Toxicity of Organic Solvent in a Young Painter Presenting as Transient Hepatitis: A Case Report

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Abstract

Organic solvents widely used in paint manufacturing, painting, and shoemaking industries may be associated with hepatotoxicity. We present a case report of a patient with prolonged occupational exposure to organic solvents who developed transient hepatitis. Monitoring contact to these chemicals and early identification of biological markers of occupational exposure should be done. More epidemiological studies on the effects of solvents on the liver should be performed so as to help the policy makers to formulate appropriate measures to prevent detrimental effects of exposure of such chemicals. Additionally, early reporting of such cases of occupational hazards will be helpful in further understanding the incidence and possible mechanisms.

Keywords: Hepatitis, hepatotoxicity, occupational hazards, organic solvents, paint, painting

INTRODUCTION

Occupational liver toxicity in workers of industries using solvents-based materials is well known.[1] Most of the organic solvents absorbed are metabolized in the liver, the toxic metabolites thus generated can damage the liver.[1] Some of the occupations where high exposure to organic solvents has been reported includes spray painting, paint manufacturing, shoemaking, and metal degreasing.[2] Liver disease from occupational exposure is rarely suspected/diagnosed;[1] hence, the exact incidence is not fully known. Nevertheless, the hepatotoxic effects of some of the solvents have been recognized as early as 1887.[4] We report a case of hepatitis following exposure to organic solvent. This case report re-emphasizes a potentially serious occupational hazard.

CASE PRESENTATION (PAINT-INDUCED HEPATOCELULAR INJURY IN A YOUNG PAINTER)

A 22-year-old male painter was admitted to hospital with history of colored urine since 1 month, yellow sclera for past 20 days, and abdominal pain on and off for past 3–4 days. No significant medical history was noted. Patient was a painter (occupied in painting the walls and hoardings for past 10 years).

On examination, patient had dense yellow discoloration of skin and sclera, nontender scaphoid abdomen with no organomegaly.

Hematological findings were normal. Biochemical parameters as follows: Random glucose: 75 mg/dl, blood urea: 35 mg/dl, creatinine: 1 mg/dl, sodium: 137 meq/l, potassium: 5.1 meq/l, protein: 7.4 g/dl, albumin: 3.6 g/dl, total bilirubin: 43.00 mg/dl, direct bilirubin: 24.60 mg/dl, indirect bilirubin: 18.40 mg/dl, aspartate aminotransferase (SGOT): 66 U/L, alanine aminotransferase (SGPT): 64 U/L, Alkaline phosphatase (ALP): 499 U/L. Serologic tests for hepatitis B, C, and HIV were all negative. Chest radiograph and electrocardiogram were normal.

Ultrasonography (abdomen): Generalized steatosis was found in the liver with no organomegaly.

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Computed tomography scan (abdomen): Crossed fused renal ectopia was seen on right side.

As the patient was a painter for almost a decade, diagnosis of occupational hepatitis was made in absence of any viral marker or any obstructive pathology finding. Patient was treated conservatively. The laboratory investigations showed a decline in the values of bilirubin and liver enzymes.

Patient was discharged after 5 days of hospitalization in a stable condition with following reports: total bilirubin 39.30 mg/dl, direct bilirubin: 24.60 mg/dl, indirect bilirubin: 14.70 mg/dl, SGOT: 51 U/L, SGPT: 58 U/L, and ALP 373 U/L. Patient was advised to refrain from smoking and painting. Patient reported in follow-up in stable condition with normal liver function tests (LFTs), biochemical profile.

**Discussion**

Occupational hepatotoxic chemicals can be classified as direct/indirect hepatotoxins or agents that cause liver injury as a result of host idiosyncrasy. The presentation of occupational liver disease may be acute/subacute or chronic but frequently it is insidious.\(^\text{[5]}\)

A number of chemical compounds are used in paint products as pigments, extenders, binders, solvents, and additives.\(^\text{[6]}\) The main solvents are toluene, xylene, chloroform, trichloroethylene, tetrachloroethylene ketones, alcohols, esters, and glycol ethers. They are readily released into the air during and after applying paint to surfaces.\(^\text{[7]}\) Painters and workers in the painting industry are potentially exposed to these chemicals both by inhalation and skin contact.

Organic solvents are known hepatotoxins; however, the effect on a worker’s liver of lower level of exposure may not be readily apparent. At high level of exposure, acute toxic hepatitis can be seen and, in some cases, fulminant hepatic necrosis.\(^\text{[1]}\)

The present report describes a case of occupational hepatitis as a result of exposure to solvents. Similar to the present case Dossing reported liver damage associated with occupational exposure to organic solvents in house painters.\(^\text{[3]}\)

Atkinson *et al.* also reported a case of acute reversible hepatotoxicity, bone marrow suppression and encephalopathy following painting in a small, nonventilated room.\(^\text{[8]}\)

Studies demonstrate that liver enzymes are elevated in painters as compared to control group.\(^\text{[6]}\) In addition to this it is also documented that levels of liver enzymes might not be sensitive enough to detect any mild changes at an early stage. Study by various authors has suggested that serum bile acid (SBA) determination might be proposed in screening of workers exposed to potentially hepatotoxic chemicals. A high SBA levels might be considered as an early sign of liver dysfunction.\(^\text{[9]}\)

To make the diagnosis of occupational toxic hepatitis, criteria’s include: Exposure to chemicals at workplace should be there. Liver enzyme must increase to at least double. Lack of an obvious nonoccupational cause.\(^\text{[10]}\) In the present patient, occupational history reveals that he is a painter who has been regularly involved in painting for almost a decade with constant exposure to paints solvents of various types. Liver enzymes were elevated. Ultrasonography abdomen did not reveal any focal lesion. Serum assay of HBsAg, HCVAg were negative.

**Factors contributing to liver toxicity by solvents**

Alcoholism, age, genetic factors, body fat, and liver blood flow.

**Pathogenic mechanism responsible for hepatotoxicity by solvent\(^\text{[1]}\)**

Inflammation, cytochrome P450 dysfunction, mitochondrial dysfunction, and oxidative stress collectively play important role in damage of the liver.

**Role of inflammation**

Kupffer cell are activated due to direct action of chemical and they produce various cytokines, such as interleukin (IL)-1, IL-6, tumor necrosis factor –Alpha. These factors are responsible for 1. Production of reactive oxygen species (ROS) from neutrophils
2. These cytokines regulate certain genes which produce factors which are responsible for promoting apoptosis and hepatocyte proliferation
3. Inflammatory cell infiltrate, lipogenesis, fibrogenesis, and cholestasis are also promoted by these cytokines.\(^\text{[1,11]}\)

**Role of cytochrome P450 dysfunction**

Cytochrome P450 is mainly responsible for detoxification of xenobiotics and endogenous substances like prostaglandins and cholesterol, etc. Genetic susceptibility of individuals differs considerably as far as detoxification by glutathione and cytochrome P450 is considered.

**Mitochondrial dysfunction**

Number of amphiphilic and cationic solvents get concentrated in the mitochondria and inhibit fatty acid beta oxidation and lead to electron transport chain damage (superoxide anion formation and steatohepatitis).\(^\text{[12]}\)

**Oxidative stress**

ROS are produced by solvents which damage the intracellular organelles and cause oxidative stress.

**Conclusion**

The present report documents a case of transient hepatitis following exposure to organic solvents. Chronic exposure to low concentrations of solvents in occupational settings can cause aberration in LFTs without any symptoms suggestive of liver disease. As these diseases lack unique findings, it is difficult to differentiate from nonoccupational causes. Hence, a cautious occupational history should always be obtained.

Careful observation with timely checkups is important for workers in the paint industry to avoid such occupational hazards. Due precaution should be taken by such workers to avoid exposure to these chemicals by aerosols/ingestion.
mode. Ventilation should be proper. Mask and protective gloves should be used by all workers. Early recognition and reporting will be helpful in further understanding the incidence and possible mechanism.

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Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest
There are no conflicts of interest.

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