Acute Lung Injury Following Accidental Exposure to LPG in a Young Adolescent Male

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Abstract
Liquefied Petroleum Gas (LPG) a common domestic and commercial fuel and being in worldwide uses, poses potential toxic health hazards. Toxicity due to LPG exposure varies between the clinical spectrum of mild symptoms to life threatening event in form of cardiac, respiratory and neurological complications as reported in literature. We presenting a case of young adolescent with accidental exposure to LPG presented as acute lung injury.

Keywords: Liquefied Petroleum Gas (LPG), acute lung injury.

Introduction
LPG a mixture of aliphatic hydrocarbon gases used as domestic fuel and in vehicles, available in pressurized cylinders. Primarily constituted by butane (80%) and propane (20%) which are inflammable gases and propylenes and butylenes added in small concentrations. As precautionary measure mercaptans are added as it has malodorous smell to detect any leakage. Various case reports with different clinical manifestations have been reported in literature¹². In present case report authors describing a case of an accidental inhalation of LPG associated with acute lung injury in adolescent male.

Case Report
A 18 year old young adolescent male presented with shortness of breath developed while entering bathroom for taking bath with history of exposure to LPG due leakage in the heating system left partially open. Patient complained of difficulty in breathing, dry cough and palpitations. Patient denied any previous history of fever, cough with expectoration, altered sensorium, seizures or drug intake.

On presentation patient was conscious able to walk with support. Examination revealed PR 120/min, BP -110/70 mmHg, RR- 28/min with normal body temperature. There was no pallor, icterus, cyanosis, peripheral edema. Systemic examination revealed fine crepitations on auscultation of chest, tachycardia on cardiac examination, rest was normal. Investigations showing TLC-8000 cells/µL, N54, L40, M2, E4, B0, LFT and RFT were within normal limits.ABG analysis showed PaO2 80% on room air, PCO2
38, PH-7.38, Spo2 86%. Chest roentgenogram PA view revealed B/L non homogenous opacities predominantly in mid and lower zones, ECG showed sinus tachycardia with generalized ST segment changes. Patient was managed with Oxygen therapy via venturimask @ 8-10L/min, propped up position, Nebulization with bronchodilators and IV Antibiotics. Patient started maintaining O2 saturation on room air after 48 hours and ECG changes also settled after same time period. Patient was discharged on 4th day.
Discussion
The initial exposure to LPG inhalation is associated with local irritation of nose, pharynx and eyes. Prolonged exposure leads to dizziness, headache and progressive shortness of breath, altered sensorium and cardio respiratory arrest. The documented causes of hypoxia has been substances released on incomplete combustion of LPG mainly carbon monoxide, Sulphur dioxide, Nitrous oxide and suspended particulate matter. Pulmonary edema and advanced circulatory failure has been demonstrated on autopsy with biochemical analysis revealing myocardial ischemia and hypoxia. Rhabdomyolysis with renal failure has also been reported\(^1\). Giddiness, headache, severe ataxia and reduced level of consciousness secondary to neurotoxic effects of gases released on incomplete combustion of LPG especially mercaptans. These hydrocarbons being lipophilic enter into circulation after inhalation and are responsible for CNS manifestations. Oxidant effects associated with methyl mercaptane can cause hemolysis particularly in G6PD deficient patients. Most of the cases reported previously were associated with history of inadequate ventilation and combustion of LPG but in our case there was exposure of LPG leakage\(^1,2\).

There are reports in literature that pure form of LPG is associated with various complications, CNS effects, Arrhythmias and Rhabdomyolysis has been reported\(^4\). Despite having low toxic potential the constituent gases of LPG- propane, butane and mercaptane can cause damage due to oxidant and direct toxic effects as in our case acute lung injury and arrhythmia was the main presentation. Cardiovascular mortality associated with butane is due to enhanced sensitization of myocardium to effects of catecholamines predisposing to life threatening arrhythmia including Ventricular Tachycardia. Hydrocarbons can cause inflammation, pneumonitis and edema in lungs\(^1,4\).

References