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Case Report

# Toxic Leukoencephalopathy Presenting with Neuropsychiatric Manifestations-A Case Report

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## Abstract

A 70-year-old male, with alcohol and nicotine dependence presented to casualty in a state of altered consciousness after accidental overnight LPG gas exposure. Over the next 2 weeks he had residual fatigability, discontinued his work, and remained abstinent from substances henceforth, otherwise was asymptomatic. In the 3rd week, he started to develop confusion, disorientation, irritability, urinary incontinence, decreased self-care, aggression and reduced oral intake. Over the next 2 days developed catatonic symptoms. Routine investigations found within normal limits. MRI-Brain revealed features suggestive of toxic leukoencephalopathy. Minimal improvement in catatonic symptoms was noted with Lorazepam. Nootropics, Steroids, Antioxidants and Multivitamins were added along with supportive care. Over the subsequent two weeks completely was bedbound, his Glasgow coma scale score deteriorated to E4V2M3. He succumbed to death.

**Keywords:** Toxic Encephalopathy; Liquid Petroleum Gas; Neuropsychiatric Manifestations

## Introduction

Hereby presenting a case of 70-year-old married male with no formal education belonging to low socioeconomic status welder by occupation. On 16th August 2022, there was an accidental Liquefied Petroleum Gas leak at his residence while he was asleep with all the windows and doors for an approximate duration of 10 hours, however the flame of stove was not on. He was found in a state of unconsciousness and unresponsiveness by the neighbours and was brought to the Emergency Department and received 2 days of In-Patient care, the details of which are not available. The family members report persisting fatigability as a result of which he was unable to go to work. He abstained from alcohol and nicotine thereafter, otherwise remained asymptomatic. He was brought to the Psychiatry Department in the 3rd week following LPG exposure with history of confusion, inability to identify family members, reduced verbal output, slowing of movements, refusal to take food, provoked and unprovoked irritability, physical aggression, poor-selfcare, urinary incontinence, impaired activities of daily living since 5 days.

History of chronic alcohol use for 30 years, daily drinker, consumed around 3 units daily. History of chronic nicotine use in the last 30 years, smoked 1 to 2 packets bidi per day. No history of prior abstinence period or prior complicated withdrawal.

Patient had no medical co-morbidities. No history of trauma to head. No significant history of any psychiatric and neurological illness in the family.

Vitals were stable.

Add on examination: Conscious, irritable, staring look present, pupils reactive, unresponsive to simple commands, emotionally unresponsive, mutism, posturing, negativism, muscle resistance noted.

Cognitive tests could not be performed.

Bush-Francis Catatonia Rating Scale revealed a score of 18.

Complete Blood Count, Renal Function Test, Liver Function Test, Serum electrolytes, Vitamin B12, Creatinine Phosphokinase, and anti-Thyroid Peroxidase were found to be within normal limits. Hepatitis C Viral marker, Human Immunodeficiency Virus, Hepatitis B Antigen titre, and VDRL were Non-reactive.

MRI-Brain revealed diffuse symmetric confluent subcortical and deep white matter T2/FLAIR hyperintensity in bilateral frontal, parietal, temporal and occipital lobes -features suggestive of toxic leukoencephalopathy. Age related neuroparenchymal atro-

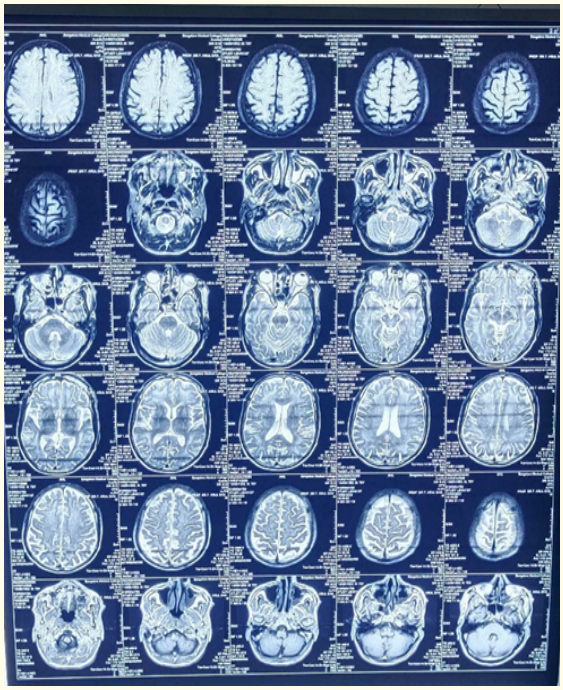

**Figure 1**

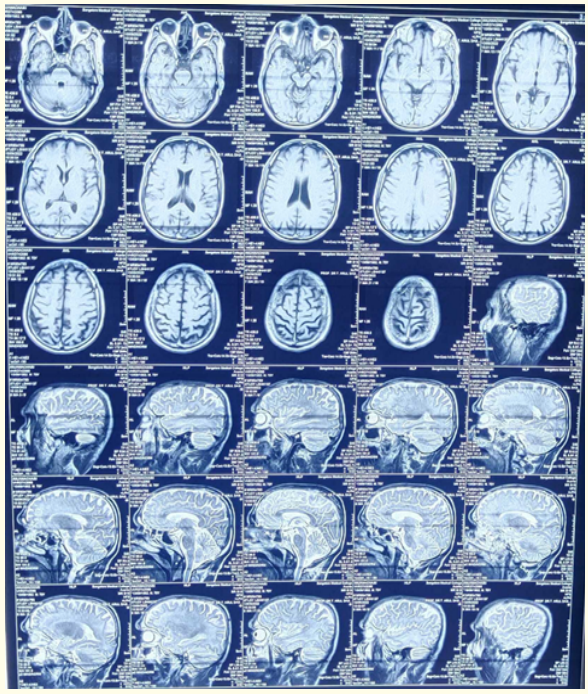

**Figure 2**

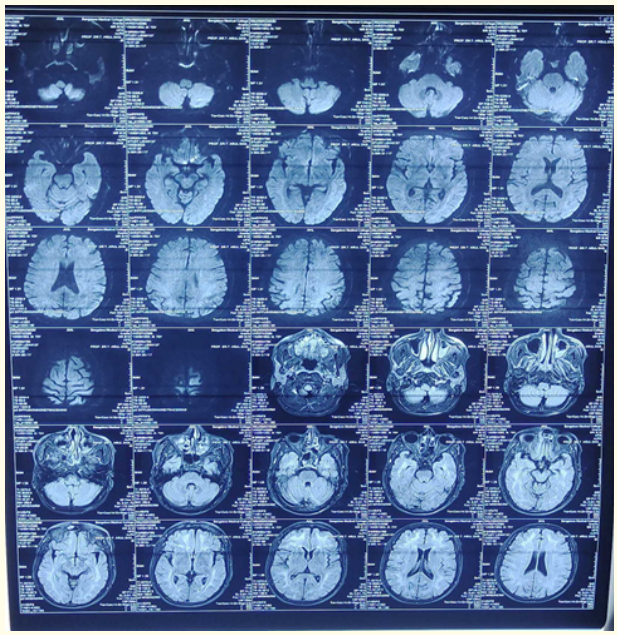

**Figure 3**

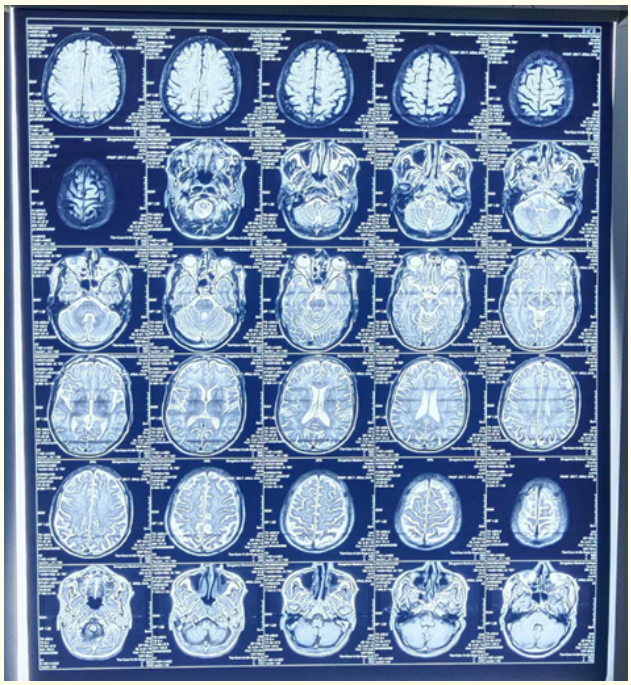

**Figure 4**

**Figure 1-4:** Images of MRI-BRAIN.

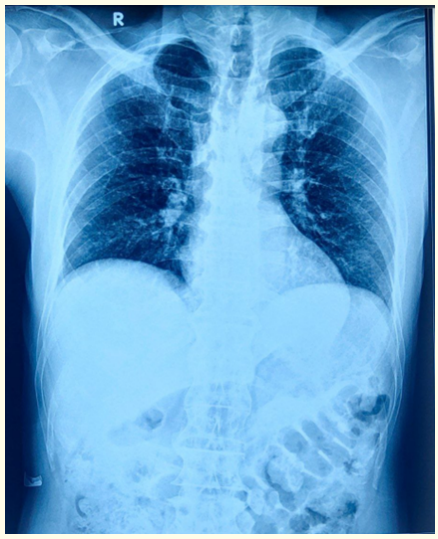


**Figure 5:** Image of chest X-ray.

phy. Small vessel ischemic changes (Fazekas Grade 1).

Chest X-Ray showed reticular opacities noted in B/L mid and lower lung zones.

ECHO-Sclerotic Aortic valve, mild Aortic Regurgitation with Left Ventricular Ejection Fraction of 61%. No resting regional wall motion abnormality seen. No effusion.

Doppler- Trivial Tricuspid Regurgitation, Mitral Regurgitation. Grade 1 Aortic Regurgitation.

Minimal improvement in catatonic symptoms noted with Lorazepam. Neurology opinion was sought and a 3 day course of Intravenous Methylprednisolone 1gm in 100ml Normal Saline, Nootropics-Intravenous Piracetam 1 ampoule QID for 5 days, Intravenous Multivitamins, Tablet Coenzyme Q 500mg BD, Tablet Amantadine 100mg BD, Tablet Pyridoxine 20mg OD given. Ryles tube feeds were given. Bladder was catheterized.

Gradual deterioration in Glasgow Coma Scale score from E4V4M5 to E4V2M3 observed over the next 4 days.

He spiked high grade fever in the second week of admission, elevated White Blood Cell counts noted, showed good response to Intravenous Piperacillin and Tazobactum 4.5 gm TID. Patient was discharged at attender's request. GCS continued to be E4V2M3.

He succumbed to death at his residence after a week of discharge i. e, 5 to 6$^{th}$ week post- LPG exposure.

## Discussion

Liquefied Petroleum Gas mainly contains Propane and Butane gas, Mercaptans are added to make leakage easily detectable. It is a colourless, odourless, highly inflammable gas which can readily evaporate into air. It is widely used both for industrial and domestic purposes.[1]Data regarding cause of LPG related deaths cited as asphyxia secondary to inhalation of asphyxiants like Carbon Monoxide, Sulphur Dioxide, Nitrous Oxide which are released due to incomplete combustion of LPG. The oxidant effects of methyl mercaptans is found to cause hemolysis especially among G6PD deficient individuals. In this case report the patient was exposed only to LPG and not to its combustion byproducts, literature reveals ini-

tial exposure causes mucosal irritation and sustained exposure of LPG can cause headache, dizziness, dyspnea, loss of consciousness and rarely cardiovascular arrest. Death due to direct toxic effects on myocytes leading to rhabdomyolysis following prolonged exposure to LPG has also been reported [1,2]. Case report of LPG gas leading to toxic encephalopathy is rarely reported.

Encephalopathy refers to any illness, injury or malfunction of brain, its symptomatology varying from sub-clinical deficits to overt clinical disorders. The significant mechanisms that cause encephalopathy are anoxic, toxic and metabolic. Toxic encephalopathy is a term generally used to indicate brain dysfunction secondary to exposure of substances like toxic chemicals, solvents, illicit drugs, toxins, poisons, paints, certain metals and medications. Though brain is protected by blood brain barrier, it is vulnerable to many toxins such as LPG. The neurons are the primary targets for toxins as they are metabolically highly active [3,4]. A detailed history, physical examination, neurological examination with radiological investigations aids in diagnosing toxic encephalopathy. With the increase in duration of exposure of toxin the severity of neurological symptoms increases. It is said that neurological symptoms are symmetrical in toxic encephalopathy. The sequelae following toxic exposure persist for a longer time even upon removal of toxic agents as the brain has limited regeneration capability [5]. Leukoencephalopathy is a structural alteration in the cerebral white matter, also caused by toxins resulting in neurobehavioral disturbances, symptoms ranging from minor memory loss, attention deficits, fatigue, confusion, subtle personality in mild disease to abulia, akinetic mutism, stupor, coma, death and severe global environment. Toxic leuckoencepalopahty following LPG exposure is rarely reported.

## Conclusion

The effect of exposure to Liquid Petroleum Gas, leading to permanent structural and irreversible damage to the brain is presented in this case report. This case report thus adds to the existing limited literature and lays emphasis on the importance of detailed history and detailed examination in arriving at diagnosis.

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