Hydrocarbon Associated Toxicities: a Case Series and Review of Literature

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Abstract

Background: Hydrocarbon associated toxicity (HAT) is an emerging threat related to wide scale industrialization and easy access to hydrocarbon-containing chemical compounds. Hydrocarbons have a unique toxicological profile and the principles of managing related toxidromes are considerably different from other toxins.

Case reports: Here, we present a case series and in-depth review of the existing literature to show the risks associated with these seemingly harmless chemicals, and the approved guidelines for treating exposed patients. In all three cases, the hydrocarbon was a diluent for a pesticide. The amount of pesticide ingested was nontoxic, while the hydrocarbons caused a dose-independent physical toxicity to the lungs.

Discussion: Hydrocarbon associated toxicities often go unnoticed because of their usage as diluents for various other toxic chemicals. Their treatment usually differs from other toxins that may have been consumed with them, albeit in insignificant quantities.

Conclusion: Recognition of a hydrocarbon diluent in a consumed toxin marks the first step in the correct treatment. Correct labeling of chemical solutions containing hydrocarbons would go a long way in identifying these toxics.

Keywords: Hydrocarbon Toxicity; Kerosene; Pesticide; Toluene; Turpentine

INTRODUCTION

Hydrocarbons are a class of organic chemical compounds composed only of the elements carbon (C) and hydrogen (H). The carbon atoms join together to form the framework of the compound, and the hydrogen atoms attach to them in many different configurations. Hydrocarbons are the principal constituents of petroleum and natural gas (1).

With the advent of the industrial age, the human-hydrocarbon interaction has become inevitable. Their versatility in utility has made them ubiquitous. From the fuel in our cars to the oil in our food, we are surrounded by hydrocarbons. This omnipresence has also made us vulnerable to their toxic effects. Whether ingested, inhaled, injected or applied, they can have toxic effects. Keeping these facts in mind, we present this case series to highlight the toxicity of these easily accessible, yet poorly understood poisons.

CASE SERIES

Patient 1

A 35-year-old male presented to the Emergency Department (ED) with a history of consuming one whole container (~45 ml) of the mosquito repellant All Out™ one hour prior. On presentation, the patient was conscious and oriented but agitated. Vital signs were all within normal limits. Considering the contents of the ingested toxin (transfluthrin, toluene, and kerosene), gastric lavage was withheld. The patient was given intravenous (IV) Lorazepam 2 mg to control the agitation. Patient was discharged in good health after 48 hours of observation.

Patient 2

A 16-year-old female presented to the ED with history of multiple episodes of vomiting four hours after intentional ingestion of less than half a cup (~100ml) of Kuber extra strong bed bug killer™ spray. On presentation, the patient was drowsy with signs of mild dehydration. IV crystalloids were started. The family had brought the bottle of the ingested insecticide to the ED, which listed the contents as alkaloids, lactone, and 92% other chemicals. The manufacturer was contacted and reported that the 92% was paint thinner (turpentine oil) with 1% pyrethroids. No gastric lavage was done. All vital signs remained stable post fluid resuscitation. Patient was kept in a semi-recumbent position throughout the duration of the hospital stay and was discharged in good health after 48 hours of observation.
health after three days of observation.

**Patient 3**
A 31-year-old female was brought to the ED by her husband who reported that after an argument she had ingested ~50 ml of Kuber extra strong bed bug killer™ spray one hour prior. The patient had persistent vomiting. She was kept in a semi-recumbent position and managed conservatively with anti-emetics and IV fluids. Hospital stay was uneventful and the patient was discharged on day 3 in good health.

Baseline electrocardiography was performed for all patients and was reported to be normal.

Chest X-rays were done on presentation and after 24 hours of hospital stay, in all of the cases, to look for signs of chemical pneumonitis. They were all reported to appear normal.

Psychiatry referrals were sought and medico-legal procedure was followed in all the cases.

All patients were reportedly healthy on telephone follow up (Table 1).

### DISCUSSION

**Epidemiology**

Hydrocarbon toxicity is a well-documented phenomenon across the world. The American Association of Poison Control Centers in a 2017 report listed a total of 17,304 reported cases of hydrocarbon associated toxicity (HAT) with 35 reported deaths(2). While HAT used to be a significant problem in the middle east, (3) the trend has seen a decline post economic growth and modernization. However, the same cannot be said about India. While there is a paucity of compiled national data, as per the National Poisons Information Centre, New Delhi reports that the most common poison exposures involve household agents (44.1%). Of these, chlorinated hydrocarbons were listed in the top six (4). There is also evidence to suggest that chronic occupational exposure to hydrocarbons can lead to a myriad of pathologies secondary to oxidative stress(5).

This data correlates with a local cross sectional study conducted in the city of Pune where household and agricultural agents were listed as the most common reported toxin (56.4%)(6).

<table>
<thead>
<tr>
<th>Table 1. The abstract of three patients</th>
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<tbody>
<tr>
<td>Ingested substance</td>
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<tr>
<td>All Out™ mosquito repellent</td>
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<tr>
<td>Hydrocarbon diluent (%)</td>
</tr>
<tr>
<td>Blood pressure (mmHg)</td>
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<tr>
<td>Pulse (beats/minute)</td>
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<tr>
<td>SpO2 on room air</td>
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<td>Gastric lavage performed</td>
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<td>Outcome</td>
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This is especially of concern among the pediatric population which can accidentally ingest these compounds. According to a study by Christian Medical College, Vellore hydrocarbons were listed as the most commonly reported toxin involved in pediatric exposures(7).

It appears that the increase in incidence of hydrocarbon associated toxicities and industrial growth have gone hand in hand. Unsurprisingly, the earliest case reports of hydrocarbon exposures can be traced back to 1897(8). A factor that has contributed to this problem has been the advent of the agricultural age and the easy availability of pesticides, many of which use hydrocarbon diluents (9). While vector borne diseases have always plagued our country, economic growth has led to drastically increased use of insect repellants and pesticides in India. (10) Since a large number of these products use hydrocarbon base, an increase in the number of HATs has mirrored the increase in the usage of these products.

An emerging concern for toxicity is inhalation of toluene-based compounds for recreational purposes which is most commonly being seen among young children and adolescents (11).

**CLINICAL PRESENTATION**

HAT most commonly happens when the route of exposure is ingestion or inhalation (12) although toxic manifestations via dermal and sub dermal exposure have also been reported. (13)(14) The effects depend on the route, amount and type of hydrocarbon. A high incidence of chemical pneumonitis has been reported in cases of hydrocarbon toxicity and it is considered a major contributor to the associated morbidity and mortality(15).

Short chain aliphatic hydrocarbons are notoriously associated with cardiac toxicity, specifically ventricular fibrillation(16). Of special note is Sudden Sniffing Death Syndrome, which is a hyperacute catecholamine surge leading to ventricular arrhythmias and sudden cardiac death(17). Systemic absorption can lead to central nervous system depression presenting as altered mentation and even coma, although paradoxical neuro-stimulation leading to hallucinations, tremors and seizures have also been reported (8). Peripheral nervous demyelination and retrograde axonal
degeneration have been associated with certain aliphatic hydrocarbon (18). Chlorinated hydrocarbons have been associated with fulminant hepatic failure (19).

**Treatment**

**Decontamination**

There is no role for gastric lavage in pure HAT, as the risk of aspiration leading to chemical pneumonitis far outweighs any benefit that it may achieve (20). The exception to this exists in cases of co-ingestions in which one of the other agents is a pesticide. Consulting the local/national toxin center in such situations is advised. In all cases, activated charcoal is strictly contraindicated (21).

**Management of systemic complications**

It is advisable to follow the Airway-Breathing-Circulation (ABC) approach which amounts to the airway taking precedence over everything else. The airway can be threatened because of thermo-chemical irritation caused by the hydrocarbon compounds. Oxygen administration is advised to treat hypoxia and positive pressure ventilation may be used if the situation demands for it. Bronchoconstriction may be treated with inhaled beta₂ agonists (22). Certain studies have recommended extracorporeal membrane oxygenation(23) and/or surfactant therapy(24) in cases with acute lung injury. However, the cost and availability of these therapeutic options may limit their usage in the Indian setting. As of now, no guidelines exist which recommend prophylactic usage of antibiotics or steroids.

Aggressive intravenous fluid therapy should be used to treat hypotension and the use of catecholamines should be avoided because of the risk of inducing arrhythmias. In case of ventricular dysrhythmias, anti-arrhythmics belonging to class la and class III should not be used to avoid the risk of QT prolongation. Instead Esmolol, Lidocaine and Propranolol are the recommended anti-arrhythmics (25). Seizures may be treated with benzodiazepines. Phenytoin must be avoided (21). Since nausea and vomiting are some of the most common symptoms that patients tend to present with, anti-emetics may be used for symptomatic relief.

**CONCLUSION**

The role of gastric lavage is a grey area of understanding where clinical gestalt has a major role to play. It is essential to consider the diluent as a toxin especially in pesticide poisonings where the latter might not even be the real threat as is evident from our cases. Psychiatric interventions play a pivotal therapeutic role in intentional toxic exposures. Strict laws need to be implemented regulating the correct labeling and sale of insecticides and pesticides. The struggle for correct identification of the toxin highlights the need for establishment of more toxin information centers across the country. We suggest that institutes with academic Emergency Medicine departments should have wider access to online toxicological databases.

**Conflict of interest:** None to be declared.

**REFERENCES**


