Introduction

LPG (liquefied petroleum gas) is a commonly used commercial and domestic fuel. Due to its common usage, unintentional prolonged exposure to LPG poses a potential health hazard to the general population, ranging from mild irritation to life threatening sequelae. Usually, LPG inhalation presents with complications like hypoxia, cardiorespiratory arrest and neurological complications as reported in earlier studies. A very rare case of accidental prolonged LPG inhalation involving three members of the same family with varied sequelae is being reported. Two family members, the child and his mother made a complete recovery while the father expired. Autopsy conducted on the latter confirmed asphyxia as the possible cause of death. Out of the three, only the mother developed massive rhabdomyolysis. Apart from this, only three cases of massive rhabdomyolysis after LPG exposure have been reported earlier. No such presentation has been reported in hitherto published Indian literature of LPG exposure.

Case Report

Acute Massive Rhabdomyolysis Due to Inhalation of LPG

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Abstract

LPG (liquefied petroleum gas) is a commonly used commercial and domestic fuel. Due to its common usage, unintentional prolonged exposure to LPG poses a potential health hazard to the general population, ranging from mild irritation to life threatening sequelae. Usually, LPG inhalation presents with complications like hypoxia, cardiorespiratory arrest and neurological complications as reported in earlier studies. A very rare case of accidental prolonged LPG inhalation involving three members of the same family with varied sequelae is being reported. Two family members, the child and his mother made a complete recovery while the father expired. Autopsy conducted on the latter confirmed asphyxia as the possible cause of death. Out of the three, only the mother developed massive rhabdomyolysis. Apart from this, only three cases of massive rhabdomyolysis after LPG exposure have been reported earlier. No such presentation has been reported in hitherto published Indian literature of LPG exposure.

Case History

A 30 years old female, non-smoker, non-alcoholic, housewife presented with history of generalised body ache since getting up from bed. There was history of exposure to LPG for the past eight hours during sleep caused by a leaking gas tube with the flame switched off. The pain was described by the patient as diffuse, dull aching type, poorly localised and without any associated swelling. It increased on moving the limbs and on applying pressure. She did not have any focal weakness except for limitation of body movement due to pain. There was no history of fever, altered sensorium, trauma, seizures, burns, alcohol or drug intake. There was no history of any urinary or fecal incontinence. There were no apparent relieving factors. Over the next few days during hospitalisation, the patient experienced gradual symptomatic relief in pain and was able to move comfortably by the eighth day.

At the time of presentation to the casualty, she was conscious, oriented, walking with support with much difficulty. Her vitals were stable. Pulse- 80/min, BP- 118/70 mm Hg, RR-16/min. Body temperature was 37.2 degree C. There was no pallor, icterus or cyanosis, JVP was not raised. There were no abnormal systemic findings. Chest, CVS, CNS and per abdomen examination were essentially normal. Severe tenderness was elicited on palpation of muscles all over the body. It increased on applying pressure and on active and passive limb movements.

On investigation, blood counts were within normal limits (Hb-10gm%, TLC-6200, DL-P52 L42 M4 E2, ESR-40, S Bil-0.8 mg/dl, SGPT-42 IU/dl, SGOT-46 IU/dl, ALP-124 IU/dl, blood urea-30 mg/dl, s creatinine-0.8 mg/dl, s uric acid-4.2 mg/dl, RBS-93 mg/dl, s Na-140 meq/l and s K-4.8 meq/l). Repeat serial blood counts including daily RFT did not show any abnormality. ABG revealed mild metabolic alkalosis (pO2 92, pCO 2 36, pH 7.48, SaO2 96%). The most significant finding was a CPK value of 11,350 IU/dl at the time of admission, the normal range being 30-170 IU/dl. Repeat serial CPK level done every other day, showed values of 10,355, 3670, 281 and subsequently 52 IU/dl. Urine routine and microscopy was normal. Urinary myoglobin was found to be positive and cardiac troponins were negative. Chest radiograph and ECG were normal. The patient was treated symptomatically with intravenous saline infusion. She did not develop any complications of rhabdomyolysis and was discharged on the ninth day of hospitalization.

The patient’s husband and son also had history of similar and simultaneous exposure to LPG. The family lived in a small single
room ill-ventilated hutment as elicited on history. They were all sleeping on the floor. The husband, possibly under the influence of alcohol, was sleeping closest to the gas stove, which was placed at ground level. Following exposure, the husband expired during sleep and was brought dead to the casualty. Autopsy was conducted on him which revealed congestion of the liver, spleen, kidneys, brain and lungs indicating hypoxia as the possible cause of death.

The patient’s three year old child, who was sleeping between his parents, presented with features of respiratory distress. Examination revealed pneumonitis. He had normal biochemical parameters. The child improved with symptomatic treatment over the next four days. He was subsequently discharged from the hospital and is doing well on follow up.

Discussion

This case report presents a unique case of LPG exposure, perhaps the first of its kind, in published literature in India. As per the literature published elsewhere, initial inhalational exposure to LPG causes local irritation in the nose, eyes and pharynx. Sustained inhalation leads to headache and dizziness progressing to difficulty in breathing, loss of consciousness and cardiorespiratory arrest. The most commonly documented cause of death is hypoxia secondary to inhalation of asphyxiant substances released by incomplete combustion of LPG. These include carbon monoxide, sulphur dioxide, nitrous oxide and total suspended particulate matter. Autopsy findings demonstrate severe pulmonary edema and suggest advanced circulatory failure. Biochemical findings reveal myocardial ischemia and hypoxia. Acute renal failure occurs in 30-40% of patients with rhabdomyolysis, especially in dehydrated patients. Headache, giddiness and rarely coma occur secondary to the neurotoxic effects of these gases especially mercaptans. Hemolysis occurs due to the oxidant effects of methyl mercaptans, especially in G6PD deficient individuals. In most of these cases, there was a history of inadequately ventilated and combustion of LPG leading to the production of asphyxiant gases like carbon monoxide. In this particular case report, the patient’s family inhaled the leaking LPG for about eight hours during sleep in a closed room and not its combustion byproducts as the gas flame was switched off.

The presentation can be attributed to the direct toxic and oxidant effects of butane, propane and mercaptans. This effect has been seen in certain animal studies performed on catfish and hen. The mother presented with features suggestive of acute massive rhabdomyolysis without any respiratory, cardiac or neurological complications. Rhabdomyolysis is a clinical syndrome in which injury to skeletal muscles results in the leakage of intracellular contents from myocytes in the plasma. Out of the several causes of rhabdomyolysis in literature, the most likely etiology is direct muscle injury and drug abuse. The diagnosis of acute massive rhabdomyolysis was based on generalized muscle tenderness, raised muscle enzyme in serum and the presence of myoglobin in urine. The clinical presentation of rhabdomyolysis is often subtle, requiring a high index of suspicion. The cause of death in such cases includes acute renal failure, hyperkalemia, acute cardiomyopathy, DIC and various other complications. Failure to recognise rhabdomyolysis on initial presentation and delay in institution of early and aggressive volume replacement are the major contributing factors in fatal cases. Our patient made a complete recovery without any sequelae. This was confirmed both clinically and biochemically. This can be attributed to prompt diagnosis and intensive symptomatic therapy including intravenous crystalloids and urine alkalinising agents. The close proximity of the patient’s husband to the leaking gases along with the possible history of alcohol exposure could have contributed to his death.

Considering the common usage of LPG by all strata of society, there is significant possibility of exposure to small leakages over a passage of time and such accidents are probably underreported. Therefore, it is important that the general population, LPG providers and healthcare professionals all should be made aware of the toxicity and its potential as a health hazard.

References