Case Report

Paint It Red! – The Terpenes Reporting

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Abstract

Turpentine is an organic solvent composed of a hydrocarbon mixture of terpenes derived from pineoil. As solvent, it serves as a thinning component for oil - based paints. Available literature exhibits few case reports of turpentine poisoning and fatal cases even more so.

We present a case of a 56 - year old female referred to Emergency Department with decreased sensorium, pin point pupils, smell of kerosene, frothing, Non-ST-Elevation Myocardial Infarction (NSTEMI), ejection fraction of 38%, bilateral creptations, right pneumothorax, acute pulmonary edema with laboratory investigation yielding low pseudocholinesterase levels and with toxicology panel positive for organophosphorus leading to sepsis with shock and renal failure. This case was gaining special interest with suspicion towards organophosphorus but not responsive to the usual line of treatment and thereby facing a diagnostic challenge. Autopsy revealed scalp contusion over parietal lobe, atherosclerotic changes of coronaries, fulminant hepatic changes, loss of cortico - medullary differentiation with perceptible odour of solvent from stomach contents. Inquiry into the case history grew as an empty bottle of paint thinner was discovered adjacent to the deceased. Qualitative methods of analysis detected the presence of 'The Turpentines'.

This case addresses few of the challenges faced when a natural ailment concurrently co - exists with a possibility of an unknown toxic substance and would require an open link to integrate departments to conclusion.

Key words:Organophosphorus; Paint thinners; rare fatality; Turpentine

Introduction:

A substance is considered to be poisonous if it damages or injures the body and poses a risk toone's life through ingestion, inhalation, or contact.[1]All around the world numerous agents such asenvironmental agents, agrochemicals or drugs are used as poisoning agents.[2] One of the major causes of mortality and morbidity on a global scale is intentional poisoning.[3]

According to the World Health Organization (WHO), 0.3 million individuals every year die as a result of various poisons.[4]Poisoning is a threat that has grown over the past ten years, not merely in India but all over the world.[5] In India, the majority of poisoning cases are caused by pesticides.[6] India has one of the highest rates of pesticide poisoning due to the country's extensive usage of pesticides for domestic and agricultural purposes. Other poisoning agents include medications, envenomation, and home products. Pesticides and medications used inagriculture, or the home are typically consumed intentionally, but animal bites and the ingestion of corrosives, kerosene, and other random substances tend to occur accidently.[7,8] The occurrence of turpentine poisoning is quite uncommon and is rarely reported.

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In few plants and pine trees, terpenes are found naturally. When the trees are tapped, an oleoresin is obtained as a sticky yellow gum. A volatile oily fraction known as turpentine is produced after the oleoresin is distillated.[9]

Monoterpenes (-pinene, -pinene, and 3-carene) make up the majority of turpentine, an oily resin that is obtained from a variety of pines.[9,10] Turpentine oil is a distillate of pine gum and, together with pine oil, it is a member of the group of hydrocarbons known as cyclic turpenes or turpene derivatives. Turpentine oil is colourless, caustic, and has a powerful, bitter taste.[11] It is a hydrocarbon derivative that is used in paint solvents, shoe polish, floor polish, waxes, varnishes, insect repellents, disinfectants, printer ink, adhesives, shampoos colognes, and as well as catarrh treatments, carminatives, and anthelmintic medicines for therapeutic purpose.[10-15]

Case Profile:

Hospital stay: We present a case of a 56 year old female referred to Casualty, Kasturba Hospital, Manipal with chief complaints being altered sensorium since 1 day. The provisional diagnosis was made as acute coronary syndrome – NSTEMI with development of acute pulmonary oedema and bilateral crepitations. On admission, patient presented with decreased responsiveness, high GRBS levels, tachypnoea, low saturation ranging from 80 – 90 %, frothing with ECG findings consistent with ST depression and ECHO findings of anterior wall hypokinesia, severe regurgitation in mitral and tricuspid valves, pulmonary arterial hypertension with development of ventricular premature complexes. The signs and symptoms pointing towards a natural entity and hence, the patient was admitted under cardiology.

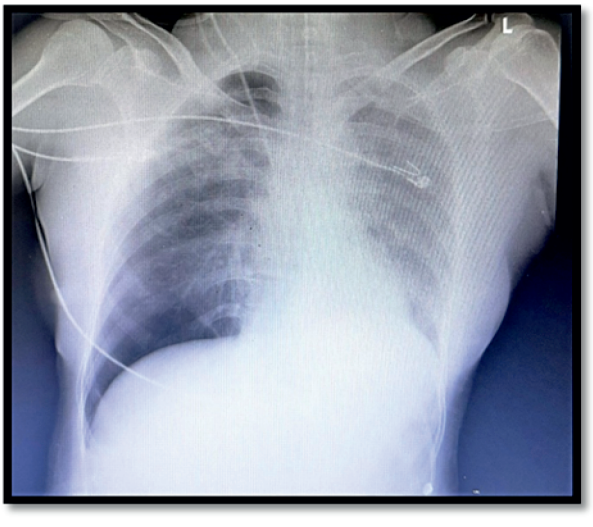
History of presenting illness was revealed to be exertional dyspnoea (on climbing stairs) since 1 week, dyspnoea with cough since 1 day and extreme fatigue. In view of deteriorating vitals, patient was intubated with consultation to medicine.

On day 2 of admission, patient was unconscious, with profuse frothing, incontinence of bowel and bladder, pin point pupils and petroleum like odour and hence police intimation was done and samples were taken for toxicology panel and taken over by medicine.

Investigations:

- Creatinine: 2.12 mg/dl (0.5 – 0.9 mg/dl)
- Sodium: 156 mmol/L (136–145 mmol/L)
- Potassium:3.0 mmol/L (3.5–5.1 mmol/L)
- Troponin–T:1.230ng/ml(upto0.020 ng/m l)
- Procalcitonin: Positive :39.32 mcg/L (<0.5 mcg/L)
- Pseudocholinesterase: 107.0 U/L (5320 – 12920 U/L)
- Urea: 55 mg/dl (10 – 40 mg/dl)
- Toxicology panel: Positive for compounds consistent with organophosphorus
- X-ray: Right mild pneumothorax(Fig 1)

Fig 1: Right pneumothorax with deep sulcus sign



On day 3, the patient succumbed to death and death summary was outlined as organophosphorus poisoning, acute coronary syndrome – NSTEMI, acute kidney injury, hypernatraemia and sepsis with septic shock. However, the case was facing a diagnostic challenge by clinicians as to

organophophorus was not responding to the usual line of treatment.

Autopsy:

The body on autopsy revealed scalp contusion, over left parietal region. (Fig 2)

Lungs were soft, oedematous and congested with varying grades of luminal narrowing in coronaries (Fig 3).

Dissection of stomach revealed perceptible odour of solvent(? Turpentine).

Fig 2: Scalp contusion

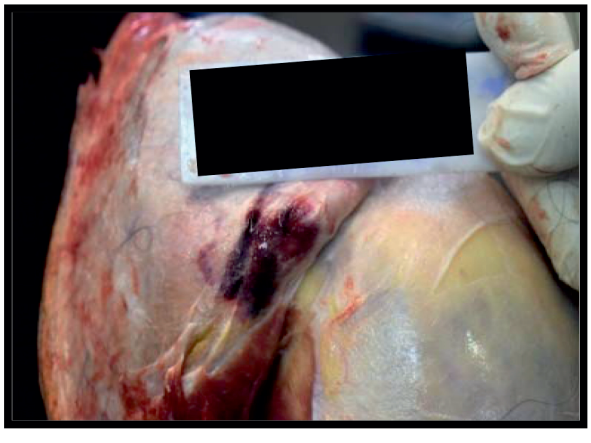
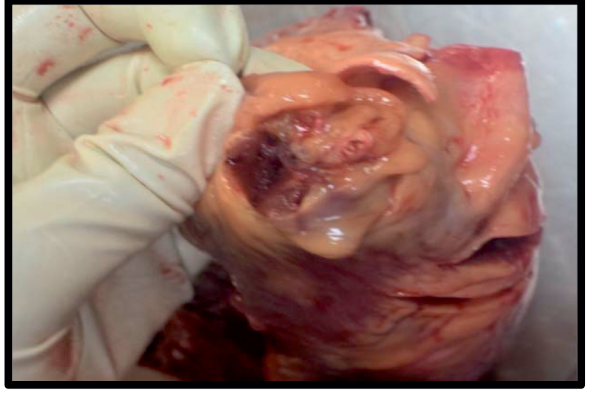


Fig 3: Luminal narrowing of left coronary



Psychological autopsy led to the deceased been discovered in fallen/sleeping state with her body leaning against the left leading to formation of scalp contusion with an empty bottle of paint thinner lying adjacent to the deceased, acess been family members are painters and hold stocks in a room, adjacent to the house.

Histopatholgy revealed lungs with diffuse alveolar damage and pulmonary haemorrhage, acute fulminant hepatitis of liver, acute kidney injury, coronary atherscleroris with features of neuronal cell injury in brainstem.

RFSL confirmed the presence of turpentine in the viscera, blood and control sample of paint thinner recovered from the scene of incidence.

Opinion as to the cause of death: Deceased died due to complications secondary to the consumption of turpentine.

Discussion:

Turpentine is formed from the steam distillation of wood resins, and it consists of hydrocarbons, nitrogen, oxygen compounds and metals. ***Product formulations contain hydrocarbons/petroleum distillates as solvent carriers eg: insecticides, pesticides, paints, wood finishes.***

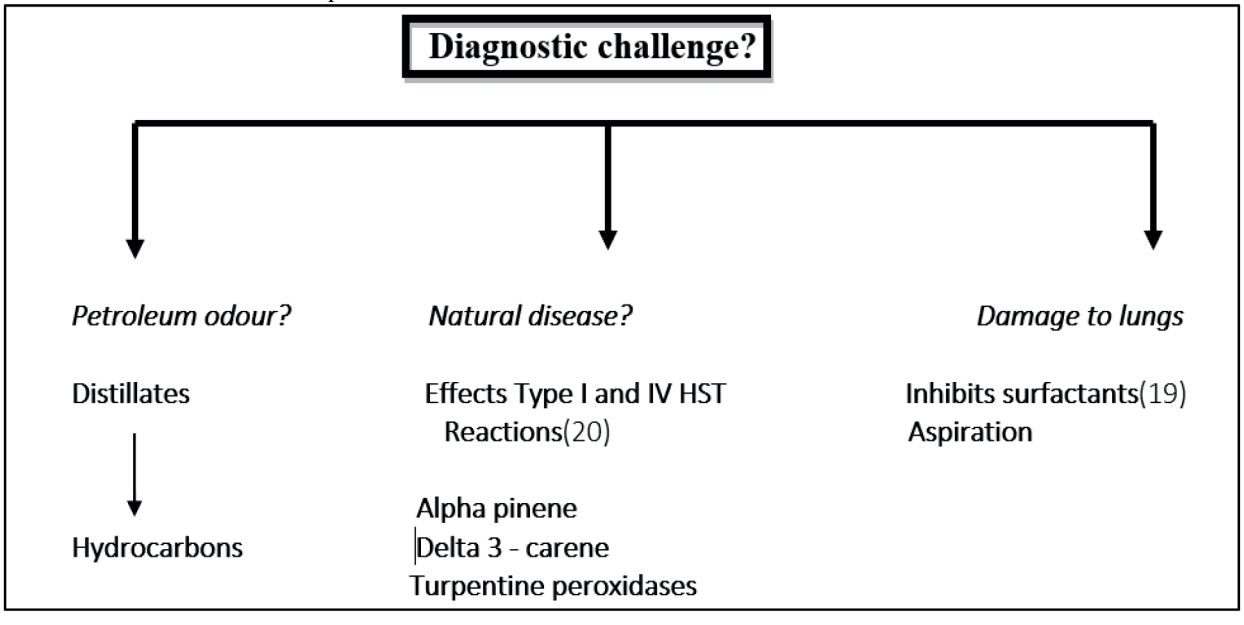
Turpentine's lethal oral dose ranges from 15 to 150 ml on average.[14]Turpentine is easily absorbed by the skin, respiratory system, and gastrointestinal system. Terpenes undergo cytochrome P450-mediated oxidation, are primarily conjugated with glucuronic acid in the liver, and are then eliminated by the kidney. Chronic turpentine exposure increased the activities of uridinediphosphoglucuronosyltransferase and liver microsomal epoxide hydrase. Through the lungs, turpentine may be expelled in its original form. Most of the turpentine and its by-products are excreted as glucuronides through the urinary tract. Turpentine's excretory output has a distinctive violet odour. The three main exposure pathways are cutaneous contact, ingestion, and inhalation.[15]

When consumed orally, turpentine oil can be hazardous when it enters the digestive system or is inhaled and reaches the lungs.[11,13]After it enters the body, clinical effects start to show after 1-2 hours. Burning sensation in the mouth, discomfort in the lips, tongue, throat, and oesophagus, along with thirst, coughing, nausea, vomiting, and diarrhoea, are among the most typical side effects of turpentine ingestion.[11,13]

Other side effects include discomfort urinating, violet-smelling urine, cold skin,

haematuria, dizziness, and drowsiness.[11,12] Aspiration pneumonitis, acute lung injury, pulmonary edema, refractory metabolic acidosis, significant hypotension, liver Supporting respiration and circulation while using a symptomatic approach are typically part of the treatment for people who have been poisoned by turpentine oil.[13] According to American and European toxicology associations, gastric lavage is not recommended after consuming hydrocarbons since it raises the risk of aspiration.[17]The increased number of eosinophils was observed in induced sputum 24 h after turpentine exposure.[18]According to some studies, treating the lung damage caused by turpentine aspiration early on with steroids and prophylactic antibiotics is futile.[16] It must be emphasized that turpentine might result in lethal poisoning due to the fact that turpentine oil is commonly used in cosmetic and household items as well as in industries.[14]

failure, renal failure, convulsions, arrhythmia, and coma are severe poisoning symptoms that can be fatal.[11-13, 16]



Conclusion:

India is primarily an agriculture-based country and we encounter most common poisoning cases to be of pesticides poisoning, whereas when variable ranges of clinical presentations are present, often the most common is most often presumed. As in our case, line of treatment for organophosphorus compounds did not respond, and it is practically a difficult task to differentiate it to turpentine as both share the same hydrocarbon components and petroleum distillates. Number of reported cases of turpentine poisoning are very rareand hence, there is no established protocol for the line of treatment of turpentine poisoning cases. In our case psychological autopsy played a pivotal role in leading to the conclusion of the Terpenes, supported by Regional Forensic Science Laboratory report confirming the presence of turpentine.

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