after the initiation of heparin therapy. The rise in the platelet count occurred some 48–72 hours later, and thus the fall in serum FDP was the first measurable index of improvement. Heparin was continued until the blood film no longer showed signs of fragmentation.

We feel that the rapid capillary method of estimating serum FDP was of great value in the management of this case, and should prove of similar value in future cases.

Summary

Serum levels of fibrin/fibrinogen degradation products have been assayed throughout the course of a case of the haemolytic-uremic syndrome. A rapid method of estimation was of value in monitoring progress and assessing the effect of heparin therapy.

REFERENCES


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Ethylene Chlorohydrin Intoxication with Fatality

Ethylene chlorohydrin is a most noxious poison that is oxidized in the body to chloroacetic acid which then inhibits the tricarboxylic cycle enzymes.

It has been used as a solvent and to prepare ethylene glycol and ethylene oxide. The germination of seed potatoes can be hastened by its application. Industrially its toxicity is well known, but the poison is also found commonly in the home as a photographic cement (Cinecol, Johnsons of Hendon).

Neither poisoning in childhood nor death in any age-group by drinking ethylene chlorohydrin have previously been described.

Case Report

The 23-month-old male patient drank approximately 2 ml. Cinecol at 1 p.m. and vomited immediately. He rapidly became pale, cyanosed, and showed respiratory difficulty, and was admitted to his local hospital for emergency treatment which included an infusion of 5% dextrose in normal saline following gastric lavage.

At 6 p.m. the child had a generalized convulsion and his pupils became fixed and dilated. 2 ml. paraldehyde were administered intramuscularly but he continued to twitch. Another convulsion occurred at 8 p.m., which responded to 30 mg. intramuscular phenobarbitone.

His systolic blood pressure fluctuated throughout 50–80 mm. Hg and his pulse rate varied from 86–140/min.

At the Regional Poisons Centre at 10 p.m. he was pale, cyanosed, and his blood pressure was unrecordable. His pulse rate was 200/min. and his rectal temperature was 36°C. The heart sounds were faint and respiration was shallow. The pupils reacted to light and he responded to stimulation.

Hydrocortisone was given intravenously in a dose of 10 mg./kg. but this did not raise his blood pressure, nor did a second prescription. Soon after this had been given the patient vomited, became apnoeic, and had a cardiac arrest.

He died less than 12 hours after drinking the chemical.

On admission here, his blood urea was 92 mg./100 ml., and his serum bicarbonate was 12.4 mEq/l. The serum electrolytes were normal and his urine was free from sugar, protein, and blood.

Necropsy. The child weighed 15·9 kg. and showed no underlying disease.

The lungs were oedematous and congested. The right lung weighed 168 g. and the left 120 g. Pulmonary haemorrhage was marked, especially posteriorly. Petechiae were present in the subepicardium, the thymus, and beneath the liver capsule. This organ weighed 600 g. The spleen weighed 55 g. and had a toxic follicular pattern. There were 23 agonal intussusceptions in the small bowel, but no other significant abnormalities were found.

Microscopically, the changes seen were non-specific. Capillary bleeding had taken place into the pulmonary alveoli. There was early necrosis of the liver parenchyma, with nuclear vacuolation, cytoplasmic swelling, and small foci of polymorph infiltration, which were also observed in the portal tracts. The changes were most obvious at the periphery of the liver lobules. Acute inflammatory cells were present in the adrenal medulla and karyorrhexis was very striking in the germinal centres of the lymph nodes and spleen. In the kidney there was tubular swelling, and the brain showed widespread neuronal enlargement with cytoplasmic
vacuolation. The Purkinje cells were severely damaged and the endothelial lining of some of the cerebral blood vessels was swollen and had separated from the surrounding tissues.

Neither ethylene chlorohydrin, nor chloracetic acid were found on toxicological examination of the blood and tissues.

Discussion

No descriptions are available of the effects in humans after drinking ethylene chlorohydrin, but extrapolating from the data obtained with rats it is likely that 1 or 2 ml. would be lethal for this exploring toddler.

Ethylene chlorohydrin is also toxic by inhalation and by absorption through the unbroken skin. Browning (1965) has reviewed the 8 reports where the chemical entered the body through these routes in industrial accidents. Carpenter, Smyth, and Pozzani (1949) found experimentally that exposure to 32 p.p.m. of ethylene chlorohydrin in air for 4 hours killed half their rats, while 9 cases of illness caused by breathing low concentrations of the substance over a long period were described by Goldblatt and Chiesman (1944). These patients complained chiefly of nausea, vomiting, and abdominal pain. Because amateur photographers might work in ill-ventilated rooms they might encounter toxic concentrations of this volatile poison. In the fatal inhalation cases symptoms appeared only insidiously, while the rats left by Ambrose (1950) in lethal concentrations of the gas died 1–2 hours after the completion of their experimental exposure but appeared well up to the time of their deaths.

Some of the industrial deaths reviewed by Browning (1965) and the 5 cases of illness described by Bleckat and Strube (1968) were caused by the chemical contaminating the unbroken skin. Similarly, Ambrose (1950) showed that small amounts of the poison applied to the intact epidermis of laboratory animals can be fatal.

Thus, the studies of the industrial accidents and the experiments on animals have shown that inhalation of the gas and skin contact with this chemical should be avoided. Ethylene chlorohydrin is also extremely poisonous when taken by mouth, and, therefore, to avoid domestic accidents, especially to children, a less toxic agent should be found as a simple film cement.

Summary

A 23-month-old boy drank 1–2 ml. of ethylene chlorohydrin and died. The clinical course of his illness and the necropsy findings are described. The dangers of the chemical are discussed, and it is recommended that less toxic products should be sought as film-base solvents for amateur use.

D. Summerfield, Esq., H.M. Coroner, Manchester, kindly gave permission to publish this case. Mr. A. Hoole of the Forensic Science Laboratory, Preston, performed the toxicological analyses, and Mr. A. R. Pippard of Johnsons of Hendon, Ltd., helped with information about Cinecol.

References

Ambrose, A. M. (1950). Toxicological studies of compounds investigated for use as inhibitors of biological processes. II. Toxicity of ethylene chlorohydrin. Archives of Industrial Hygiene and Occupational Medicine, 2, 591.

Bleckat, G., and Strube, G. (1968). Die Klinik der akuten percuta-


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Cryptorchidism, Chest Deformities, and other Congenital Anomalies in Three Brothers

A family is described in which all male offspring displayed an identical clinical picture which included cryptorchidism, chest deformities, and pulmonary anomalies, abnormal shape of skull, hypoplasia of fatty and muscle tissue, and severe mental retardation.

Family Investigation

Details of the family tree are given in Fig. 1. There is no consanguinity. None of the other relatives showed any congenital anomalies of the kind described here. All living members of the family have been examined.