SHORT REPORT

Diagnostic radiopacity and hepatotoxicity following chloroform ingestion: a case report

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Background: Diagostic imaging can help in the management of toxicologic emergencies. The authors report on a patient who presented to the emergency department with coma and suppressed respirations after ingestion of an unknown substance.

Methods: Ingestion of chloroform with radiopaque material in the bowel on abdominal radiograph was documented. The patient was treated with haemoperfusion, activated charcoal, and laxatives to decrease the toxicities.

Results: Hepatotoxicity occurred on post-ingestion day 3 and elevation of liver enzymes reached peak levels on post-ingestion day 5. The patient received N-acetylsystein and supportive care during hospitalisation. The patient improved from hepatic dysfunction and was discharged without complication on post-ingestion day 11.

Conclusion: Radiographic studies in toxicology may confirm a diagnosis and assist in therapeutic intervention.

Halogenated hydrocarbons such as chloroform are radiopaque. If ingested, this toxin may be seen on an abdominal radiograph. Poisoning after oral chloroform ingestion, however, remains rare.[1–3] To date, there has been no report of chloroform ingestion with both diagnostic radiopacity and hepatotoxicity. We describe a case of chloroform intoxication that was diagnosed by a simple abdominal radiograph and had associated hepatotoxicity. This case supports the effectiveness of conventional radiography in the diagnosis and management of intoxication.

CASE REPORT

A 20 year old woman presented unresponsive after attempting suicide by ingestion of an unknown substance one hour before presentation. Paramedics found the patient unresponsive and lying on the floor. A search of the surroundings revealed an empty 100 ml bottle with unknown contents and vomitus with sweet smell. Her partner reported that she purchased chloroform several days earlier.

On arrival to the emergency department, the patient's vital signs revealed: blood pressure of 100/80 mm Hg, a tachycardia of 130 beats/minute, no spontaneous respiration, and a rectal temperature 36.2℃. Neurological examination showed a patient in deep coma with an intact corneal reflex. Her pupils were dilated to 6 mm and were sluggishly reactive to light. She was intubated and mechanically ventilated. The initial blood gas study showed a respiratory acidosis that was improved following mechanical ventilation. The initial liver function test, blood glucose, levels of blood urea nitrogen, and electrolytes were normal. We ordered chest and abdominal radiograph. There were contrast materials noted in the small bowel (fig 1). After physical examination and conventional abdominal radiographs, we believed that the radiopaque material was chloroform and was the cause of the coma and respiratory depression of the patient.

The chloroform concentration was not obtained because our laboratory could not perform this assay. We infused activated charcoal 50 g and 250 ml of 70% sorbitol through a nasogastric tube. In addition the patient received haemoperfusion on the day of exposure to the toxin. We did not use N-acetylcystein, preventively.

On post-ingestion day 1, the patient's prothrombin time was prolonged. The patient was medicated with N-acetylcystein of 600 mg/day until she was discharged. Thirty six hours later she was awake and alert. She stated that she ingested a bottle of chloroform about one hour before her admission. Abnormal liver enzymes were noted on post-ingestion day 3. The elevation of the liver enzymes reached peak levels on post-ingestion day 5. However, she had normal blood urea nitrogen level. On post-ingestion day 9, the prothrombin time was normalised. On post-ingestion day 11, she was discharged in stable condition. At six weeks after discharge, her liver enzymes returned to normal. The liver function abnormalities during hospitalisation are shown in the table.

DISCUSSION

Chloroform is a potent central nervous system (CNS) and respiratory depressant.[1] Depression of CNS appears soon after exposure. In the case here reported, the patient progressed to coma and apnoea for about one hour. The signs and

Table 1 Liver function tests following ingestion of chloroform in a 20 year old woman

	Days following ingestion							
Liver function test (normal range, unit)	0	1	3	5	7	9	11	42
AST (0–50, IU/l)	34	18	58	1513	474	173	80	28
ALT (0–45, IU/l)	9	9	51	2717	1540	802	421	29
Alkaline phosphatase (35–160, IU/l)	45	31	55	71	81	73	62	50
Total bilirubin (0–1.6, mg/dl)	0.9	1.4	2.4	5.4	4.5	1.9	0.9	0.4
Direct bilirubin (0–1.0, mg/dl)	0.3	0.2	0.5	1.8	1.6	0.7	0.4	0.2
Prothrombin time (seconds)	–	14.1	18.4	15.8	13.9	11.8	–	–
% of control		66.1	35	50	68.5	102		

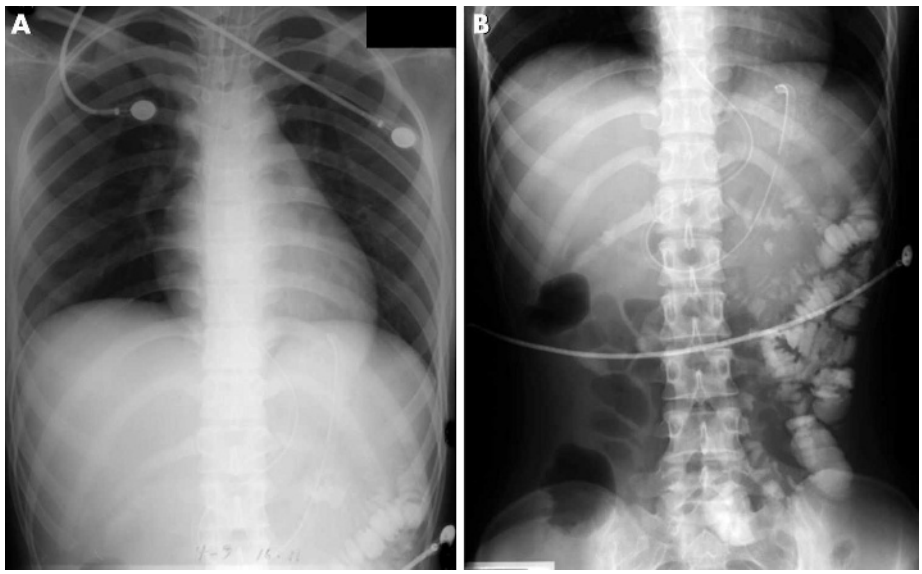

Figure 1 (A) chest radiograph shows endotracheal tube and nasogatric tube. There are no abnormal findings in the lung fields. (B) Abdominal radiograph shows contrast material in the bowel.

symptoms of CNS and respiratory depression, sweet odour, and delayed liver toxicity are highly suggestive of intoxication with a chlorinated hydrocarbon. Therefore, a useful clinical pearl is that many halogenated hydrocarbons are radiopaque.[4–6] Chloroform is a clear, colourless, and volatile liquid with radiopacity. Consideration of a potentially radiopaque substance is useful in suggesting diagnostic possibilities when a radiopaque substance is discovered on an abdominal radiograph. More common radiopaque substances are iron tablets, heavy metals, halogenated hydrocarbons such as carbon tetrachloride, methylene iodide, and chloroform.[6] However, these ingestions are rare and the quantity ingested is usually too small to show this effect. Our patient ingested about 100 ml of chloroform for the purpose of suicide. We predicted the amount of ingested chloroform from patient's history, empty bottle size, and abdominal radiograph. A large amount of contrast materials in the small bowel were discovered on the abdominal radiograph. The radiograph helped to confirm the diagnosis of the toxin and to quantify the amount of toxin involved.

Although gastrointestinal decontamination is probably indicated after chloroform ingestion, the toxin is rapidly absorbed and the utility of decontamination would be expected to be limited with delayed presentation. However, in our patient, because radiopaque chloroform was observed in the abdominal radiograph, we used activated charcoal, sorbitol, and magnesium oxide. Although its use was not followed by rapid recovery from CNS and respiratory depression, gastrointestinal decontamination including whole bowel irrigation may decrease the absorption of chloroform.

The toxicities associated with chloroform frequently occur in inhalation. Reported ingestions are rare.[6] Ingestion of 30 ml of chloroform has been suggested as the minimal lethal adult dose, but survival after hepatorenal failure has been reported after ingestion of 120 ml.[2] In our case, the patient ingested a large amount of chloroform and experienced delayed hepatotoxicity. The mechanism of hepatotoxicity is thought to resemble that of acetaminophen hepatotoxicity in that the production of a toxic metabolite by the cytochrome P450 system is required before the onset of toxicity.[7 8] Abnormalities in the liver enzymes were observed to peak at 72–96 hours after a chloroform exposure and return to normal within 6–8 weeks. In the case here reported, liver enzymes reached a peak on post-ingestion day 5 and returned to normal without complications at six weeks after discharge. N-acetylcystein may be effective in preventing hepatotoxicity in a manner similar to acetaminophen.[7 8] However, to date no clinical data are available to support its use. In our case, we used N-acetylcystein 600 mg/day after the prothrombin time was observed to be prolonged. However, we do not know the mechanisms of the protective effects of low dose of N-acetylcystein on hepatotoxicity after chloroform ingestion. In one report, haemodialysis has been used with a favourable outcome where no hepatorenal toxicity developed.[9] We used haemoperfusion; however, we could not determine its clinical effects on CNS and respiratory depression as well as liver toxicity.

In conclusion, this case supports the effectiveness of conventional radiography in the diagnosis and management of chloroform intoxication.

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