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SEVERE DIABETIC STUPOR WITHOUT KETOSIS

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R. D. Lawrence wrote: '. . . the term diabetes mellitus includes a variety of glycosuric conditions separate both in clinical type and aetiology.' The case reported here may represent a condition differing in aetiology from the usual type of diabetes mellitus.

CASE REPORT

An African male age 32 was admitted to the Baragwanath Hospital on 3 June 1957, in a state of extreme shock and dehydration. He was stuporose, but not in deep coma. The pulse rate and blood pressure were not recordable on admission, and his breathing was not of the Kussmual type.

Examination of the urine showed specific gravity 1,020, albumin absent, sugar ++++ (Benedict's test).

On testing the urine for ketone bodies with Rothera's test there was only a slight purplish colour, which developed on standing for 5 minutes. This was recorded as a 'slight trace'. Similarly the test for serum ketone bodies was negative. The test for diacetic acid in the urine with Gerhardt's reagent was negative.

The rest of the clinical examination was negative except for mild bilateral cataracts. The patient is a thin individual, weighing 113 lb. (at present) and height 5ft. 7 in. There was no clinical evidence of hyperthyroidism or of Cushing's disease.

When his condition improved after a few hours we were able to obtain history. There was no record of previous treatment for diabetes. He had been complaining of polyuria, and general weakness for the past month. His appetite had been very poor for the same time, and he had had very little to eat for 2 days before admission. He did not give a history of a large carbohydrate meal such as might have caused a starvation hyperglycaemia and glycosuria. However, he had no recollection of events for 12 hours before admission.

Treatment

Treatment was begun with soluble insulin, 100 units intravenously and 50 units intramuscularly. An intravenous drip was set up. Insulin was given at 2-hourly intervals and 4 blood-sugar estimations were carried out at hourly intervals as follows:

Insulin 3 *June*

8.45 p.m., 100 units i.v. and 50 units i.m.
10.45 p.m., 80 units i.v. and 40 units i.m.
12.45 a.m. next day, 100 units i.v.

Blood Sugar, 3 *June*

8.45 p.m., 1,568 mg.%
9.45 p.m., 1,140 mg.%
10.45 p.m., 924 mg.%
11.45 p.m., 700 mg.%.

From the above it can be seen that it required only 270 units of insulin to reduce the blood sugar from 1,568 to 700 mg.%.

After the 3rd intravenous dose of insulin the patient was given 40-50 units of insulin subcutaneously at 3-hourly intervals. The dosage was subsequently reduced and at present he is maintained on 70 units of 'Novolente' insulin a day.

Fluids. Four vacolitres of normal saline were given intravenously during the first 2 hours. Subsequently he received 5% dextrose in water alternating with 5% dextrose in saline with 2g. of potassium chloride in alternate vacolitres. Thus within the first 5 hours he had received 7 litres of fluids.

Progress. Within the first 12 hours he had received 470 units of insulin and within the first 24 hours 650 units. There was no ketonuria on all subsequent examinations after admission. After 2 hours the patient showed a remarkable improvement. He was mentally clear. His blood pressure was 110/70 mm. Hg.

Other Investigations

3 June, on admission: Blood urea 100 mg.%, serum potassium 6·9 mEq/l., serum sodium 128 mEq/l., Plasma CO_2 content 17·7 mEq/l.

Total protein 8·1g.%, albumin 3, globulin 5·1.

Thymol turbidity 3·5 units. Thymol flocculation negative. Colloidal red ++++. Plasma cholesterol 232 mg.%. Total lipids 710 mg.%. Alpha 1 and alpha 2 lipoprotein 31%, beta lipoprotein 69%.

4 *June*. Haemoglobin 17·7 g.%. Leucocytes 16·400 per c.mm. neutrophils 72%, lymphocytes 37%, monocytes 1%.

14 *June*. Blood urea 18 mg.%. Plasma CO_2 content 17 mEq/l. (same as on admission).

20 *June*. Lumbar puncture. The CSF showed no abnormalities; there was 1 lymphocyte per c.mm. and protein was 16 mg.%.

27 *June*. Glucose tolerance test: *Fasting blood*-sugar 215 mg.%. After ingestion of 50g. of glucose the blood sugar rose to a maximum of 320 mg.% after 2 hours, and fell to 119 mg.% after 9 hours.

These investigations were carried out by the South African Institute for Medical Research at Baragwanath Hospital.

DISCUSSION

The unusual features in this case are the following:

1. The absence of ketonuria although the blood sugar was 1,568 mg.%
2. The patient was not in deep coma, although the blood sugar level was extremely high and he became mentally clear after only a few hours of therapy.
3. There was rapid improvement and he required a smaller amount of insulin than one would expect.

The absence of ketosis is the most important feature of this case. Lawrence[1] discusses two types of diabetics who do not develop ketosis—the common 'lipoplethoric' or fat diabetic and the rare 'lipo-atrophic' type. The explanation offered is that 'insulin turns labelled glucose not only into glycogen but almost equally rapidly into stored fat . . . The lipo-atrophic subject cannot store fat and therefore cannot store ingested sugar, which circulates in excess. Perhaps the over-full stores of the lipoplethoric patient prevent the easy storage of glucose . . . ' Therefore, the glycosuria is not due to lack of insulin but to failure of conversion of glucose to fat. He mentions that in the lipo-atrophic variety 'all phases of hyperglycaemia produced an intense lipaemia.' This did not occur in our case although he is also a thin individual. There is also no hepatosplenomegaly, which was a feature in Lawrence's case.

Root and Leech[2] describe a case of diabetic coma with ketonaemia but no diacetic acid in the urine and they attribute this anomaly to failure of liver function.

Another type of diabetes that may be relevant to this case is the transient starvation glycosuria that develops in experimental animals and humans after periods of starvation or fat feeding. The explanation given is that in these individuals there is a depression of liberation of insulin and a marked hyperglycaemia and glycosuria may develop on administration of carbohydrate.

Lukens and Dohan, as quoted by Cecil and Loeb,[3] have produced permanent diabetes in normal cats through the maintainance of hyperglycaemia by the intraperitoneal injection of glucose. This procedure, it is postulated, exhausts the beta cells of the pancreas and eventually leads to their degeneration. It would be interesting to postulate that a similar mechanism is involved in the production of the type of diabetes described above, or may precipitate it. That is to say: This patient as far as can be ascertained has a high carbohydrate and low fat intake; this prolonged high carbohydrate ingestion stimulates the beta cells at first, but finally they become exhausted, with the resultant production of diabetes. The absence of ketosis is more difficult to explain; possible factors may be the low fat storage due to low intake of fats, or liver dysfunction resulting in their abnormal metabolism.

The other features mentioned above—the fact that the patient was not in deep coma and responded rapidly, and required relatively small amounts of insulin—may perhaps also be explained by the absence of ketonuria. It appears that the mental state in diabetic coma is associated with the acidosis rather than the hyperglycaemia, and the presence of acidosis indicates a severe type of metabolic disturbance requiring a larger amount of insulin.

As regards therapy in diabetic coma Root and Leech point out the danger of excessive administration of glucose. If glucose is given before it can be utilized, it will result in further dehydration, which in turn may cause irreversible cerebral damage. We feel that this would apply particularly to the type of stupor described above, where the initial blood-sugar level was extremely high. It would be preferable to administer glucose only when the patient is well hydrated with intravenous saline.

SUMMARY

A case of diabetic stupor without ketosis and with blood sugar of 1,568 mg.% is described. Certain possible theories for the absence of ketosis are mentioned. It is also pointed out that premature administration of glucose may be of danger in a case of this nature, and it is important to note that this type of diabetes required relatively small amounts of insulin despite the high blood sugar level.

We wish to thank the Superintendent of Baragwanath Hospital for permission to publish this case.

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