4.17% (v/v), color (QS). She was diagnosed with schizophrenia 3 years ago and was on regular antipsychotic medication (tablet risperidone 2 mg once daily). The patient was well educated with no history of drug dependence or alcoholism. There was no similar previous history of hospital admission. On arrival at the ED, she was unconscious and gasping. Glasgow Coma Scale (GCS) was 3 with a blood pressure of 150/80 mm Hg, pulse rate of 78 beats/minute, respiratory rate of 28/minute, and oxygen saturation of 99% on room air. The initial arterial blood gas

© The Author(s). 2023 Open Access This article is distributed under the terms of the Creative Commons Attribution 4.0 International License (https://creativecommons. org/licenses/by-nc/4.0/), which permits unrestricted use, distribution, and non-commercial reproduction in any medium, provided you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons license, and indicate if changes were made. The Creative Commons Public Domain Dedication waiver (http://creativecommons.org/publicdomain/zero/1.0/) applies to the data made available in this article, unless otherwise stated. (ABG) indicated a severe metabolic acidosis with a high anion gap and normal lactate levels [pH, 7.05; partial pressure of oxygen, 78 mm Hg; partial pressure of carbon dioxide, 11.9 mm Hg; bicarbonate (HCO₃), 3 mmol/L; lactate, 1.91 mmol/L; sodium (Na) ions, 140 mmol/L; K⁺, 5.2 mmol/L; chlorine ions, 115 mmol/L]. The patient was immediately intubated in view of low GCS and severe metabolic acidosis. Blood alcohol concentration (BAC) was 280 mg/dL. She has resuscitated with 500 mL of 0.9% normal saline over 15 minutes and 100 mEq of NaHCO₃ bolus, followed by a continuous infusion of the same. She was also started on an antiepileptic injection of levetiracetam 500 mg intravenous twice daily. Post-resuscitation, her ABG improved with adequate urine output of around 30–40 mL/hour. An urgent NCCT brain was done, which was normal. For further management, she was shifted to the medical intensive care unit.

She was managed with empirical antibiotics, multivitamins, low-molecular-weight heparin, and nasogastric tube feeding. On the second day of admission, her GCS improved, and her ABG normalized. She was hemodynamically stable and maintained adequate urine output with routine enteral feeding. No additional fluids were administered. Antibiotics were stopped on day 3 as cultures were sterile. Subsequently, on day 5, absent motor power and sluggish deep tendon reflexes in bilateral upper and lower limbs were noted. The bilateral plantar reflex was normal. She was irritable with a GCS of 15. Following this, an urgent NCCT brain showed symmetrical hypodensities in bilateral frontal, medial occipital cortical & subcortical white matter, and bilateral putamina with effaced cortical sulci suggestive of toxic leukoencephalopathy. NCCT cervical spine was normal. As part of a further neurological evaluation, an electroencephalogram (EEG), NCS, and MRI brain was done. EEG was suggestive of a normal drowsy profile, but interestingly NCS showed low amplitude in the right median and ulnar nerves, nonrecordable compound muscle action potential in the left median, ulnar, bilateral tibial, and peroneal nerves, and decreased conduction velocity in both sides' ulnar nerves suggestive of motor axonal polyradiculoneuropathy affecting both upper and lower limbs. MRI brain showed hypointensities in bilateral putamen, caudate nucleus, frontal cortical, and subcortical white matter in T1-weighted (T1W) and hyperintensity in the same areas in T2-weighted sequences (Figs 1 and 2). Susceptibility-weighted imaging (SWI) showed an abnormal hyperintense area with central areas of blooming of bilateral putamina and adjoining external capsule

regions, which was suggestive of hemorrhagic necrosis (Fig. 3). MRI findings were also suggestive of toxic leukoencephalopathy NCS findings were also attributed to hand sanitizer ingestion. Rheumatoid factor (RF) and antinuclear antibody (ANA) were negative. A working diagnosis of toxic leukoencephalopathy with motor axonal polyradiculoneuropathy was made, and the patient was managed with supportive care. After discussion with the patient's family, an elective percutaneous tracheostomy was done, given the anticipated prolonged ventilation. She was weaned from ventilatory support and subsequently transferred to a high-dependency unit. Her motor power improved to 2/5 in bilateral upper limbs and 3/5 in bilateral lower limbs, with sluggish deep tendon reflexes in bilateral upper and lower limbs.

She was discharged home with a GCS of 7/15 (E4VTM2).

Around a 1-month post-discharge, over a telephone conversation, the patient was doing well with no further improvement in motor power. No adverse or unanticipated events have been reported post-discharge till now. All procedures performed in this study were in accordance with the ethical standards of the Institutional and/or National Research Committee(s) and with the Helsinki Declaration (as revised in 2013).

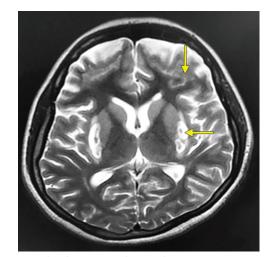


Fig. 2: T2-weighted (T2W) MRI showing hyperintensities suggestive of toxic encephalopathy (yellow arrows)

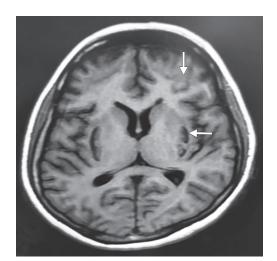


Fig. 1: T1-weighted (T1W) MRI showing hyperintensities suggestive of toxic encephalopathy (white arrows)

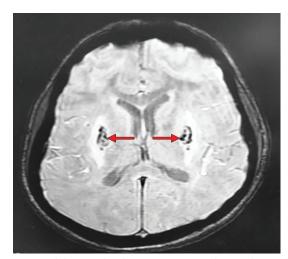


Fig. 3: Susceptibility-weighted imaging (SWI) MRI showing hemorrhagic necrosis in bilateral putaminal (red arrows)

DISCUSSION

As per the Centers for Disease Control and Prevention reports, alcohol-based hand sanitizer formulations containing either 80% ethanol or 75% isopropanol act to inactivate the SARS-CoV-2 virus. Hand hygiene with sanitizers plays a pivotal role in reducing the transmission of COVID-19.³ Due to easy availability, it has become a potential agent for either intentional suicidal or unintentional accidental ingestion in children during this pandemic.⁴

Our patient, during her stay in ICU, developed leukoencephalopathy with polyradiculoneuropathy. Differential diagnoses considered were leukoencephalopathy secondary to hand sanitizer, compressive myelopathy, Guillain-Barre syndrome, and vasculitic neuropathy. She had no history of fever, diarrhea, any viral infection, or trauma. Her initial motor examination was normal, and the symptoms were acute in nature. MRI brain and spine were also normal. RF and ANA tests were negative. Hence, we managed her as a case of toxic leukoencephalopathy with polyradiculoneuropathy secondary to the ingestion of a large volume of hand sanitizer. Ethanol was the predominant component of the hand sanitizer. Although we cannot rule out the effects of other components of the hand sanitizer, we believe ethanol to be the most probable cause of the patient's clinical features. Glycerol is a nontoxic compound, and hydrogen peroxide is toxic only at a concentration of >35%.⁵

The toxic BAC is around 150–250 mg/dL, while a concentration of 400 mg/dL or more is often fatal, even though death has happened at the lower range.² The fatal medical consequences of acute ethanol intoxication include hypothermia, respiratory depression, hypotension, cardiac arrhythmias or arrest, lactic acidosis, acute liver injury, and acute kidney injury.^{2,6} However, neurological manifestations are uncommon following such intoxication. Although our patient had a BAC of 280 mg/dL, we suspect that actual concentrations at the time of intoxication would have been much higher. This may be the reason for such extensive neurological involvement.

Mahajan et al. reported a case of combined isopropanol (70%) and ethyl alcohol intoxication in a 60-year-old male who presented with headache, dizziness, and disorientation postconsumption.⁷ Although immediate NCCT brain was normal, MRI of the brain and spine during the course of the hospital stay revealed T2-weighted (T2W) and diffusion-weighted images of hyperintensities involving bilateral cerebral and cerebellar cortices and white matter, basal ganglia, thalami, brainstem and cervical spinal cord with cerebellar tonsillar herniation. The patient was treated with symptom-directed supportive care.

We would like to highlight a few salient points of difference compared to the above case reported by Mahajan et al.⁷ Compared to their case, where the patient had an intake of 473 mL of isopropanol along with ethanol, our patient consumed a much lesser quantity (250 mL) of ethanol-based hand sanitizer. Even though there was severe high anion gap metabolic acidosis, there was no hyperlactatemia, hyperkalemia, and acute kidney injury, mandating urgent renal replacement therapy in our case. Secondly, although neurological involvement in their case was characterized by findings as mentioned above, our case had extensive brain involvement. Thirdly, our patient had polyradiculoneuropathy, as evidenced by abnormalities in NCS, which makes this case a unique one.

Two more cases of ethanol-based hand sanitizer consumption have been reported by Hanna et al. and Stevens et al.^{4,8} However, such severe neurological manifestations were not reported by them. Hand hygiene and alcohol-based hand sanitizers are a crucial part of infection control in today's era. Our case highlights that patients with psychiatric disorders can ingest sufficient doses to bring about lethal intoxication. Neurological manifestations evident on MRI and NCS following ingestion of alcohol-based hand sanitizers have been reported in a limited number of cases.⁹ To the best of our knowledge, this is the first case report with such extensive involvement of the brain evident on MRI along with polyradiculoneuropathy after acute intoxication of hand sanitizer.

CONCLUSION/**H**IGHLIGHTS

Hand sanitizer ingestion in adults may have varied presentations from asymptomatic to lethal. Neurological presentations like leukoencephalopathy with polyneuropathy may be likely sequelae following acute hand sanitizer poisonings, as seen in our case. Vigilance should be exercised over access to ethanol-based hand sanitizers, particularly in children, adults with psychiatric disorders, and patients with a history of substance dependence. This case report emphasizes the importance of hand sanitizer ingestion as a differential diagnosis for patients presenting with neurological manifestations in the era of COVID-19.

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