Acute Alcoholic Hypoglycaemia in Two 4-year-olds

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Hypoglycaemia is a well-recognized complication of alcohol poisoning in adults, and over 120 cases have been recorded since 1941. Hypoglycaemia as a result of alcohol ingestion by children, however, has been recorded on very few occasions (Peluffo et al., 1958; Cummins, 1961; Ramon et al., 1963). This paper describes the cases of two children who developed hypoglycaemic convulsions after alcohol ingestion.

CASE 1

A 4-year-old boy was admitted to hospital via the casualty department in July 1968. Three hours earlier he had consumed 540 ml of sherry. He had vomited several times and had gradually become drowsy. On examination he was unconscious and did not respond to painful stimuli. There were no neurological signs and respiration was not depressed. His breath smelled strongly of alcohol. Gastric lavage was not performed.

Five hours after ingestion he developed six right-sided convulsions, each lasting two minutes, though by this time he was conscious but drowsy, the blood sugar was less than 25 mg./100 ml. and blood alcohol was 100 mg./100 ml. He was given 300 ml of milk containing 30 g of glucose by mouth. Within 10 minutes the convulsions ceased and full consciousness was regained. He then drank a further 300 ml of milk containing 10 g of glucose. The convulsions did not recur and there was no residual neurological deficit.

Next day he was his usual self, apart from a slight headache and symptoms suggestive of an alcoholic “hangover.” Blood sugar levels were normal. He was discharged two days after admission.

CASE 2

A 4-year-old boy was admitted to hospital in October 1969. Four hours previously he had consumed approximately 400 ml of gin. He had become drowsy over the next three hours and had convulsed for five minutes previous to admission.

On examination he was deeply unconscious and convulsing. His capillary blood sugar was less than 40 mg./100 ml. (Dextrostix), and in venous samples. Blood sugar was found to be 12 mg./100 ml. An intravenous drip of 10% dextrose was set up and 20 ml of 50% dextrose was injected intravenously at once. The convulsions ceased and consciousness was regained within two minutes of this injection. Venous blood sugar levels were normal after four and eight hours. The intravenous 10% dextrose was then discontinued.

The boy behaved normally thereafter and physical examination was negative. Subsequent blood sugars and an oral glucose tolerance test were normal. He was discharged on the third day after admission.

COMMENT

It is now accepted that alcohol can produce hypoglycaemia in normal subjects, though the exact mechanism is not clear. In acute alcoholism islet cell response is normal, but there is thought to be a failure of normal glycogenolysis and gluconeogenesis.

In the two cases described here the convulsions developed within five hours of ingestion of the alcohol and were clearly due to hypoglycaemia. Prolonged and severe hypoglycaemia can be fatal (Cummins, 1961) or produce irreversible brain damage.

If a child thought to have ingested alcohol develops drowsiness, coma, convulsions, or bizarre central nervous system signs, blood sugar levels should be estimated and an immediate therapeutic trial of intravenous glucose is justified.

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REFERENCES