

LINDANE-INDUCED HEPATOTOXICITY IN HUMAN: REPORT OF A RARE CASE

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ABSTRACT

Background

Lindane is an insecticide which is used in commercial insecticides and food containers.

Case presentation

Herein, we reported on a 62-year-old man with liver cirrhosis 12 years after continuous exposure to lindane gas. The diagnostic workup showed no other possible cause, and findings of liver scintigraphy and clinical manifestations were compatible with substance-induced cirrhosis.

Conclusion

Chronic exposure to lindane may cause hepatotoxicity and cirrhosis in human.

Keywords: Lindane, human, hepatotoxicity

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INTRODUCTION

Lindane, the gamma isomer of hexachlorocyclohexane (γ -HCH) is a white solid substance used as an insecticide, scabicide, pediculicide, and ectoparasiticide. It is also used in commercial insecticide and food containers. It may evaporate into the air with colorless vapor and musty odor.(1)

Exposure to lindane is possible from ingesting contaminated drinking water, breathing contaminated air, or having contact with it.(1), Limited animal study is available on lindane toxicity by inhalation. The most acute effects in human have been due to accidental or intentional

ingestion of the substance, although inhalation toxicity occurred (especially among children) when it was used in vaporizers. Workers may be exposed to the product through dermal contact and through inhalation.(2)

To the best of our knowledge, no report has been published on lindane-induced hepatotoxicity in man. Herein, we reported on a patient with lindane-induced hepatotoxicity presented with liver cirrhosis.

CASE PRESENTATION

A 62-year-old man presented with six months history of malaise, abdominal discomfort mostly in the right upper quadrant, jaundice and dyspnea. The patient was working in a food preserving factory for 12 years where he has been exposed to lindane gas. He left his job due to his illness since six months before. He mentioned two episodes of tonic-clonic seizure that was happened almost six years before. There was no other positive point in his past medical history including other medical

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illnesses, drug abuse, transfusion, surgery or smoking. He was pale with icteric sclera. On physical examination, the abdomen was soft with no tenderness or rebound tenderness. Shifting dullness was detected. Bilateral lower limb edema up to knees was also detected. Cardio-pulmonary and other examinations were normal.

He had a oral temperature of 37.4 °C; his heart rate, respiratory rate, and blood pressure were normal. The patient was admitted with the possibility of viral hepatitis but all viral markers were negative. Liver function tests revealed an SGOT of 187 U/L, SGPT of 260 U/L, ALK P of 460 IU/L, direct bilirubin of 10.2 mg/dL, total bilirubin level of 5.4 mg/dL, total protein of 6.8 g/dL, serum albumin of 2.7, PT of 19.3 s (INR of 1.4), and a PTT of 48 s.

Abdominal sonography detected hepatomegaly (154×83 mm), splenomegaly, ascitic fluid in abdominal and pelvic cavity and increased diameter of the portal vein (17 mm). Findings of liver scintigraphy, besides clinical findings, were consistent with parenchymal liver disease (cirrhosis). The patient scheduled for liver biopsy but he refused to do so.

DISCUSSION

Lindane is a lipid-soluble substance. It is stored in fat and repeated small exposures may result in accumulation and eventual clinical toxicity.(3), Many studies have shown the oxidative effects of lindane on various organs of mammals such as rat blood (4), brain (5), testis (6), and liver (4); all these effects were duration- and age-dependent.(4-6), Metabolism of lindane occurs mainly in the liver by cytochrome P450

enzyme system in the smooth endoplasmic reticulum. Lipid peroxidation has been proposed as a major molecular mechanism involved in tissue injury induced by lindane. It is also mentioned that lindane hepatotoxicity is largely dependent on Kupffer cell function, which may involve mediators leading to pro-oxidant and inflammatory processes.(3)

Most reports of acute toxicity from lindane involve blood levels of 130 ng/mL or greater, with the most severe and fatal cases involving levels exceeding 500 ng/mL.(7), Signs and symptoms such as nausea, vomiting, headache, tremor, ataxia, tonic and clonic convulsion, alteration in EEG pattern, weakness, wheezing, arrhythmia, agranulocytosis, rhabdomyolysis, disseminated intravascular coagulation, lactic acidosis, hypotension and hepatic and renal damage can be occurred in lindane toxicity.(2, 3), Lindane has been demonstrated to be a liver carcinogen in mice via oral exposure.(1), Chronic exposure to lindane by inhalation in humans has been associated with effects on the liver, blood, and nervous, cardiovascular, and immune systems.(1)

However, there is no solid evidence supporting lindane-induced hepatotoxicity in human. We could not detect any viral or metabolic cause for cirrhosis in our patient. Therefore, the possibility that lindane might cause the cirrhosis in our patient is strongly suggested.

CONCLUSION

Chronic exposure to lindane may cause hepatotoxicity and cirrhosis in human.

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