Diagnosis of chlorinated hydrocarbon poisoning by x ray examination

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A 45 year old man was admitted to hospital with vomiting, abdominal pain, and diarrhoea. There was no coma or confusion but the patient was agitated and suffering from headache. Arterial blood pressure was 120/80 mm Hg, heart rate 80/min, temperature 36°C, and no abnormalities were found at clinical examination.

The patient was a construction worker. He had taken his lunch on the building site during which he had drunk a colourless liquid from an unlabelled bottle by mistake. He was unable to say what was in the bottle but his vomit and breath smelt strongly of solvent.

Because of his abdominal pain, an x ray examination was performed which showed a radio-opaque liquid in the stomach and ileum (fig 1). ECG was normal and few biological disturbances were found: ALAT 36 mU/ml ($N < 40$); ASAT 53 mU/ml ($N < 40$); serum creatinine 170 μ mol/l; white blood cells 17 000/mm³; and haemoglobin 17.6 g/dl. Blood electrolytes were normal.

After a gastric lavage the patient was transferred to our hospital. His clinical course was uneventful. The third day, his serum creatinine was 65 μ mol/l and he was discharged a few days later in good condition; he was unable to remember the circumstances of the accident.

Evidence of exposure to chlorinated hydrocarbons was searched for because of the loss of memory. Trichloroethanol and trichloroacetic acid were measured by a colorimetric method derived from the Fujiwara method. No trichloroethanol was found in the blood (limit of detection; 0.01 mmol/l) but blood trichloroacetic acid level was 0.09 mmol/l on the first day. Urinary trichloroethanol concentrations were 0.85 mmol/l on the first day and 0.29 mmol/l on the fifth; urinary trichloroacetic acid concentrations were 1.5 mmol/l and 0.89 mmol/l on the same days. There were no evidence of arsenic, lead, mercury, or bismuth poisoning.

The actual product is unidentified but it was a chlorinated hydrocarbon able to metabolise to trichloroethanol and trichloroacetic acid. Trichloroethylene was not likely as there was no renal, cardiac, or cerebral impairment despite the important amount of solvent found in the stomach (fig 1). Methylchloroform or tetrachloroethylene which are often used for industrial purposes are less toxic and could be responsible for that type of clinical pattern.

To confirm the x ray findings three doses of 1,1,1-trichloroethane, 0.2 ml, 1 ml, and 2 ml, were administered to male Sprague Dawley rats weighing 250 g. In all cases 1,1,1-trichloroethane was radio-opaque (fig 2).

Fig 1 Abdominal x ray film showing radio-opaque liquid in stomach and ileum.

Fig 2 1,1,1-Trichloroethane administered to male Sprague Dawley rats (250 g): 0.2–1 and 2 ml.

Many toxic substances are radio-opaque and during acute intoxications, abdominal x ray examinations may be useful for diagnosis. Furthermore, they may provide evidence for the effectiveness of gastric lavage.

Abdominal opacities have already been described in carbon tetrachloride poisoning.^{1–3} That other chlorinated hydrocarbons are radio-opaque is less well known. The radio-opacity, however, seems to be strictly correlated to the ratio:

$$\frac{\text{Number of chlorine atoms}}{\text{Molecular weight}} \quad (\text{fig 3})$$

References

- 1 Bagnasco FM, Stringer B, Muslim AM. Carbon tetrachloride poisoning—radiographic findings. *NY State J Med* 1978; **78**:646–7.
- 2 Mathieson PW, Williams G, McSweeney JE. Survival after massive ingestion of carbon tetrachloride treated by intravenous infusion of acetylcysteine. *Human Toxicol* 1985; **4**:627–31.
- 3 Truss CD, Killenberg PG. Treatment of carbon tetrachloride poisoning with hyperbaric oxygen. *Gastroenterology* 1982; **82**: 767–9.

Fig 3 Radiograph of bottle containing (from left to right): methylene chloride, carbon tetrachloride, chloroform, trichloroethylene, tetrachloroethylene, methylchloroform, water, monochlorobenzene, and orthodichlorobenzene.