

# Acute Lindane Poisoning in a Child

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Lindane, an organochlorine insecticide belonging to the hexachlorocyclohexane family, is widely used as a therapeutic insecticide for humans and animals. It can be absorbed by ingestion, inhalation, or dermal exposure. The primary target of action is the central nervous system. Lindane toxicity has been reported to occur mostly by way of dermal exposure. This report presents a case where intoxication occurred through ingestion.

## Patient Description

A 3½ year old male Bedouin child from a rural area was admitted to the Children's Emergency Room because of a short attack of seizures that occurred at home. A short while before, he had ingested an unknown quantity of an unidentified liquid insecti-

cide used for treating animals and passed spontaneously. A day after admission the child's parents showed us the material responsible for the poisoning. It was identified as a 35% lindane solution used as an animal insecticide. They had bought the insecticide somewhere on the West Bank; there was no manufacturer's name on the label, only a sticker stating the lindane concentration.

On admission the child was lethargic and irritable. He exhibited nausea, vomiting and tremor. His temperature was 37.2°C, pulse 150 beats/minute, blood pressure 110/70 mmHg, and respirations 23/minute. Initial arterial blood gases showed pH 7.28, pCO<sub>2</sub> 41 mmHg, pO<sub>2</sub> 95 mmHg, and HCO<sub>3</sub> 20.0 mmol/L. The hemoglobin level was 11.3 g/dl and white blood cell count 14,400/mm<sup>3</sup>.

Other laboratory results were: serum sodium 140 mEq/L, potassium level 3.8 mEq/L, chloride 105 mEq/L, blood urea nitrogen 35 mg/dl, creatinine 0.9 mg/dl, creatine phosphokinase 50 U/L, alanine aminotransferase 41 U/L, and aspartate aminotransferase 35 U/L. X-ray films of the chest and electrocardiogram were normal. In the emergency room there was a repeat occurrence of general clonic-tonic seizures that were arrested by intravenous injection of diazepam solution (0.2 mg/kg). The child was hospitalized in the General Pediatric Department for further observation. He continued to be lethargic, irritable and nauseous, was still vomiting and had tremors. The child developed a third episode of general clonic-tonic seizures that lasted for about 2 minutes and passed

spontaneously without anticonvulsant therapy. Electroencephalogram revealed a typical 3/sec spike and generalized wave discharge. A repeat analysis of blood gases was absolutely normal. The patient's lethargic and irritable state continued for about 18 hours following ingestion of the toxic material. He was observed for another 24 hours during which he remained stable and alert, without any further episodes of seizure. A repeated EEG was normal, and the child was discharged home without any sequelae.

### Comment

Lindane is the most active, effective and toxic gamma-isomer of benzene hexachloride; its oral LD<sub>50</sub> in rats is reported to be 60 mg/kg body weight. The toxic effect of lindane occurs mainly by suppression of the GABA-mediated synaptic inhibition [1]. The clinical picture of acute lindane poisoning is characterized by irritability, seizures, tremor, twitching, headache, nausea, vomiting, diarrhea, weakness, dyspnea, cyanosis, cardiac arrhythmia, circulatory collapse, and coma with fatal outcome [2]. The intensity of these symptoms depends on the absorbed dose. The treatment of lindane poisoning is symptomatic and supportive and there is no specific antidote.

Most of the reported cases of lindane poisoning occurred by means of excessive dermal exposure. Poisoning by ingestion is uncommon and usually occurs because of unintended ingestion of common 1% lindane

solutions that are used for the treatment of scabies and pediculosis in humans. Aks et al. [3] described three cases of lindane (1% Kwell lotion) ingestion in children who recovered completely. Two children developed seizures and vomiting, and the third child manifested only sleepiness. In our patient, intoxication was due to accidental ingestion of a highly concentrated lindane preparation intended for animals. Like us, Starr and Clifford [4] reported a case of lindane pellet ingestion by a 2½ year old girl. The clinical picture was characterized by irritability and grand mal seizures. The patient received supportive treatment and recovered within 24 hours.

Nordt and Chew [5] described oral ingestion of lindane in three toddlers. Nausea and CNS toxicity (seizures or listlessness) were the main signs of poisoning, and all the children recovered without any complications. CNS redistribution of lindane to the blood and then to fat may account for the apparent rapid CNS recovery in spite of persistent substantial total body burden [2]. This may explain why all signs of intoxication in our patient disappeared within 24 hours. In addition, our patient developed mild metabolic acidosis. While lactic acidosis has been reported after ingestion of lindane, metabolic acidosis may be a consequence of severe convulsions. The combination of lactic acidosis and severe convulsions could explain the metabolic acidosis observed in our patient. However, we did not measure blood lactic acid.

The mild leukocytosis that was reported in our patient's laboratory data is not normally observed in lindane poisoning and may have been due to the convulsions and stress, which increase the white blood cell count.

In summary, this report describes a case of acute lindane toxicity by way of oral ingestion of a highly concentrated lindane preparation used for animals. Lindane toxicity should be considered when a case of seizures of unknown etiology occurs in a child from a rural area, especially in surroundings where insecticides are stored. Parent education and awareness is the key to primary prevention of such incidents.

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### References

1. Narahashi T, Frey JM, Ginsburg KS, Roy ML. Sodium and GABA-activated channels as the targets of pyrethroids and cyclodienes. *Toxicol Lett* 1992;64-5:429-36.
2. Howland MA. Chlorinated hydrocarbons, pyrethrins, and DEET. In: Goldfrank LR, ed. *Goldfrank's Toxicologic Emergencies*. 5th edn. Connecticut: Appleton & Lange, 1994:1121-6.
3. Aks S, Krantz A, Hryhorczuk D, et al. Acute accidental lindane ingestion in toddlers. *Ann Emerg Med* 1995;26:647-51.
4. Starr HG, Clifford NJ. Acute lindane intoxication. *Arch Environ Health* 1972; 25:374-5.
5. Nordt SP, Chew G. Acute lindane poisoning in three children. *J Emerg Med* 2000;18(1): 51-3.

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