POST-ALCOHOLIC HYPOGLYCAEMIA AND TOXIC HEPATITIS IN AN AFRICAN CHILD

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In a previous article[1] the findings in patients suffering from post-alcoholic hypoglycaemia were presented. A concomitant, mild, and transient toxic hepatitis was noted in all patients where adequate biochemical tests were performed. Among the group of 23 patients there was a 5-year-old African child. Because death occurred within 16 hours of admission, inadequate biochemical results were obtained. In this report we describe the biochemical findings in the case of a 4-year-old African child admitted to King Edward VIII Hospital in hypoglycaemic coma following the accidental intake of gavine (distillate of *shimeyane*).

CASE REPORT

An African male child, aged 4 years, was admitted in coma to King Edward VIII Hospital in June 1961. The mother stated that the child was well the day before, but had not wakened on the morning of admission. Her husband was known to be a heavy gavine drinker, and she thought that it was possible that the child had taken some of it.

Examination and Treatment

Semicomatose child of fair nutrition, with breath smelling of stale alcohol. Bradycardia was present. Deep tendon reflexes were absent. There were no other abnormalities. Post-alcoholic hypoglycaemia was suspected. After intravenous administration of 25 ml. of 50% dextrose solution the child responded immediately and was able to talk coherently.

Biochemical Findings

Before the administration of the intravenous dextrose solution blood was taken for biochemical and haematological examination. The biochemical tests included investigation of blood glucose ('true' blood sugar), alkaline-phosphatase activity, total bilirubin, serum glutamic oxaloacetic acid trans-

aminase (SGOT), prothrombin index, urinary urobilin and bilirubin, and blood volatile reducing substances. The first three tests were estimated by standard routine laboratory methods,[2] the SGOT by Hergt and Langin's[3] simplified modification of the original method of Karmen *et al.*,[4] the prothrombin index by Quick's method, and the volatile reducing substances by the method of Bowden and McCallum.[5] The SGOT, serum bilirubin, alkaline phosphatase and urinary urobilin and bilirubin tests were repeated until normal results were obtained.

The findings on admission are shown in Table I, while Table II shows the results of tests performed daily. From

TABLE I. BIOCHEMICAL FINDINGS ON ADMISSION

Blood sugar (mg. per 100 ml.)	*Volatile reducing substances (mg. per 100 ml.)*	*SGOT (Karmen units per ml.)*	*Bilirubin (mg. per 100 ml.)*	*Alkaline phosphatase (K-A units)*	*Prothrombin index*	*Urobilin*
19	14	92	0·8	30	92*	Negative

* Test was done a day after admission.

TABLE II. DAILY BIOCHEMICAL ESTIMATIONS

Days	*SGOT (Karmen units per ml.)*	*Serum bilirubin (mg. per 100 ml.)*	*Serum alkaline phosphatase (K-A units)*	*Urinary urobilin*
1	92	0·8	30	Negative
2	162	0·8	25	—
3	220	—	—	Negative
4	136	0·7	—	Positive
5	130	0·2	—	Negative
6	—	—	—	Negative
7	—	—	—	—
8	—	0·3	14	—
9	58	—	—	—
10	—	—	—	—
11	40	0·2	—	—

these results it was apparent that the patient had been admitted in hypoglycaemic coma and suffered from a transient and mild liver dysfunction.

A glucose-tolerance test, performed after recovery from the hepatitis, showed a normal, but 'flat', glucose-tolerance curve.

DISCUSSION

The findings in this patient are thus similar to those in the adult group described in our previous article.[1] In that article it was suggested that the intake of ethyl alcohol had resulted in the hypoglycaemia and the consequent liver dysfunction. In cases where gavine has been consumed, it is possible that impurities, produced during its distillation, could play a part in the production of the toxic hepatitis.

In our previous article[1] it was stated that we felt the majority of the patients could not be termed chronic alcoholics. This case confirms the fact that chronic alcoholism is not a necessary predisposing factor. The poor nutritional state of the average chronic alcoholic probably results in the liver becoming more vulnerable to mild toxins such as ethyl alcohol. Nevertheless, in the majority of our African patients it is probably the long-continued inadequate dietary intake — the result of poverty and poor dietary habits and not of chronic alcoholism — that results in the liver becoming more vulnerable to mild toxins.

The mechanism of post-alcoholic hypoglycaemia is not fully understood. It is known from liver biopsy findings that there is a depletion of liver glycogen.[1] This probably results from the increased utilization of carbohydrate that occurs during the metabolism of ethyl alcohol,[6] from lack of intake of food during alcoholic coma, and from decreased synthesis of liver glycogen during this period. The last might be the result of failure of gluconeogenesis, which might take place because of liver dysfunction produced by the toxic hepatitis. Further investigation is being made into the adrenal and pituitary function of patients suffering from post-alcoholic hypoglycaemia. At this stage it can be said that some of the malnourished patients appear to have associated pituitary dysfunction. It is possible that this factor plays a part in the decreased synthesis of liver glycogen.

Finally, it should be emphasized that prolonged coma following the intake of alcohol, in adults or children, can result from hypoglycaemia. Early recognition and treatment is essential since, in our experience, long-standing hypoglycaemia in children often results in permanent damage to the central nervous system, or in death.

SUMMARY

The clinical and biochemical findings in a 4-year-old African child suffering from post-alcoholic hypoglycaemia and toxic hepatitis are described.

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